

Massport CAC Environment and Health Sub-Committee Meeting

Feb. 12, 2024

Topic: Health Effects of Transport Air and Noise Pollution

Present:

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Mora Zlody, City of Boston
Stephen Fox, City of Boston, South End
Aaron Toffler, Exec Director of MCAC
Alan Wright, Chair of Board of MCAC
Alan Stein, Swampscott
Heidi Porter, Town of Bedford
Chris Hart, Milton
Alex Chatfield, Town of Lincoln
Irene Walczak, City of Boston, Hyde Park
Mica

Agenda

9-10

Aviation Noise Annoyance Math (from Schultz FICON to Miller NES)

- Who is most affected?
- Simple math you can do
- Graphic DNL and Noise Annoyance Exercise

10-10:30

Transportation Noise (in General)--beyond annoyance from WHO to You

- Local-scale side effects of transport noise beyond annoyance.
- All transportation noise is integrated biologically; it doesn't matter where it derives from. The impact in the body is the same Your body is reacting to total of transportation and probably other).
- Using the framing of the **WHO Europe - Systematic Review of Environmental Noise (2018)**. It is the single best summary from Europe on the health impact of noise. Have had some small updates. Europe and North America are different in how they handle health. More social, more public health in Europe, and the countries cooperate quite well (although there are some great cul de sacs of research in North America, too).
- Learning disabilities, sleep disturbance, cardiovascular disease
- Must we integrate aviation and surface transport noise impacts?
- Use WHO Europe Data (disability weightings, etc.)
- YES, integration of WHO Noise Environmental Epi from Europe
- Somerville Leaffer STEP work/Volpe noise model/Matt Simon paper
- PLUS can apply WHO concentration response functions (CRFs) from across US
- YES, biology is integrated and cumulative – Volpe again
- Acute is most noticed, versus chronic most health effects via VSL

- 10:30-11 **Transportation-related Air Pollution (TRAP) at Multiple Scales**
- Particulate matter of all sizes – and gasses like Ozone, NOx, GHGs (a quick and selective history—PM sizes from big to small)
 - Brief history of U.S. air pollution—PM and gasses and lead – UFP
 - Primary and secondary PM – both in PM regulations and PM creation
 - PM – TSP to PM10 to PM2.5 Harvard Six Cities—Ozone (MA) and Lead
- 11-11:15 **Leveraging Science to Drive Environmental and Health Protection**
- How the US EPA looks at air pollution. Environmental regulations are driven only by health outcomes.
 - US EPA frame for primary versus secondary—Pollutants and Regulations. They use primary and secondary in two different ways.
 - One reason this is important for this conversation is that particles in the air can be immediately formed from semi-volatile exhaust, especially in cold weather, or they can be formed by photo-chemical regional pollution. For example, PM2.5 does not vary between the upwind and downwind side of a highway. It's regional. It is not local. And a lot of people only focus on PM2.5 because it's really easy.
 - How will we be able to mobilize and drive health outcome evidence?
- 11:15-11:30 **Integrating Regions – Land Use, Transport, Environment, Health, SES**
- Think of the whole regional, community, and hyper-local planning, by considering land use and transportation, and environment and health. And then, on the back end of that, too often is socio-economics, and environmental justice.
 - Research scales vary with the subject – Census Block Groups base works well
 - No region can succeed alone – Boston, LA, Seattle, and Detroit could work
 - How will we integrate land use, transport, environment, and health data?
- 11:30-11:45 **Extending Environmental Health Research into Advanced Causal Pathways**
- The NLRP3 inflammazone, one of 20 in humans, integrates inflammation.
 - **Emerging causal pathways. NLRP3** driving health effects of particles for better part of a decade. (Will discuss reasons why for a long while the environmental epidemiology community believed that was not true, which has perhaps slowed progress.) NLRP3 is the match that lights inflammation and integrates all inflammation. And that's why, if you keep yourself in shape (diet good, weight good), these things do not cascade on you so much, because NLRP3 is integrating all of them. It can help cure things, if you have an acute threat. But past the tipping point, it is going to get rid of you and recycle your elements.
- Ion Channels**, especially K⁺ (Potassium) and Ca⁺⁺ (Calcium), plus a few others, run everything. There has been a lot of research into ion channels. 4 Nobel prizes awarded to different teams working on ion channels. 300,000 papers at National Library of Medicine. Potassium is by far the most important, and Calcium is second. There are just a few other ions that drive health outcomes. There is a **relationship between ions and NLRP3**. And there are no environmental epidemiologists who know anything about this. So that's a problem.
- How much work remains to integrate NLRP3 and key ions with PM and noise?

