

Preventable Deaths from PM2.5 Air Pollution in Fairbanks, AK

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Summary: Fine particulate matter (PM2.5) air pollution is associated with an 11.5 to 23.5 percent increased number of deaths in Fairbanks, Alaska. The higher end of the range applies to areas with higher PM2.5 concentrations, such as North Pole. Each year, an estimated 50 to 101 deaths in the Fairbanks North Star Borough (FNSB) are associated with PM2.5 air pollution. Reducing ambient PM2.5 concentrations and ambient exposure would prevent these premature deaths.

A strengthening body of scientific studies supports a causal, essentially linear relationship between PM2.5 exposure and morbidity and mortality. Particles less than 2.5 micrometers in diameter (PM2.5), referred to as “fine” particles, have been found to pose the greatest health risks. Because of their small size (approximately 1/30th the average width of a human hair), fine particles can lodge deeply into the lungs, trigger acute cardiovascular events, and promote chronic disease.

Implications: Lives are lost year after year as air pollution is prolonged. Taking every action possible to reduce air pollution will save lives now.

Methods

Mortality Formulas

1. Each $1 \mu\text{g}/\text{m}^3$ increase in PM2.5 is associated with 1% increased mortality.
2. Each $1 \mu\text{g}/\text{m}^3$ increase in PM2.5 is associated with 1.4% increased mortality.

Formula 1 - Brook 2010: “The overall evidence from the cohort studies demonstrates **on average an approximate 10% increase in all-cause mortality per 10- $\mu\text{g}/\text{m}^3$ elevation in long-term average PM2.5 exposure.**” (Bold added.)

Brook, Robert D, et al. “American Heart Association Scientific Statement on Particulate Matter Air Pollution and Cardiovascular Disease.” *Circulation* 121 (2010): 2331-78.

<http://circ.ahajournals.org/content/121/21/2331.full>>

Formula 2 - Lepeule 2012: “Each **10- $\mu\text{g}/\text{m}^3$ increase in PM2.5 was associated with a 14% increased risk of all-cause death** [95% confidence interval (CI): 7%, 22%], a 26% increase in cardiovascular death (95% CI: 14%, 40%), and a 37% increase in lung-cancer death (95% CI: 7%, 75%).” (Bold added.)

Lepeule, Johanna, et al. “Chronic Exposure to Fine Particles and Mortality: An Extended Follow-up of the Harvard Six Cities Study from 1974 to 2009.” *Environ Health Perspect* 120.7 (2012): 965-70. <<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3404667>>

Applying Mortality Formulas to Local Data

Formula range: **1 to 1.4%** increased mortality for each $1 \mu\text{g}/\text{m}^3$ annual average PM2.5 increase.

Death rate: **430.8** = Three-year crude death rate by residence for FNSB 2010-2012, per 100,000

Population: **100,320**= Population of Fairbanks North Star Borough, 2012

Annual average PM2.5, Fairbanks and North Pole, AK:

11.5 $\mu\text{g}/\text{m}^3$ = annual average PM2.5, State Office Building FRM, downtown Fairbanks, 2010-2012 (Hartig to McLerran correspondence 12/10/2013)

16.8 $\mu\text{g}/\text{m}^3$ = annual average PM2.5, North Pole Fire Station FRM, 2012
(http://www.epa.gov/airquality/airdata/ad_rep_mon.html)

Results

11.5 to 23.5% increased mortality associated with PM2.5 air pollution exposure, with the higher rates of mortality expected in areas of higher PM2.5 concentrations, such as North Pole.

Calculation of increased mortality range using mortality formulas:

11.5% increased mortality = 11.5 annual average Fairbanks x 1% mortality increase
(Brook 2010)

23.5% increased mortality = 16.8 annual average North Pole x 1.4% mortality increase
(Lepeule 2012)

50 to 101 premature deaths each year in FNSB associated with PM2.5 air pollution.

