

BUILDING EVIDENCE



FOR HEALTH

Editor in Chief | Joseph G. Allen, Harvard T.H. Chan School of Public Health



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PARTICULATE MATTER

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What is it?

Particulate matter refers to liquid and solid particles that are suspended in the air. In one cubic centimeter of air, there are typically over 5,000 of these particles. Particulate matter is comprised of a variety of compounds from both natural and anthropogenic sources. The toxicity of particulate matter depends on its composition; natural substances such as sand tend to be larger and benign while anthropogenic compounds such as those emitted from automobiles and power plants tend to be more toxic. Anthropogenic particles are a combination of compounds that are directly emitted by power plants and mobile sources such as black carbon and polycyclic aromatic hydrocarbons (PAHs) and particles that form through chemical reactions with gaseous compounds emitted into the atmosphere such as sulfur dioxide (SO₂), nitrogen oxides (NO_x), and volatile organic compounds (VOCs). Natural and anthropogenic particles have different size distributions as well, with anthropogenic particles generally being smaller.¹ For this reason particulate matter is often characterized by its size. PM₁₀ refers to particles less than 10 μm in diameter, PM_{2.5} for particles less than 2.5 μm in diameter and so on. Concentrations of particulate matter are reported in units of particle mass or particle counts, the former usually being in units of micrograms of particulate matter per cubic meter of air and the latter being the number of particles per cubic centimeter of air.² As smaller particles tend to be more numerous, particle counts are predominantly an indicator of ultrafine particles (UFP), or particles with a diameter less than 0.1 μm.

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Why do we care?

Humans have evolved to have several layers of protection from respirable hazards. The first is the hair-like vibrissae and mucus in the nasal cavity, which filter particles out of incoming air, and the second is the blood-air barrier in the alveolar region of the lungs, which serves to exchange fresh oxygen with CO₂ in the blood and prevents most other compounds from entering the blood stream. Larger particles, such as those that come from natural sources, are filtered out through the nasal passage, but particles smaller than 2.5 microns in diameter can penetrate into the lungs.³ Once there, they can cause a host of respiratory ailments including asthma, respiratory inflammation, decreased lung function and cancer.⁴⁻⁶ These damages are caused by two main pathways: 1) free radicals and free radical precursors (such as heavy metals and PAHs) in PM_{2.5} can lead to oxidative stress of the lung tissues⁷⁻¹⁰ and 2) the inflammatory response in reaction to PM_{2.5} can over time lead to irreparable damages in lung function.¹¹⁻¹³ The magnitude of the health burden from exposures to PM_{2.5} became apparent in the landmark Six Cities study, which found mortality rates increased by 16% for every 10 μg/m³ increase in PM_{2.5} concentration.¹⁴ For reference, the National Ambient Air Quality Standard (NAAQS) regulating PM_{2.5}, which was enacted shortly after the six cities study, is 12 μg/m³ while the average concentration in Beijing since 2008 is approximately 100 μg/m³.¹⁵ Studies published since the Six Cities study have related PM_{2.5} to a host of pulmonary and cardiovascular diseases.^{5,16-18}



Of increasing concern are UFP, which not only can penetrate to the alveolar region of the lungs but also are small enough to pass through the blood-air barrier and enter the blood stream.¹⁹⁻²² Recent research suggests that the impacts of PM_{2.5} may be driven in part by the particles on the small end of the size distribution. UFP, as measured by particle counts, have been linked to inflammatory markers in the blood, which are a precursor for cardiovascular disease.²³⁻²⁷

In a study of six U.S. cities, mortality rates increased by 16% for every 10 µg/m³ increase in ambient PM_{2.5} concentration.¹⁴

How does the building contribute to this issue?

Buildings can both exacerbate and attenuate particulate concentrations indoors. Mechanically ventilated buildings typically have filters in the heating, ventilation and air conditioning (HVAC) systems that collect particles from outdoor air entering the building. Filters are rated on a minimum efficiency reporting value (MERV) scale ranging from 1 to 20. Filters with higher MERV ratings remove smaller and a higher percentage of particles. For example, a MERV 14 filter will remove between 75-85% of particles in the 0.3-1 µm diameter range and over 90% of larger particles. HEPA filters are MERV 17-20 and remove 99.97% of particles greater than 0.3 µm in diameter.²⁸

Buildings are also home to many indoor sources of particulate matter, which can lead to higher levels indoors than out. Gas stoves generate combustion byproducts during cooking that cause dramatic spikes in particulate concentrations.²⁹ Other common indoor sources include personal care products, candles, incense and smoking. These indoor sources are most prevalent in residential environments, which are also more likely to be naturally ventilated (i.e. without filtration) than commercial buildings. In a study of two residential apartment buildings in the US, apartments with combustion sources had an average PM_{2.5} concentration of 94 µg/m³ compared to 53 µg/m³ in apartments without. The building that was mechanically ventilated had concentrations that were half of outdoor levels, while the building that was naturally ventilated had concentrations that were 30% higher than outdoor levels.³⁰

What can I do?