- NLRP3 is a molecular complex that is especially common in immune cells of different types. NLRP3 and the other inflammasomes all generate interleukin-1 cytokines. Cytokines are small proteins crucial in controlling growth and the activity of immune system cells. NLRP3 and other inflammasomes assemble in response to the detection of stress- or infection-related stimuli. They activate the inflammatory response and can restrict pathogen replication.¹

1. Health Effects of Transport Air and Noise Pollution

FICON study in 1992 found that 0% of adult population are highly annoyed at 50 DNL, and 12.5% are highly annoyed at 65 DNL. Study done by Schultz, working for US Airforce. Base housing with families after WWII was built near runways, before they had jets. Airforce got pushback from families that the jets overhead were very noisy. Shultz was engaged to survey noise annoyance levels.

NES study in 2021 (Neighborhood Environmental Survey) looked at populations near 20 U.S. airports. Researcher of MIT-associated businesses named Miller. Study found that 20% of adult population were highly annoyed at 50 DNL, and 62% are highly annoyed at 65 DNL.

Noise Exposure is measured by DNL (Day Night Levels), dB, a log-based measurement with night-time penalty (might be order of magnitude—not sure). Created a National Dose-Response Curve from this. Average of noise exposure over 24-hour period. Problem is that this doesn't account for variation. Might be one very noisy plane during that period (but looks like less because averaged), or multiple planes with less noise.

Interpretation of the chart comparing 1992 and 2021: Difference in “highly-annoyed” population between 1992 FICON and 2021 NES is staggering. More people are now highly annoyed at every DNL Level, and the “highly annoyed” population is increasing at a lower DNL level in 2021, compared to 1992.

One reason why may be noise exposure. Wig thinks transportation noise is lower than it was in 1992. Noted that aircraft are less noisy now than in 1992, especially big jets closer to Logan Airport. Difference could be due to people using headphones and exposing themselves to loud music? Alan thought that some parts of transportation noise are louder (motorcycles).

In every case, there are local differences. But these are small compared with the differences within the population.

There are quite sizable populations that have 100 times different sensitivity to aviation and surface transportation noise.

Aaron concluded that the amount and scale of noise that people experience today is greater than years ago.

Why was 12.5% at 65 DNL an appropriate regulatory level? Answer: it was a practical methodology for the U.S. Airforce. It was an arbitrary level set at the levels that government could afford to incur through

¹ Inflammasomes <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4292152/> Interleukin-1 <https://inflammregen.biomedcentral.com/articles/10.1186/s41232-019-0101-5>

noise insulation. **But it doesn't make sense to use the average to determine when a community needs help.** If you are 100 times as sensitive, you need help, but if people are not sensitive, they don't need help. Sound proofing housing that is at 12.5% makes no sense. (Example, do you give kids inhalers when their school has 10% of kids with asthma, and no inhalers to other people in the town?)

Stephen: At Massport, we are investing an enormous amount of money in terms of sound insulation. Given this questionable methodology for setting level, shouldn't we be re-thinking this investment? Wig thinks yes.

Alan. The statement that aviation noise has decreased is debatable. Thirty years ago, a stage 2 jet engine (which was the most common engine on a commercial jet) was emitting decibel levels that were deafening. Now most of the jets have Stage 4 and Stage 5 engines that are highly efficient, both in terms of how the fuel is burned and in the dynamics of air movement through the engine and across the wings. So, the noise level has gone way down.

BUT, the volume of traffic has gone way up if we compare the number of flights from 30 years ago to 2019 (before COVID). And for those of us living under flight paths, the flight corridors from departures and arrivals are more concentrated because of RNAV. The problem in many ways has increased. More persistent exposure. RNAV has concentrated noise above certain well-defined areas—less variation than there used to be from aviation noise. The plains go over the same point every time.

RNAV. Also called area navigation, RNAV refers to a method of navigation that is instrument based. It permits aircraft operation on any desired flight path within the coverage of ground- or space-based navigation aids. Allows more precise departure and arrival configurations. This in turn can result in savings on time and fuel, fewer Air Traffic Control transmissions, and more efficiency. RNAV is a part of Next Generation Air Transportation System (NextGen), a large-scale FAA initiative to modernize the U.S. national airspace system. NextGen includes airport infrastructure improvements, new air traffic technologies and procedures, and safety and security enhancements. It is also meant to help reduce aviation's effects on the environment.²

Stephen: If we have quieter aircraft, why are people getting more annoyed at lower levels (e.g. 50 DNL versus 65 DNL)?

Mike(?): The amount of noise is more persistent and constant, but not necessarily the level of noise (decibels).

Wig: There are less planes than 5-10 years ago.

Alan: Note that it is all noise, not just aviation noise.

Eireen: Old way to manage, with pilot flying visually, was like a flashlight. There is a lot of variation in that kind of radio signal. But the next generation is GPS. It is like a laser pointer, with less variation. Planes go over same point all the time. Level of noise is exponential. Also planes used to be able to climb to 12K feet. This helped to alleviate some of the decibels that people were exposed to. Now they are not climbing as high anymore (only 6K feet). 2015, some new technology came into play. More people were

² NextGen <https://www.faa.gov/nextgen> RNAB
https://www.faa.gov/air_traffic/publications/atpubs/pham_html/chap20_section_5.html

exposed. And number of planes coming in and out was increasing. Especially because they affected different people by the new flight paths—people weren't used to the noise.

