Health Effects of Wood Smoke

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Wood Smoke - Developing Countries

- About ½ of world population (3 billion) use biomass (crops, animal dung, wood, charcoal) and coal as fuel
- Indoor air levels of PM of 6800mcg/m^3
- WHO estimate – 1.6 million premature deaths/yr due to smoke from solid fuels
- # 10 on list of major preventable risk factors

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Wood Smoke -Developed Countries

- Varies by region, season, and trends
- Increasing in some communities; U.S. (especially NE, NW) and Canada
- Winter estimates of % contribution to $PM_{2.5}$
  - 90% Seattle, 20-30% Denver, 44% Portland, 65% Elverum Norway, 17% Atlanta
- "…no reason to assume that the effects of particulate matter in areas polluted by wood smoke are weaker than elsewhere."

Bonan C, et. al. Scand J Work Env Health 2003:29:251-60

Studies of Health Effects of Wood Smoke

- Few focused specifically on wood smoke
- More in developing countries
- Evidence of effects of other sources likely similar
  - Outdoor air pollution, ETS, tobacco smoke, etc.
  - Real world exposures are mixed

Components of Wood Smoke

- Particulate matter –
  - $PM_{2.5}$ fine– alveolar deposition
  - PM_{10} coarse– nasopharyngeal/tracheal deposition
- Gases: NOx, SOx, CO
- Five classes of carcinogenic compounds (IARC)
- PAH– benzo[a]pyrene
- Volatile organic compounds, dioxins
- Metals – Arsenic, lead, chromium, etc.

Chronic Obstructive Pulmonary Disease (COPD)

- **Strong evidence**
  - **Mexican cohort study of COPD patients: "Women exposed domestically to biomass develop COPD with clinical characteristics, quality of life, and increased mortality similar in degree to that of tobacco smokers."**

    (Ramirez-Venegas, et al AJRCCM 2006;173:393-7)
  - **Case control study in Spain: Wood and charcoal smoke strongly associated with COPD**
- **Proposed mechanisms - activation of inflammatory pathways, release of oxidants**

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Acute Lower Respiratory Infection (ALRI)

- Strong evidence: especially in children
- American Indian/Alaskan Native infants in SW U.S. and Alaska especially at risk
- "Cooking with wood-burning stoves was associated with higher indoor air concentrations of respirable particles and with an increased risk of ALRI in Navajo children." Robin LF, et. al. Ped Inf Dis J 1996;15:859-865
- Proposed mechanism – inflammation, destruction of alveolar macrophages, suppression of immunity

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Other Respiratory Disease

- Otitis media – few direct studies, well documented for ETS
- Lung fibrosis and interstitial lung disease: suggested by a few studies but silica dust may explain
- Asthma – some evidence that wood smoke exacerbates asthma in sensitive persons
- Tuberculosis – 3 studies with increased risk
- Potential mechanisms – impaired immunity, systemic inflammation

Cardiovascular Disease

- Many studies, worldwide, consistently show increased risk for CV events from PM
  - Both short- and long-term exposures
  - Current PM levels
  - At least one specifically wood smoke
- Possible mechanisms: accelerated atherosclerosis, enhanced thrombosis, systemic inflammation, acute vasoconstriction, arrhythmias, effects on autonomic nervous system

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Cancer

- **Lung cancer – strong evidence, especially in women**

- **Cancer of mouth, pharynx and larynx: Brazilian case – control study showed risk increased 2-4 times with use of woodstoves**

  Pintos J et al. Int J Epi 1998;27:936-40

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Limitations of Current Science

- Hard to distinguish wood smoke from other PM
- Lack of measures of personal exposures
  - Level of pollutant
  - Duration of exposure
  - Lack of biomarkers of exposure
- Lack of control for confounding variables
- Study design
  - Most studies are observational / ecologic
  - Need for intervention studies
- Findings may not be generalizeable to other regions

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Selected results from some CDC investigations of the health effects of forest fires

Humboldt County, California, 2000

Bitterroot Valley, Montana, 2001

Malaysia, 2002

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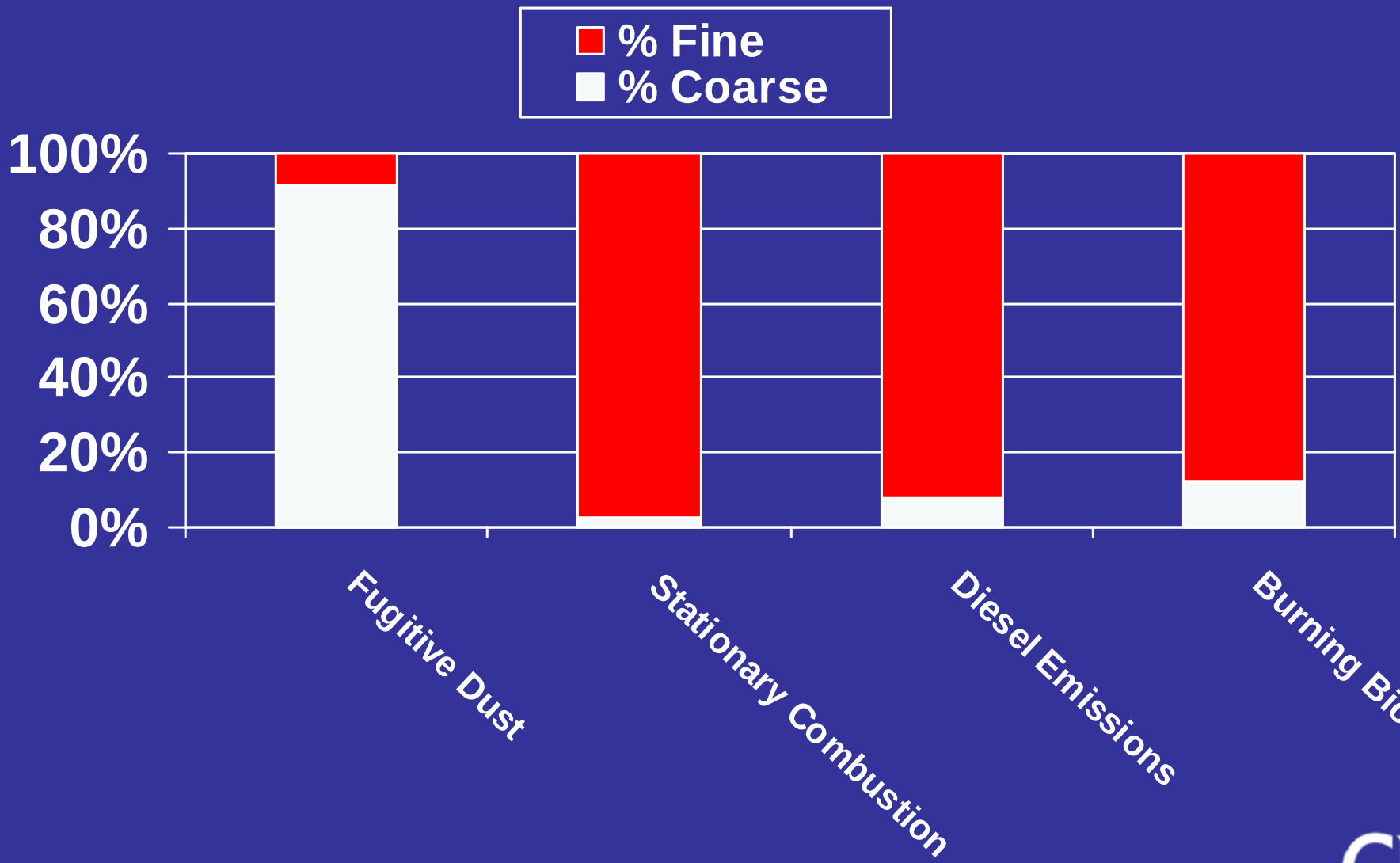
Particle Size Distribution of Primary Sources of Ambient Particulate Matter



