Adverse Health Effects of Air Pollution and Particulate Matter

Introduction:

EPA Regulations: Air Permits allow private and public sector facilities to emit pollutants within specific limits that may often be lagging behind the most current science data. Updating emission limits is a slow process hampered by the need to move cautiously since tighter regulations result in potential increased costs for government regulators and start-up operational costs for the permit applicants. Political resolve, lobbying efforts, and public opinion play significant roles in determining environmental regulations.

Most importantly at stake is our environmental and public health. After all the health of each of us is personal. This brief discussion is focused on the adverse health effects of air pollution and especially particulate matter from major mobile (internal combustion engines) and stationary sources (power plants).

http://www.stateoftheair.org/2013/health-risks/health-risks-particle.html

—U.S. Environmental Protection Agency, Integrated Science Assessment for Particulate Matter, December 2009. EPA 600/R-08/139F.conservation of other natural resources and materials.

An EPA review of particle pollution released in December 2009 led by the Clean Air Scientific Advisory Committee examined research published between 2002 and May 2009. The EPA concluded that particle pollution caused multiple, serious threats to health**:**

-Causes early death (both short-term and long-term exposure)
-Causes cardiovascular harm (e.g. heart attacks, strokes, heart disease, congestive heart failure)
-Likely to cause respiratory harm (e.g. worsened asthma, worsened COPD, inflammation)
-May cause cancer
-May cause reproductive and developmental harm

In October of 2013, the International Agency for Research on Cancer (IARC), which is an arm of the WHO, concluded after studying over 2000 peer reviewed journal articles, that outdoor air pollution and particulate matter both cause lung cancer in humans.

The IARC cited one 2010 study that estimated fine particles (PM2.5) alone contribute to 223,000 deaths from lung cancer worldwide.

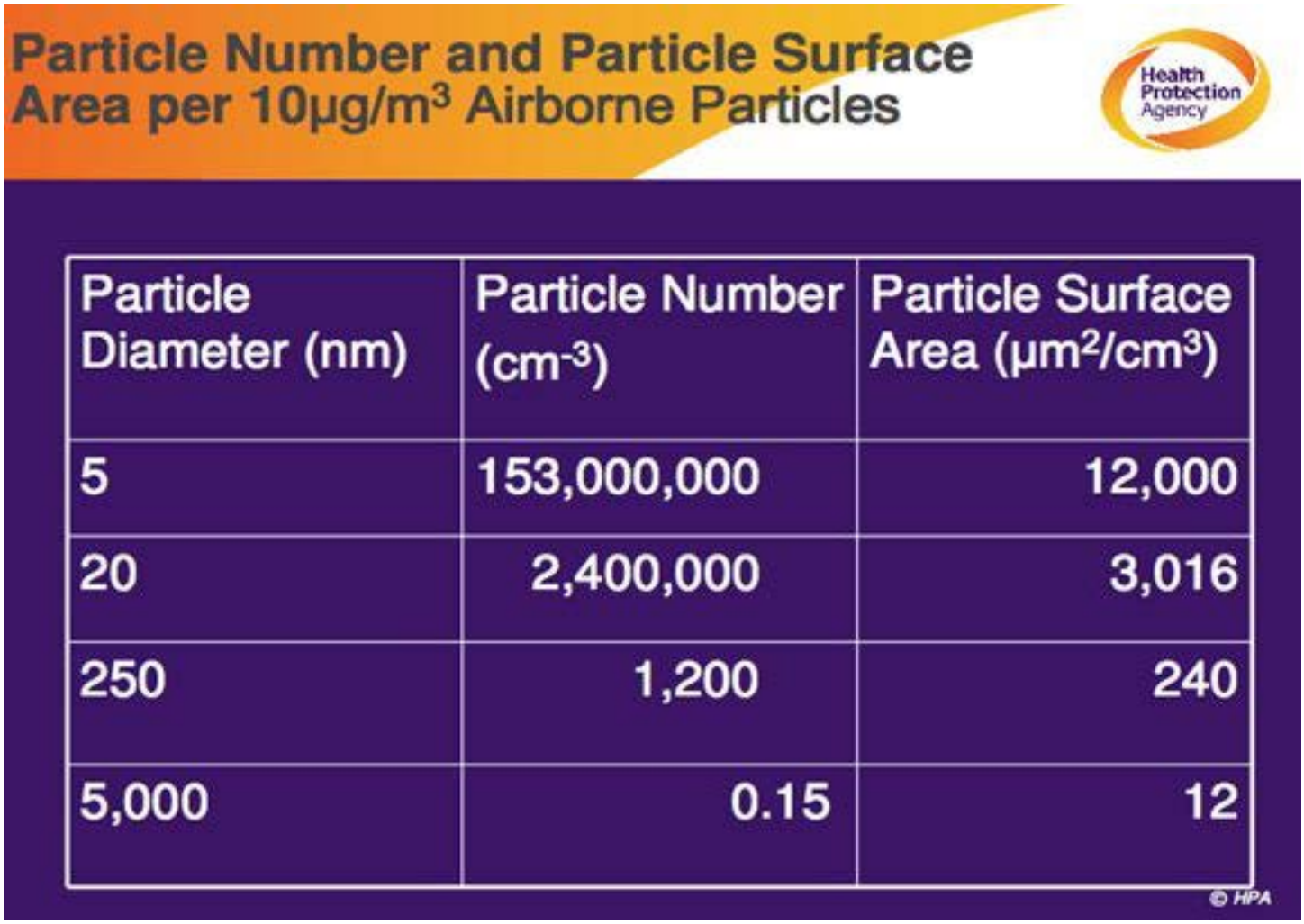
The WHO has additionally addressed indoor air pollution as a major source of illness and death (http://www.who.int/indoorair/en/).