Calculation of premature deaths per year range:

50 premature deaths = 11.5% increased mortality x 430.8 crude death rate

101 premature deaths = 23.5% increased mortality x 430.8 crude death rate

Regulatory Background

In 2012, the health-based Federal standard for annual average PM_{2.5} was lowered from 15 to 12 micrograms per cubic meter and the Federal standard for daily average PM_{2.5} of 35 µg/m³, established in 2006, was retained. In 2009, the most populated areas of the Fairbanks North Star Borough (FNSB) were designated as a nonattainment area for violation of the daily standard. The Environmental Protection Agency recently determined that, beginning in 2015, data collected at the North Pole Fire site will be used in nonattainment calculations. [EPA letter 11/19/2014](#). [KUAC news on EPA decision 12/12/2014](#).

To meet Federal requirements, the State of Alaska has established new regulations and proposed additional regulations and a State Implementation Plan (SIP) to meet PM_{2.5} attainment. [Proposed regulations and SIP](#). To be considered, comments must be received by 5:00 pm on Dec. 19, 2014. [Electronic comment form](#). The proposed regulations and SIP do not consider increased deaths, injuries, or associated costs that could be reduced or prevented with effective PM_{2.5} controls.

Data Sources

Mortality: Brook 2010 and Lepeule 2012 studies were written by experts in the field, published in credible periodicals, and have been widely cited. Lepeule 2012 is the most current.

Crude death rate, Fairbanks North Star Borough: Three-year crude death rate by residence for FNSB 2010-2012, Alaska Bureau of Vital Statistics, personal communication, 8/28/2014. [Alaska Bureau of Vital Statistics (907)465-8604, BVSResearch@alaska.gov]

Population, FNSB: 2012 estimates, Alaska Dept. of Labor and Workforce Development <http://laborstats.alaska.gov/pop/estimates/data/TotalPopulationBCA.xls> (accessed 12/10/2014)

Annual average PM_{2.5}:

Fairbanks 2010-2012, ADEC Commissioner Hartig to EPA Regional Administrator McLerran correspondence 12/10/2013

North Pole 2012, North Pole Fire Station #3, site ID 02-090-0035, 96 samples from Mar. 1 to Dec. 29, 2012, EPA AirData, http://www.epa.gov/airquality/airdata/ad_rep_mon.html (accessed 9/30/2014)

A more complete annual average for North Pole may be calculated using data from the second to fourth quarter of 2012 and the first quarter of 2013, giving an annual average of **20.4 µg/m³**. EPA Air Trends, Table 5

http://www.epa.gov/airtrends/pdfs/PM25_DesignValues_20112013_FINAL_08_28_14.xlsx (accessed 12/10/2014). Relying on a more conservative annual average of **16.8 $\mu\text{g}/\text{m}^3$** likely underestimates mortality.

Annual average PM2.5 data is from 1-in-3-day Federal Reference Method (FRM) monitors, the class of monitor used in the referenced health studies and regulatory attainment.

Limitations

Following are notes regarding the limitations of this analysis. Each represents an area for additional examination. Further, each indicates that the method applied in this analysis may underrepresent local PM2.5 impact on human health and lives. If true, the health benefit of reducing PM2.5 is far greater than presented by our initial analysis.

The mortality estimates in this analysis are all cause and do not break out increased mortality associated with short-term PM2.5 exposure of days, hourly spikes, or deaths due to a specific cause. For example, the North Pole Fire monitor recorded hourly spikes of 329 $\mu\text{g}/\text{m}^3$ in 2014 and 843 $\mu\text{g}/\text{m}^3$ in 2013. Neighborhood impacts on health from one coal boiler or, as identified in a key study, one wood-fired hydronic heater can spike PM2.5 over 1,000 $\mu\text{g}/\text{m}^3$.¹ Studies have found significant associations between short-term PM2.5 exposure and cardiovascular death. In 2010, the state conducted a study of 5,718 hospital visits with mean 24-hour PM2.5 concentration of 20.1 $\mu\text{g}/\text{m}^3$.^{2,3} For each 10 $\mu\text{g}/\text{m}^3$ increase in the mean 24-hour PM2.5 concentration one day prior to a hospital visit, the 2010 study found a 6 to 7 percent increased rate of hospitalization for cerebrovascular or respiratory tract infection-coded visits.

Reductions in life expectancy are not considered in this analysis. Mortalities associated with PM2.5 occur with every stage of life. Individuals at higher risk of injury and death from PM2.5 include those with heart and lung disease, older adults, children, diabetics, pregnant women and fetuses, athletes, smokers, and anyone with higher or prolonged exposures (such as individuals who work in an area with polluted air).