PM_{2.5} is a regional pollutant, where emissions from one state may influence concentrations in another. Therefore, ambient levels of particulate matter must be regulated at the state and national level, and since 2000 average PM_{2.5} concentrations have fallen from 13.5 µg/m³ to 8.5 µg/m³ in response to the NAAQS.³¹ While your surrounding context may be out of your control, exposures to particulate matter for residents in the same location differ substantially. In the same building, indoor levels can range from 75% lower than outdoor levels to 60 times larger.³⁰ In mechanically ventilated buildings, you can increase the MERV rating of the filters in your HVAC system to remove a larger percentage of the smaller, more toxic particles. In naturally ventilated spaces, standalone filtration units can provide the same benefit. In residential environments, source control – using electric over combustion cooking appliances, smoking outside the residence, reducing the use of spray products, etc. – can lower concentrations, in particular attenuating peak concentrations. In the case of gas stoves, ventilation hoods can exhaust combustion fumes outside during cooking events. Developers and building managers can investigate proximity to major outdoor sources such as highways and power plants to determine if other control techniques are necessary to manage indoor particulate levels.



REFERENCES

1. Das, R., B. Khezri, B. Srivastava, S. Datta, P.K. Sikdar, R.D. Webster, and X. Wang. 2015. Trace Element Composition of PM_{2.5} And PM₁₀ From Kolkata – A Heavily Polluted Indian Metropolis. *Atmospheric Pollution Research*, (5): p. 742-750.
2. EPA. Particulate Matter (PM) Basics, E.P. Agency, Editor. 2016.
3. Le, J., E.D. Ashley, M.M. Neuhauser, J. Brown, C. Gentry, M.E. Klepser, A.M. Marr, D. Schiller, J.N. Schwiesow, S. Tice, H.L. VandenBussche, G.C. Wood, and F. Society of Infectious Diseases Pharmacists Aerosolized Antimicrobials Task. 2010. Consensus Summary of Aerosolized Antimicrobial Agents: Application of Guideline Criteria. *Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy*, 30(6): p. 562-584.
4. Pope, C.A., 3rd, R. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, and G.D. Thurston. 2002. Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution. *JAMA*, 287(9): p. 1132-1141.
5. Katanoda, K., H. Satoh, K. Tajima, T. Suzuki, H. Nakatsuka, T. Takezaki, T. Nakayama, H. Nitta, K. Tanabe, and S. Tominaga. 2011. An Association Between Long-Term Exposure To Ambient Air Pollution and Mortality from Lung Cancer and Respiratory Diseases in Japan. *J Epidemiol*, 21(2): p. 132-143.
6. Yadav, A.K., K. Kumar, A.M.b.H.A. Kasim, M.P. Singh, S.K. Parida, and M. Sharan. 2003. Visibility and Incidence of Respiratory Diseases During the 1998 Haze Episode in Brunei Darussalam. *Pure and Applied Geophysics*, 160(1): p. 265-277.
7. Greenwell, L.L., T. Moreno, T.P. Jones, and R.J. Richards. 2002. Particle-Induced Oxidative Damage is Ameliorated by Pulmonary Antioxidants. *Free Radic Biol Med*, 32(9): p. 898-905.
8. Rahman, I. and W. MacNee. 1996. Role of Oxidants/Antioxidants in Smoking-Induced Lung Diseases. *Free Radic Biol Med*, 21(5): p. 669-681.
9. Kelly, F.J. 2003. Oxidative Stress: Its Role in Air Pollution And Adverse Health Effects. *Occup Environ Med*, 60(8): p. 612-616.
10. Valavanidis, A., K. Fiotakis, E. Bakeas, and T. Vlahogianni. Electron Paramagnetic Resonance Study of the Generation of Reactive Oxygen Species Catalysed by Transition Metals and Quinoid Redox Cycling by Inhalable Ambient Particulate Matter. (1743-2928 (Electronic)).
11. Gripenback, S., L. Lundgren, A. Eklund, C. Liden, L. Skare, G. Tornling, and J. Grunewald. 2005. Accumulation of Eosinophils and T-Lymphocytes in the Lungs After Exposure to Pinewood Dust. *Eur Respir J*, 25(1): p. 118-124.
12. Gordon, S. 2003. Alternative Activation of Macrophages. *Nat Rev Immunol*, 3(1): p. 23-25.
13. Park, E.J., J. Roh, Y. Kim, K. Park, D.-S. Kim, and S.-D. Yu. 2011. PM_{2.5} Collected in a Residential Area Induced Th1-Type Inflammatory Responses With Oxidative Stress in Mice. *Environ Res*, 111(3): p. 348-355.
14. Laden, F., J. Schwartz, F.E. Speizer, and D.W. Dockery. 2006. Reduction in Fine Particulate Air Pollution and Mortality: Extended Follow-up of the Harvard Six Cities Study. *American Journal of Respiratory and Critical Care Medicine*, 173(6): p. 667-672.
15. AQICN. Beijing Air Pollution: Real-time Air Quality Index (AQI). 2016.
16. Dominici, F., P. R., M.L. Bell, L. Pham, A. McDermott, S.L. Zeger, and J.M. Samet. 2006. Fine Particulate Air Pollution and Hospital Admission for Cardiovascular and Respiratory Diseases. *JAMA*, 295(10): p. 1127-1134.
17. Peters, A., D. Dockery, J.E. Muller, and M.A. Mittleman. 2001. Increased Particulate Air Pollution and the Triggering of Myocardial Infarction. (1524-4539 (Electronic)).
18. Lee, S.L., Y.L. Wong Wh Fau - Lau, and Y.L. Lau. 2006. Association Between Air Pollution and Asthma Admission Among Children in Hong Kong. (0954-7894 (Print)).
19. Knol, A.B., J.J. de Hartog, H. Boogaard, P. Slottje, J.P. van der Sluijs, E. Lebret, F.R. Cassee, J.A. Wardekker, J.G. Ayres, P.J. Borm, B. Brunekreef, K. Donaldson, F. Forastiere, S.T. Holgate, W.G. Kreyling, B. Nemery, J. Pekkanen, V. Stone, H.E. Wichmann, and G. Hoek. 2009. Expert Elicitation on Ultrafine Particles: Likelihood of Health Effects and Causal Pathways. *Particle and Fibre Toxicology*, 6: p. 19-19.
20. WHO: Global Atlas on Cardiovascular Disease Prevention and Control. Edited by: Mendis S, Puska P, Norrving B. 2011, WHO, Geneva, http://whqlibdoc.who.int/publications/2011/9789241564373_eng.pdf.
21. Li, N., S. Georas, N. Alexis, P. Fritz, T. Xia, M.A. Williams, E. Horner, and A. Nel. 2016. A Work Group Report on Ultrafine Particles (American Academy Of Allergy, Asthma & Immunology): Why Ambient Ultrafine and Engineered Nanoparticles Should Receive Special Attention for Possible Adverse Health Outcomes in Human Subjects. *J Allergy Clin Immunol*, 138(2): p. 386-96.
22. Oberdorster, G. 2001. Pulmonary Effects of Inhaled Ultrafine Particles. *Int Arch Occup Environ Health*, 74(1): p. 1-8.
23. Fuller, C.H., P.L. Williams, M.A. Mittleman, A.P. Patton, J.D. Spengler, and D. Brugge. 2015. Response of Biomarkers of Inflammation and Coagulation to Short-Term Changes in Central Site, Local, and Predicted Particle Number Concentrations. *Annals of Epidemiology*, 25(7): p. 505-511.