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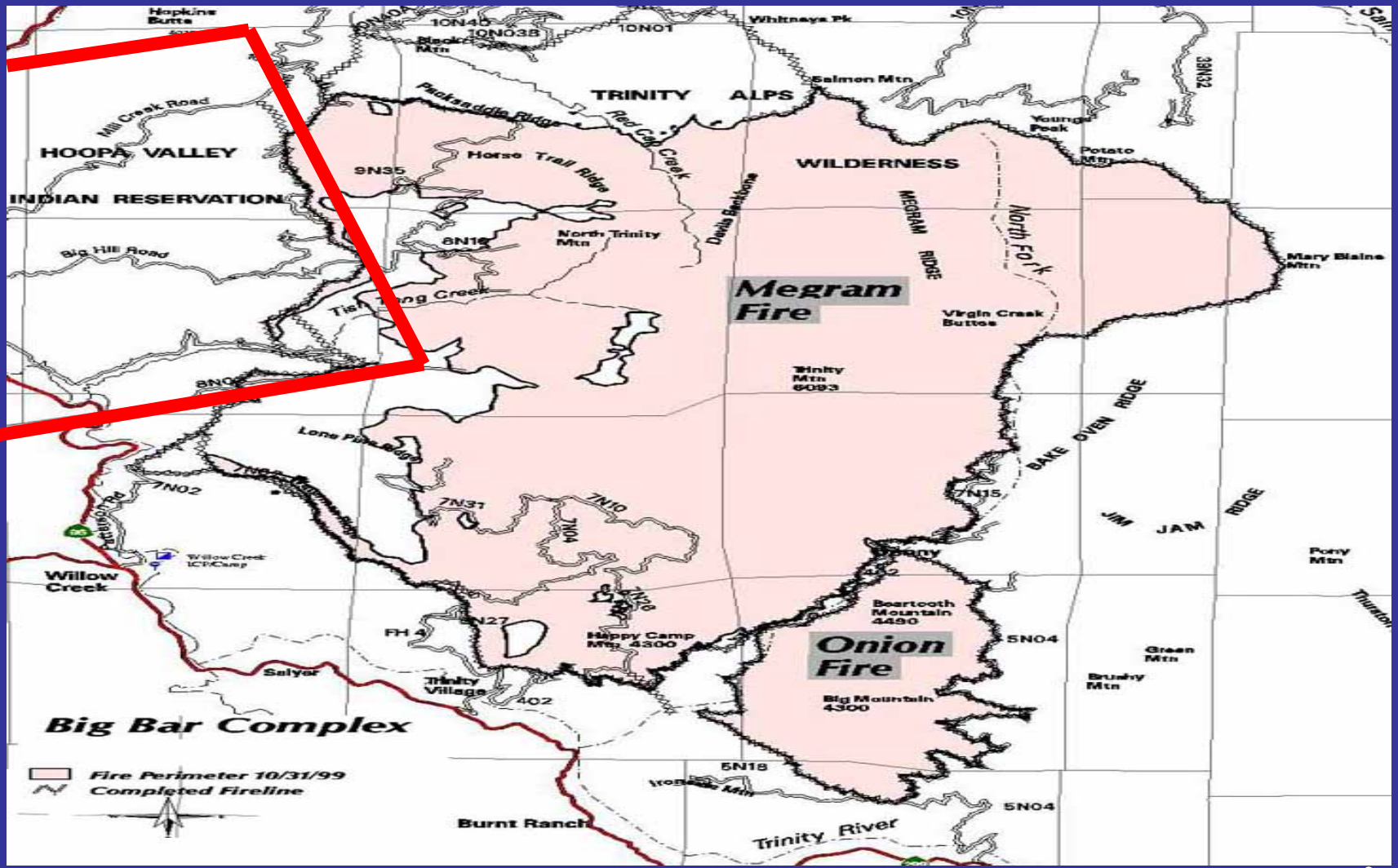
The Big-Bar Fires, Shasta-Trinity Forest, 10/31/99



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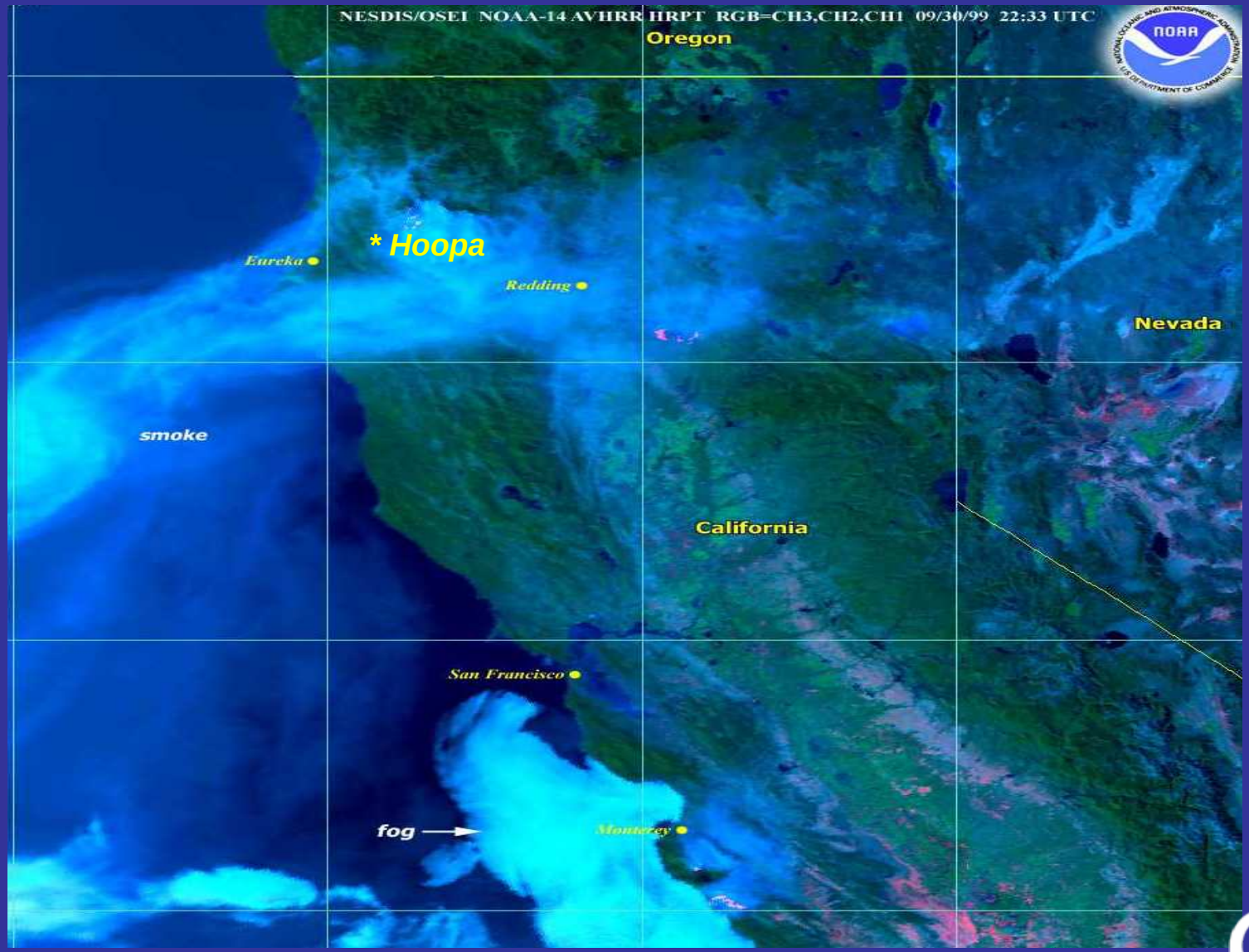
Smoke from Wildland Fires in Northern California September 30th,1999