Wig: If flights are more concentrated, and there are less people under the flight paths, why are there so many more people highly annoyed? **Eireen** thought there are more people under the flight path. Wig thinks it is the biology. Thinks it is the people's biology that has changed. Alan thinks it is more than just aviation.

Mike: Maybe it is about the sampling.

Wig: The study is meant to be representative poll of whole population.

Mike: Issue could be related to sampling. Is it just the people under the flight paths? Are they over-sampling denser areas? Is sampling same as in 1992? Could be biased.

Eileen: Has population density changed?

Alan: Doesn't think population density has changed. In post-WWII up to 80s, Boston lost 200K people, but have now recovered that. Since Boston proper is so close to airport, and we have these flights going over, we have a larger population exposed now than there used to be.

Alice: What does this chart look like, compared to what Europe is tracking?

Wig: The 20 airports sampled in U.S. did not have the same outcomes. Variability was different. Could be due to lifestyles or urbanization. O'Hara is much bigger than Logan, but it is farther from the city. In terms of comparison to Europe, Schiphol airport is in very dense population. Places with two parallel runways, there are people who are slammed.

Where should you go (as an individual) if you have a noise problem? Mass Eye and Ear. They have HST graduate program (Hearing and Speech Therapy).

Wig: At what DNL level in Boston do you find the most people who are highly annoyed by noise. Answer: you have to know the population at each DNL. If you do the math using DNL curve, it is 51-55 DNL. The reason is that most of the population is there. Even though the noise levels are less, the population overwhelms the answer. Shouldn't just look just at soundproofing above 65 DNL. If we do that, we're missing a massive amount of the population who is highly affected by this, because most of the population is at the lower decibel levels. There are so many more people there, that by far there's the highest number of people feeling highly annoyed about noise there.

Alan: Science can show distributions that you have, and you can describe the deficiencies in the science. But what's going on in making the decision to put in soundproofing, or other interventions, is a policy decision made at the political/legislative level. At what point are we going to say "we are going to put in soundproofing at this level, and we're not going to do anything below that." For example, in Winthrop, and parts of East Boston, a decision was made to provide insulation into houses and new windows for populations that were exposed to very high levels of noise very frequently. I would wager, not knowing the details of that decision, that it was partly based on how much can government afford to do. Because it is an enormous expense. As Gary (Gerry?) says, there's a push to make sure that others who were not included in the original sound proofing are included. But there's no way that it could be

affordable to soundproof all of the population of Roxbury that's exposed to departures at runway 27, or all the population of South Boston that's exposed to arrivals on the 4th.

Noise is a problem. And there's more and more research being done on it. I think it is something that, as a committee, we should focus on. But not first. We need to be focusing on particulate air pollution.

Wig: We'll get to that.

Alan: Wrapping up the noise discussion, points to us in the future, as a committee, commissioning studies, collaborating with noise researchers, to begin to get a handle on the problems that the noise is causing. We know that for children, noise exposure in the early morning and when they are in school is delaying their learning ability and their development and leads to social problems. It is something that has to be addressed.

Aaron: Noise causes health problems, more than "annoyance"—see our comment letter on the FAA Noise Policy Review

?: Regarding noise and its impacts. Even if it was totally affordable, it also has to do with knowing what we can do to help. It is not just people who are affected, but also the natural world. Do we have any data on the impact on the environment? It seems harder to know the impact on environment. This point holds for particulate matter also.

Wig: EPA primary air pollution standards have to do with effects on humans. Secondary has to do with all other living things. Acid rain regulations are secondary effects. Primary and secondary doesn't specify priority, but they are categories. So EPA does care about the impact on the environment (animals, plants, watersheds, estuaries).

Aaron: Wildlife, **ecosystem impacts of noise should be included in the discussion of mitigation measures.**

In terms of interventions, we want long-term interventions. **What other interventions are possible on top of sound insulation? For example, what about making changes to the building codes?** Not just insulation and windows. Need to look at this holistically, about commercial real estate and residential. Noise and hazardous particulates.

Wig: Massachusetts has been at the forefront of building insulation regulations --really tight building and triple insulated windows, you have to have make up air from outside, and that gets filtered. **Need to have MER 16 filtration systems.** It is quieter and helps with particles. (Renovations are part of the code.) There are ways that the regulations are helping each other.

Stephen: Is there a correlation between "highly annoyed" with noise, and health impacts?

Wig: Yes, but the way it was framed in WHO systematic review of noise, they didn't look at it that way. They looked at the categories of outcomes of environmental noise: **annoyance, learning disabilities, sleep disturbance as separate categories.** Those are chronic. If people are not learning because of noise, this has life-long impacts. Sleep disturbance also, has a ripple affect. And lastly, they looked at noise (aviation and other forms), and cardiovascular disease. They didn't say that it was necessarily going through annoyance (i.e. noise → annoyance → health outcome). Learning disabilities and sleep disturbance are more serious than annoyance, and cardiovascular disease can lead to death, so that is very serious. The disability weightings come into play here.