4.3 million deaths /3 billion people heating and cooking at home:
The WHO reports that of the 3 billion people who cook and heat their homes using solid fuels such as wood, charcoal, coal, dung, and crop wastes on open fires or traditional stoves, 4.3 million people die annually from exposure to household air pollution.

Background Information
The number of alveoli in the human... [Am J Respir Crit Care Med. 2004] - PubMed - NCBI.pdf

Am J Respir Crit Care Med. 2004 Jan 1;169(1):120-4. Epub 2003 Sep 25.
The number of alveoli in the human lung.
Ochs M, Nyengaard JR, Jung A, Knudsen L, Voigt M, Wahlers T, Richter J, Gundersen HJ.

Summary of Abstract:
In six adult human lungs, the mean alveolar number was 480 million (range: 274-790 million; coefficient of variation: 37%). Alveolar number was closely related to total lung volume, with larger lungs having considerably more alveoli. The mean size of a single alveolus was rather constant with 4.2 x 10(6) microm3 (range: 3.3-4.8 x 10(6) microm3; coefficient of variation: 10%), irrespective of the lung size. One cubic millimeter lung parenchyma would then contain around 170 alveoli.



Particle Diameter (nm)	Particle Number (cm^{-3})	Particle Surface Area ($\mu m^2/cm^3$)
5	153,000,000	12,000
20	2,400,000	3,016
250	1,200	240
5,000	0.15	12

