Air Pollution and Cardiovascular Conditions

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Air Pollution

Any visible or invisible particle or gas found in the air that is not part of the original, normal composition.
Classification

- **Natural:** forest fires, pollen, dust storm

- **Unnatural:** man-made; coal, wood and other fuels used in cars, homes, and factories for energy

Air Quality Index (AQI)

- Indicates whether pollutant levels in air may cause health concerns.
- Ranges from 0 (least concern) to 500 (greatest concern)
### Main Pollutants

- **Ozone**
- **CO**
- **Particulate Matter**
  - Thoracic particles*: PM10
  - Coarse particles [PM10 to 2.5]*
  - Fine particles* : PM2.5
- **Sulphur Dioxide**
- **Mercury**
- **Lead**

### Air Quality Chart

<table>
<thead>
<tr>
<th>Air Quality</th>
<th>Air Quality Index</th>
<th>Protect Your Health</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good</td>
<td>0-50</td>
<td>No health impacts are expected when air quality is in this range.</td>
</tr>
<tr>
<td>Moderate</td>
<td>51-100</td>
<td>Unusually sensitive people should consider limiting prolonged outdoor exertion.</td>
</tr>
<tr>
<td>Unhealthy for Sensitive Groups</td>
<td>101-150</td>
<td>Active children and adults, and people with respiratory disease, such as asthma, should limit prolonged outdoor exertion.</td>
</tr>
<tr>
<td>Unhealthy</td>
<td>151-200</td>
<td>Active children and adults, and people with respiratory disease, such as asthma, should limit prolonged outdoor exertion. Everyone else, especially children should limit outdoor exertion.</td>
</tr>
<tr>
<td>Very Unhealthy (Alert)</td>
<td>201-300</td>
<td>Active children and adults, and people with respiratory disease, such as asthma, should limit prolonged outdoor exertion. Everyone else, especially children, should limit outdoor exertion.</td>
</tr>
</tbody>
</table>
Impact

These pollutants are associated with increased hospitalization and mortality due to cardiovascular disease, especially in persons with congestive heart failure, frequent arrhythmias, or both.

Who is at Risk?

- Children
- Elderly
- Prior heart or lung disease patients sp. those with history of Asthma.
- Diabetics
- Persons who work/exercise outdoors
- Otherwise healthy adults and children
Health Problems

- Impaired fertility
- Birth Defects
- Respiratory Infections
- Asthma
- Emphysema
- Lung Cancer
- Heart attacks
- Strokes
- Premature Death

"Pyramid of Effects"

- Lung function changes, immune cell responses, heart rate or heart rate variability responses
- Asthma attacks, medication use, symptoms
- Doctor visits
- Hospital Admissions
- Death
WHO

- PM 2.5: 76
- PM 10: 179
- Pollution Index: 95.68
- PM 10 pollution level:

Extremely high

Air Pollution in Major Cities

<table>
<thead>
<tr>
<th>City</th>
<th>PM2.5</th>
<th>PM10</th>
<th>Index</th>
<th>Pollution level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cairo</td>
<td>76</td>
<td>179</td>
<td>95.68</td>
<td>Ext. High</td>
</tr>
<tr>
<td>Paris</td>
<td>18</td>
<td>28</td>
<td>69.94</td>
<td>Low-Mod</td>
</tr>
<tr>
<td>NY</td>
<td>9</td>
<td>16</td>
<td>59.35</td>
<td>Low</td>
</tr>
<tr>
<td>Tokyo</td>
<td>15</td>
<td>28</td>
<td>44.37</td>
<td>Low-Mod</td>
</tr>
<tr>
<td>Rio</td>
<td>36</td>
<td>67</td>
<td>71</td>
<td>High</td>
</tr>
<tr>
<td>Moskow</td>
<td>20</td>
<td>33</td>
<td>62.65</td>
<td>Moderate</td>
</tr>
<tr>
<td>Riadh</td>
<td>165</td>
<td>368</td>
<td>71.64</td>
<td>Ext. High</td>
</tr>
<tr>
<td>Johannesburg</td>
<td>41</td>
<td>85</td>
<td>71.06</td>
<td>High</td>
</tr>
</tbody>
</table>
Potential Biological Mechanisms

- Direct Effect:
  - of pollutants on the cardiovascular system, blood, and lung receptors,

- indirect effects:
  - mediated through pulmonary oxidative stress and inflammatory responses.
Direct Effect

- Direct effects may occur via agents that readily cross the pulmonary epithelium into the circulation, such as gases and soluble constituents of PM2.5.
- 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 direct effects is the explanation for the occurrence of rapid (within a few hours) cardiovascular responses, such as increased myocardial infarctions.

Indirect Effects

- 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:
  - Activating hemostatic pathways
  - Impairing vascular function, and
  - Accelerating atherosclerosis.
Possible biological mechanisms linking PM with cardiovascular disease.