Stephen: Young people disregard “annoyance”. Would be good if we could show it was more serious.

Wig: This is what WHO systematic review was trying to do.

?: What about environmental stress. This creates a bad environment. If you are stressed out consistently and at high levels, you are going to have bad health outcomes. Stress is an impact to measure. Might be good to show these connections to lay people (river and tributaries).

Wig: It is fine to look at stress in general terms. When you come to epidemiology, they will do the statistics on how direct and strong is the connection. They will have measures (possible causal factors or mediators) like presence of inflammatory agents, blood pressure, etc. to show how stress from noise affects health.

2. Transportation Noise beyond annoyance

Wig: Consideration of other transportation, surface transportation. Consider noise levels from surface transportation for people living in a triple decker in Somerville. They are close to Logan, but also close to Rt. 93 and Rt. 33. 200K vehicles a day including a lot of trucks. What are the decibels contributed by surface transportation versus aviation.

Answer: People living along highways experience primarily noise from surface transportation. Noise is 100 times as high for people living next to highways, compared to any flight pattern. The surface transport noise is constant, 5 days a week.

Wig: Who has most noise? Which floor?

Answer: The higher you go, the higher the noise. If you are on first floor you get noise from the lane of traffic right next to you, but not the others. But the higher you are, the more direct noise you get from all the lanes of traffic. The third floor will have most noise in a triple-decker.

The surface transportation has an effect not just on noise annoyance, but on the other outcomes as well that we’ve been talking about. When you see the summaries from WHO Europe on total number of deaths from surface transportation noise versus aviation noise, it is staggeringly higher for surface transportation noise versus aviation.

?: What will this mean for policy of creating affordable housing in high density areas.

Putting more affordable housing in high density zones could expose them to more transportation noise. The biggest low-income housing projects are built next to the highways. The richer communities don’t want affordable housing in their quaint downtown. Communities vary in how much they care about these things. Is there a way to be proactive to get communities to care more? People come to reps when they are annoyed, but is that too late—just to be reacting to people who are annoyed? Might need to be community-by-community. Ask the councilor to consider.

Alan: We know that the people of Milton voted against a proposal for a zoning change the town needed to comply with the MBTA Communities Act.³ The biggest source of the “no” on that vote was East Milton. What is going on in East Milton? About 60-70 years ago, East Milton was divided in half by Rt. 93, Southeast Expressway. They did some concessions and buried it down, but is not much below ground level, and it divided the very old village of East Milton. Has a lot of surface transportation noise from constant traffic. They are also getting a lot of aviation noise and pollution from arrival flight paths heading into Runway 4. So, all that aviation traffic coming into Logan is consolidating in Milton and then heading down over Dorchester and South Boston. East Milton is an aggrieved community. This is where the “no” vote came from on MBTA Communities Act. Larger point that I am making is that **noise is a social stressor, in addition to being an individual physiological stressor.**

Kendall Square has the **Volpe National Transportation Centre**. Volpe, including Matt Simon (BUSPH), Kevin Lane, Jon Levey. Volpe has a national transportation model that is really detailed. It has all the highways, all the regional (jet noise footprint), etc. All that data. Matt Simon wrote a paper (last 2 years) looking at transportation noise exposure and particulate exposure from aviation and surface transportation for the whole U.S. at a very granular scale over one year. What he found was that the exposures were greater among **newest immigrant** populations (Hispanic population).

3. Transportation-related air pollution (TRAP)

Nitrogen Oxides (tailpipe NO and NO² – in combination NO_x). NO_x are very good indicator of transportation pollution (aviation or surface) for air pollution research. NO₂ is more regional, NO closer to source.

“**Criteria air pollutants**” are defined as “air pollutants for which acceptable levels of exposure can be determined and for which an ambient air quality standard has been set.”⁴ Examples include: ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, and PM₁₀ and PM_{2.5}. They are regulated by the [Clean Air Act](#).

MBTA diesel commuter rail system is extensive. As of 10 yrs ago, that system burned 20 m. gallons diesel per year (metropolitan Boston). Logan burned 20 m. of “jet A” (which is like diesel, but not same) on tarmac, and 20 m. in the first 3,000 feet above the tarmac. This cut off is the level to estimate proximal impact because above 3,000 feet, it is not coming down immediately. It is processed and becomes part of the general atmosphere. Logan by itself, had double the total fuel consumption as the MBTA system. (Some disagreement between Alan and Wig: Alan said jets don’t burn diesel. Wig said it was almost the same.)

There are two other things you can do: 1) could electrify take-offs and landings; 2) Could build an off-shore airport in connection with a barrier, and put the port out. This is similar to what they do in Asia. Prompted by increasing flood events (Mystic River).