These estimates do not consider the costs of increased deaths and injuries associated with PM2.5 air pollution. The health benefits of improved air quality have not been considered as an offset to any possible increased cost of controls on residential and commercial heating with wood, waste oil, and coal. Savings from reducing PM2.5 air pollution include reduced economic costs from premature deaths (including lost productivity), medical expenses, and mitigation such as relocation and air filters. Social costs, such as lost care and affection, are significant impacts of premature death. In 2006, EPA placed the economic value of one human life as \$7.4 million (\$8.4 million in 2014 dollars).⁴ In 2012, the US Department of Transportation placed the value of one human life as \$9.1 million (\$9.3 in 2014 dollars).⁵ In 30 years, one person's

earnings would total \$1.4 million, using an average per capita personal income of \$45,432 for Fairbanks in 2012.⁶

Also not considered in this analysis is the relationship between cardiac death and the lowest PM2.5 concentrations. At low doses, the relationship between PM2.5 and sudden death, the signature adverse outcome of PM2.5 exposure, appears supralinear, not linear.⁷ On many days during the summer, PM2.5 concentrations range between 2 and 4 $\mu\text{g}/\text{m}^3$, among the cleanest ambient air in the world. The significance of a supralinear relationship is that even more benefit can be derived from improving air quality when PM2.5 is low.

Another aspect not considered in this analysis is that PM2.5 from wood smoke is likely more toxic than Lepeule 2012 or Brook 2010 suggest. Health studies have generally not been conducted in communities with such a high proportion of wood smoke. Source apportionment studies for the Fairbanks PM2.5 nonattainment area identified between 57.9 and 85.5 percent of winter PM2.5 is wood smoke, depending on location.^{8,9} Toxicological studies suggest that wood smoke particulate pollution is more toxic to lung macrophages than an equivalent concentration of similar-sized particulate pollution found in typical urban smog.^{10,11,12,13} The very small size of the particulate emissions and high levels of polycyclic aromatic hydrocarbons and free radicals from wood smoke may account for its excessive toxicity compared to fossil fuel generated PM.¹⁴ Ultrafine particles are more potent in inducing inflammatory responses than fine particles.^{15,16,17,18} Compared to cigarette smoke, free radicals from wood smoke stay chemically active up to forty times as long.¹⁹ Health consequences from Fairbanks PM2.5 were likely worse than estimated in this analysis, due to the concentrations of highly toxic wood smoke.

Use of Federal Equivalent Methods (FEM), rather than Federal Reference Method (FRM) monitor data, would increase samples threefold, reducing potential sampling error. FEM monitors are less expensive and require less labor to operate than FRM monitors, and “would provide enhanced spatial coverage for PM2.5 measurements and should improve the linkages (e.g., concentration-response functions) between ambient concentrations of PM2.5 and health outcomes such as respiratory and cardiovascular disease.”²⁰ More PM2.5 samples from a greater number of locations should allow improved predictions of health effects. FRM data takes four to twelve weeks to return from the lab and represents at best one out of three days, just one-third of the days each year. In contrast, FEM data updates hourly every day. Semi-continuous hourly/daily FEM monitors inform the public of current air conditions, and agencies use FEM monitors to determine air emergencies and forecasts.

In addition, use of FRM monitor data in health studies may reduce estimates of harm. FEM monitors have long been known to capture a higher proportion of the PM2.5 mass than FRMs.²¹ The largest reason for this difference appears to be because FEM monitors capture the

semivolatile component of PM_{2.5}, “known to exceed 30 percent of measured concentration levels.”²² Semivolatile particles volatilize off FRM filters before lab analysis is conducted. Combustion of wood and coal are important sources of semivolatile components. “Nitrate and OC [organic carbon] are the two components that make up most, if not all, of the semivolatile fine particle mass lost from the FRM.”²³ A 2012 Fairbanks speciation study found organic carbon was the greatest contributor to PM_{2.5} mass, between 42 and 58 percent.²⁴ The 2012 study provided as background, “Compared with PM_{2.5} concentrations in the lower 48, Fairbanks has some of the highest wintertime ambient PM_{2.5} concentrations measured throughout the United States.” Further, semivolatiles, such as the lost FRM mass, appear more toxicologically potent.²⁵ FEM data available to the public on Envistaweb and other sites has been adjusted downward to make it more FRM-like.²⁶ FRM monitor data, or downward-adjusted FEM data that fail to count as much as nearly one-third of the PM_{2.5} mass, can confound health studies and emergency response. Underestimating exposure and harm to health is of greatest concern in areas with high PM_{2.5} concentrations like Fairbanks and especially North Pole.

