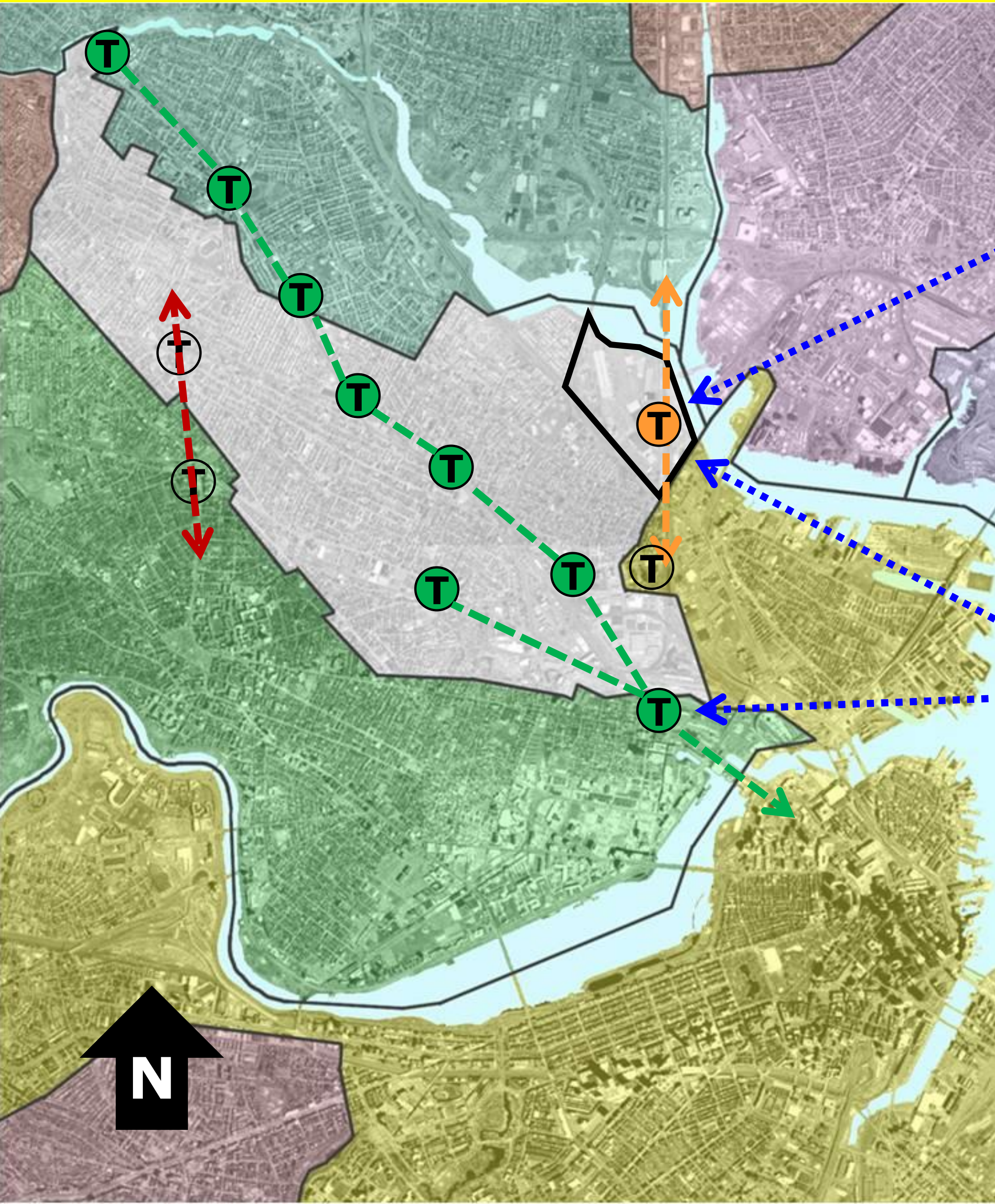
What you see in this picture is the least of it!

I93 has 150,000 vehicles per day overhead

New housing proposed with retail to the right

MBTA diesel commuter rail lines (3) under

FOCUS of STEP and Mystic View Task Force



Land Use and Economic Development

Assembly Square - 145 acres

- Old underutilized industrial site (FORD plant)
- Developers preferred Big Boxes
- Community wanted dense mixed use (TOD)
  - 30 new acres open space
  - 30,000 jobs
  - 30,000,000 net annual taxes
- Settled December 2006 after years of battle

Transportation

- Orange Line ~ $50M first T-stop 33 years
- Green Line Extension ~ $1B ~ first light rail
  branches in several generations
- Solid T circles represent new stations
- Community Path to connect from Bedford
  through Davis Square to Green Line and Boston

STEP and Mystic View used air quality and health in their advocacy for local and regional changes

Recent Afternoon on I93 in Somerville MA – Citizens still learning to drive

Somerville has 200,000 VMT per square mile per day and most diesel rail

Action-oriented focus of volunteer groups Mystic View and STEP in Somerville evolved as follows:

1998 to 2000 and Beyond

LIVE WORK BALANCE

Jobs, Taxes & Open Space

2001 to 2003 and Beyond

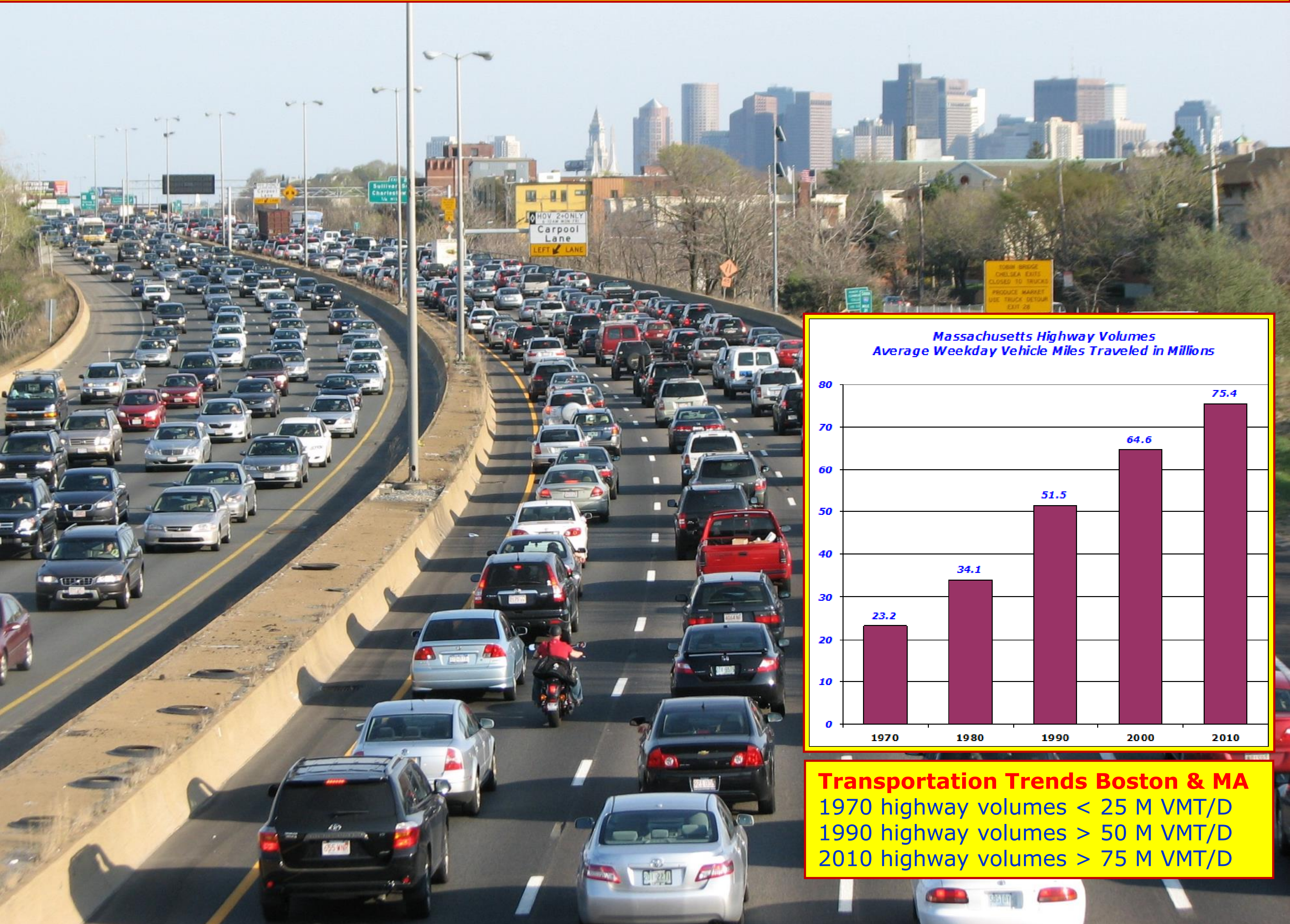
Transportation Capacity

2004 to 2006 and Beyond

Air Quality & Public Health

These focal areas turned out to be highly interactive!

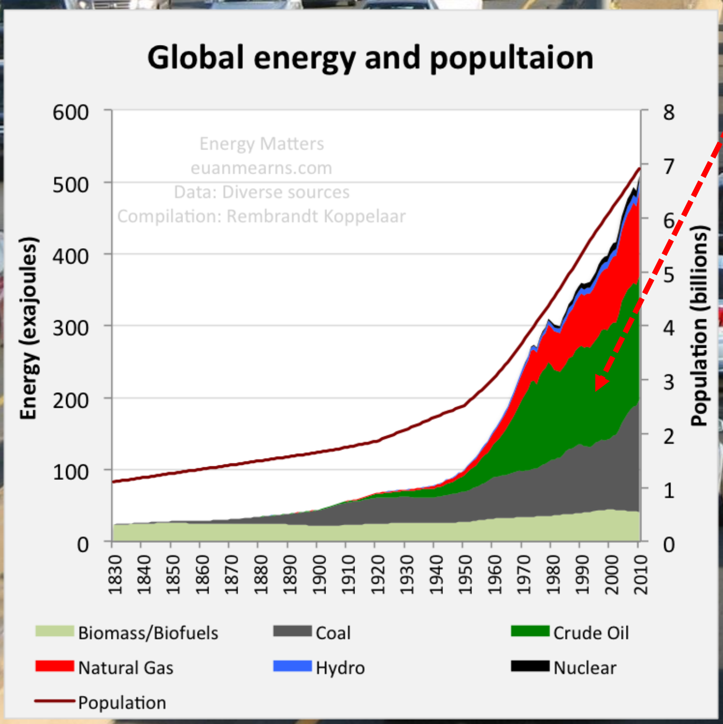
Recent Afternoon on I93 at Somerville Medford MA city line – Citizens still learning to drive!