³ The law requires that an MBTA community shall have at least one zoning district where multi-family housing is permitted--meaning no special permits and no town zoning boards holding things up--located within a half mile of public transit and suitable for families with children. See <https://www.cbsnews.com/boston/news/mbta-communities-law-housing-zoning-question-everything/>

⁴ <https://ww2.arb.ca.gov/our-work/programs/criteria-air-pollutants#:~:text=Criteria%20air%20pollutants%20are%20air,dioxide%2C%20and%20PM10%20and%20PM2.5>

Ozone. How much will you find on a highway? Will find zero. No Ozone on the downwind side of highway because NO has more affinity for an Oxygen atom, than Ozone does. The NO grabs the Oxygen atom and becomes NO₂. NO₂ is much stronger than Ozone at being able to hold on to oxygen. Ozone formed from photo-chemicals and smog. No Ozone to speak of in Manhattan (allows some trees to do really well). Get 20 miles outside Manhattan on long island, and you get a lot more Ozone, and chemicals dropping out of the air.

It also gets to coastal Maine. All the pollution (polluted air) from the corridor, including from Boston, is sent out over the water and bakes in sun. Then in the evening, with an off-shore breeze, it goes in and hits the population on the coast. Blue mountains are blue because of tree pollution.

What does it mean, practically speaking? Ozone is an extreme irritant. Can make you short of breath. Serious for kids with asthma.

Particles

Story is one of regulating increasingly smaller particles.

Initially, regulations applied to “total suspended particulates” (TSP). Didn’t matter what size. Then, the first size regulation was PM₁₀. If you have a really dirty diesel and construction equipment (out of tune bulldozer, etc.), that’s what you see, the cloud of black smoke. Construction companies provide fuel to themselves. They have #2 oil that is taxed less than motor oil. They use this if they aren’t caught. Visible smoke is large particles (PM₁₀ or higher).

Also, visible smoke having to do with cooking. If you don’t have a stove hood. Not good to have a lot of cooking unventilated, as housing gets tighter.

PM₁₀ was in original Clean Air Act of 1970. The only pollutant added after that 1970 Act was lead. Great studies showing permanent effect of lead on children’s brains. One report found that blood lead in children in NYC went up and down with season. As they added lead to gas in the wintertime to get higher burning capacity, children’s lead levels went up within a month. A couple of other great studies, last one was on IQ, showed lead poisoning affects IQ and you can’t recover the lost IQ. It is permanent. One study (1979) in Chelsea and Somerville tested children’s baby teeth for lead testing. They found that the kids who were most exposed to lead gasoline, their IQ was done for life. Study stopped by family (upset by findings). Small number study. Great scientists, Belenger from Harvard, Needleman from Carnegie Mellon.⁵ EPA worked very quickly after this came out.

PM_{2.5} does not vary between upwind and downwind side of a highway. It is a regional pollutant, not a local pollutant. Lots of people focus on PM_{2.5} because it is easy to measure.

⁵ Needleman HL, Gunnoe C, Leviton A, Reed R, Peresie H, Maher C, Barrett P. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. N Engl J Med. 1979 Mar 29;300(13):689-95. <https://doi.org/10.1056/NEJM197903293001301> (online copy not available)

How did we get a PM2.5 regulation? There was one brilliant study in 1970 (? Should be 1993) that led to PM2.5 regulation. **Harvard six-cities study.** Doug Dockery first author, Frank Spizer, Jack Spengler.⁶ Chose six cities in U.S. to look at PM10. Had different climates, populations, sources of pollution. They happened to have PM2.5 monitors and PM10 monitors. Cohort study that ran 10 years or so. One monitor in center of each city. Average size of cities was 27 sq miles, and they only had one monitor (annual average PM10 and annual average PM2.5). Studies included Watertown, MA, a town in Wisconsin (turned out to be cleanest), and Stubenville OH (turned out to be dirtiest city because of industrial air pollution). Other follow-up studies were done that verified these results.

Found extraordinary correlation between PM2.5 and cardiovascular death (correlation is almost straight line). Spizer didn't want to publish immediately because outcomes were so stunning; said they needed to reanalyze before publishing to be sure. But no mistakes were found, so they published. Simple study with extraordinary outcome.

EPA knew immediately that they had to act to put in place PM2.5 regulation. They did this in record time, within a couple of years. Also started Particulate Matter Research Center Program. Ever since there have been five centers. Always has been one in Boston, one in Los Angeles—place with most mobile pollution—and one in Rochester (because there was a nanoparticle scientist there). Other centers have moved around, e.g. Seattle (UW), Baltimore (JHU), Detroit.

From that study, **PM2.5 took over most of the focus on human health effects of air pollution.** Global Burden of Disease now has data on PM2.5. They also measure pollution from indoor cooking. This has been addressed through electrification—a great success story in India and other countries with unventilated indoor cook stoves.

PM2.5 regulation stuck, because they didn't have technology to measure smaller molecules.

Recently there has been a **new lowering of the PM2.5 national ambient air quality standard.** This is because newer technology allows. Lower regulation will affect the West Coast mostly.

Discussion of wild fires. Fine particulate matter (under PM2.5) is main component of wildfire smoke, along with larger particulates (incl. soot). Wild fires can be intense. Recently affected NY Metro area (not Boston). Most effects will be acute. Extra heart attacks, people with asthma, people who are frail. Chronic health effects have vastly more negative health impact than acute. Last 30 years in most Western societies, PM2.5 has dominated.