¹ Lisa Rector, et al, “Assessment of Outdoor Wood-Fired Boilers.” NESCAUM, 2006, 5-4. <<http://www.nescaum.org/documents/assessment-of-outdoor-wood-fired-boilers>>.

² Joe McLaughlin, “Association between Air Quality and Hospital Visits — Fairbanks, 2003–2008.” ADHSS, Bulletin No. 26 (2010). <http://www.epi.hss.state.ak.us/bulletins/docs/b2010_26.pdf>.

³ Bill Hogan, “Fact Sheet: Air quality and hospital visits – Fairbanks study.” Commissioner’s Office, ADHSS. 2010. <http://dhss.alaska.gov/News/Documents/press/2010/FBXstudy_fs_083010.pdf>.

⁴ “Frequently Asked Questions on Mortality Risk Valuation.” National Center for Environmental Economics. N.p., Web. 11 Dec. 2014. <<http://yosemite.epa.gov/EE%5Cepa%5Ceed.nsf/webpages/MortalityRiskValuation.html#whatisvsl>>.

⁵ US Dept of Transportation. “Guidance on Treatment of the Economic Value of a Statistical Life in the U.S. Department of Transportation Analyses,” Polly Trottenberg. Office of the Secretary. 2013. <<http://www.dot.gov/sites/dot.dev/files/docs/VSL%20Guidance.doc>>.

⁶ AK Dept of Labor and Workforce Development. “Income Data for Alaska and U.S.” Research and Analysis. N.p., Web. 11 Dec. 2014. <<http://laborstats.alaska.gov/income/income.htm>>.

⁷ Annette Peters, “Air Quality and Cardiovascular Health: Smoke and Pollution Matter.” *Circulation* 120 (2009): 924-27. <<http://circ.ahajournals.org/content/120/11/924.full.pdf>>.

⁸ Tony Ward, Fairbanks, “Alaska PM_{2.5} Source Apportionment Research Study.” Univ of MT, Center for Environmental Health Sciences (2013): 12. <http://dec.alaska.gov/air/anpms/comm/docs/fbxSIPpm2-5/Fairbanks_CMB_Report_Univ_MT_Final_030113.pdf>.