Transportation Trends Boston & MA
1970 highway volumes < 25 M VMT/D
1990 highway volumes > 50 M VMT/D
2010 highway volumes > 75 M VMT/D

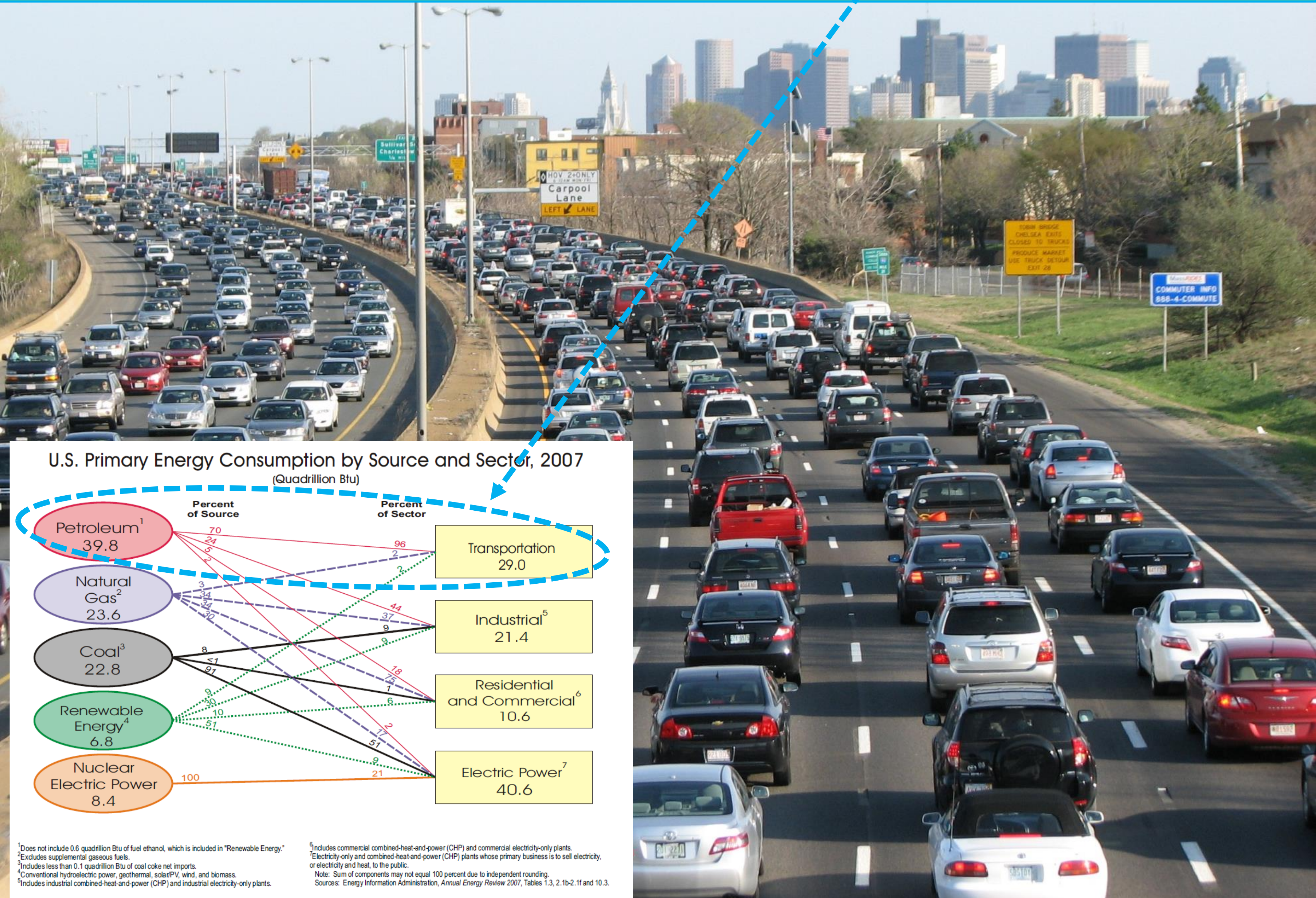
Recent Afternoon on I93 in Somerville MA – Citizens still learning to drive

Transportation dominates petroleum consumption , but could be multi-source electric



Recent Afternoon on I93 in Somerville MA – Citizens still learning to drive

One big issue is that transportation dominates petroleum consumption (70%) but could be multi-source electric




Is this a healthy place and time for a young mother and daughter to exercise?


Are these healthy places for housing?
Are HEPA filter interventions possible?
TO NORTH USE EXIT 28
93
TUNNEL 3 MILES


Urban alternatives can provide respite
NO MOTOR VEHICLES
Somerville Community Path near Davis Square


Is Route 28 a good place for a bicycle commuter in morning rush hour?

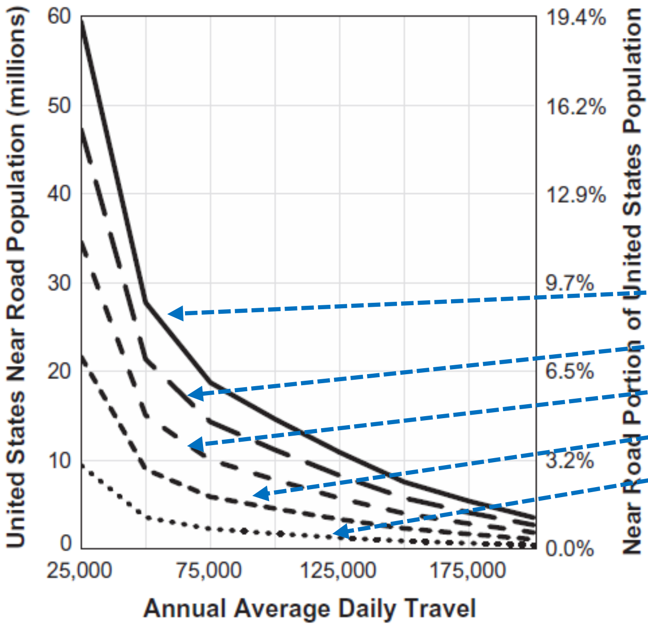


Fig. 1. US population living near high volume roads during the year 2010.

A census of the US near-roadway population: Public health and environmental justice considerations

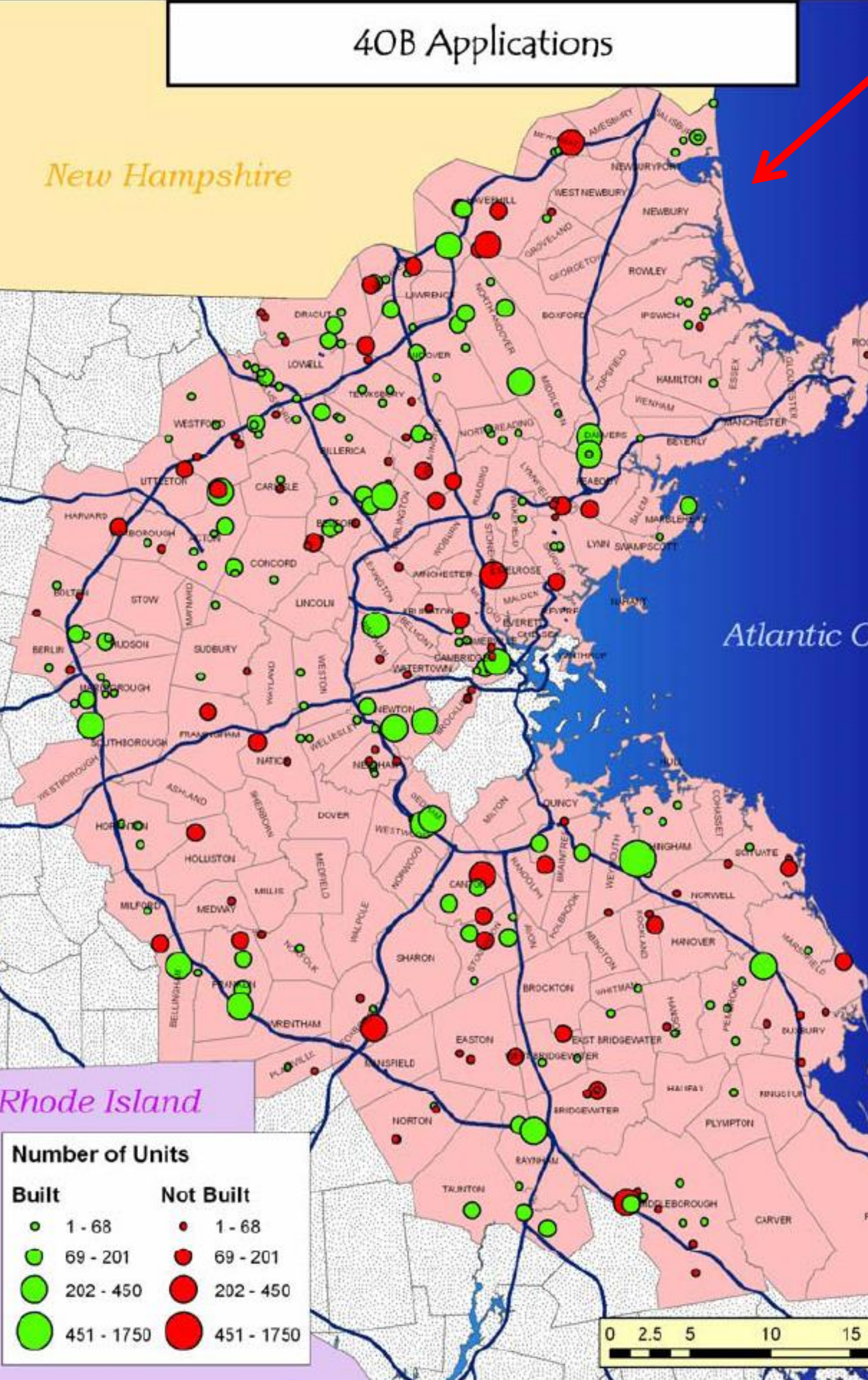
Gregory M. Rowangould *

Civil Engineering Department, MSC01 1070, University of New Mexico, Albuquerque, NM 87131, USA

A B S T R A C T

This study estimates the size and distribution of the population living near high volume roads in the US, investigates race and income disparities in these near roadway populations, and considers the coverage of the national ambient air quality monitoring network. Every US census block is classified by traffic density and proximity to roads falling within several traffic volume ranges using year 2008 traffic data and the 2010 and 2000 US Census. The results indicate that 19% of the population lives near high volume roads. Nationally, greater traffic volume and density are associated with larger shares of non-white residents and lower median household incomes. Analysis at the county level finds wide variation in the size of near roadway populations and the severity of environmental justice concerns. Every state, however, has some population living near a high volume road and 84% of counties show some level of disparity. The results also suggest that most counties with residents living near high volume roads do not have a co-located regulatory air quality monitor.

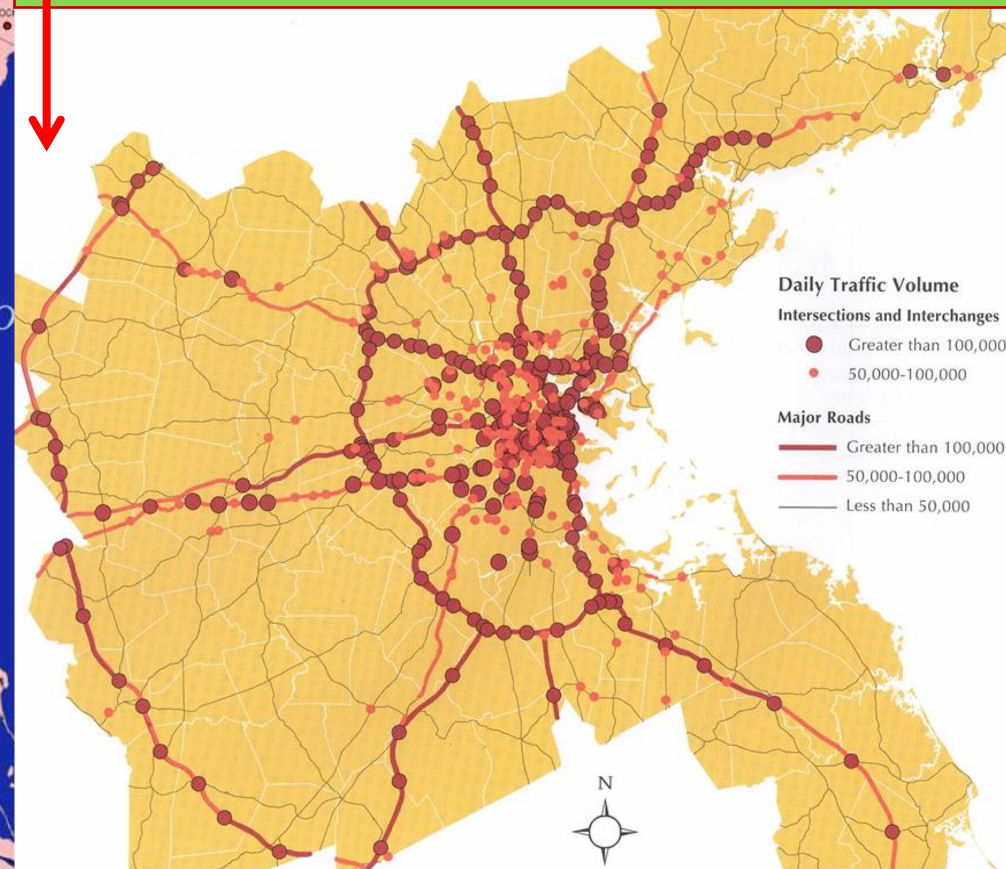
Near Roadway Exposure
~60 Million US Residents
~20% of US Population