MA was last state in the U.S. to have every acre in violation of Clean Air act because of Ozone. We were the last state to be out of compliance. This helped create air quality and policy groups like NESCAUM.

Why? We are downwind from the urban centers from DC, Baltimore, NYC, New Haven. All that highly polluting stuff came in airstreams toward NE and MA, with a lot of Ozone created by photo-chemical baking of those emissions.

⁶ Dockery DW, Pope CA, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG, and Speizer FE. An Association between Air Pollution and Mortality in Six U.S. Cities. The New England Journal of Medicine. 1993 Dec 9; 329(24):1753-1759. <https://doi.org/10.1056/NEJM199312093292401>

That is no longer true. There has been enough progress with both transportation precursor emissions and industrial precursor emissions. In many ways, Manufacturing and industry have been easier to control than mobile sources because you can manage energy demand in a factory more easily. For factory emissions with consistent air pollution flow, you can put scrubbers in smoke stacks. (Easier to do than with cars, which need differential power.)

Wasn't until Moody figured out the 3-way catalytic converter that we could manage tail pipe emissions. Amazing feat. There are different things you need to do to the emissions in the tail pipe to control things that have very different characteristics from each other.

Near Source Precursor Studies. There has also been series of studies, starting in Scandinavia, looking at lung cancer in cities, at a very fine grain. The first one was done in Oslo. These scientists also created the first ultrafine particle model. It is cold in Oslo. The way you form primary ultrafine particles is that you take hot exhaust gas and put it into very cold air, and you get instant ultrafine particles. Happens most readily on cold, still winter mornings. Can look clear to a normal person in the city, but the semi-volatile chemicals in the hot exhaust, they become particles instantly when they hit the cold air. There is an explosion of particle creation from tailpipes, in wintertime, if there are precursors in the tailpipe.

That's the other "**primary versus secondary**" definition of EPA. EPA considers exhaust in the vehicle, and for two minutes outside vehicle, to be primary pollution. Takes 2 minutes for the exhaust to cool down to ambient temperature and humidity. After that, it is considered secondary pollution. (Remember they also used "primary versus secondary" to refer to pollution that affects people (primary) versus the rest of the natural world (secondary).

Question: how does snow affect pollution.

Ultrafines versus fine particles. Ultrafine particles (UFP) are particulate matter of nanoscale size (less than 0.1 μm or 100 nm in diameter). Fine particles are 1000 times bigger (diameter of 2.5 μm or less). The general population, the general medical community, presume that ultrafines come from same process as fine particles. But they don't come from same process.

Ultrafines are not created from photo-chemical processing, they are formed by the cooling of semi-volatiles in the exhaust. Photo-chemical is baking air for hours and changing the chemicals. One is regional (PM 2.5) the other is not regional—it is hyper local (ultrafines). Primary ultrafines formed by combining hot exhaust gas and cold air—winter is a good time to monitor for UFPs—turns from gas to particles.

The Northeast States for Coordinated Air Use Management ([NESCAUM](#)) is a good air quality and policy group in this region. Functions as a "coalition of state air agencies" and "promotes regional cooperation and action by its member states in support of effective programs to reduce the adverse public health and environmental impacts of air pollution and climate change."

Alan: Experience in Chelsea: 1) traffic going over bridge (pollution coming down; PM2.5); 2) Runway 33 used for departures so you see massive amounts of jets coming over; 3) Asphalt roads. Population is experiencing all this air pollution from these different sources. Beginning of COVID, they had highest mortality rates in Chelsea.

Wig: Chelsea is second to Somerville in mobile pollution per square mile, but it has great challenges.

Ultrafines are high every day on cold winter days. Seattle is doing best ultrafine particle research today. They have a lot of rain (but not as cold). Elaine Austin, UW Seattle, "MOV-UP" studies (noise, ultrafine). They have national ultrafine particle model. Could be paired with Volpe's model. Noise is much easier to model. For particles, it is much more difficult with regard to where they are, how high they are. Makes it difficult to model.

Tufts has been leader in ultrafine particle research. John Durant (environmental engineer) wrote doctorate on Mystic River pollutants. He thought it would be heavy industry, the legacy of old chemical plants would be affecting water, but it was mostly transportation-related pollutants affecting water by the time he did his work.

Developing multi-family housing, can use air filtration. Recommend MERV 16.

MERV rating is an engineering pressure calculation used to measure an air filter's effectiveness at decreasing particles and pollutants. The unit is microns and rating goes from 1 to 20. The number goes up as a filter is able to improve indoor air quality. Below MERV rating 13, they don't get the particles between 50-150 microns in size. A 5 micron particle is acting like a gas, zooms around and will stick to walls and curtains. Can't filter those, but the in-between ones (50-150) are hard to filter. MERV 16 is more recommended, has 6-inch filter generally. In California it is not allowed to use MERV13 with less than a 2-inch filter. Hugely cleaner. US State Department used air filtration units for their staff living in China and India.

