



Particulate Matter Standards in Context: Trends and Health Effects

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Overview

- North Carolina is already near or in attainment of current and proposed PM2.5 standards
 - Although several areas would violate standard if EPA goes lower than current proposal
- Long-term PM2.5 non-attainment problem has been addressed by already-adopted measures
- Harm from PM2.5 has been greatly exaggerated
 - Federal PM2.5 standards are costly, but will provide minimal health benefits

Current and Future PM2.5 Standards

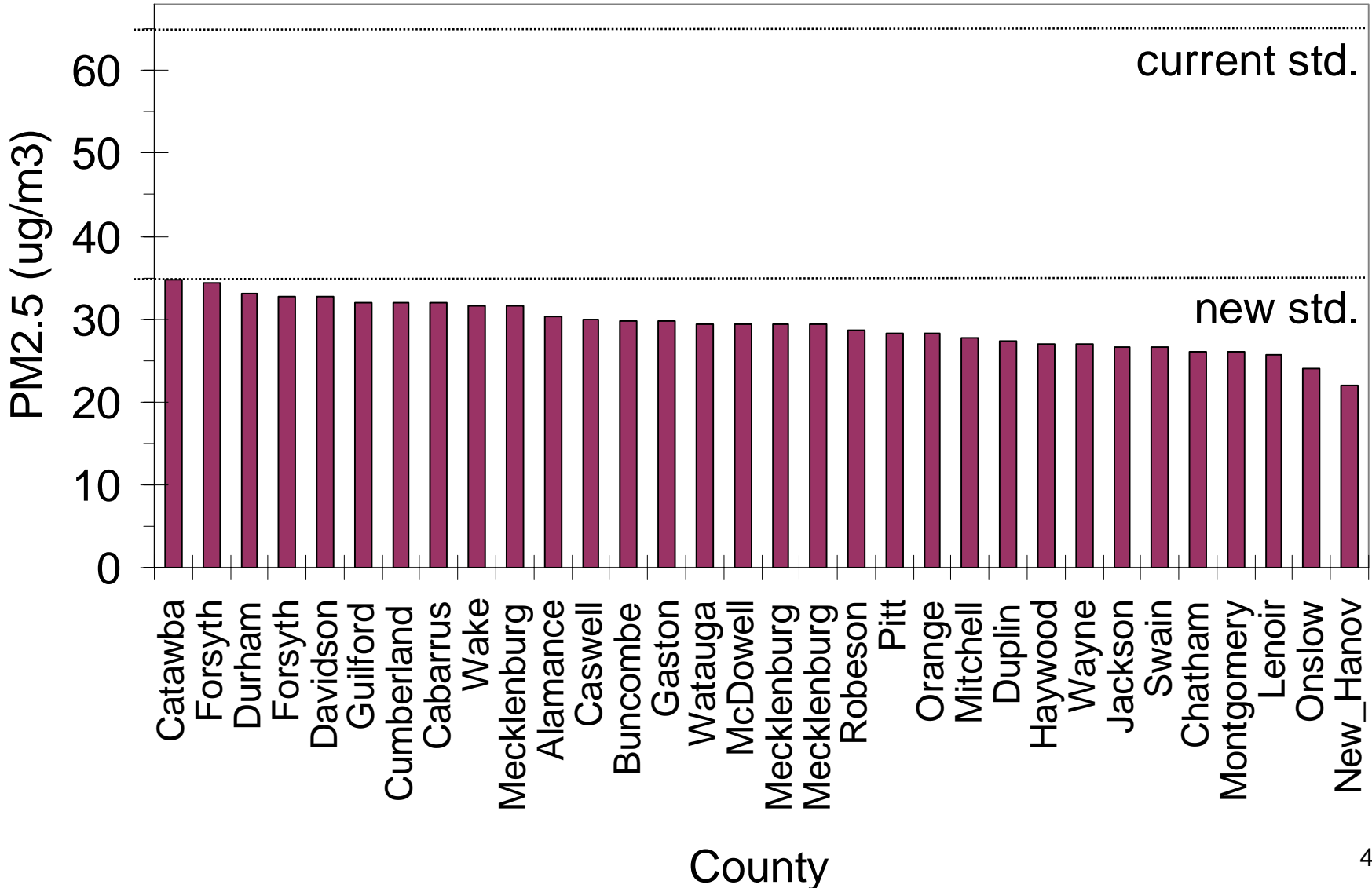
○ Current Standards

- Annual Standard, 15 ug/m³
 - North Carolina is near full attainment
- 24-hour Standard, 65 ug/m³
 - North Carolina is in full attainment with room to spare