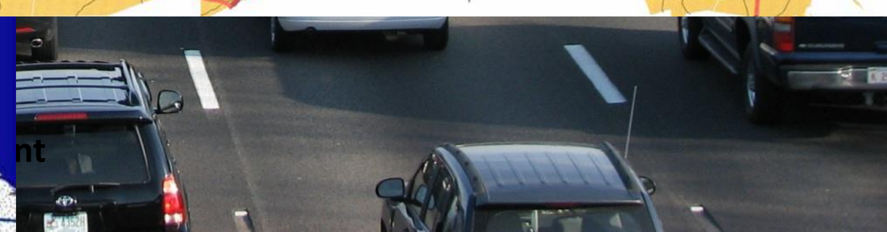


MIT's Map of 40B Affordable Housing Projects and MAPC's Map of Highway Pollution Health Hazards

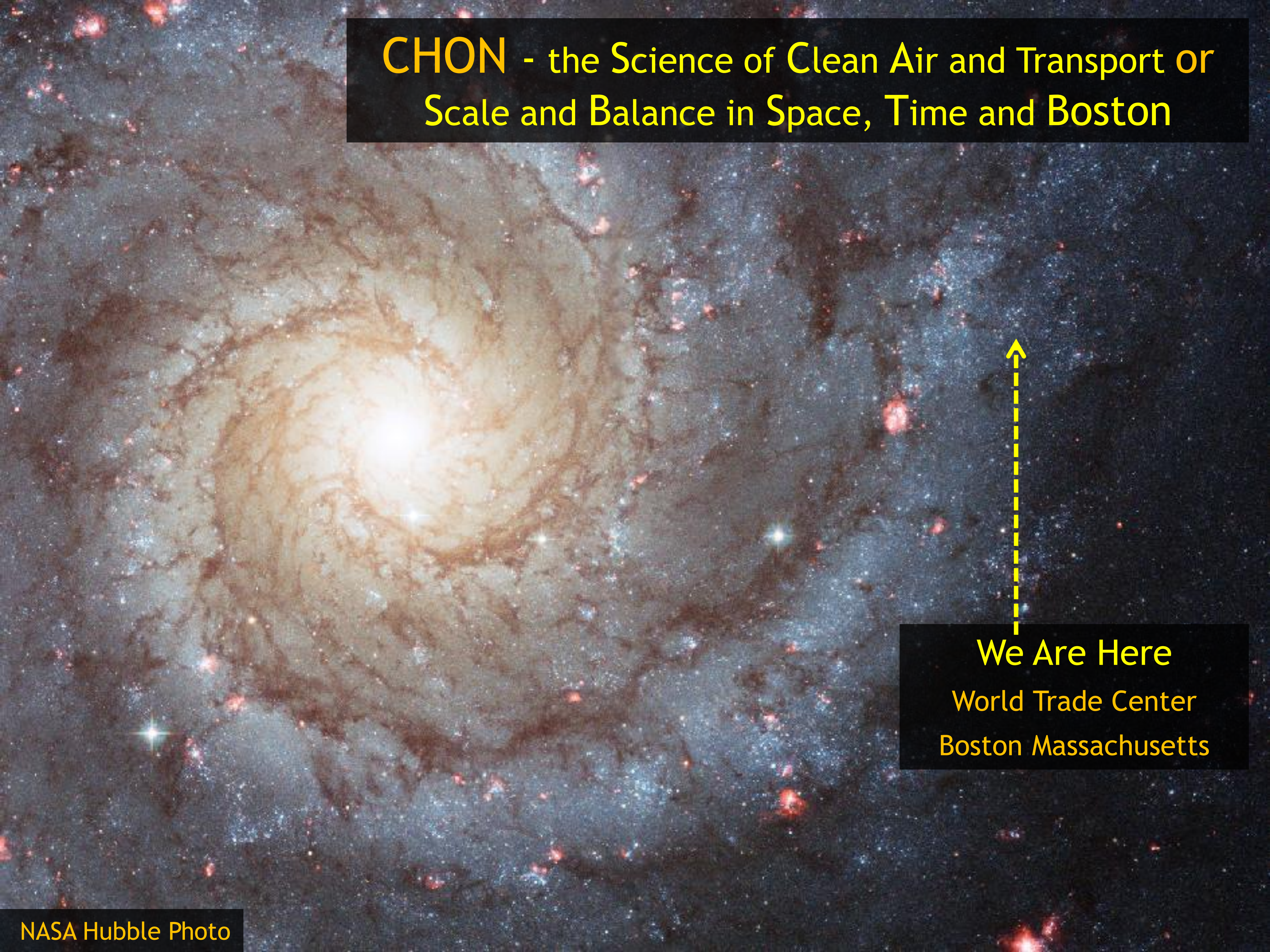
Are virtually THE SAME MAP!

In the US, 9% of all people and 18% of all renters live within a few hundred feet of highways or other large mobile air pollution sources.

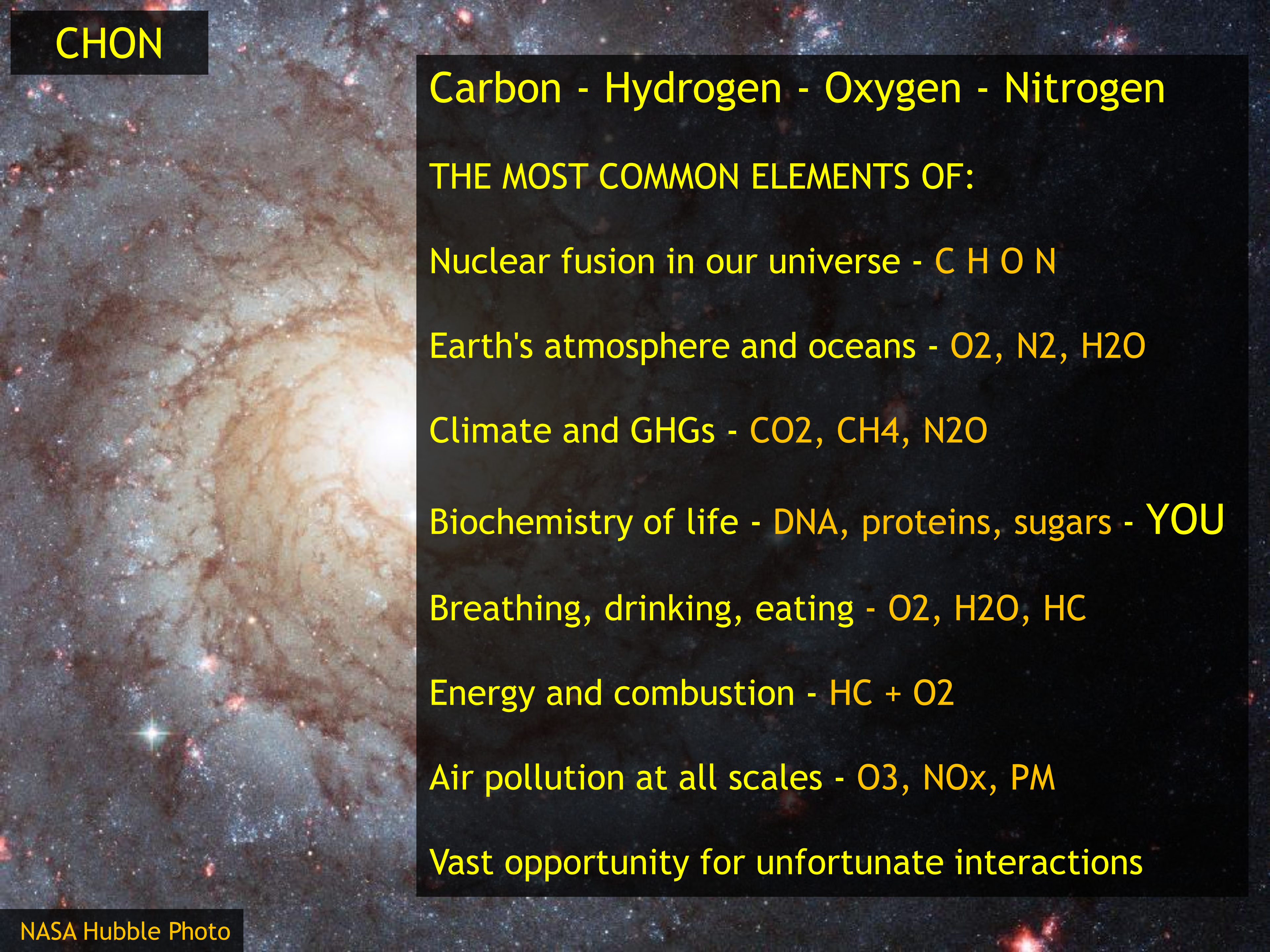




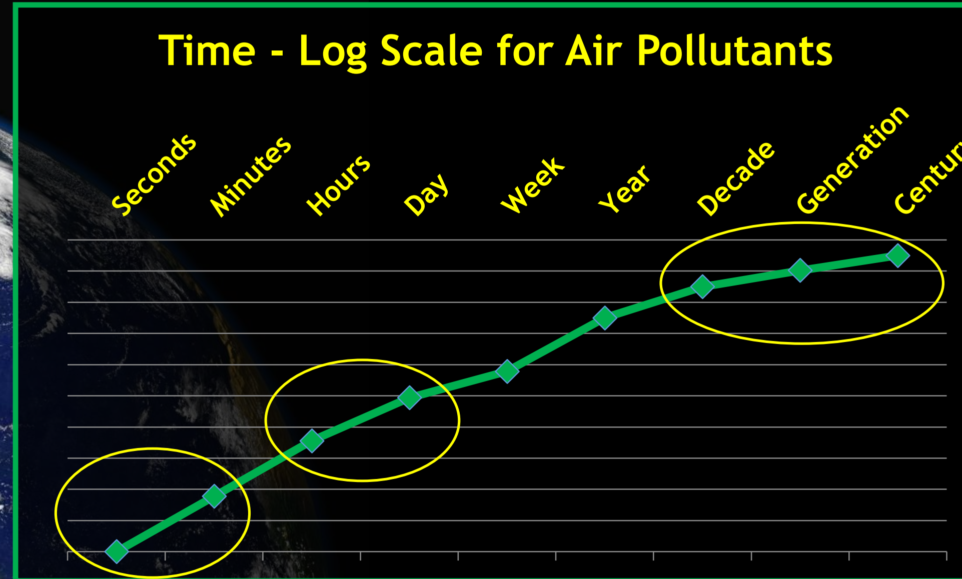
CHON - the Science of Clean Air and Transport or Scale and Balance in Space, Time and Boston



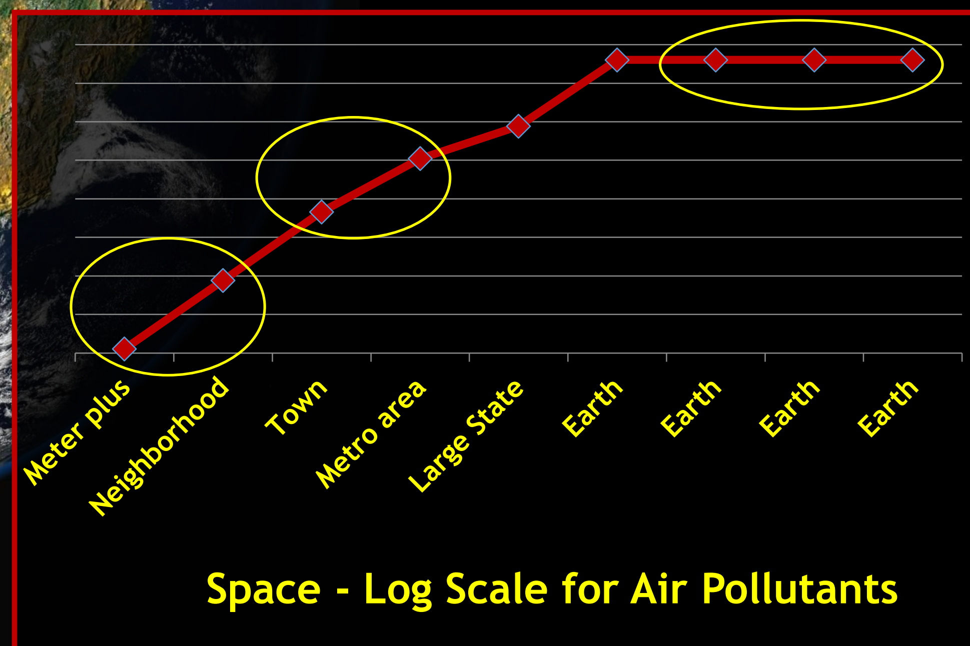
NASA Hubble Photo


CHON
Carbon - Hydrogen - Oxygen - Nitrogen
THE MOST COMMON ELEMENTS OF:
Nuclear fusion in our universe - C H O N
Earth's atmosphere and oceans - O2, N2, H2O
Climate and GHGs - CO2, CH4, N2O
Biochemistry of life - DNA, proteins, sugars - YOU
Breathing, drinking, eating - O2, H2O, HC
Energy and combustion - HC + O2
Air pollution at all scales - O3, NOx, PM
Vast opportunity for unfortunate interactions
NASA Hubble Photo