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Hoopa Valley Indian Reservation

- Trinity River Valley, northern California
- 770 tribal households
- 57% poverty
- 32% unemployment

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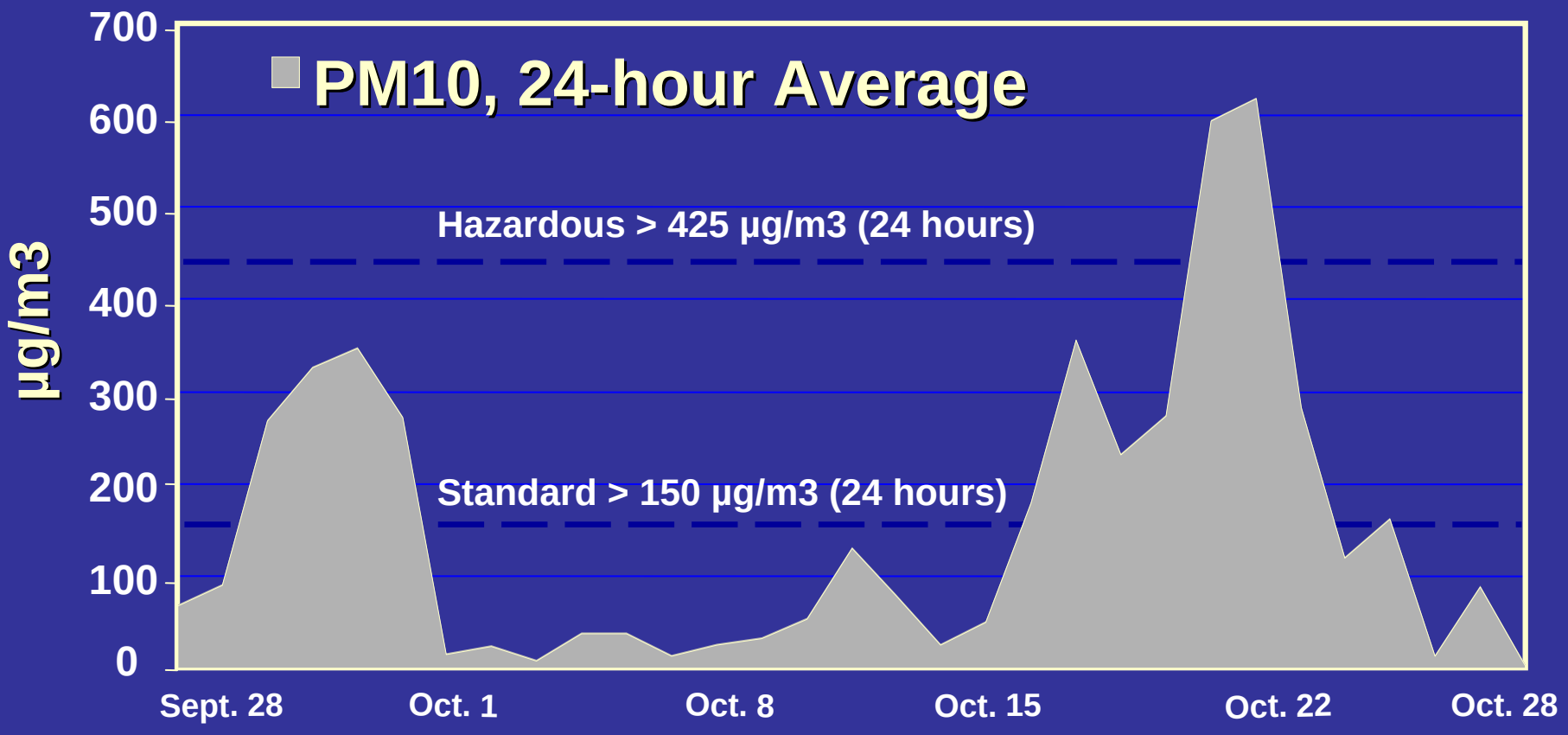
Ambient Particulate Matter < 10 Microns (PM_{10}), Hoopa Valley Indian Reservation, September 28-October 28, 1999



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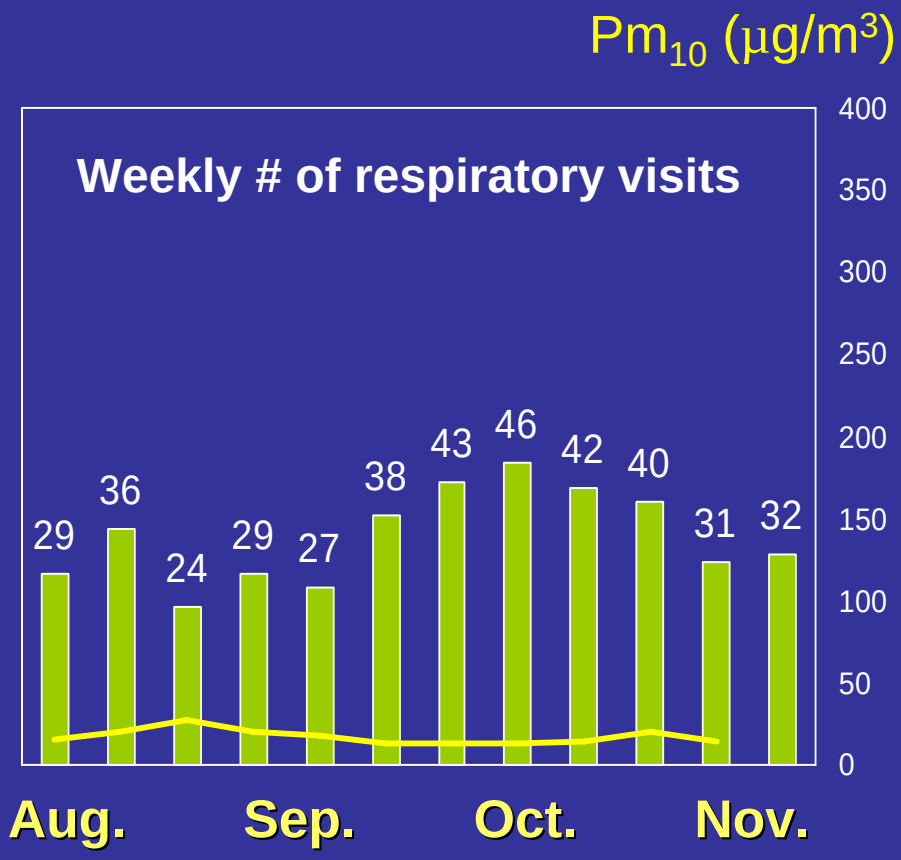
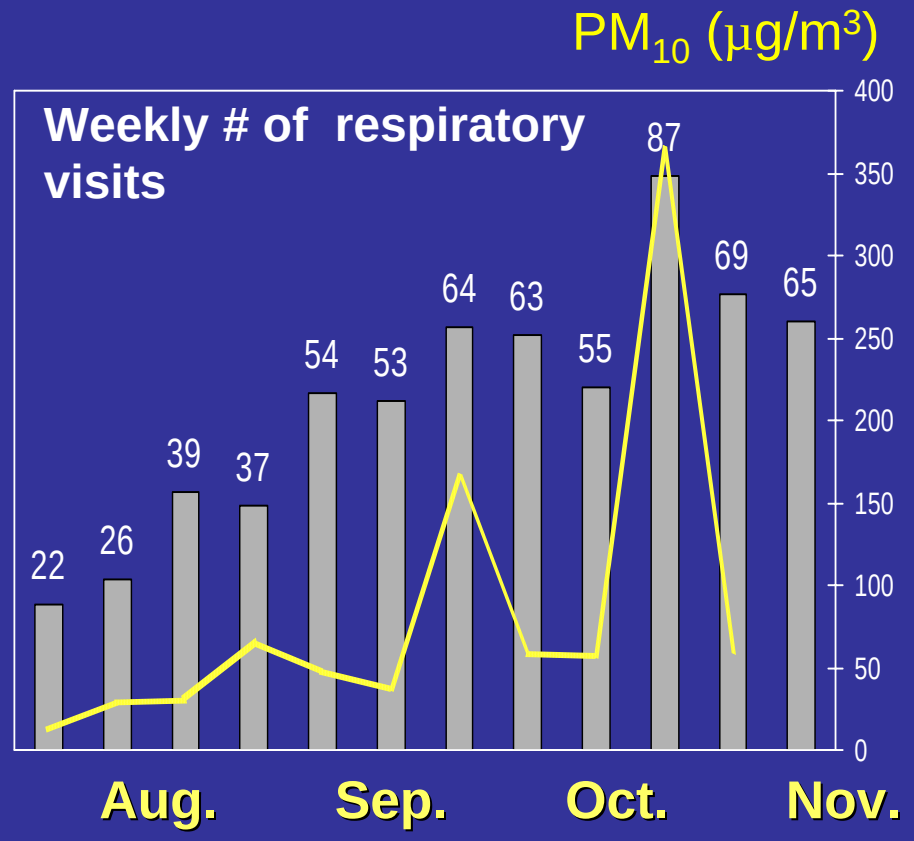
Average Weekly PM_{10} Levels and Number of Respiratory Visits to K'ima:w Medical Center, By Week, August-November, 1998,1999