PM_1 Particles at Coal- and Gas-Fired Power Plant Work Areas

Hicks J B et al. Ann Occup Hyg 2012;56:182-193

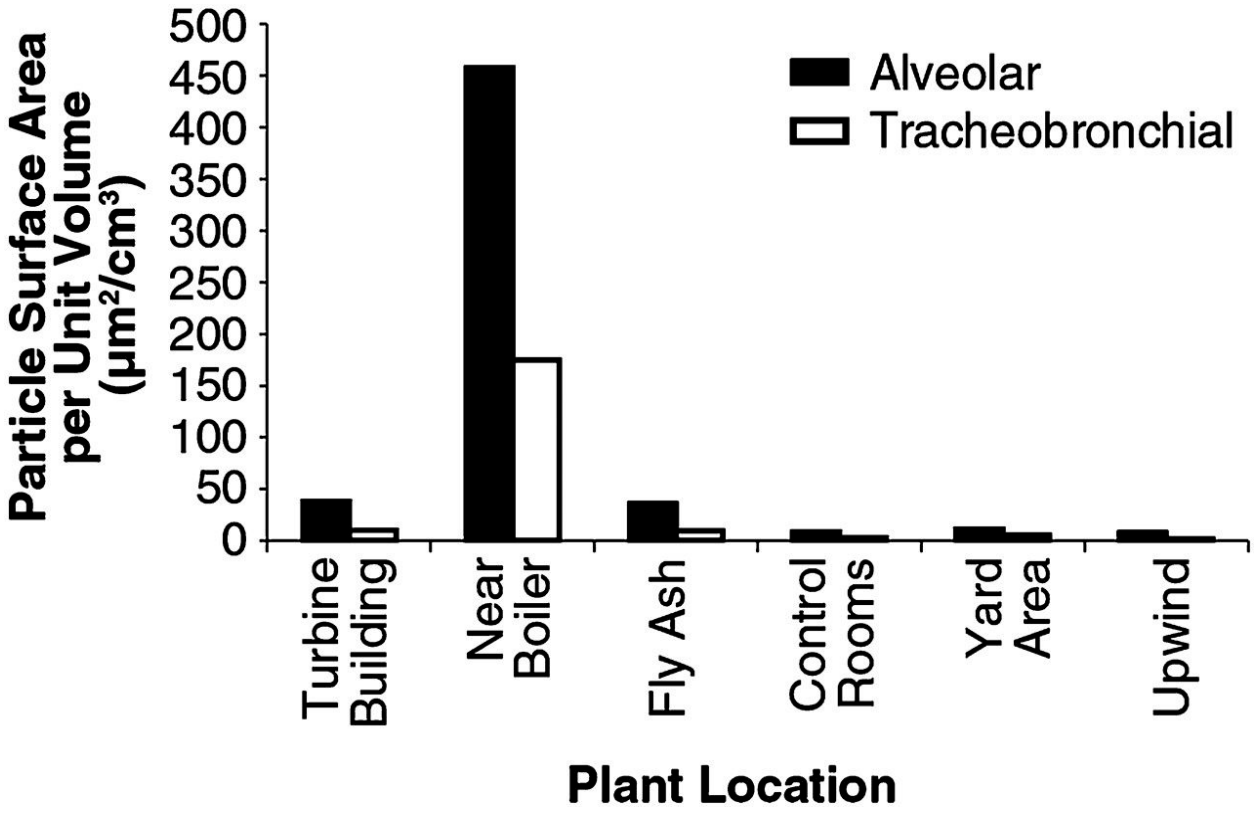


Fig. 2.
Plant Site 1, mean particle surface area (alveolar and tracheobronchial fractions) by plant location.

1) In Vivo Studies of the Deposition and Biokinetics of Inhaled Nanoparticles

Future determinations of adverse health effects from fine and ultra fine particles, in particular the ultra fine forms will be partially largely determined by studies of particle biokinetics which examines the biophysical and biochemical nature of the particles and how they pass through airways and biological tissues. These politically charged studies will no doubt fuel many regulatory disputes.

Biokinetics Factors include:
- The physical size and chemical characteristics of the particles
- How far they enter the breathing passages based largely on size,

- Whether the particles are retained or expelled by muco cilliary or cough mechanisms
- How they are transported across membranes passively or via bronco alveolar macrophages
- The retention time and the particles' chemical reactivity
- Whether retained particles are transported into the blood or further into target organs

The **macro**scopically assessed amount of particles in the respiratory tract and secondary target organs provide dose estimates for toxicological studies in the level of the whole organism.

From a **micro**scopic analysis level, looking at individual particles' effect on cell and subcellular components helps create adverse health effects inventories which can serve as regulatory guidelines.

It's vital that quantitative analysis and the use of particle doses from real world exposures be used whenever possible so that the time consuming and disputable toxic equivalent methods be avoided.