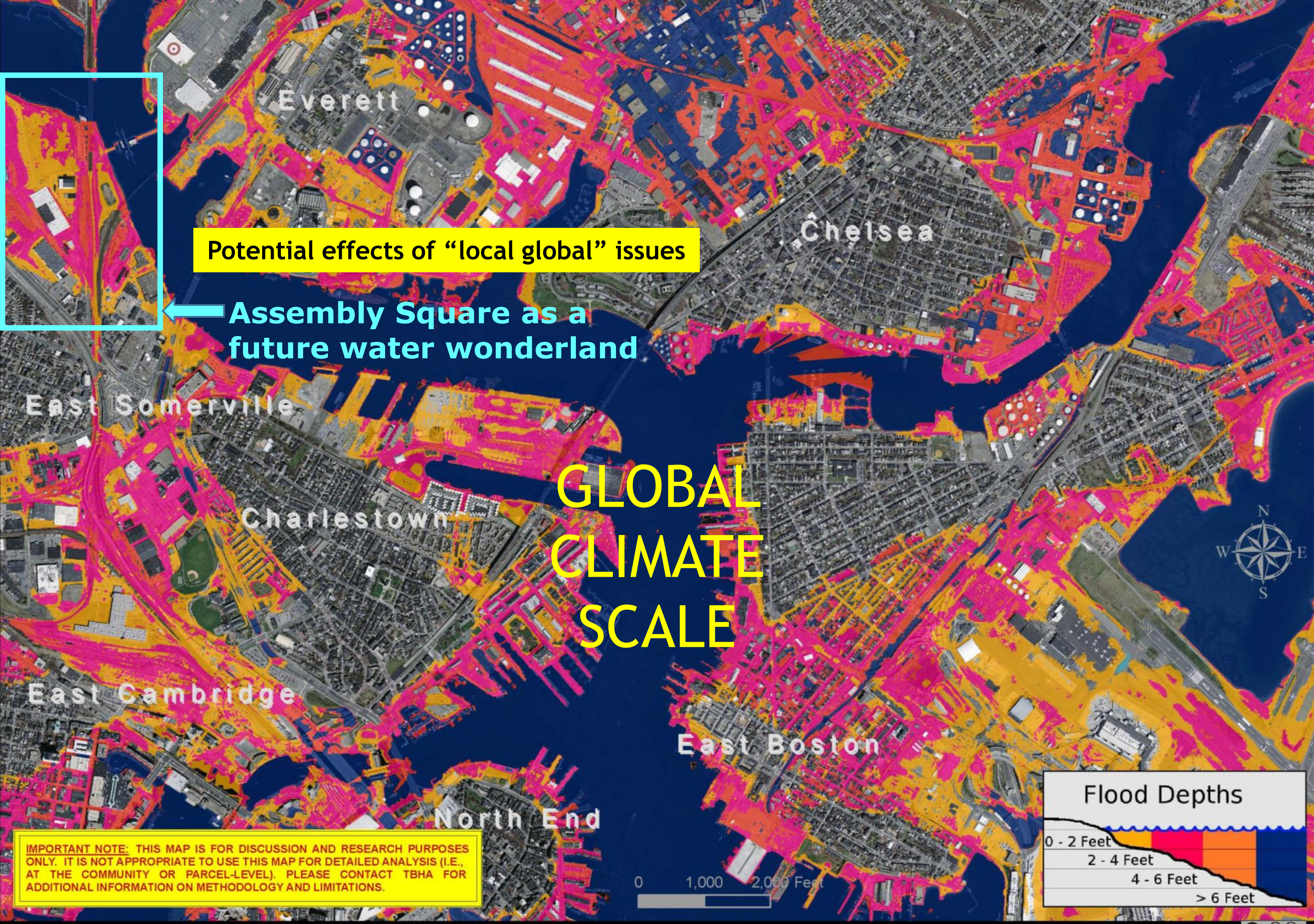
Scale of Time and Space on Earth



Near Highway EPA Regional Climate GHGs



NASA



Battelle The Business of Innovation | University of Massachusetts Boston | the boston harbor association

Chelsea, Everett, EastBoston
Mean Higher High Water Plus 7.5 Feet
Sources: MassGIS, NOAA, USGS

Map Development:
Chris Watson, Ellen Douglas - UMass Boston
Paul Kirshen - Battelle

Notice the difference in PM2.5 scale

US – 0 to 18

Europe – 0 to 35

SE Asia – 0 to 100

~ 7 Million Deaths per Year due to PM, mostly China and India

Between 140,000 & 360,000 in the US Out of total of 2,500,000

Between 1990 and 2010 a huge shift from communicable diseases to environmental

REGIONAL PM2.5 SCALE

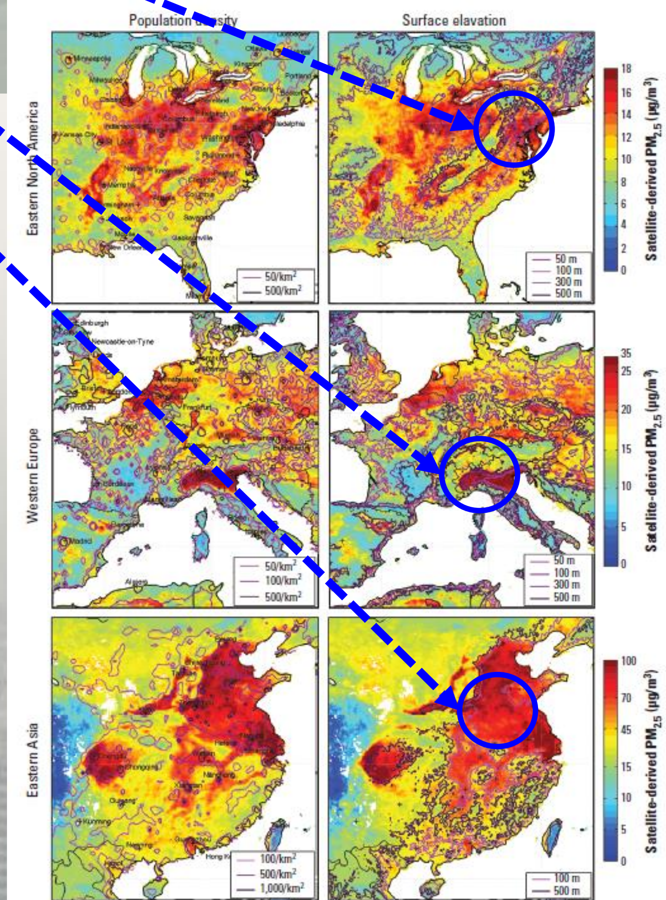


Figure 5. Regional satellite-derived $PM_{2.5}$ concentrations. Columns show mean satellite-derived $PM_{2.5}$ for 2001–2006 at locations that contain at least 50 measurements. Contours denote population density (left) and surface elevation (right). Crosses indicate city centers. Note the different color scales for each region. Altitude data are from the U.S. Geological Survey (1996).

PETER PARKS via Getty Images

Shanghai a while back

From Lim LANCET December 2012 WHO
Global Burden of Disease Mortalities

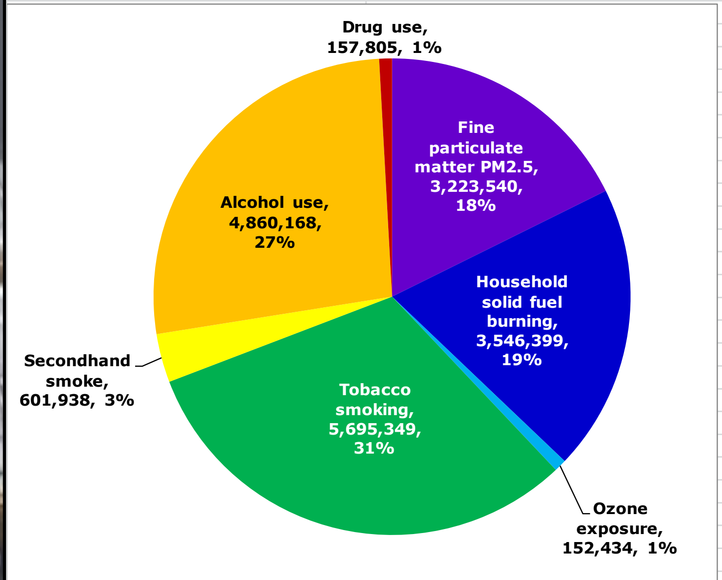
Millions of Global Deaths:

3.2 PM2.5 Regional Air Pollution

3.6 Household Cooking

5.7 Tobacco

2010 GBD Attributable Deaths from Air Pollution and Drugs Only	
Fine particulate matter PM2.5	3,223,540
Household solid fuel burning	3,546,399
Ozone exposure	152,434
Tobacco smoking	5,695,349
Secondhand smoke	601,938
Alcohol use	4,860,168
Drug use	157,805



2010 GBD Attributable Deaths from Air Pollution and Smoking Only	
Fine particulate matter PM2.5	3,223,540
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Tobacco smoking	5,695,349
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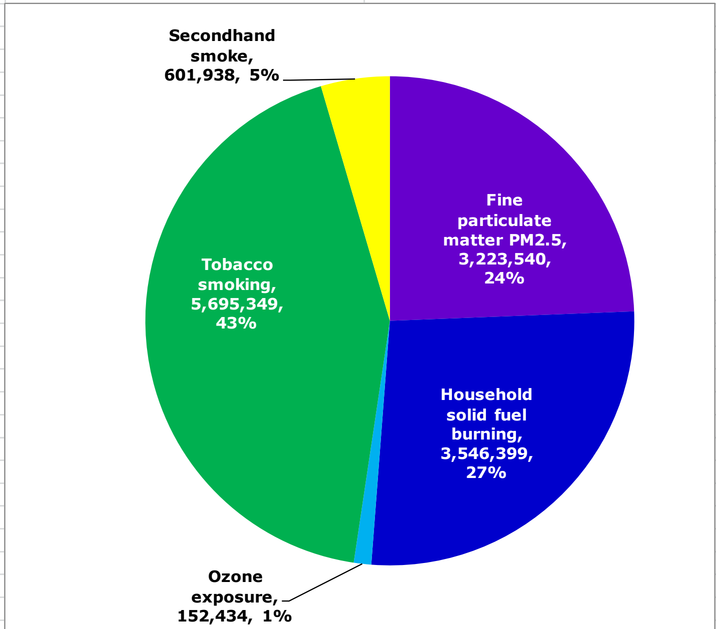
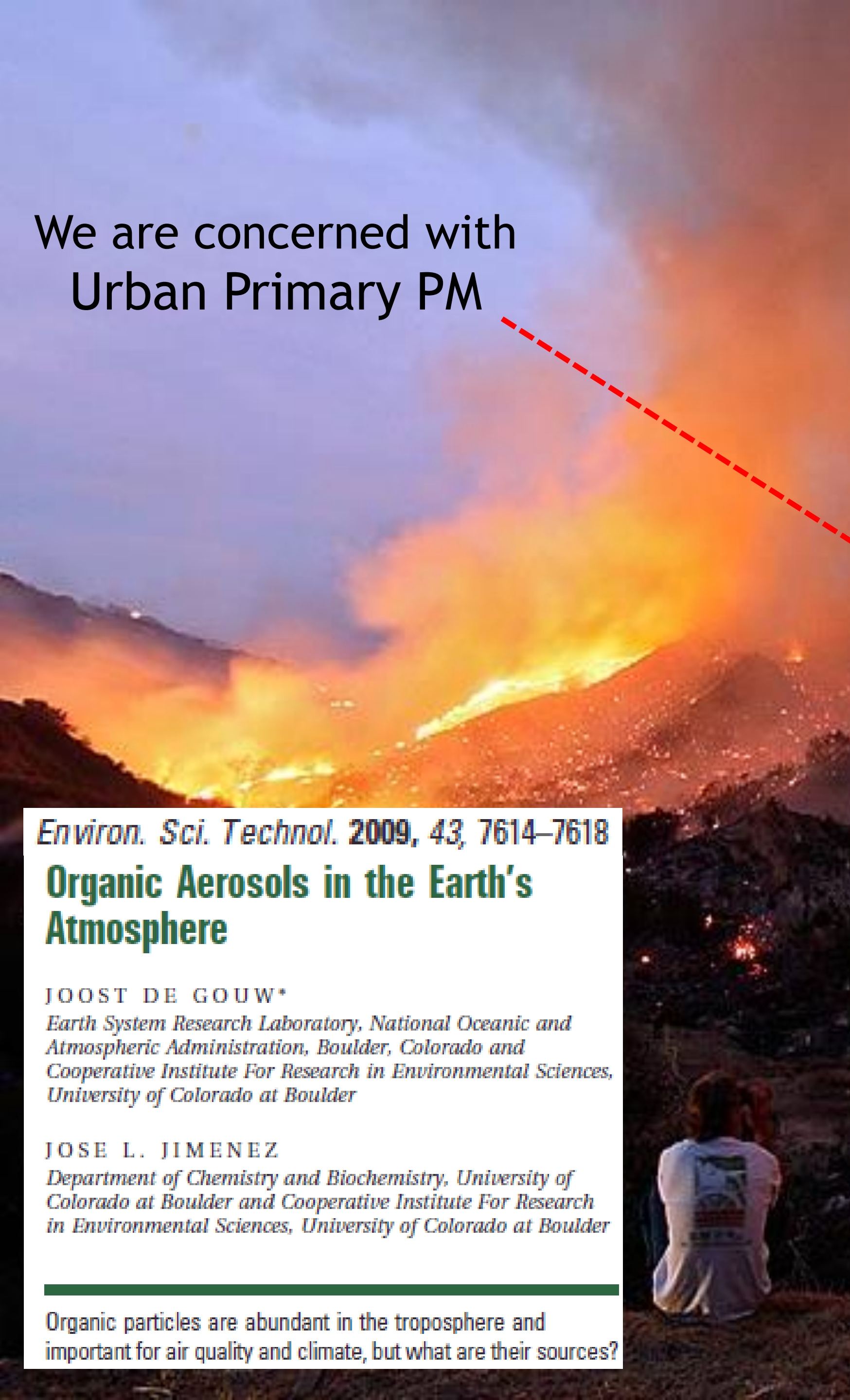