1998



1999

Interventions

- Mask Use: Ineffective
- PSA's: Effective, but mechanism unclear
- HEPA Cleaners: Effective, need validation
- Evacuation: Ineffective, not feasible

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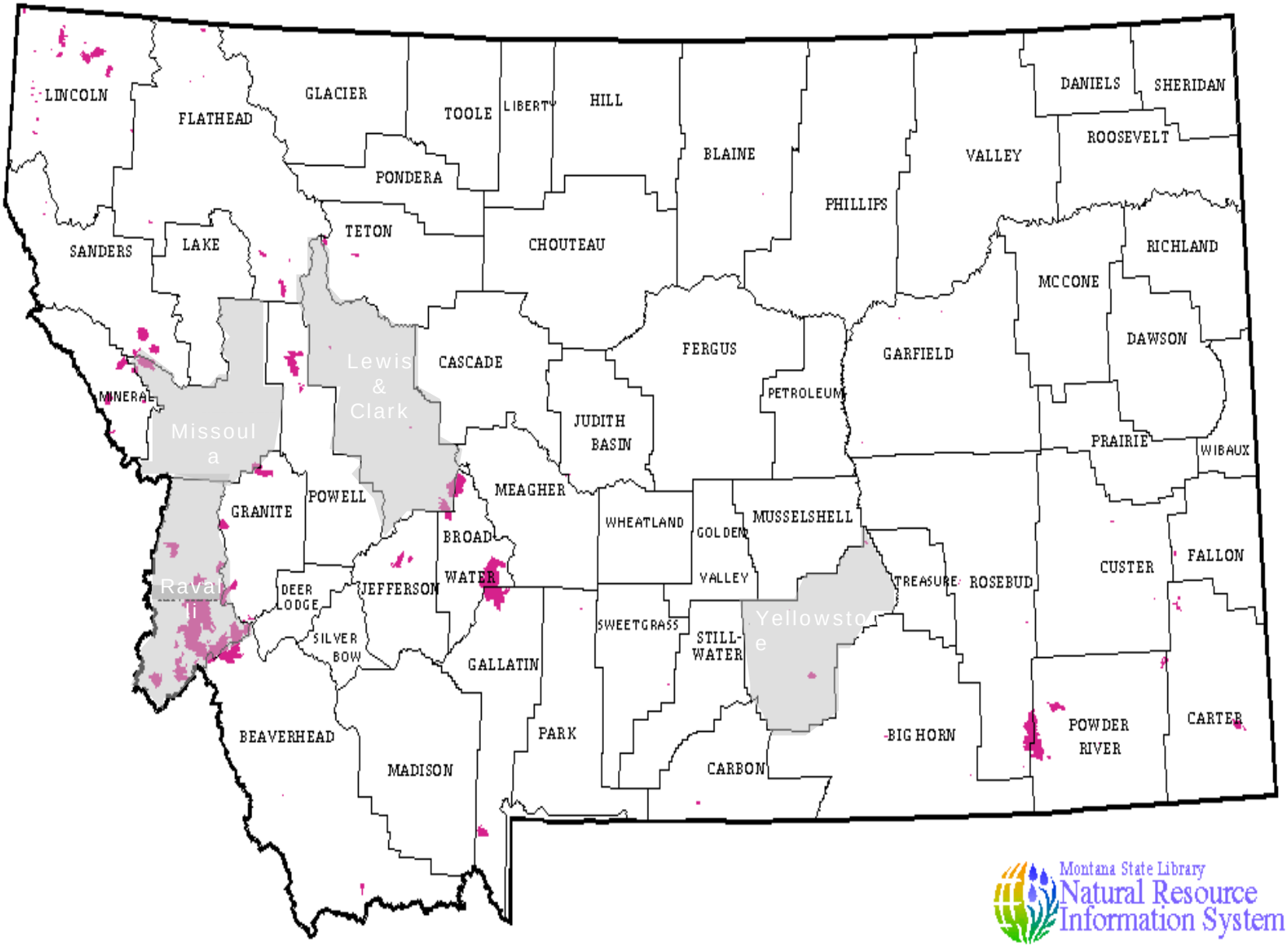
Respiratory and Circulatory Hospital Admissions Associated with Forest Fires - Montana, July-September, 1999 & 2000