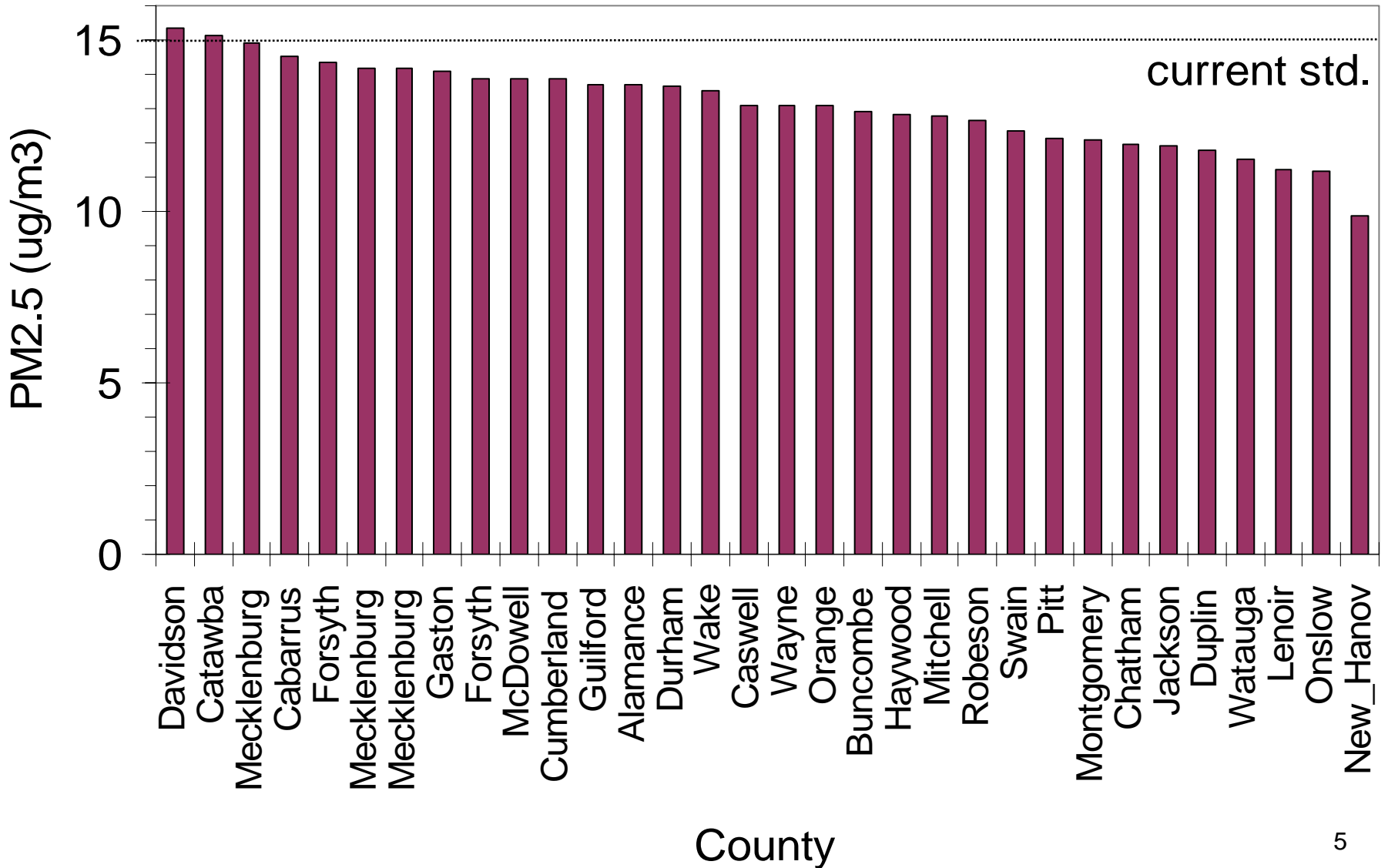
○ Proposed Standards

- Annual standard remains the same
- 24-hour Standard lowered to 35 ug/m³
 - North Carolina barely in attainment
- EPA pressured by CASAC, media, activists to clamp down further
 - Annual 13-14 ug/m³?
 - 24-hour hour 30 ug/m³?