Eyjafjallajokull
April 2010 - LA Times

Second hand smoke

Cooking indoors and out

Pasadena Burns 2009
LA Times Photo



We are concerned with
Urban Primary PM

Environ. Sci. Technol. **2009**, *43*, 7614–7618

Organic Aerosols in the Earth's Atmosphere

JOOST DE GOUW*
Earth System Research Laboratory, National Oceanic and Atmospheric Administration, Boulder, Colorado and Cooperative Institute For Research in Environmental Sciences, University of Colorado at Boulder

JOSE L. JIMENEZ
Department of Chemistry and Biochemistry, University of Colorado at Boulder and Cooperative Institute For Research in Environmental Sciences, University of Colorado at Boulder

Organic particles are abundant in the troposphere and important for air quality and climate, but what are their sources?

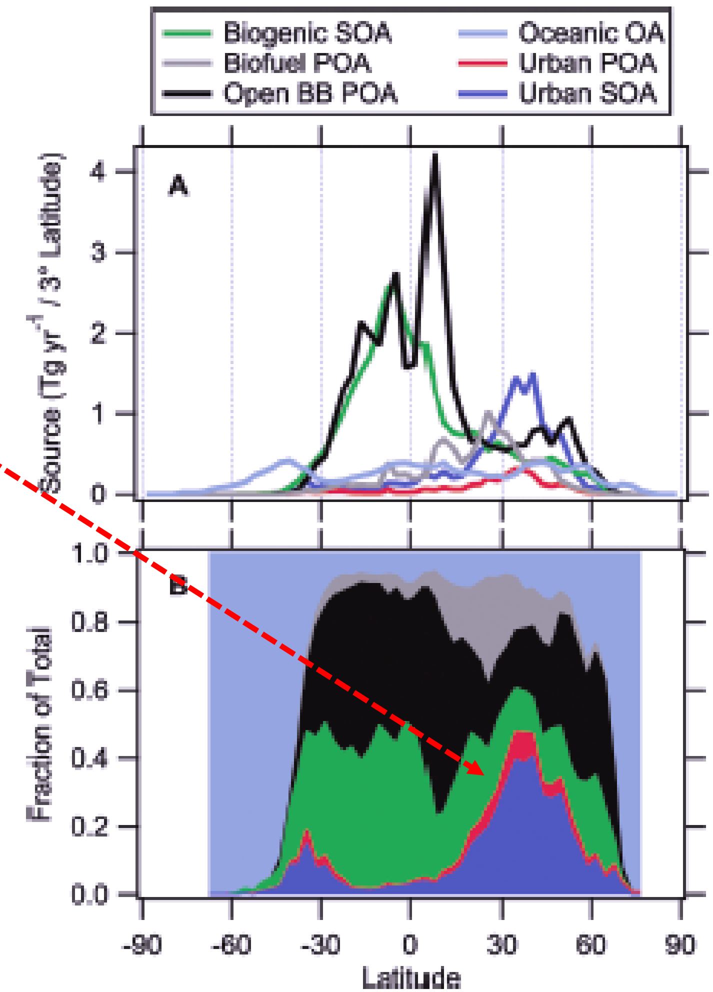
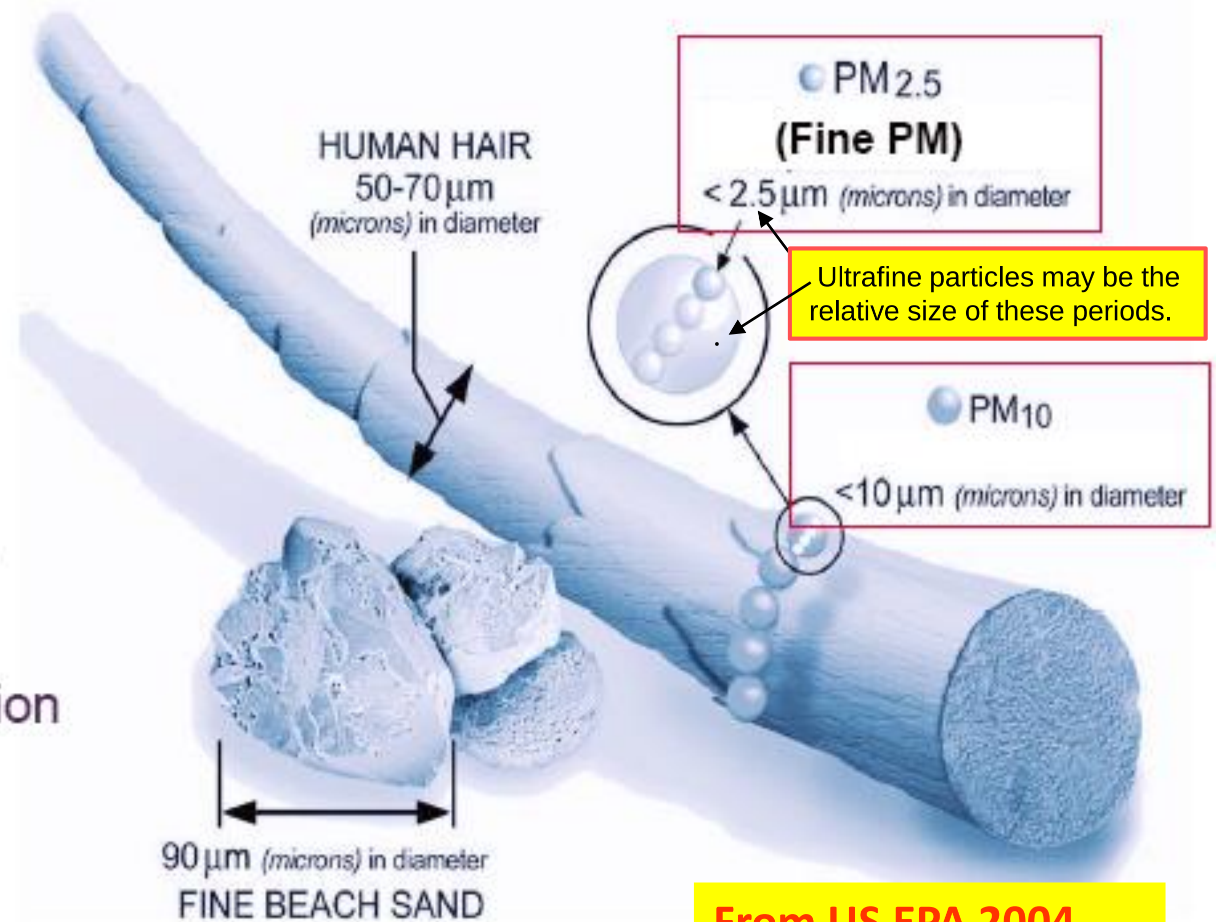


FIGURE 3. Estimated mean zonal distribution of organic aerosol sources. Biogenic SOA (*4*, *40*, *52*), oceanic OA (*53*), and POA (*32*) are taken from the literature. Urban SOA is calculated here. Note that the zonal distribution of concentrations may deviate from that of sources due to transport.

What is Particulate Matter (PM)?

Ultrafine particles can be 100 to 1000 times smaller than coarse and fine particles.

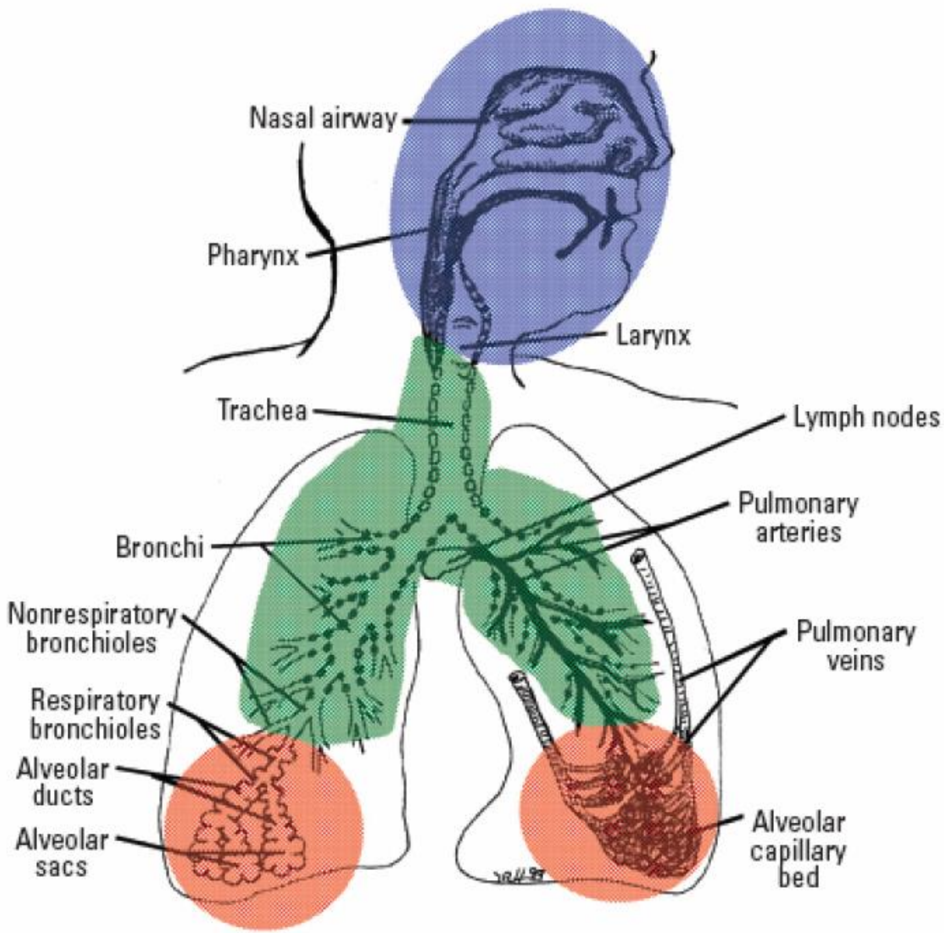
- PM is a complex mixture of solid, semi-volatile and aqueous materials of various sizes found in the air.
- When inhaled, smaller particles generally penetrate more deeply into the lung.
- The size and composition of PM have important implications regarding health outcomes.