Eireen: Curious about metrology of how to measure ultrafine particles. Do standards exist for measuring ultrafine particles? NISTIS?

Answer: Tufts has most TSI ultrafine particle monitors in the Northeast. These are engineers really good at QAQC. The monitors are sent back to TSI every few years to be recalibrated or rebuilt. Highly calibrated by environmental engineers. Not amateur instruments.

Aaron: WHO Europe has a "good practice statement." But in U.S., UFPs are not regulated by the Clean Air Act and there are no regulatory standards for UFPs, so there is no standard to measure against.

Wig: There is only one ultrafine particle standard in the U.S., and it is a very recent standard, and it has to do with jet engines. Cannot manufacture jet engines that have ultrafine particles (and now these are defined to be one component of ultrafine particles that is more easily measured). Two people were given the courtesy of speaking to EPA and FAA before they instituted this standard; Debbie Wagner from Seattle, and Wig. (Erin and some others sat in on this meeting.) They were going to pass this anyway. But it is not a standard that applies to the amount of ultrafines coming out of jets or other things at Logan. Only applies to the manufacturing process, and the total emissions from a new jet engine. There is a standard (it is not zero). Even though it only applies to one component of the ultrafine particles, it helps. Older jet engines are grandfathered.

Stephen: New manufacturing of jet engines doesn't control emissions? Or it does?

Wig: The only standard is a standard applied to FAA and it has to do with qualifications of the engine at the date of manufacture and sale. It mimics a European standard. Will manufacture same jet engines in

Western Europe as they do here, so having the additional standard isn't as big a deal as it may seem. But it is the camel's nose under the tent.

Wig: Series of community-based participatory research studies called the Community Assessment of Freeway Exposure and Health (CAFEH).⁷ Started 15+ years ago with funding from the National Institute for Environmental Health Sciences. From the literature, Wig knew that particles had more health effects than gasses (since 1970). Also clear that people who lived next to the largest sources of ultrafine particles (including roadways) had unexpectedly high health effects. First of these studies were done in Oslo, then Stockholm. Looked at lung cancer and correlated this outcome with industrial pollution or mobile pollution.

Found that lung cancer was much higher in people who lived in the portions of Oslo and Stockholm that had the 10% highest mobile pollution. They weren't expecting that. Stockholm is warmer, so you would expect more effect in Oslo. And they found this. But the major finding was seeing 50% increase in lung cancer for people living in the top 10% of mobile polluted areas, both among smokers and non-smokers.

The lead author, Nosted in Oslo, and Niberg lead author in Stockholm. Top epidemiologist at Asta Zeneca.⁸

Later, in Vancouver, study by Michael Brower (UBC Vancouver) and Wenji Gam, looked at cardiovascular mortality and found same outcome. Five times as much death for people closest to the busiest roadways than people in the dirtiest regional areas. Much larger outcome than from PM2.5 exposure.

Finally, California did study of connection between mobile sources of air pollution and childhood asthma. Results in few premature deaths, but still a big problem. Came out with same outcomes.

These studies happened a long time ago, and there is no suspect other than ultrafine particles.

Yet, EPA has done nothing. And that's common in most countries, though a lot of countries are focused on filtration. The reason is, in part, the difficulty of the studies. I mentioned that in the Harvard six studies, the average city size was 27 sq mi., and they had only annual reading from one monitor per city. Yet, they still found a strong correlation of PM2.5 and cardiovascular deaths. This shows how regional the effect is.

Compare this to the CAFEH studies. CAFEH did studies with Aerodyne (private company), the best air pollution scientists in the world, with the fanciest equipment. Have equipment to study ultrafine particles. Aerodyne air chemistry consultants and equipment were lent to do research in pine forests in Finland, in China to look at particulate matter in big cities, in Mexico City studies (with Molina). Unparalleled in their level of skill. Very high security. Generous with equipment and mobile apps.

Wig asked to use Aerodyne's equipment to study ultrafines and other size particles and gasses in Somerville. Shared cost (\$5K for community group, and \$5K for Aerodyne). Took ½ a day. After Tufts researchers left, used proton transfer mass spec. in Big Dig tunnels. Aerodyne worked with John Durant

⁷ <https://tischcollege.tufts.edu/tisch-college-community-research-center-tcrc/research-projects/community-assessment-freeway-exposure-and-health-cafeh>

⁸ An estimated 19% of lung cancer deaths are associated with exposure to air pollution, making it the second largest risk factor. Holme et al. (2023). <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10543654/>

and the other co-authors (including Wig), put a lot of staff time in, and got a good paper out (in Atmospheric Chemistry and Physics). Allowed us to do studies in Somerville, Chelsea, Roxbury. Those studies have continued. John Durant and Doug Brugge had never worked together and had never studied ultrafine particles or cardiovascular disease. And now they are focusing whole careers on ultrafine particles, looking at cardiovascular disease, blood pressure, air filter efficacy. HUD studies in public housing.