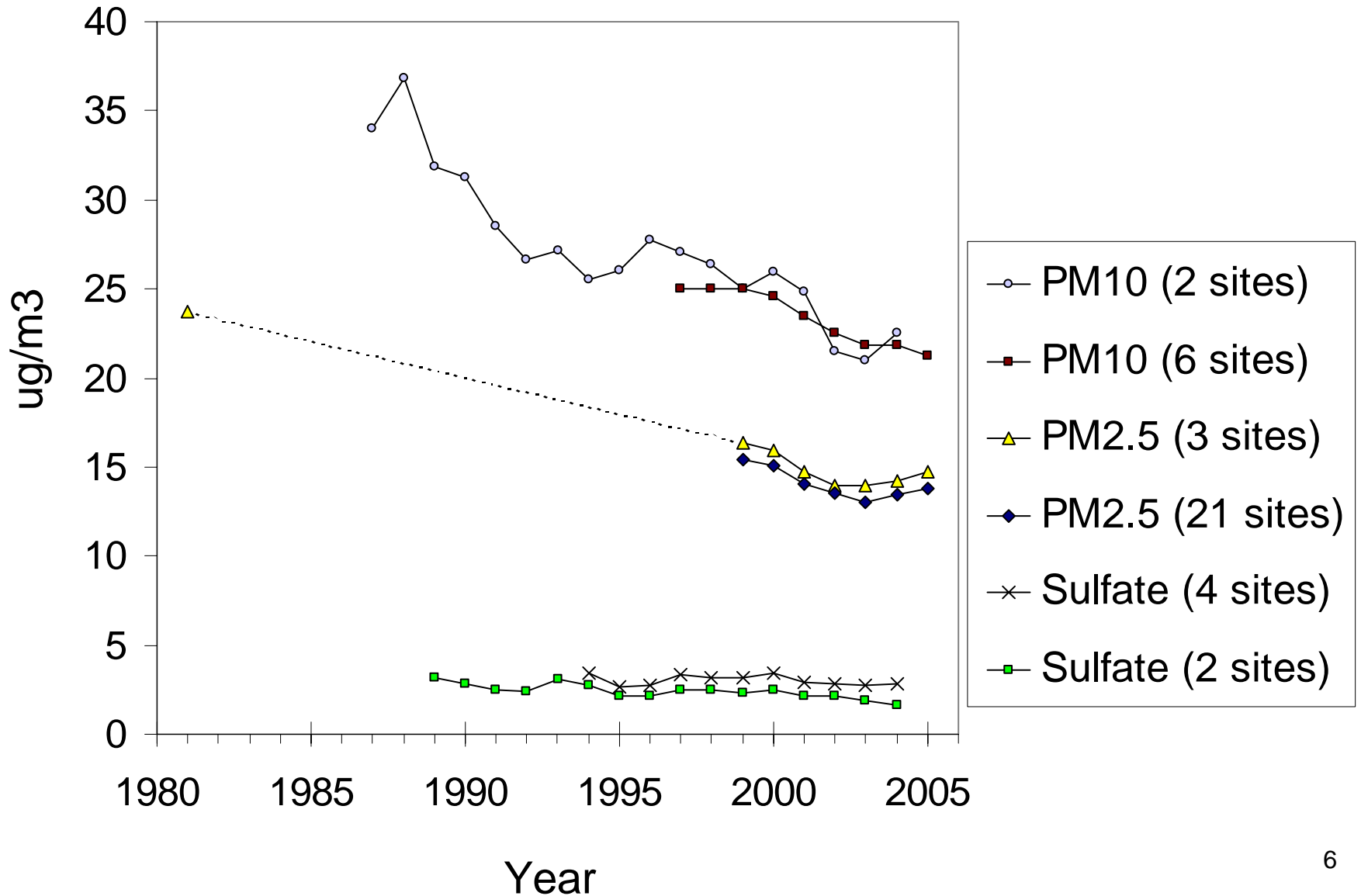
24-hour PM2.5, 2002-04



Annual PM2.5, 2002-04



North Carolina PM Trends



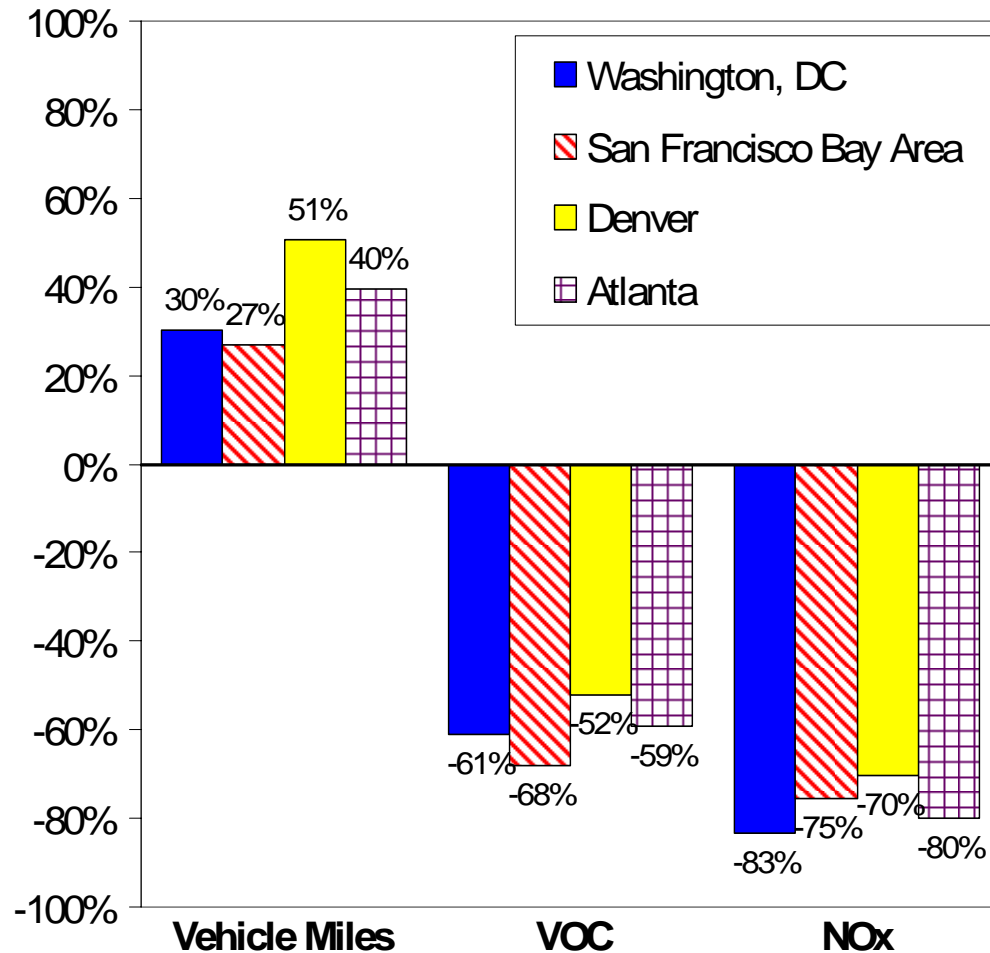
Emissions will continue to decline

- Current requirements will eliminate most remaining PM- and ozone-forming emissions
 - Emissions of average automobile dropping about 10% per year (on-road data).
 - Diesel soot from average truck dropping about 7%-8% per year (on-road data).
 - During next 20 years: 90% reduction in per-mile emissions of NO_x, VOC, diesel soot; >80% reduction after accounting for VMT growth. (Tier 2 light-duty; Tier IV non-road; HD truck stds.
 - Power plants: >30% reduction since 1990. 60% reduction in ozone-season NO_x from 1998-2004. CAIR eliminates most remaining emissions.
 - MACT standards eliminate most industrial VOC
- Long-term problem of air pollution has already been solved