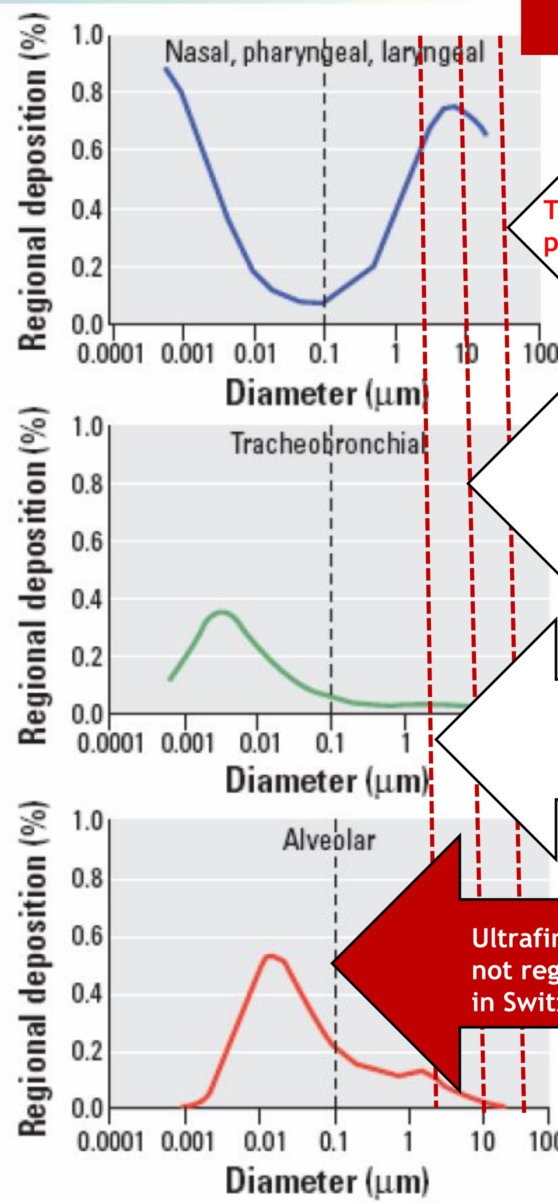
From US EPA 2004

EPA PM NAAQS applied to smaller particles over time

Translocation, cont.



Oberdorster et al., EHP, 2005



Total suspended particulates 1970

PM 10 1987

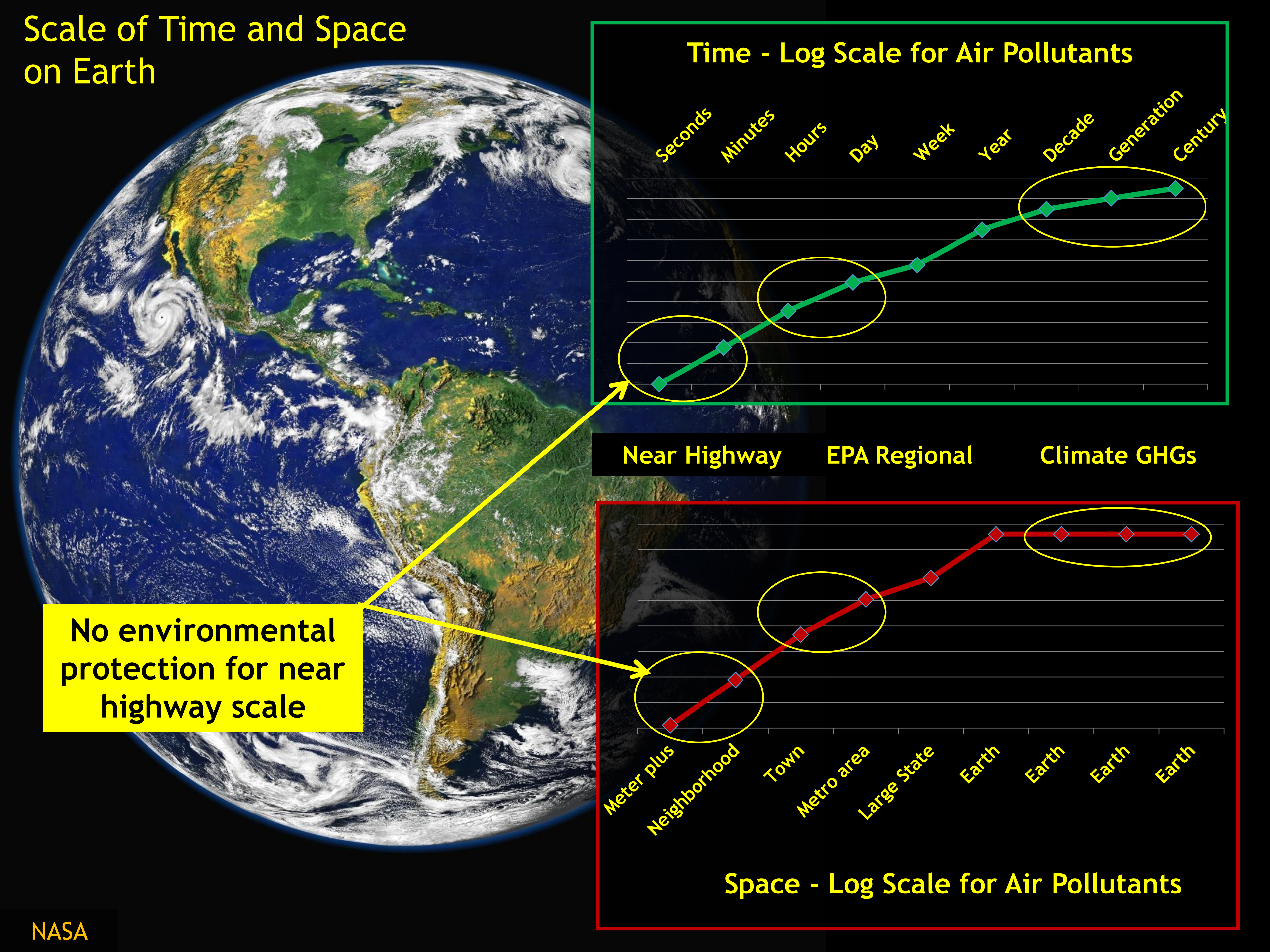
PM 2.5 1997

Ultrafine Particles not regulated except in Switzerland

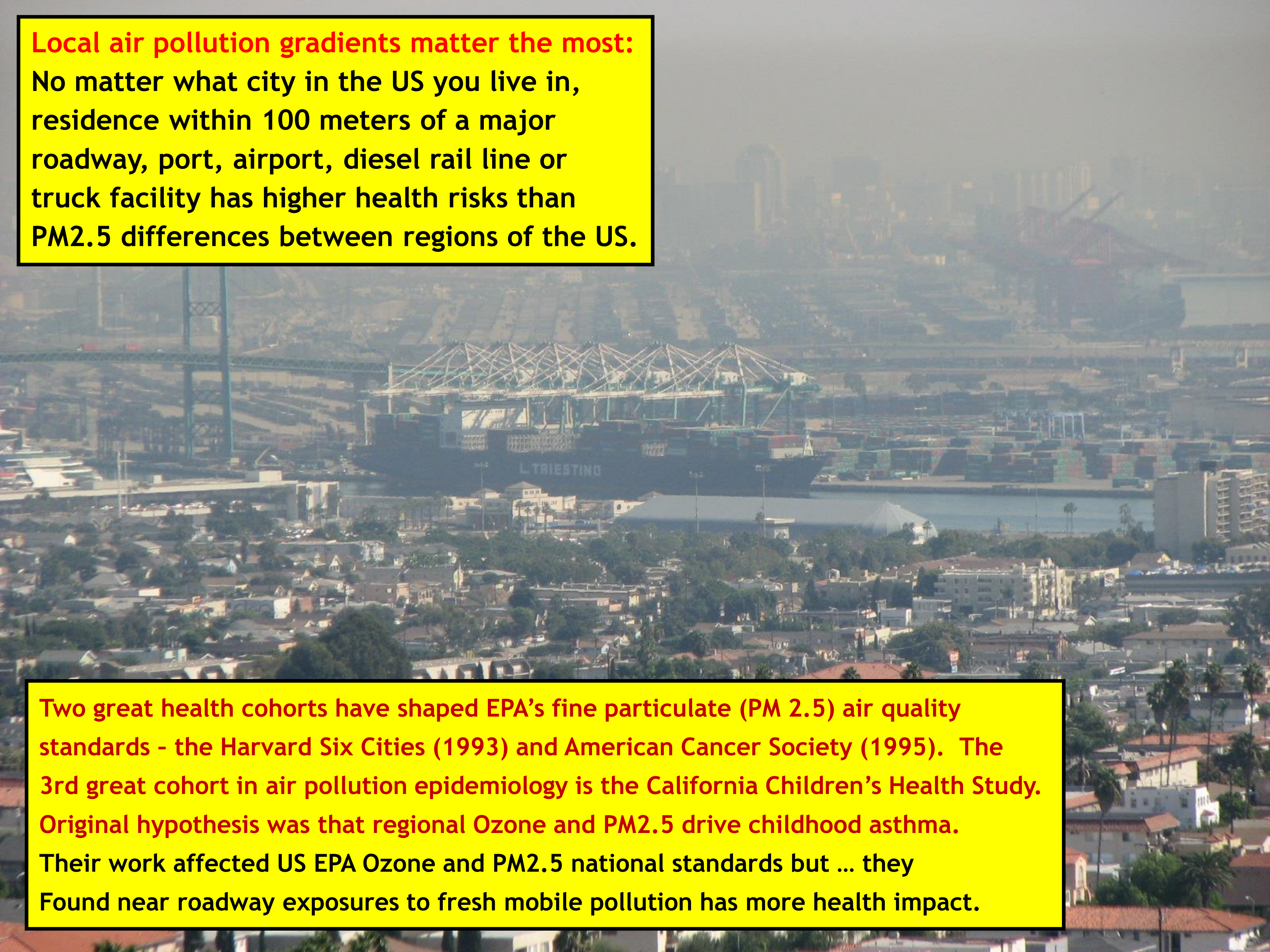
Smallest particles have deepest penetration and high deposition rates in all areas, especially alveolar and nasal/pharyngeal.

Over time smaller particles = bigger concerns

John Froines slide


Scale of Time and Space on Earth
Time - Log Scale for Air Pollutants
Seconds
Minutes
Hours
Day
Week
Year
Decade
Generation
Century
Near Highway
EPA Regional
Climate GHGs
No environmental protection for near highway scale
Meter plus
Neighborhood
Town
Metro area
Large State
Earth
Earth
Earth
Earth
Space - Log Scale for Air Pollutants
NASA

Local air pollution gradients matter the most:
No matter what city in the US you live in, residence within 100 meters of a major roadway, port, airport, diesel rail line or truck facility has higher health risks than PM2.5 differences between regions of the US.



Two great health cohorts have shaped EPA's fine particulate (PM 2.5) air quality standards – the Harvard Six Cities (1993) and American Cancer Society (1995). The 3rd great cohort in air pollution epidemiology is the California Children's Health Study. Original hypothesis was that regional Ozone and PM2.5 drive childhood asthma.
Their work affected US EPA Ozone and PM2.5 national standards but … they
Found near roadway exposures to fresh mobile pollution has more health impact.

Changes in Residential Proximity to Road Traffic and the Risk of Death From Coronary Heart Disease

Wen Qi Gan,[a] Lillian Tamburic,[b] Hugh W. Davies,[a] Paul A. Demers,[a,c] Mieke Koehoorn,[a,c] and Michael Brauer[a]

Heart Disease Mortality highest for those living near highways

In a related Gan multi-pollutant paper PM2.5 lost all statistical significance when compared with NO2 and BC

55% Higher Cardiovascular Mortality Near Highways
Vancouver

Background: Residential proximity to road traffic is associated with increased coronary heart disease (CHD) morbidity and mortality. It is unknown, however, whether changes in residential proximity to traffic could alter the risk of CHD mortality.
Methods: We used a population-based cohort study with a 5-year exposure period and a 4-year follow-up period to explore the association between changes in residential proximity to road traffic and the risk of CHD mortality. The cohort comprised all residents aged 45–85 years who resided in metropolitan Vancouver during the exposure period and without known CHD at baseline (n = 450,283). Residential proximity to traffic was estimated using a geographic information system. CHD deaths during the follow-up period were identified using provincial death registration database. The data were analyzed using logistic regression.
Results: Compared with the subjects consistently living away from road traffic (>150 m from a highway or >50 m from a major road) during the 9-year study period, those consistently living close to traffic (≤150 m from a highway or ≤50 m from a major road) had the greatest risk of CHD mortality (relative risk [RR] = 1.29 [95% confidence interval = 1.18–1.41]). By comparison, those who moved closer to traffic during the exposure period had less increased risk than those who were consistently exposed (1.20 [1.00–1.43]), and those who moved away from traffic had even less increase in the risk (1.14 [0.95–1.37]). All analyses were adjusted for baseline age, sex, pre-existing comorbidities (diabetes, chronic obstructive pulmonary disease, hypertensive heart disease), and neighborhood socioeconomic status.
Conclusions: Living close to major roadways was associated with increased risk of coronary mortality, whereas moving away from major roadways was associated with decreased risk.