Charon Gwynn, Joshua A. Mott, Todd Damrow
David Mannino, Stephen Redd

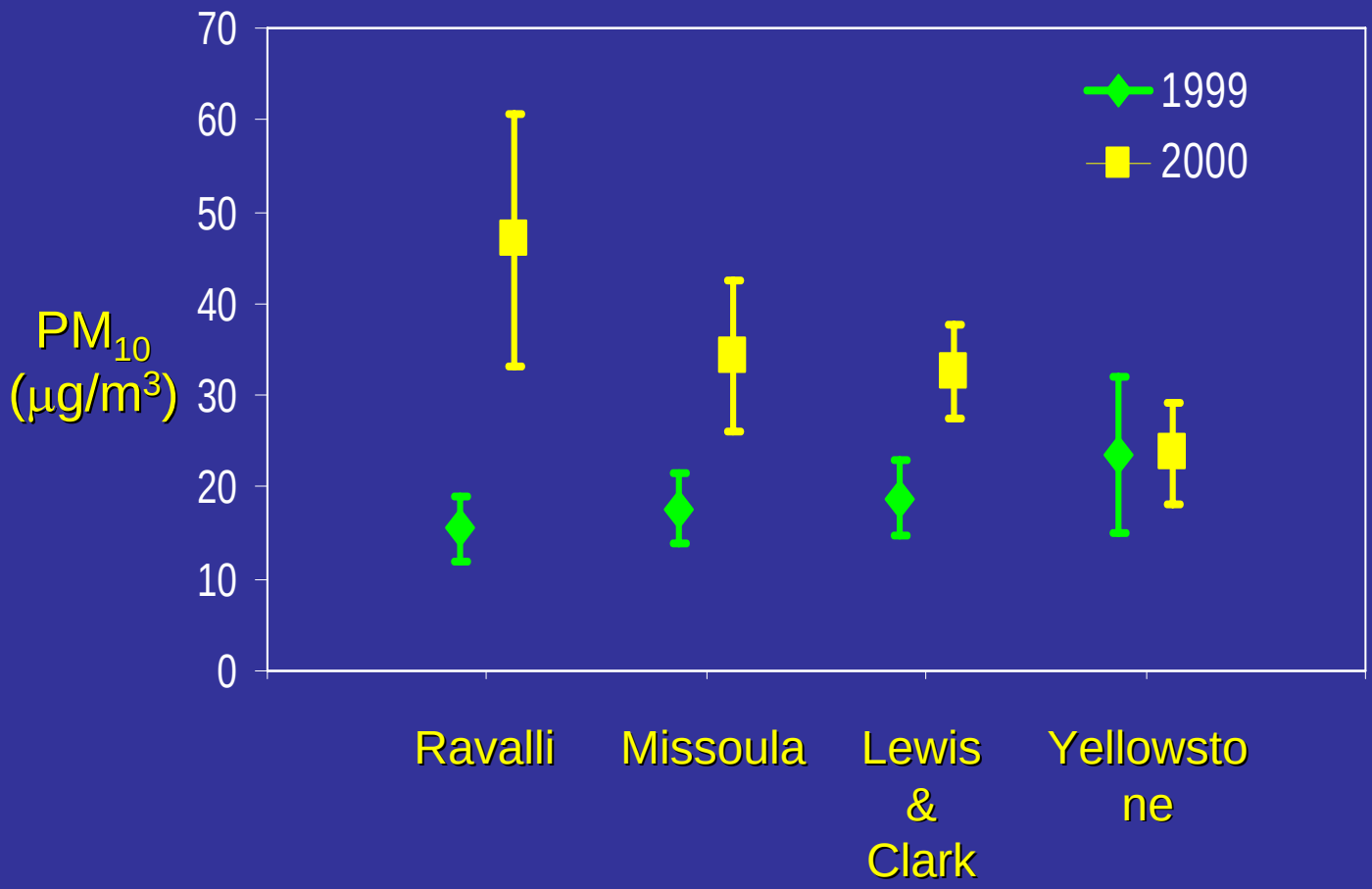
EPI-AID 2001-07

Year 2000 Fire Perimeters in Montana

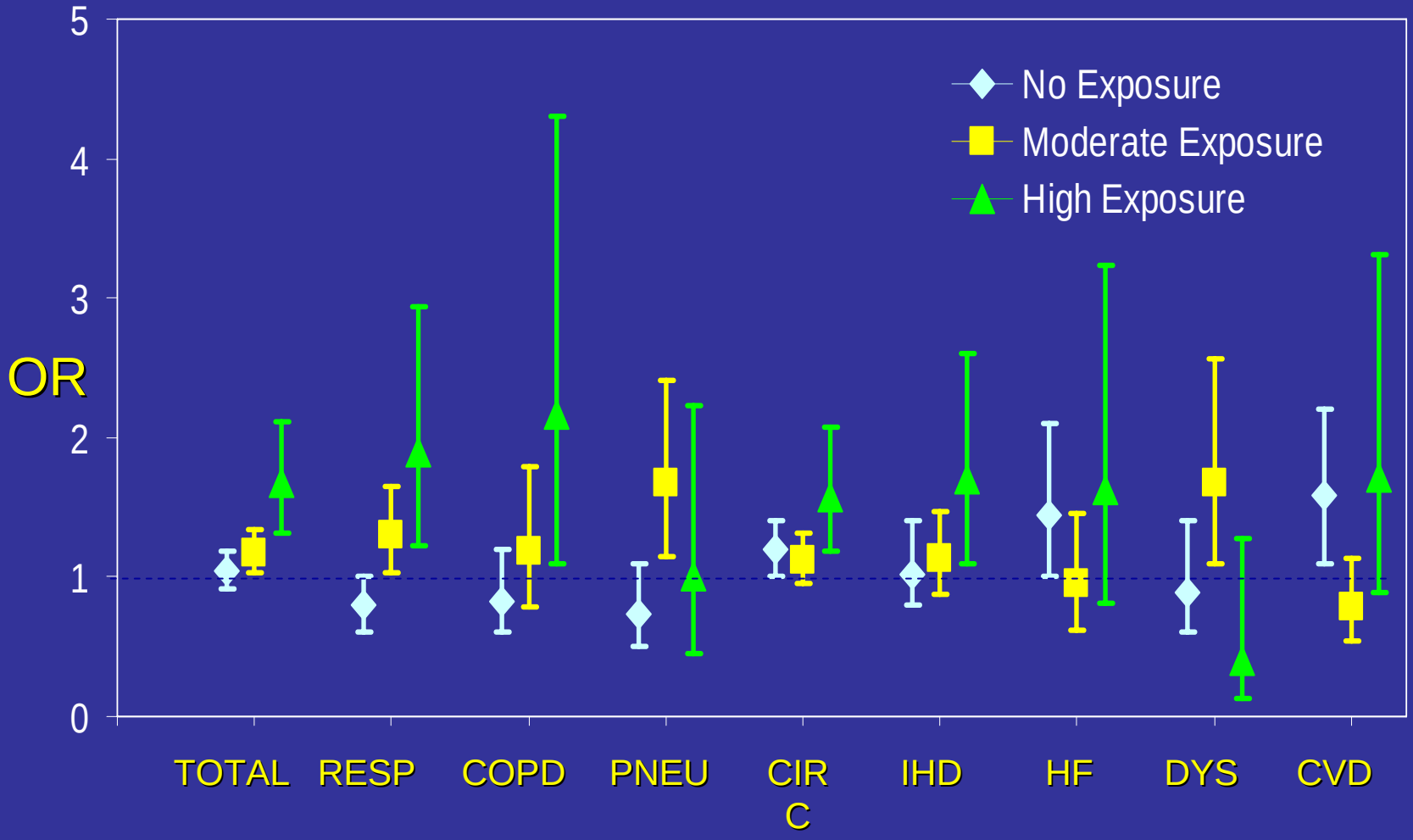


Montana State Library
Natural Resource Information System

Increase in Average PM Concentration Between the 1999 & 2000 Study Periods



Odds Ratios for Admission in 2000 Compared to 1999 for Each Exposure Level



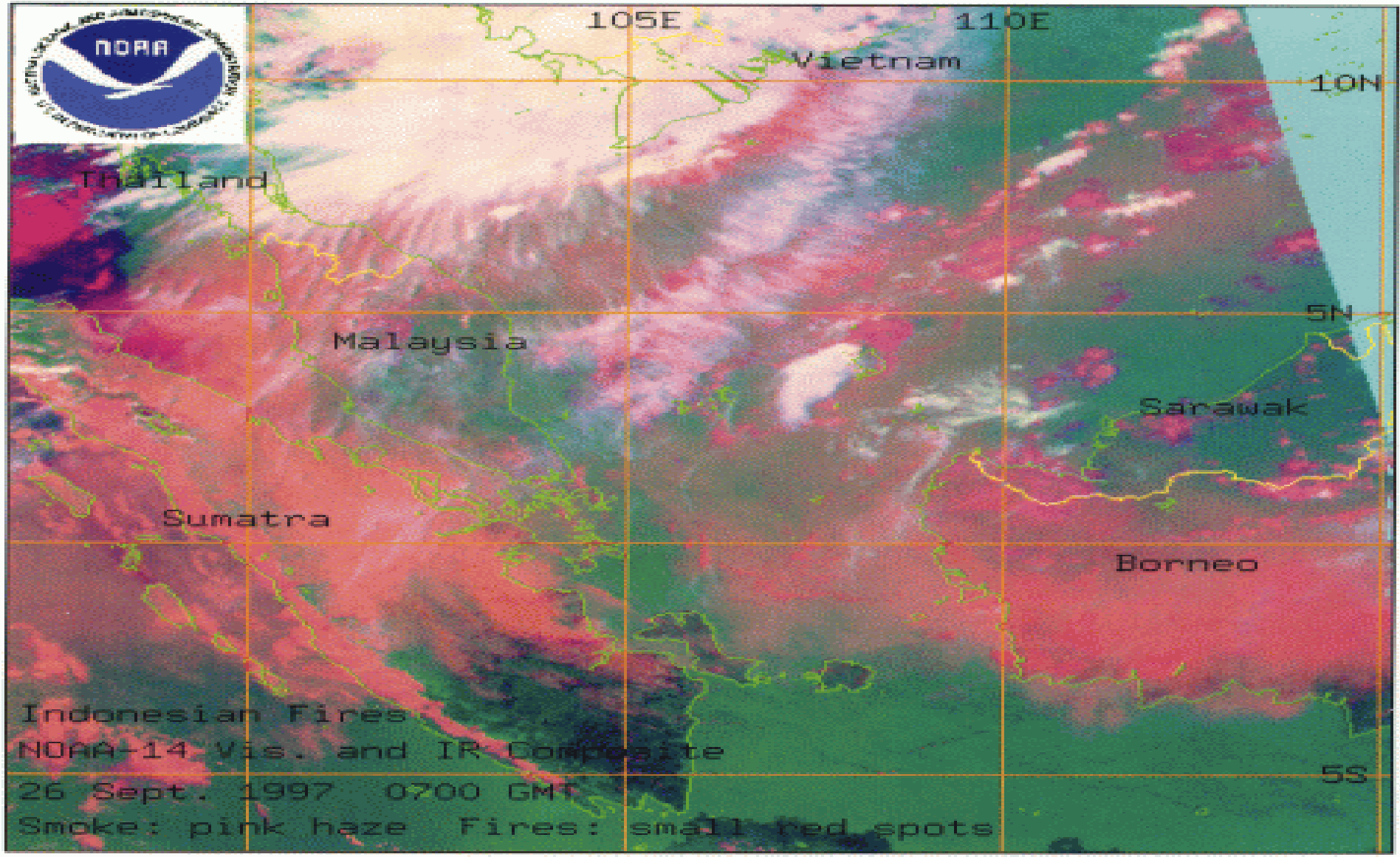
Conclusions

- Risk of admission for circulatory and respiratory illness was greater:
  - in highly exposed area during the 2000 fire than the unexposed area
  - in 2000 than 1999 in smoke exposed areas
- From 1999 to 2000, risk of admission generally increased with exposure
- Evidence of the influence of biomass smoke exposure on more severe health endpoints.