Regulators make a similar prediction

- Look at conformity findings
 - Predict large decreases in VOC and NOx, despite large VMT increases
- All based on MOBILE6, which *underestimates* rate of automobile emissions decline
 - On-road data show MOBILE6 overestimates emissions of newer cars relative to older cars
 - Tier 2 requires lower emissions than MOBILE6 predicts
- Real improvements will be even greater than regulators predict

Percent change: 2025 vs. 2005



PM2.5 and health: Less than meets the eye

- Regulators, environmentalists, and health experts justify PM2.5 standards based on purported health benefits
 - Claim tens of thousands of premature deaths each year due to PM, as well as other harms
- No question that high levels can kill: “London Fog” of December 1952 killed ~4,000 people
 - Smoke and SO₂ soared to tens of times current peak levels (visibility as low as 20 feet).
- Question now whether current, low PM levels also kill.
- Evidence is much weaker and less consistent
 - Uncontrolled confounding, rather than real causal connections.
 - Publication bias: research reporting harm is more likely to be published
 - Political economy: regulators are major funders of the research used to justify their powers and budgets
- Popular accounts create false appearance of large and certain PM risks

How do we learn about health effects?

- Randomized, controlled study (“experimental” study): gold standard
 - Remove all sources of variation except the exposure of interest
 - Assess serious PM effects in animals; mild PM effects in humans
- “Observational” study: the most common type
 - Confounding is a concern, because people are not randomly assigned to pollution levels. Examples:
 - If people in higher PM areas are more likely to smoke, could attribute deaths to pollution that were actually caused by smoking
 - Higher ozone is associated with higher temperatures, so could attribute deaths to ozone that were caused by heat stress
 - Try to measure potential confounders and statistically control for them, leaving a residual correlation between pollution and health outcomes.
 - The implicit assumption is that this residual correlation (if greater than zero) represents a genuine causal relationship.
- Air pollution mortality claims are based mainly on results of observational studies
 - Cohort study: follow people in different cities over time
 - Time-series study: correlation of daily deaths and PM levels

Limits of observational epidemiology (1)

- Can only control for what you measure, so there is always the risk of additional uncontrolled confounding
 - Recent evidence suggests this is the norm, rather than the exception
- Observational studies gave spurious results in a number of recent high-profile reversals of conventional medical wisdom:
 - Hormone-replacement therapy (HRT): Observational studies suggested not being on HRT increased cardiovascular risk by a factor of 2 (i.e., 100%). But later randomized trials showed that HRT does not reduce CV risk and might increase it.
 - Beta-carotene went from protective to harmful (30% decrease in risk in observational studies; 12% increase in risk in randomized trials)
 - Other recent reversals based on randomized trials include:
 - (1) low-fat diet did not reduce cardiovascular or cancer risks;
 - (2) calcium and Vitamin D did not reduce osteoporosis risk

Limits of observational epidemiology (2)

- The moral is that it's difficult to get reliable results on the causes of health outcomes based on data from non-randomly-selected and non-randomly-assigned people, even with careful efforts to control for biases
- Observational air pollution studies face an even greater risk of spurious results. They are trying to tease out ostensible risks of a few tenths of a percent to a few percent, and therefore suffer an even greater risk of uncontrolled confounding
 - Yet these observational studies claim to be teasing out these tiny risks from among a sea of confounders with much larger health risks than the putative risks of air pollution

Spurious PM health effects claims (1)

- EPA based annual PM_{2.5} standard on the ACS cohort study
 - Reported 4% increase in mortality for each 10 ug/m³ increase in PM_{2.5} (Pope et al. 2002).
- But odd results suggest there's no causal connection
 - PM kills men, but not women; people who say they are moderately active, but not those who say they are very active or sedentary; those with no more than a high school degree, but not those with some college
 - Adding migration into and out of cities eliminated PM_{2.5}-mortality correlation
 - Midwest rust-belt cities lost population during the 1980s as people sought jobs elsewhere. Midwest areas also had higher PM_{2.5} levels.
 - But people who work and have wherewithal to migrate are also healthier than average. So differential migration is likely the actual explanation for excess deaths that were spuriously attributed to PM_{2.5}.
 - Adding other variables to the statistical model also reduced or eliminated PM_{2.5} effects