(*Epidemiology* 2010;21: 000–000)

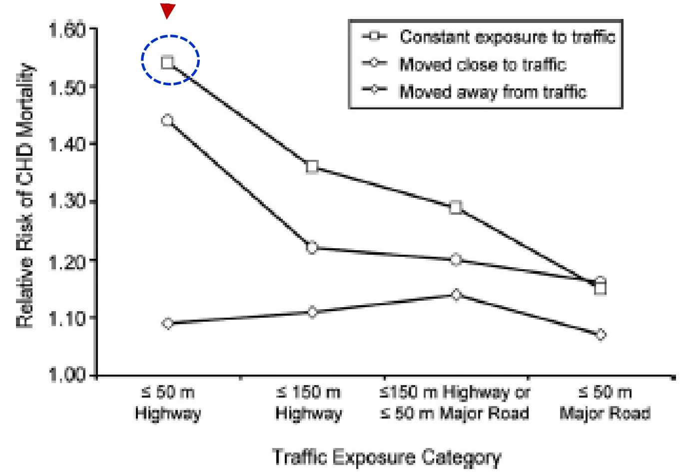


FIGURE 1. Association of road traffic exposure with coronary heart disease mortality by road types and distances. RRs adjusted for age, sex, neighborhood SES, and pre-existing comorbidities.

Nyberg Stockholm 2000 – All statistical significance is in highest NO2 decile when viewing association of lung cancer with long term residential exposures Nafstad's Oslo NOx lung cancer study had higher concentration response

60% Higher Lung Cancer Mortality in those exposed to Traffic Stockholm

TABLE 4. Relative Risk of Lung Cancer (and 95% Confidence Interval) Associated with 10-Year Averages of Two Exposure Indicators for Air Pollution (NO_2 for Traffic-Related Air Pollution and SO_2 for Air Pollution from Heating) Lagged 20 Years

Variable	Cases	Controls	One Pollutant*		Both Pollutants†	
			RR‡	95% CI‡	RR‡	95% CI‡
NO_2 from road traffic						
Continuous variable (per 10 $\mu g/m^3$)			1.10	0.97–1.23	1.15	0.97–1.35
Quartiles and 90th percentile						
$<12.78\ \mu g/m^3$§	243	608	1		1	
≥ 12.78 to $<17.35\ \mu g/m^3$	264	588	1.15	0.91–1.46	1.19	0.91–1.56
≥ 17.35 to $<23.17\ \mu g/m^3$	250	601	1.01	0.79–1.29	1.11	0.83–1.48
≥ 23.17 to $<29.26\ \mu g/m^3$	165	346	1.07	0.81–1.42	1.19	0.86–1.66
$\geq 29.26\ \mu g/m^3$	120	221	1.44	1.05–1.99	1.60	1.07–2.39
SO_2 from heating						
Continuous variable (per 10 $\mu g/m^3$)			1.01	0.98–1.03	0.99	0.95–1.02
Quartiles and 90th percentile						
$<66.20\ \mu g/m^3$§	239	612	1		1	
≥ 66.20 to $<87.60\ \mu g/m^3$	270	581	1.16	0.91–1.47	1.07	0.83–1.40
≥ 87.60 to $<110.30\ \mu g/m^3$	259	593	1.00	0.79–1.27	0.90	0.67–1.19
≥ 110.30 to $<129.10\ \mu g/m^3$	151	360	0.92	0.70–1.21	0.80	0.58–1.12
$\geq 129.10\ \mu g/m^3$	123	218	1.21	0.89–1.66	0.95	0.64–1.39

Estimated time weighted average air pollution exposure 21–30 years before end of follow-up.

* Estimate obtained when only one pollutant was entered into the regression model.

† Estimate obtained when the corresponding variable for the other pollutant (SO_2 or NO_2) was entered separately into the same regression model as a confounder. For example, point estimates 1.15 (NO_2) and 0.99 (SO_2) for the continuous air pollution variables are obtained from the same model, and similarly for the categorical variable results.

‡ Adjusted for age, selection year, smoking, radon, socioeconomic grouping, occupational exposure to diesel exhaust, other combustion products and asbestos and employment in risk occupations.

§ Referent category.

Childhood Asthma and Exposure to Traffic and Nitrogen Dioxide

W. James Gauderman, Edward Avol,* Fred Lurmann,† Nino Kuenzli,* Frank Gilliland,* John Peters,* and Rob McConnell**

California Children's Health Study

Childhood Asthma 100% Higher for homes near intense traffic

Background: Evidence for a causal relationship between traffic-related air pollution and asthma has not been consistent across studies, and comparisons among studies have been difficult because of the use of different indicators of exposure.

Methods: We examined the association between traffic-related pollution and childhood asthma in 208 children from 10 southern California communities using multiple indicators of exposure. Study subjects were randomly selected from participants in the Children's Health Study. Outdoor nitrogen dioxide (NO_2) was measured in summer and winter outside the home of each child. We also determined residential distance to the nearest freeway, traffic volumes on roadways within 150 meters, and model-based estimates of pollution from nearby roadways.

Results: Lifetime history of doctor-diagnosed asthma was associated with outdoor NO_2; the odds ratio (OR) was 1.83 (95% confidence interval = 1.04–3.22) per increase of 1 interquartile range (IQR = 5.7 ppb) in exposure. We also observed increased asthma associated with closer residential distance to a freeway (2.22 per IQR; 1.36–3.63) and with model-based estimates of outdoor pollution from a freeway (1.89 per IQR; 1.19–3.02). These 2 indicators of freeway exposure and measured NO_2 concentrations were also associated with wheezing and use of asthma medication. Asthma was not associated with traffic volumes on roadways within 150 meters of homes or with model-based estimates of pollution from nonfreeway roads.

TABLE 3. Associations Between Exposure to Traffic at Home and Asthma History

Exposure Metric	Odds Ratio per IQR OR* (95% CI)
Distance to freeway	1.89 (1.19–3.02)
Traffic volume within 150 meters	1.45 (0.73–2.91)
Model-based pollution from:	
Freeways	2.22 (1.36–3.63)
Other roads	1.00 (0.75–1.33)
Freeways and other roads	1.40 (0.86–2.27)

*Odds ratio per change of 1 IQR. For distance to freeway, OR for the 25th percentile compared with the 75th percentile (ie, living closer compared with farther from the freeway). For remaining traffic variables, OR for the 75th percentile compared with the 25th percentile. All models were adjusted for sex, race, Hispanic ethnicity, cohort, and community.

Traffic-Related Air Pollution, Particulate Matter, and Autism

Rapidly Emerging Issue

Heather E. Volk, PhD, MPH; Fred Lurmann; Bryan Penfold; Irva Hertz-Picciotto, PhD; Rob McConnell, MD

Risk of Autism 200% Higher
For children with high traffic exposures in 1st year of life
Southern California

Results: Children with autism were more likely to live at residences that had the highest quartile of exposure to traffic-related air pollution, during gestation (AOR, 1.98 [95% CI, 1.20-3.31]) and during the first year of life (AOR, 3.10 [95% CI, 1.76-5.57]), compared with control children. Regional exposure measures of nitrogen dioxide and particulate matter less than 2.5 and 10 µm in diameter ($PM_{2.5}$ and PM_{10}) were also associated with autism during gestation (exposure to nitrogen dioxide: AOR, 1.81 [95% CI, 1.37-3.09]; exposure to $PM_{2.5}$: AOR, 2.08 [95% CI, 1.93-2.25]; exposure to PM_{10}: AOR, 2.17 [95% CI, 1.49-3.16) and during the first year of life (exposure to nitrogen dioxide: AOR, 2.06 [95% CI, 1.37-3.09]; exposure to $PM_{2.5}$: AOR, 2.12 [95% CI, 1.45-3.10]; exposure to PM_{10}: AOR, 2.14 [95% CI, 1.46-3.12]). All regional pollutant estimates were scaled to twice the standard deviation of the distribution for all pregnancy estimates.

Conclusions: Exposure to traffic-related air pollution, nitrogen dioxide, $PM_{2.5}$, and PM_{10} during pregnancy and during the first year of life was associated with autism. Further epidemiological and toxicological examinations of likely biological pathways will help determine whether these associations are causal.

Arch Gen Psychiatry.
Published online November 26, 2012.
doi:10.1001/jamapsychiatry.2013.266

Table 2. Risk of Autism for 524 Children, by Quartile[a] of Modeled Traffic-Related Air Pollution Exposure From All Road Types

	Odds Ratio (95% CI)		
Time Period	4th Quartile	3rd Quartile	2nd Quartile
First year of life			
Crude	2.97 (1.71-5.27)	1.00 (0.63-1.60)	0.88 (0.55-1.42)
Adjusted[b]	3.10 (1.76-5.57)	1.00 (0.62-1.62)	0.91 (0.56-1.47)
All pregnancy			
Crude	1.99 (1.22-3.28)	1.10 (0.67-1.78)	1.20 (0.74-1.95)
Adjusted[b]	1.98 (1.20-3.31)	1.09 (0.67-1.79)	1.26 (0.77-2.06)
First trimester			
Crude	1.91 (1.67-3.14)	1.28 (0.80-2.06)	1.28 (0.77-2.14)
Adjusted[b]	1.85 (1.11-3.08)	1.28 (0.79-2.08)	1.28 (0.77-2.15)
Second trimester			
Crude	1.69 (1.04-2.78)	1.15 (0.71-1.87)	0.89 (0.54-1.47)
Adjusted[b]	1.65 (1.00-2.74)	1.13 (0.69-1.84)	0.90 (0.54-1.49)
Third trimester			
Crude	2.04 (1.25-3.38)	0.92 (0.57-1.48)	1.12 (0.68-1.84)
Adjusted[b]	2.10 (1.27-3.51)	0.91 (0.56-1.46)	1.17 (0.71-1.93)

ENVIRONMENTAL HEALTH PERSPECTIVES
http://www.ehponline.org

A Study of the Combined Effects of Physical Activity and Air Pollution on Mortality in Elderly Urban Residents: The Danish Diet, Cancer, and Health Cohort

Zorana Jovanovic Andersen, Audrey de Nazelle, Michelle Ann Mendez, Judith Garcia-Aymerich, Ole Hertel, Anne Tjønneland, Kim Overvad, Ole Raaschou-Nielsen, and Mark J. Nieuwenhuijsen

http://dx.doi.org/10.1289/ehp.1408698

Received: 15 May 2014
Accepted: 26 January 2015

Those who live in the cleanest 50% of Aarhus & Copenhagen & cycled over 4 hours per week, had lower total & cardiovascular mortality.

Danish Cyclists who live in 10% most polluted parts of Copenhagen compared with least traffic polluted 50%:

~50% Higher Total Mortality

~100% Higher Cardiovascular Mortality

Table S1. Adjusted associations[a] of total and cause-specific mortality with cycling among 52,061 participants in Diet, Cancer and Health cohort, by intensity of cycling and different levels of NO_2.