2) Acute inflammatory responses in the airways and peripheral blood after short-term exposure to diesel exhaust in healthy human volunteers.
Sundeep Salvi Et Al. Am J Respir Crit Care Med 1999 Mar;159(3):702-9
- To determine the impact of DE on human airways, we exposed 15 healthy human volunteers to air and diluted DE under controlled conditions for 1 h with intermittent exercise.
- Lung functions were measured before and after each exposure.
- Blood sampling and bronchoscopy were performed 6 h after each exposure to obtain airway lavages and endobronchial biopsies.
- While standard lung function measures did not change following DE exposure, there was a significant increase in neutrophils and B-lymphocytes in airway lavage, plus increases in histamine and fibronectin.

- The bronchial biopsies obtained 6 h after DE exposure showed a significant increase in neutrophils, mast cells, CD4+ and CD8+ T lymphocytes along with upregulation of the endothelial adhesion molecules ICAM-1 and VCAM-1, with increases in the numbers of LFA-1+ cells (cells that induce binding of immune cells for recruitment to a site of infection) in bronchial tissue.

Conclusion: At high ambient concentrations, acute short-term DE exposure produced a well-defined and considerable systemic and pulmonary inflammatory response in healthy human volunteers, while still retaining standard lung function measurements. This inflammatory response is often associated with heart disease and cardiovascular events.

3) Translocation of Inhaled Ultrafine Particles to the Brain
G. Oberdorster, Et. Al. Inhalation Technology 2004, Vol. 16, No. 6-7, Pages 437-445
- UFP (<100nm) have already been detected in extrapulmonary organs such as the liver and the olfactory bulb in the brain within 4 to 24 hr post exposure.
- The current study exposed rats for 6 hours to 34nm ^{13}C UFP at a $160ug/m^3$ concentration. Rats were necropsied 1, 3, 5, & 7 days after exposure.
- Pulmonary and lung tissues had an average ^{13}C concentration of 1.39 ug/g of tissue on day 1 and by day 7 mucocilliary and macropage clearance reduced the concentration to 0.59 ug/g of tissue. The olfactory bulb concentration went up from 0.39 ug/g on rat

tissues sampled on day 1 to 0.43ug/g on day 7. Depending on particle size, >50% of inhaled UFP can be deposited in the nasopharingealregion during nasal breathing. About 20% of the UFP deposited on the olfactory mucosa can translocate to the olfactory nerve to the olfactory bulb in the brain.

The UFP increases in olfactory bulbs are consistent with earlier studies in nonhuman primates and rodents that demonstrated that intranasally instilled solid UFP translocate along axons of the olfactory nerve into the CNS.

Conclusion: The CNS can be targeted by airborne solid ultrafine particles and that the most likely mechanism is from deposits on the olfactory mucosa of the nasopharyngeal region of the respiratory tract and subsequent translocation via the olfactory nerve.

Whether this translocation of inhaled UFP can cause CNS effects needs to be determined in future studies.

4) Correlation between particle size, in vivo particle persistence, and lung injury.
G Oberdörster, J Ferin, and B E Lehnert - Environ Health Perspect. Oct 1994; 102(Suppl 5): 173–179.

Particle translocation to the pulmonary interstitium and persistence there of the ultrafine TiO2); greater epithelial effects (Type II cell proliferation; occlusion of pores of Kohn) and the beginning of interstitial fibrotic foci with ultrafine TiO2; significant sustained impairment of alveolar macrophage function after ultrafine TiO2 exposure as measured by the clearance of test particles.

5) Air pollution and inflammation in type 2 diabetes: a mechanism for susceptibility
M S O'Neill[1], A Veves[2], J A Sarnat[3], A Zanobetti[4], D R Gold[4], P A Economides[5], E S Horton[6], J Schwartz[4] *Occup Environ Med* 2007;**64**:373-379

Particulate air pollution has been associated with several adverse cardiovascular health outcomes, and people with diabetes may be especially vulnerable. One potential pathway is inflammation and endothelial dysfunction

6) **Long-term Exposure to Ambient Fine Particulate Pollution Induces Insulin Resistance and Mitochondrial Alteration in Adipose Tissue**
Xiaohua Xu, et al Toxicological Sciences, Volume 124, Issue 1 > Pg 88-98.

Long-term ambient $PM_{2.5}$ exposure induces impaired glucose tolerance, insulin resistance, inflammation, and mitochondrial alteration both in number and average size, and thus, it is a risk factor for the development of type 2 diabetes.