Indonesian/Malaysian Haze Episode of 1997 and 1998

London, England, Dec. 5-9, 1952: PM_{10} estimated at 500-1000 $\mu g/m^3$
Kuching, Sarawak, Sep. 15-22, 1997: PM_{10} estimated at 600-850 $\mu g/m^3$

A Chance to Understand Intense Biomass Haze Effects in (Relatively) Less Urban Environments



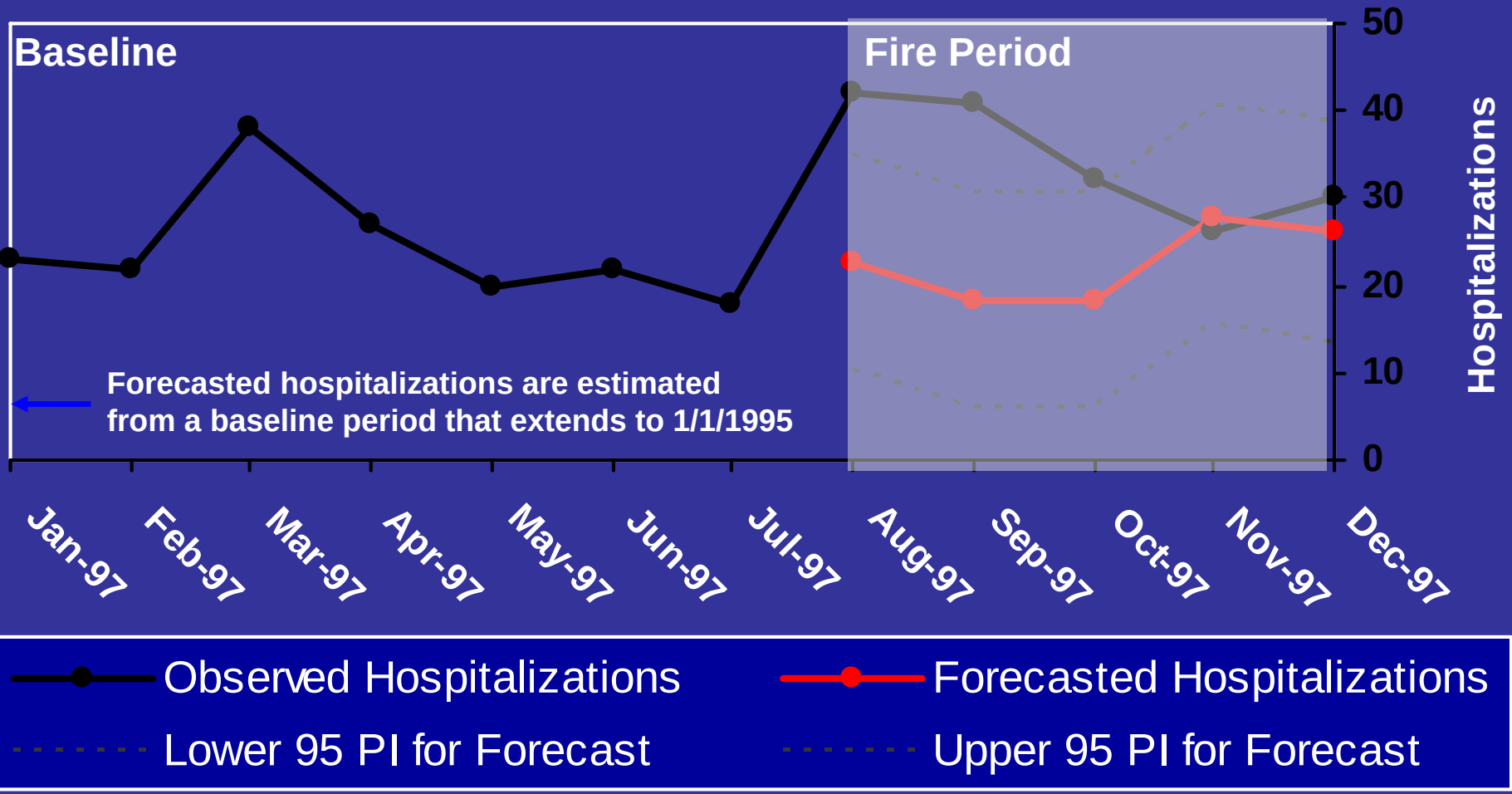
An estimated 87.8 million people were exposed to PM_{10} concentrations >= 150 $\mu g/m^3$ on a single day during the episode (Brauer et al., 2001)

24-hour PM_{10} was 666 μg/m³ on 4/30/1998 (peaked at night)

24-hour PM_{10} was 411 and 420 μg/m³ on 9/14 and 9/15/1997

24-hour PM_{10} was 852 μg/m³ on 9/22/1997

Figure 3. Time Series Analyses: Comparison of observed and forecasted hospitalizations from August 1 to December 31, 1997 using the Holt-Winters' forecasting method*—Asthma Hospitalizations, ages 40-64.