24. Lane, K.J., J.I. Levy, M.K. Scammell, J.L. Peters, A.P. Patton, E. Reisner, L. Lowe, W. Zamore, J.L. Durant, and D. Brugge. 2016. Association of Modeled Long-Term Personal Exposure to Ultrafine Particles with Inflammatory and Coagulation Biomarkers. *Environment International*, 92–93: p. 173-182.
25. Ostro, B., J. Hu, D. Goldberg, P. Reynolds, A. Hertz, L. Bernstein, and M.J. Kleeman. 2015. Associations of Mortality With Long-Term Exposures to Fine and Ultrafine Particles, Species And Sources: Results from the California Teachers Study Cohort. *Environ Health Perspect*, 123(6): p. 549-56.
26. Viehmann, A., S. Hertel, K. Fuks, L. Eisele, S. Moebus, S. Mohlenkamp, M. Nonnemacher, H. Jakobs, R. Erbel, K.H. Jockel, and B. Hoffmann. 2015. Long-Term Residential Exposure to Urban Air Pollution, and Repeated Measures of Systemic Blood Markers of Inflammation and Coagulation. *Occup Environ Med*, 72(9): p. 656-63.
27. Li, Y., K.J. Lane, L. Corlin, A.P. Patton, J.L. Durant, M. Thanikachalam, M. Woodin, M. Wang, and D. Brugge. 2017. Association of Long-Term Near-Highway Exposure to Ultrafine Particles with Cardiovascular Diseases, Diabetes and Hypertension. *Int J Environ Res Public Health*, 14(5).
28. NAFA. Understanding MERV. 2017 [cited 2017 May 9th]; Available from: <https://www.nafahq.org/understanding-merv/>.
29. Wallace, L. and W. Ott. 2011. Personal Exposure to Ultrafine Particles. *J Expo Sci Environ Epidemiol*, 21(1): p. 20-30.
30. Patton, A.P., L. Calderon, Y. Xiong, Z. Wang, J. Senick, M. Sorensen Allacci, D. Plotnik, R. Wener, C.J. Andrews, U. Krogmann, and G. Mainelis. 2016. Airborne Particulate Matter in Two Multi-Family Green Buildings: Concentrations and Effect of Ventilation and Occupant Behavior. LID - 10.3390/ijerph13010144 [doi] LID - E144 [pii]. *International Journal of Environmental Research and Public Health*, 13(1).
31. EPA. Particulate Matter (PM2.5) Trends. 2017 [cited 2017 4/14/2017]; Available from: <https://www.epa.gov/air-trends/particulate-matter-pm25-trends>.