Spurious PM health effects claims (2)

- Recent time-series study showed that more complete control for weather eliminates mortality associations with ozone and PM10 (Keatinge & Donaldson, 2006)
 - Heat stress increases mortality risk, so time-series studies include controls for weather.
 - But at any given temperature, greater sunshine and lower winds add to heat stress. However, greater sunshine is also associated with higher ozone, while lower winds are associated with higher PM10.
 - In other words, since sunshine and wind are correlated with both pollution levels and mortality, failing control for them created a spurious correlation between air pollution and risk of death.
 - This effect had not previously been addressed in air pollution epidemiology studies.

Publication bias/model-selection bias inflate pollution effects

- Studies reporting larger effects are more likely to be published
 - “Publication bias arises because there are more rewards for publishing positive or at least statistically significant findings. It is a common if not universal problem in our research culture. In the case of time-series studies using routine data there are particular reasons why publication bias might occur. One is that the data are relatively cheap to obtain and analyse, so that there may be less determination to publish “uninteresting” findings. The other is that each study can generate a large number of results for various outcomes, pollutants and lags and there is quite possibly bias in the process of choosing amongst them for inclusion in a paper. In the field of air pollution epidemiology, the question of publication bias has only recently begun to be formally addressed.” (Anderson et al. World Health Organization, 2004)
- NMMAPS (HEI-sponsored study) showed publication bias inflates apparent pollution-mortality association by more than a factor of 3
 - NMMAPS applied same modeling strategy to 95 U.S. cities, avoiding publication bias inherent in single-city studies.
 - Higher ozone and PM were associated with *lower* mortality risk in more than one-third of cities.
 - Removing a few outlier cities (in both “positive” and “negative” directions) reduced “pooled” PM effect to statistical insignificance

- Observational studies can become statistical fishing expeditions, turning up chance correlations in noisy data, rather than real causal relationships
 - Recent caution from air pollution epidemiologists: “Estimation of very weak associations in the presence of measurement error and strong confounding is inherently challenging. In this situation, prudent epidemiologists should recognize that residual bias can dominate their results. Because the possible mechanisms of action and their latencies are uncertain, the biologically correct models are unknown. This model selection problem is exacerbated by the common practice of screening multiple analyses and then selectively reporting only a few important results.” (Lumley and Sheppard, *Epidemiology*, 2003)
- Accounting for model-selection bias eliminates association between air pollution and mortality (Koop and Tole, *J Env Econ and Mgmt*, 2004)

The results you don't hear about

- Even without accounting for weaknesses of observational epidemiology, selective omission creates appearance of more consistent and robust effects than reflected by weight of evidence
- Evidence is not consistent from study to study
 - Cohort study of 50,000 veterans with high blood pressure found negative PM2.5-mortality correlation (Lipfert et al., *Inhalation Toxicology*, 2000)
 - Cohort study of 50,000 Californians found no association of PM2.5 and mortality (Enstrom, *Inhalation Toxicology*, 2005)
- Declining PM2.5 effects in ACS study
 - 6.9%/10 ug/m³ in first ACS report (1982-89 follow-up)
 - 4%/10 ug/m³ in second report (1982-98 follow-up)
 - Means effect size must have declined—not mentioned in ACS reports, but can be backed out from data provided
 - PM2.5 effect declined 55% from 1980s to 1990s—trend is toward statistical insignificance.
 - Enstrom (2005), Lipfert et al. (2000) also show declining effects over time.

2,000 wrongs don't make a right

- When EPA proposed tougher PM_{2.5} standards in December, a number of health scientists and environmental activists argued that 2,000 studies published since 1997 demonstrated the need for even lower standards.
- But these are observational studies that suffer from confounding, publication bias, and model-selection bias.
- Repeating an invalid approach 2,000 times doesn't improve its validity.

Toxicology studies provide little support for health effects claims

- If the effects claimed based on observational epidemiology studies were real, we should see supporting evidence in controlled toxicology studies.
- But toxicology studies with human volunteers or animals provide little support for harm from PM at contemporary pollution levels.