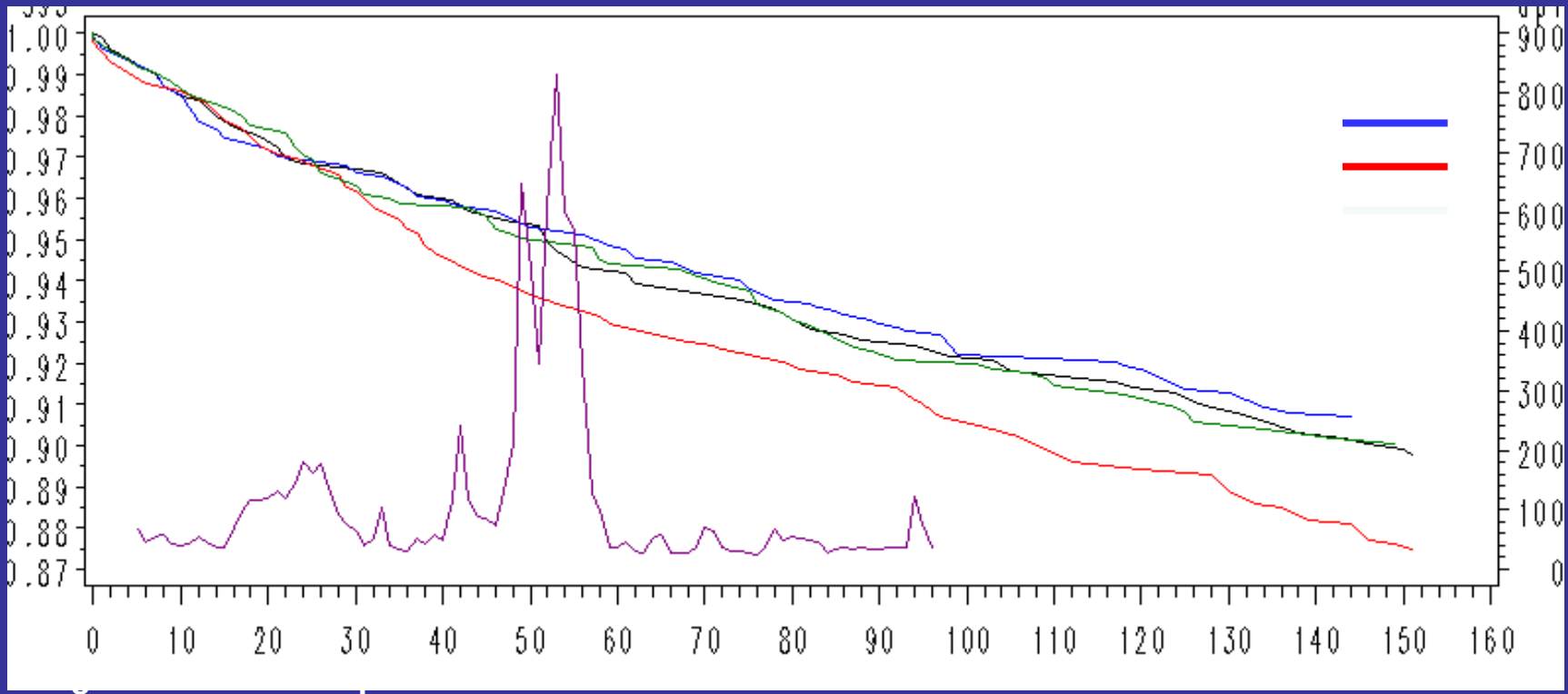


* Time-series forecasting uses a baseline period of January 1,1995 to July 31, 1997.
For ease of presentation, only data from January, 1997 to December, 1998 are included in this figure.
PI = Prediction Interval.

Figure 7. Kaplan-Meier survival curves of time to hospital re-admission during follow-up, cardio-respiratory admissions (ICD-9 390-519), persons >= 65 years of age

Survival Function S(t)

Kuching API, 1997



* = Year of the Forest Fires

Major Conclusion

- Exposure to wood smoke appears to be related to adverse health effects (e.g. self-reported symptoms, ED visits, hospitalizations)

Next Steps?

1) Improve our measures of exposure
- Direct (individual monitors or biomarkers) vs. Indirect
- Capture of different microenvironments
- Better measurement of confounders (e.g. smoking)

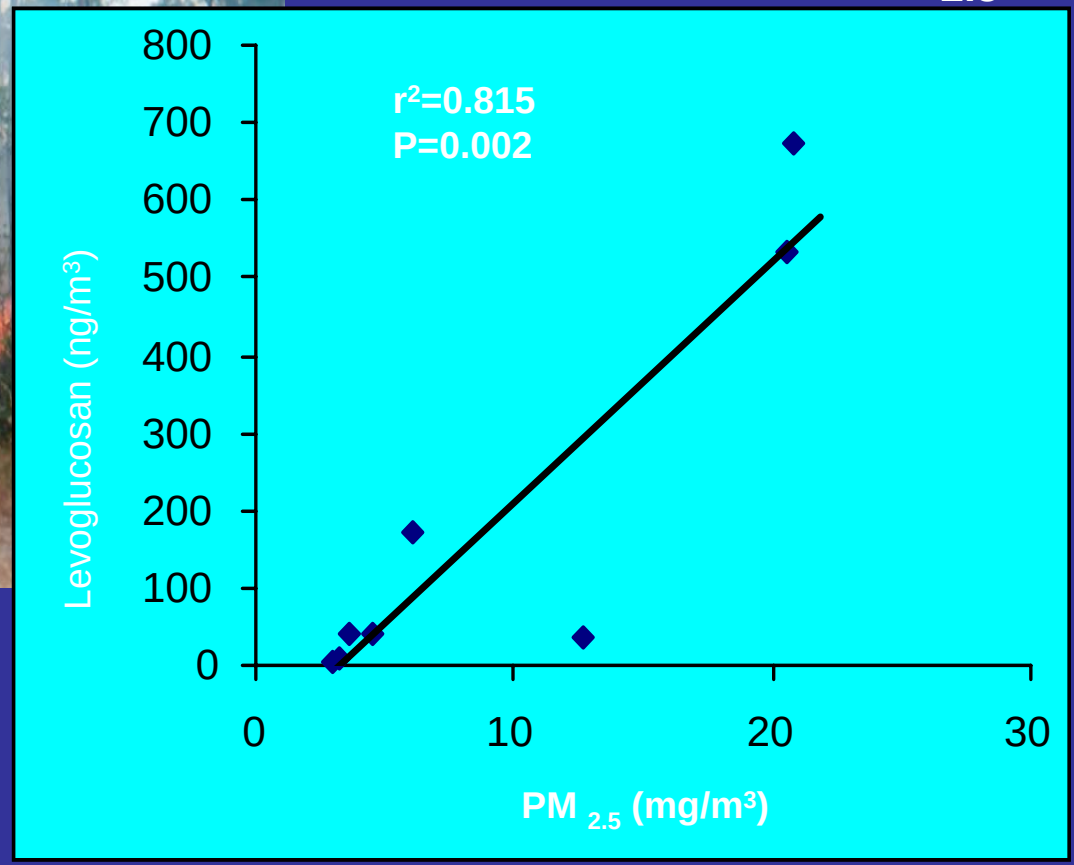
2) Use more objective indicators of health endpoints (e.g. lung function vs. symptom reporting)

3) Demonstrate the effectiveness of policy on humanhealth, not simply concentrations of pollutants