Exposure to inhaled particles induces: **Alveolar inflammation**, leading to exacerbation of preexisting lung disease,

Subsequently, several studies of controlled exposures to particles demonstrate increases in both **cellular and biochemical markers** of pulmonary and systemic inflammation.
Effects of Inflammation, Oxidative Stress, and Alterations in Blood-Borne Factors on the Cardiovascular System

Exposure to PM increases fibrinogen, 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 disease and established as an important independent risk factor for myocardial infarction and stroke.

- Serum II-6, IL-1, and granulocyte macrophage colony stimulating factor are increased in healthy male subjects after exposures to increased air pollution due to forest fires.

- They are increased in vitro with exposure of human lung macrophages to urban PM10. II-6 is directly involved in regulation of the synthesis of C-reactive protein in the liver.
**Effects of Inflammation, Oxidative Stress, and Alterations in Blood-Borne Factors on the Cardiovascular System**

CRP impairs endothelial vasoreactivity in individuals with preexisting coronary artery disease. In addition, CRP may contribute directly to the development and progression of atherosclerosis via:

- 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.

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

Inflammation (proinflammatory cytokines, CRP, and components of innate immunity) plays a significant role in the genesis of atherosclerosis and in plaque instability.
Short term PM exposure

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>% of Total Deaths</th>
<th>Cause-specific ↑ per 10 µg/m³ ↑ in PM₂.₅</th>
<th>Approximate % of excess deaths due to PM exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>All cause</td>
<td>100%</td>
<td>1.0%</td>
<td>100%</td>
</tr>
<tr>
<td>Respiratory</td>
<td>8%</td>
<td>0.5-1.5%</td>
<td>12%</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>45%</td>
<td>0.5-1.5%</td>
<td>68%</td>
</tr>
</tbody>
</table>

Source: C. Arden Pope III, as quoted by Robert D. Brook, Air pollution and cardiovascular disease. Available [here](http://www.epa.gov/agingepa/pdfs/press/AHA_PMtalk_Brook_12_11.pdf)

Vascular tone

Alterations in vascular tone due to air pollution exposure have also been demonstrated.

The inhalation of PM and ozone for 2 hours caused conduit arterial vasoconstriction in healthy adults.

It is possible that vasoconstriction occurs as a result of endothelial dysfunction caused by acute systemic inflammation and oxidative stress.
Vascular tone

- Ambient air pollution increases blood pressure in cardiac rehabilitation patients and in adults with lung disease.

- 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. This finding suggests that air pollution exposure conveys a greater susceptibility to myocardial ischemia.

Disturbances of the Cardiac Autonomic Nervous System

- 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 occurs rapidly and is inversely proportional to the increase in the concentration of PM. It predicts an increased risk of cardiovascular morbidity and mortality in the elderly and those with significant heart disease.
Disturbances of the Cardiac Autonomic Nervous System

- Decreased parasympathetic input to the heart may provide an important mechanistic link between air pollution and cardiovascular mortality by promoting fatal tachyarrhythmias.

- It is conceivable that in certain populations, air pollution-mediated bradyarrhythmias may also contribute to sudden death.

Environmental Pollution and Congenital Heart Disease

- Congenital heart disease is associated with genetic defects, infections (eg, rubella), radiation, medications, and environmental exposures.

- 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 also observed were valvular, aortic, and truncal defects associated with O3 levels.
Environmental protection agency (EPA)

- EPA is evaluating tools and methods to help communities, states control and reduce air pollution’s health risks, including those to the heart.
- Scientists are studying intervention strategies such as roadway barriers (walls or tree lines) to minimize pollutant exposure.
- One EPA study found that dietary additions of omega-3 fatty acids, found in fish, can reduce some biochemical and physiological responses after exposure to air pollution.

THE ASSOCIATION ADVOCATES

- Everyone in the world would benefit from improved air quality, whether they currently have CVD or not. The American Heart Association therefore advocates for a comprehensive approach to clean air, including:
  - Supporting the full implementation of the Clean Air Act.
  - Tightening regulations on sources of particulate matter and ozone to improve the quality of our air.
THE ASSOCIATION ADVOCATES

Configuring and designing cities and communities to provide greater separation between residents and pollution sources such as highways and power plants.

Encouraging physicians and other health care professionals to talk to their patients about the cardiovascular risks of exposure to significant air pollution and provide tips for reducing this exposure, such as avoiding prolonged or heavy outdoor exertion during times when the air quality may be dangerous.

- http://edugreen.teri.in/explore/air.htm
- http://www.oneworld.net/penguin/pollution/pollution_home.html
- http://www.rcc.org/oem/agindex.html
- www.lungusa.org (Annotated Bibliography of Recent Studies...)
- www.lungnc.org
- www.healtheffects.org (Understanding the Health Effects of Components of the Particulate Matter Mix: Progress and Next Steps)
- www.epa.gov/airnow
- www.nga.org
- www.landofsky.org/airquality
- www.cleartheair.org
- www.airtrust.org
- www.saminet.org