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- ⁹ Tony Ward, et al, "Source Apportionment of PM2.5 in a Subarctic Airshed - Fairbanks, Alaska," *Aerosol and Air Quality Research*, 12 (2012): 539. <http://aaqr.org/VOL12_No4_August2012/8_AAQR-11-11-OA-0208_536-543.pdf>.
- ¹⁰ L.M. Franzi, et al, "Why is Particulate Matter Produced by Wildfires Toxic to Lung Macrophages?" *Toxicol Appl Pharmacol*. 257.2 (2011): 182-8. doi: 10.1016/j.taap.2011.09.003. Epub 2011 Sep 16. <<http://www.ncbi.nlm.nih.gov/pubmed/21945489>>.
- ¹¹ Teresa C. Wegesser, et al, "California Wildfires of 2008: Coarse and Fine Particulate Matter Toxicity." *Environ Health Perspect*. 117.6 (2009): 893–897. Epub 2009 Feb 2. doi: 10.1289/ehp.0800166. <<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2702402>>.
- ¹² Carol Potera, "Air Pollution: The Oxidative Punch of Wildfires." *Environ Health Perspect*. 117.2 (2009): A58. <<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2649241>>.
- ¹³ C.T Migliaccio, et al, "Adverse Effects of Wood Smoke PM(2.5) Exposure on Macrophage Functions." *Inhal Toxicol*. 2 (2013):67-76. doi: 10.3109/08958378.2012.756086. <<http://www.ncbi.nlm.nih.gov/pubmed/23363038>>.
- ¹⁴ P.H. Danielsen, et al, "Oxidative Stress, DNA Damage, and Inflammation Induced by Ambient Air and Wood Smoke Particulate Matter in Human A549 and THP-1 Cell Lines." *Chem Res Toxicol*. 24.2 (2011):168-84. doi: 10.1021/tx100407m. Epub 2011 Jan 14. <<http://www.ncbi.nlm.nih.gov/pubmed/21235221>>.
- ¹⁵ D. M. Brown, et al, "Increased Inflammation and Intracellular Calcium caused by Ultrafine Carbon Black is Independent of Transition Metals or Other Soluble Components." *Occup Environ Med* 57 (2000):685-91. doi:10.1136/oem.57.10.685. <<http://oem.bmj.com/content/57/10/685>>.
- ¹⁶ S. A. Murphy, et al, "Bioreactivity of Carbon Black and Diesel Exhaust Particles to Primary Clara and Type II Epithelial Cell Cultures." *Occup Environ Med* 56(1999):813-19. <<http://oem.bmj.com/content/56/12/813doi:10.1136/oem.56.12.813>>.
- ¹⁷ D. Höhr, et al, "The Surface Area Rather than the Surface Coating Determines the Acute Inflammatory Response after Instillation of Fine and Ultrafine TiO₂ in the Rat." *Int J Hyg Environ Health*. 205.3 (2002): 239-44. <<http://www.ncbi.nlm.nih.gov/pubmed/12040922?dopt=Abstract&holding=npg>>.
- ¹⁸ C. Monteiller, et al, "The Pro-inflammatory Effects of Low-toxicity Low-solubility Particles, Nanoparticles and Fine particles, on Epithelial Cells in Vitro: the Role of Surface Area." *Occup Environ Med*. 64.9 (2007): 609-15. Epub 2007 Apr 4. Abstract. <<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2092561>>.
- ¹⁹ Thoms M. Lachocki, et al, "Persistent Free Radicals in Woodsmoke: an ESR Spin Trapping Study." *Free Radical Biology and Medicine*, 7.1 (1989): 17-21. Abstract. <<http://www.ncbi.nlm.nih.gov/pubmed/2546867#>>
- ²⁰ Luther Smith, et al, "Equating Semi-Continuous (SC) PM2.5 Mass Monitor Measurement Values with Federal Reference Method (FRM) PM2.5 Monitor Measurement Values: Final Report." EPA/600/R-11/039, EPA. 2011. 3. <http://www.epa.gov/heasd/documents/cdc/AnnualReports/29640nerl11SC_PM_MonitorValues.pdf>.
- ²¹ NYS Department of Environmental Conservation. *NYSDEC In-house Discussion: the Implications of Approved Continuous PM2.5 FEMIII Instruments*. Dirk Felton. NESCAUM. 2008. <http://www.nescaum.org/documents/mac/mac-committee-meeting-2/felton_decinhousefem.pdf>.

²² Gobeli, David, Herbert Schloesser, and Thomas Pottberg. *Met One Instruments BAM-1020 Beta Attenuation Mass Monitor US-EPA PM2.5 Federal Equivalent Method Field Test Results*. Grants Pass: Met One Instruments, 2008. *Met One Instruments*. Web. 11 Dec. 2014. <<http://www.metone.com/documents/AWMA2008-Final-2.pdf>>.

²³ Schwab, James J, Henry D. Felton, Oliver V. Rattigan, and Kenneth L. Demerjian. "New York State Urban and Rural Measurements of Continuous PM2.5 Mass by FDMS, TEOM, and BAM." *Journal of the Air & Waste Management* 56.4 (2006): 372-83. <<http://dx.doi.org/10.1080/10473289.2006.10464523>>.

²⁴ Ward, Tony, et al. "Source Apportionment of PM2.5 in a Subarctic Airshed - Fairbanks, Alaska." *Aerosol and Air Quality Research* 12 (2012): 538. <http://aaqr.org/VOL12_No4_August2012/8_AAQR-11-11-OA-0208_536-543.pdf>.

²⁵ Kleinman, Michael. "Cardiopulmonary Health Effects: Toxicity of Semi-Volatile and Non-Volatile Components of PM." *California Air Resources Board and the California Environmental Protection Agency* No. 07-307. 2013. 1-35. <<http://www.arb.ca.gov/research/apr/past/07-307.pdf>>.

²⁶ ADEC, Envistaweb <<http://dec.alaska.gov/applications/air/envistaweb>>.