Toxicology of Particulate Matter (1)

- Nitrate and sulfate PM are not toxic to humans, even at concentrations tens of times higher than peak ambient concentrations
 - Ammonium sulfate is used as an “inert control” in studies of the effects of acid aerosols.
 - Inhaled magnesium sulfate is used to open constricted airways in asthmatics.
 - Yet justification for coal-fired power plant SO₂ regulations is reduction of sulfate PM, and most of claimed NO_x-reduction benefits are due to nitrate PM reductions
- PM of all kinds does not kill animals at concentrations anywhere near levels found in ambient air

Toxicology of Particulate Matter (2)

- Health Effects Institute (HEI) study exposed healthy and asthmatic volunteers to 100 ug/m³ of diesel PM for 2 hrs while they exercised intermittently. Little evidence of any lung inflammation in healthy participants, and the study “did not find inflammatory changes in asthmatic participants.”
- Another HEI study exposed healthy and asthmatic volunteers to 200 ug/m³ of concentrated ambient PM_{2.5} collected in Los Angeles for 2 hours with intermittent exercise.
 - No changes in symptoms or lung function in either healthy or asthmatic subjects. Little evidence of inflammatory responses. Since many inflammatory markers were measured, and only a few changed, authors pointed out that changes could be due to chance.

Toxicology of Particulate Matter (3)

- “It remains the case that no form of ambient PM—other than viruses, bacteria, and biochemical antigens—has been shown, experimentally or clinically, to cause disease or death at concentrations remotely close to US ambient levels. This lack of demonstration is not for lack of trying: hundreds of researchers, in the US and elsewhere, have for years been experimenting with various forms of pollution-derived PM, and none has found clear evidence of significant disease or death at relevant airborne concentrations.”

(Green and Armstrong, *Regulatory Toxicology and Pharmacology*, 2003)

New mouse study claims PM2.5 advances heart disease

- Mice on high-fat diet had 50% greater arterial plaque when exposed to PM
 - Published in JAMA; NIH press release; many news stories
- NIH press release: "Test results with laboratory mice show a direct cause-and-effect link between exposure to fine particle air pollution and the development of atherosclerosis...[The study] may explain why people who live in highly polluted areas have a higher risk of heart disease."
 - "the fine particle concentrations used in the study were well within the range of concentrations found in the air around major metropolitan areas."
- In reality, study is irrelevant for both humans and mice
 - Unrealistic mice: genetically engineered to have stupendous cholesterol levels—14 times the level in normal mice. For comparison, only one-in-50 men has cholesterol even 1.5 times normal; only one-in-500 exceeds two times normal.
 - Unrealistic PM2.5 doses: 85 ug/m³ for 6 hours each weekday (30 hours/week), for 6 months (1/4 of a mouse lifespan), and filtered air otherwise.
 - Researchers claimed this was like real-world, because annual average was 15 ug/m³.
 - But areas that average 15 ug/m³ rarely reach 85 ug/m³. For example, Modesto, CA averaged 16 ug/m³ last year, but averaged only 1.5 hrs/week \geq 85 ug/m³; 8 hrs/week \geq 50 ug/m³.
 - The very reason for using such unrealistic, artificial mice, is that PM2.5 doesn't kill real mice.

Spinning the JAMA mouse study

- The scientists who performed the study played down the unrealistic nature of the mice. NIH press release said the mice were “genetically programmed to develop atherosclerosis at a higher-than-normal rate.” That’s like doing a study of people who weigh 500 pounds and referring to them merely as “overweight.”
- 7 of 10 news stories didn’t even mention that the mice were bred for high cholesterol.
- NIH scientists also misled about PM2.5 doses: “the fine particle concentrations used in the study were well within the range of concentrations found in the air around major metropolitan areas.”

Conclusions

- Trusted sources—regulators, health scientists, activists, and journalists—have created a false impression of much greater and certain harm from PM2.5 than the evidence warrants
- This false impression results from
 - Exaggeration of harm
 - Omission of contrary evidence
 - Misleading descriptions of research results and their implications
 - Reliance on techniques that are not capable of providing reliable information on risks
- PM2.5 is unlikely to be deadly at current, historically low, concentrations
- Federal PM2.5 standards are based on false premises