7) **Association Between Fine Particulate Matter and Diabetes Prevalence in the U.S.**
John F. Pearson, BS,1,2 Chethan Bachireddy, BS,1,3 Sangameswaran Shyamprasad, MS,1 Allison B. Goldfine, MD,4,5 and John S. Brownstein, PHD1,4,6,7 Diabetes Care. Oct 2010; 33(10): 2196–2201.

Diabetes prevalence increases with increasing PM2.5 concentrations, with a 1% increase in diabetes prevalence seen with a 10 µg/m3.

Even for counties within guidelines for EPA PM2.5 exposure limits, those with the highest exposure showed a >20% increase in diabetes prevalence compared with that for those with the lowest levels of PM2.5, an association that persisted after controlling for diabetes risk factors.

Conclusion: Our results suggest PM2.5 may contribute to increased diabetes prevalence in the adult U.S. population. These findings add to the growing evidence that air pollution is a risk factor for diabetes.

8) **Epigenetic Changes Through Airborne Pollutants**
Hyand-Min Byun, Harvard School of Public Health - Clinical Epigenetics Society March 6, 2014

Effects	Disease
IL-6 >	-Asthma
IL-8>	-Heart Disease
TNF>	-Neurological disorders
Blood Pressure>	-Lung Cancer
ROS generation>	-Leukemia
Pro-inflamatory signaling>	
Hypercoagulability>	
Coronary flow<	

9) **Ultrafine particle emissions: Comparison of waste-to- energy with coal- and biomass- fired power plants** Lital Yinon Columbia University Jan 2010

Combustion of coal in thermoelectric power plants is a major source for direct emission of UFP. In 2007, almost 50% of the approximately 4.16 billion MWh generated in the United States, corresponding to 1.047 billion tons of coal, were produced by coal-fired power plants (DOE).

The particle size distribution at the ESP inlet showed a high number concentration of particles with the mode around 75nm and number concentration of about 10^8 particles/cm^3. At the ESP outlet, the number concentration of particles larger than 70 nm decreased about three orders of magnitude. However, the reduction in the number concentration of particles smaller than 70 nm decreased significantly at particle sizes below 70 nm. Wang et al (2008) observed a number concentration of $6x10^8$ particles/cm^3 for particles in the size range of 5.6-560nm in a coal-fired power plant in China.

Enrichment of metals in UFP- summary of various studies

Study	UFP Enriched
Seames and Wendt, 2000	As, Cd, Se
Yoo et al, 2005	Cd, Cr, Mg, Mn, Ni, Pb,
Sui et al, 2007	Ca, Cr, Cu, Fe, Mg, Mn, Pb, Zn
Wang et al, 2008	As, Cd, S, Sb, Se, Pb, and to a lesser extent: Zn, Cr, Ni, Cu, V, Co, Cr, Ni, Mg, Mn

UFP emission from biomass- fired power plants

Biomass contains various trace elements such as Zn, Cd, Cu, Cr, Pb, and Hg, which vary considerably, depending on the biomass type and source (Demibras, 2005). Biomass combustion leads to relatively high PM emissions and the particle size distribution varies for different fuels and combustor systems (Nussbaumer, 2003). Combustion of wood in different laboratory scale combustion systems showed that the majority of the particles emitted were in the size range of 30 to 300 nm with a mode of 80 nm (Wieser and Gaegauf, 2000). The particle number concentration varied widely from 10^7-10^{10} particles/cm^3. Chemical analysis of particles emitted from wood combustion showed that metals such as Zn and Mg were concentrated in the UFP (Johansson et al, 2003; Tissari et al, 2008).

The number concentration of particles in the size range of 17 to 300 nm emitted from boilers operating on three different biofuels (sawdust, wood pallets, and forest residues) ranged from 6.3×10^7 to 7.7×10^7 particles/cm^3 with a particle size mode ranging between approximately 70-100 nm (Weirzbicka et al, 2005). The concentration of Zn, Cd, Pb and Cr in the particles depended on the type of fuel, but was generally significantly higher in the smaller particles.