1) Improve our measures of exposure

- CDC Firefighter studies and Mexican Stove Project

Levoglucosan vs $PM_{2.5}$



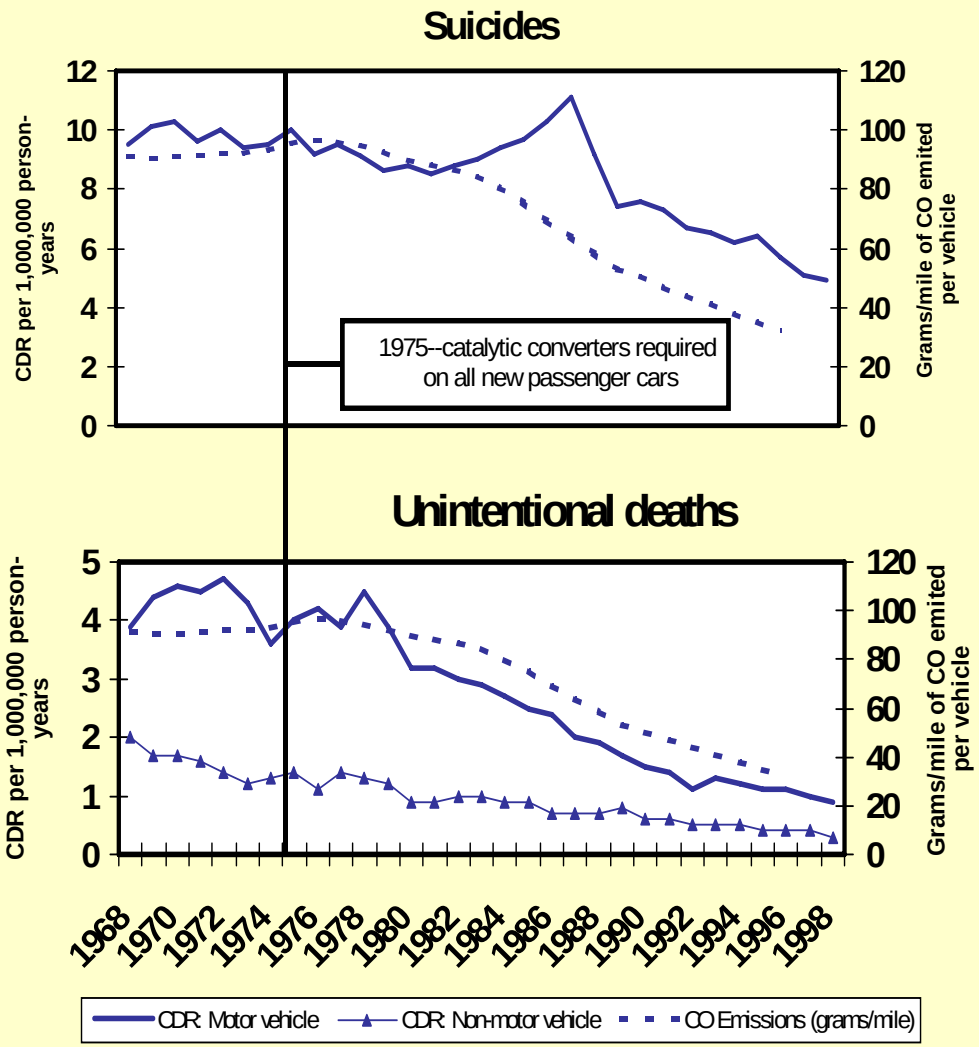
2) More objective health endpoints

- CDC Summertime Ozone Study

3) **Perhaps an opportunity to collaborate and demonstrate the impact of an air pollution reduction policy on human health outcomes?**

- **(e.g. CDC/EPA Evaluation of the Clean Air Act on CO poisoning deaths)**

Annual crude death rates from carbon monoxide poisoning, and average annual estimated CO emissions per light duty motor vehicle (grams/ test mile), United States, 1968-1998.



CO Emissions Control Timeline

1970: Congress enacts Clean Air Act. CO emissions standard at 34.0 grams/mile.

1975: Catalytic converter introduced on new passenger cars to meet new CO emissions standard of 15 grams/mile.

1978: 1975 and newer model year cars make up 34% of the U.S. passenger vehicle fleet.

1980: All new passenger cars required to meet new CO emissions standard of 7.0 g/mile. 1975 and newer model year cars make up 50% of US. passenger vehicle fleet.

1981: All new cars required to meet new CO emissions standard of 3.4 g/mile.

1990: 1975 and newer model year cars make up 91% of the U.S. passenger vehicle fleet.

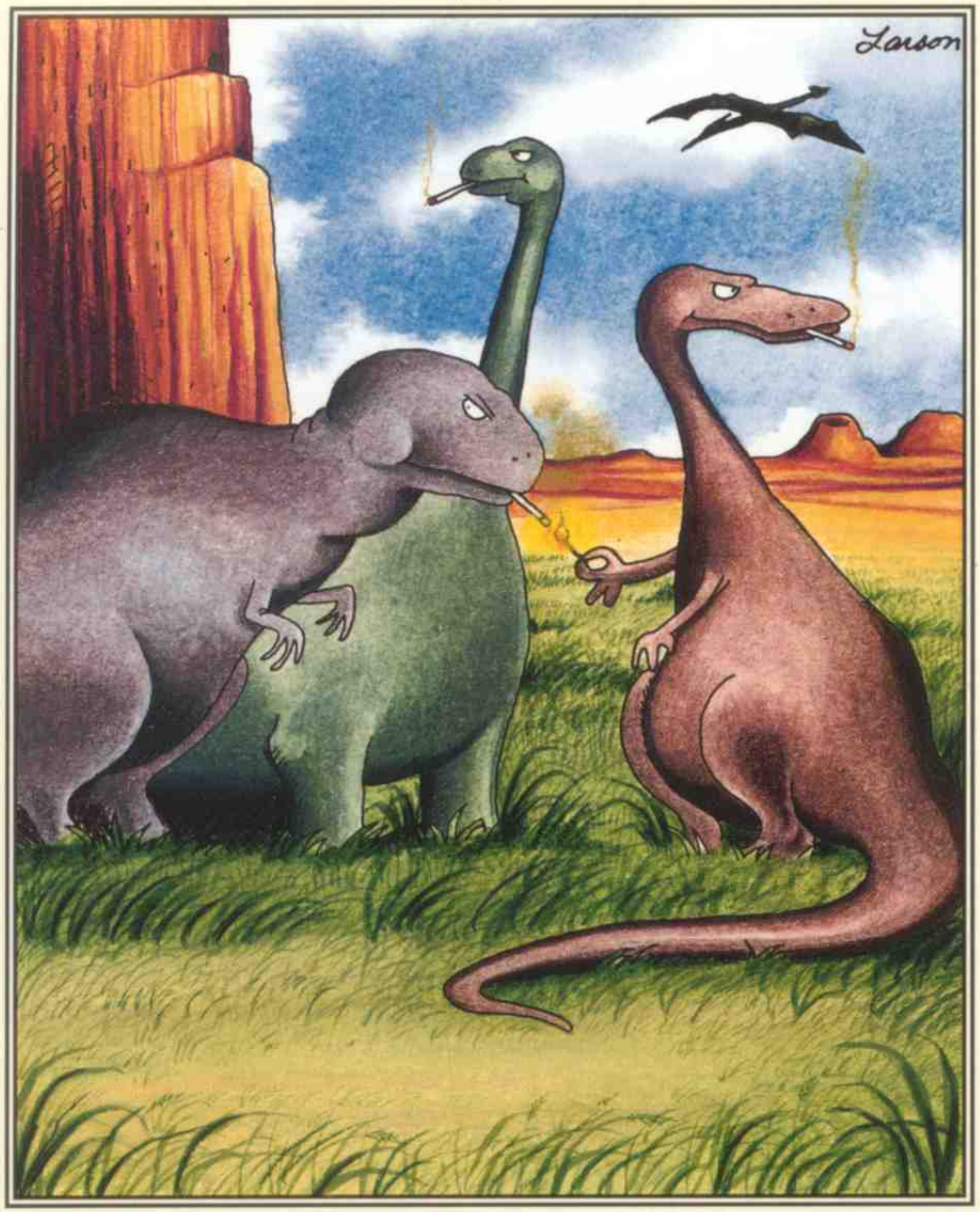
1992: Standards setting emission limits for carbon monoxide at temperatures < 20 ° F are established. Oxygenated gasoline is introduced in cities with high CO levels.

Source:EPA CO emissions inventory data, and Fact Sheet OMS-12.

Did you know? Replacing 20 non-certified, older stoves with 20 EPA certified stoves can prevent the emissions of one ton of particulate matter (PM2.5) into our environment per year.

What is the health benefit for those who live in homes with new stoves?

12/15/82



The real reason dinosaurs became extinct