Physical Activity	Low NO_2 (< 15.1 µg/m^3) HR (95% CI	Moderate NO_2 (15.1-23.9 µg/m^3) HR (95% CI	Very high NO_2 (≥ 23.9 µg/m^3) HR (95% CI	*p-value*[b]
Total mortality (*n* = 5,534)				
Does not cycle	1.00	1.26 (1.15, 1.39)	1.39 (1.22, 1.58)	
Cycles 0.5-4 h/week	0.87 (0.79, 0.95)	1.00 (0.91, 1.10)	1.10 (0.96, 1.26)	
Cycles >4 h/week	0.82 (0.72, 0.93)	1.02 (0.92, 1.14)	1.19 (1.01, 1.40)	*0.52*
Cancer mortality (*n* = 2,864)				
Does not cycle	1.00	1.22 (1.07, 1.39)	1.36 (1.13, 1.64)	
Cycles 0.5-4 h/week	0.97 (0.86, 1.10)	1.09 (0.96, 1.23)	1.19 (0.98, 1.45)	
Cycles >4 h/week	0.91 (0.76, 1.08)	1.14 (0.99, 1.33)	1.16 (0.92, 1.47)	*0.71*
Cardiovascular mortality (*n* = 1,285)				
Does not cycle	1.00	1.36 (1.13, 1.64)	1.78 (1.39, 2.29)	
Cycles 0.5-4 h/week	0.83 (0.68, 1.01)	1.09 (0.90, 1.31)	1.21 (0.91, 1.61)	
Cycles >4 h/week	0.73 (0.55, 0.96)	0.98 (0.78, 1.23)	1.38 (1.00, 1.91)	*0.78*
Respiratory mortality (*n* = 354)				
Does not cycle	1.00	1.02 (0.74, 1.40)	0.73 (0.45, 1.18)	
Cycles 0.5-2 h/week	0.56 (0.39, 0.81)	0.72 (0.51, 1.02)	0.48 (0.26, 0.89)	
Cycles >4 h/week	0.49 (0.28, 0.85)	0.57 (0.37, 0.88)	0.57 (0.29, 1.12)	*0.78*
Diabetes mortality (*n* = 122)				
Does not cycle	1.00	1.36 (0.79, 2.37)	1.20 (0.56, 2.53)	
Cycles 0.5-2 h/week	0.69 (0.35, 1.34)	0.86 (0.46, 1.61)	0.69 (0.25, 1.84)	
Cycles >4 h/week	0.55 (0.21, 1.47)	0.75 (0.36, 1.56)	0.56 (0.16, 1.91)	*0.98*

HR hazard ratio; CI confidence interval.

[a]Adjusted for NO_2, gender, calendar year, and mutually for other three physical activities, occupational physical activity, smoking status, smoking intensity, smoking duration, alcohol intake, environmental tobacco smoke, education, fruit and vegetable intake, fat intake, risk occupation, mean income in municipality, and stratified by marital status. [b]*p*-value for interaction.

Exposure to Traffic and the Onset of Myocardial Infarction

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N Engl J Med 2004;351:1721-30.

Heart attacks elevated (2.9 to 3.9) after exposure to traffic

BACKGROUND

An association between exposure to vehicular traffic in urban areas and the exacerbation of cardiovascular disease has been suggested in previous studies. This study was designed to assess whether exposure to traffic can trigger myocardial infarction.

METHODS

We conducted a case–crossover study in which cases of myocardial infarction were identified with the use of data from the Cooperative Health Research in the Region of Augsburg Myocardial Infarction Registry in Augsburg, in southern Germany, for the period from February 1999 to July 2001. There were 691 subjects for whom the date and time of the myocardial infarction were known who had survived for at least 24 hours after the event, completed the registry's standardized interview, and provided information on factors that may have triggered the myocardial infarction. Data on subjects' activities during the four days preceding the onset of symptoms were collected with the use of patient diaries.

RESULTS

An association was found between exposure to traffic and the onset of a myocardial infarction within one hour afterward (odds ratio, 2.92; 95 percent confidence interval, 2.22 to 3.83; P<0.001). The time the subjects spent in cars, on public transportation, or on motorcycles or bicycles was consistently linked with an increase in the risk of myocardial infarction. Adjusting for the level of exercise on a bicycle or for getting up in the morning changed the estimated effect of exposure to traffic only slightly (odds ratio for myocardial infarction, 2.73; 95 percent confidence interval, 2.06 to 3.61; P<0.001). The subject's use of a car was the most common source of exposure to traffic; nevertheless, there was also an association between time spent on public transportation and the onset of a myocardial infarction one hour later.

CONCLUSIONS

Transient exposure to traffic may increase the risk of myocardial infarction in susceptible persons.

Table 2. Odds Ratios for the Onset of Myocardial Infarction (MI) after Time Spent in Traffic, According to the Means of Transportation.*

Type of Transportation and Hours before MI	No. of Subjects	Frequency of Exposure in Case Period on Day of MI (%)	Odds Ratio (95% CI)	P Value
Any means of transportation†				
Concurrent	585	8.0	1.50 (1.07–2.09)	0.02
1 hr	625	12.1	2.92 (2.22–3.83)	<0.001
2 hr	634	8.9	2.01 (1.49–2.72)	<0.001
3 hr	635	5.5	1.15 (0.79–1.66)	0.47
4 hr	638	5.6	1.27 (0.89–1.83)	0.19
5 hr	639	6.8	1.64 (1.17–2.30)	0.004
6 hr	640	6.1	1.34 (0.93–1.92)	0.11
Cars				
Concurrent	585	5.6	1.33 (0.90–1.99)	0.15
1 hr	625	8.3	2.60 (1.89–3.57)	<0.001
2 hr	634	6.5	1.94 (1.37–2.76)	<0.001
3 hr	635	4.2	1.16 (0.76–1.78)	0.49
4 hr	638	4.0	1.21 (0.79–1.86)	0.38
5 hr	639	5.3	1.73 (1.19–2.54)	0.005
6 hr	640	5.0	1.55 (1.04–2.30)	0.03
Bicycles				
Concurrent	585	1.8	2.59 (1.27–5.29)	0.009
1 hr	625	2.4	3.94 (2.14–7.24)	<0.001
2 hr	634	1.6	2.70 (1.37–5.33)	0.004
3 hr	635	1.0	1.66 (0.74–3.74)	0.22
4 hr	638	0.7	1.16 (0.45–2.96)	0.76
5 hr	639	0.9	1.49 (0.63–3.54)	0.37
6 hr	640	0.7	1.02 (0.36–2.87)	0.97
Public transportation				
Concurrent	585	0.5	1.08 (0.33–3.55)	0.90
1 hr	625	1.2	3.09 (1.41–6.75)	0.005
2 hr	634	0.9	2.13 (0.91–5.23)	0.08
3 hr	635	0.3	0.69 (0.17–2.88)	0.62
4 hr	638	0.9	2.27 (0.95–5.41)	0.06
5 hr	639	0.6	1.54 (0.55–4.37)	0.41
6 hr	640	0.3	0.73 (0.17–3.06)	0.67

N Engl J Med 2007;357:1075-82.

Doubling of heart muscle stress when exercising with diesel pollution present

Ischemic and Thrombotic Effects of Dilute Diesel-Exhaust Inhalation in Men with Coronary Heart Disease

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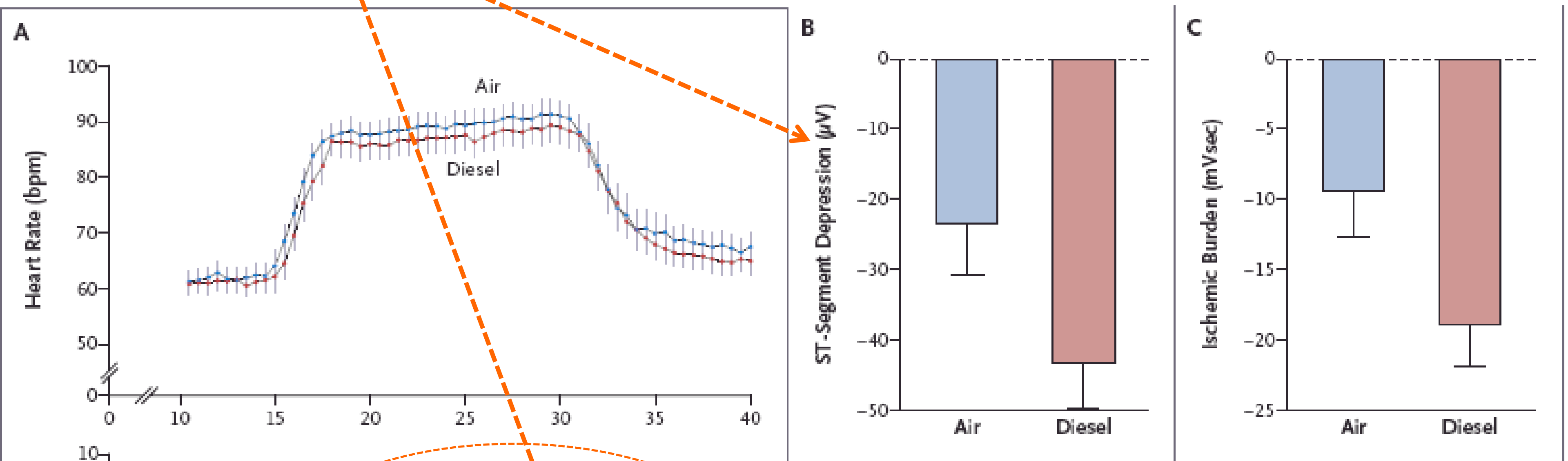

Figure 1. Myocardial Ischemia during 15-Minute Intervals of Exercise-Induced Stress and Exposure to Diesel Exhaust or Filtered Air in the 20 Subjects.

Panel A shows the average change in the heart rate and in the ST segment in lead II. Panel B shows the maximum ST-segment depression during inhalation of diesel exhaust as compared with filtered air ($P=0.003$), and Panel C shows the total ischemic burden during inhalation of diesel exhaust as compared with filtered air ($P<0.001$); the values in Panels B and C are averages of the values in leads II, V_2, and V_5. In all three panels, red indicates exposure to diesel exhaust, and blue exposure to filtered air. T bars denote standard errors, and mVsec millivolt seconds.

Relative risks in best spatial epidemiology studies on high mobile pollution exposures at residence:

Cardiovascular deaths - 50% + higher (solid)

Lung cancer deaths - 50% + higher (solid)

Childhood asthma - 50% + higher (solid)

Childhood autism – 100% + higher (emerging)

PM2.5 Particles <= 2.5 micrometers diameter
UFP Particles <= 100 nanometers diameter
PAH Polycyclic aromatic hydrocarbons

Questions? wigzamore@gmail.com

Direct traffic exposures (small acute studies):

Heart attacks – 3X higher - but cyclists – 4X higher

Cyclist dose – 4X to 6X higher due to ventilation

EKG ST segment depression - 2X higher – oxygen crisis in heart

Rapid cardiovascular signaling due to diesel emissions particulates DEP

Air toxics and garden vegetables:

Aerial plant to root PAH ratio – 4X to 6X higher

Cardiovascular Disease and Lung Cancer relative risks appear to be similar for truck industry workers, diesel rail engineers and near highway residents - these elevated risks are <u>not</u> driven by PM2.5 mass, but may be driven by UFP

Individual Exposure is determined by:
How big is the source – e.g., vehicles per day
Meteorology – sun, wind, temperature, etc.
Built or geographic ... protection or traps
How close are you, how many hours per day
Activity based ventilation and metabolic rates

Work remains to refine these engineering / design elements that can be used for charrette focus

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All of the following are engineering tactics:

- Residential and school HEPA filtration and other protective building systems
    - 90 to 95% reductions possible, maybe 80% after human behavior included
- Air intake locations could be chosen more carefully
    - Good idea but little good quantification in literature
- Sound proofing through extra window glazing, insulation and other features
    - Large reductions possible as seen in FAA noise proofing

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All of the following are design tactics with potentially strong co-benefits:

- Land use buffers ala California ARB Handbook – distance between sources and receptors
    - 50% or greater exposure reductions from 500 foot buffers for sensitive uses
- Vegetation or built wall barriers to absorb or block pollution
    - 10 to 25% reductions possible, especially with height, but geometric trade-offs are complex
- Street trees, hedges and pleasant vegetation
    - -25 to +25% increases / reductions but also heat island and green space co-benefits
- Decking over of highways to link urban areas and block pollution
    - 20 to 50% reductions (rough est.) of long term urban design as in Back Bay, Freeway Park, etc.
- Urban design such as healthy placement of buildings and open space
    - 10 to 25% reductions (est.) but not much literature, site planning used by San Francisco
- Garden locations, including healthy vegetables
    - Small but strong literature, including from mainland China, about near highway air toxins
- Park locations for active recreation and for susceptible people
    - Important due to human ventilation, susceptible populations – children, seniors, co-benefits
- Active travel locations, including bicycling and walking paths (reductions of 50% possible)
    - Important because of human ventilation rates which yield high effective biological doses


NO MOTOR VEHICLES
Somerville Community Path
near Davis Square
THANK YOU