We put together a team, including doctoral and master students, [John Durant](#), [Doug Brugge](#) (UConn Medical), [Christina Fuller](#) (health expert, a member of the Clean Air Scientific Advisory Committee (CASAC) of the US Environmental Protection Agency, one of the people who in the future will determine significant air pollution regulations) and [Allison Patton](#) (now with Health Effects Institute, Boston; degree from MIT) as air pollution scientist.

Remember the Harvard Six Cities study: one measurement, 27 sq mile areas, one year. Allison asked Wig, **what spatial-temporal density does my analysis have to be at, based on our mobile labs, permanent labs on either side of highway, in order for us to match this with cardiovascular health in the population in Somerville?**

Wig answered: We need to know **level of every pollutant we are measuring** (all the traditional pollutants, plus ultrafine particles) **in every 20-meter square, for every one of the 8,760 hours per year**. What I asked her to do was 500 million times as hard as what the Harvard Six Cities scientists did. Literally 500 million times the spatial-temporal density of the HSC study. And she just went and did it. That allowed us to tie the primary ultrafine particles to cardiovascular inflammation. Produced 30-40 peer-reviewed NLM papers on this, in environmental and health journals.

Alan: This was correlation, not causal. You were able to show correlation.

Wig: We measured **c-reactive protein (CRP)** (a pentraxin) and **interleukin-6 (IL-6)**. Those are the two inflammatory biomarkers for cardiovascular disease. Our statistical significance was dead on. There are no medical studies...

Alan: Who is doing the causal pathway research?

Wig: Cardiovascular researchers are doing research on NLRP3 and Ion channels. NLRP3 is the causal pathway driving the health effects. Integrates all the inflammation.

Wig: [For this presentation], I went and grabbed abstracts for the first 5 documents. All papers are 140 pages of abstracts. All of those papers supported the findings. EPA regulations are based on the same kind of studies, and the same kind of outcomes. People look at other things, that they may think are important, but these measures are the ones most cardiovascular researchers will include.

Alan: Let's turn this into a paper—not a publishable academic paper. Include links. Need for three purposes:

- 1) document we give to new members, or spread among stakeholders
- 2) as we move forward, and get to point of pushing information out to Massport, constituent communities, we need a background document, including links to papers.

Wig: We have the recording. You write it, and I'll work with you on anything that's not ship shape. There are no regulations coming out of Massachusetts. There are only regulations coming from EPA. (MCAC member: But we want to be able to move ahead, pre-regulation, about things that cities are talking about, including zoning, etc.)

The homerun is **national regulation**. And I don't want to waste time on other stuff—not a waste of time for you, but I don't have that much time. I work 4,000 hours a year, have worked 60,000 hours pro bono.

Need a national partnership to move regulations from EPA. We are not going to succeed unless we partner with other regions of the country who are 1) competent in EH research and these topics; 2) are varied, 3) have good experience in this, 4) have academic communities. Suggest that Boston, Los Angeles, Seattle, and Detroit are the four best places, for following reasons:

- **Boston/Worcester** have most academics. Between Worcester and Boston, the Universities here are almost unparalleled. We have some good inflammasome scientists in Boston area, including Children's Hospital (associated with Harvard), Broad Institute, Kendall Square scientists.
- **SoCal** has important history with mobile pollution and has great academic institutions and researchers
- **UW Seattle** has a lot of possible resources to fund activities in the longer term. Gates Foundation very supportive of environmental health and the global burden of disease. Great group of mobile pollution people there. Connected with SoCal too (so have experience collaborating). Elena is there. Nulackshi Hudda is there.
- **Detroit**. Two best immune cell labs in the U.S. include **University of Michigan Medical**, and **UMASS Worcester Medical**. Best inflammasome labs in country.

Problem with U.S. Federal reference diesel particulates

A 2008 paper published by NLRP3 scientists in Europe looked at different kinds of particles to see if there was NLRP3 reaction and production of ION-1 β (beta). They included federal reference diesel particulates from the United States. These are U.S. immune cell biologists (not air pollution scientists). And they didn't find much reaction. They concluded that, surprisingly, unlike almost all other particles, diesel emission particulates did not trigger NLRP3 and IOB-1 beta reaction. But there was a problem with assumptions made about the federal reference diesel particulates.

Constantinos (Costas) Sioutas, Los Angeles (USC), with post-doc Ferra Hami, have been testing U.S. Federal reference diesel particulates against actual real-world diesel particulates over past two years. Found that the U.S. Federal reference doesn't react.

Wig pressed the scientists at the National Institute of Science and Technology (NIST) about their federal reference standard, and they said "Well, we just created it so that people could measure a few of the PAHs. We weren't trying to recreate actual diesel emission particulate." So they hadn't meant for it to be used as a reference for diesel particulates in research.

But because the inflammasome scientists in Europe didn't know that, until the Sioutas and Hami research, the whole world was misled.

But that has now been nailed shut. There have been studies on particulate matter, including ambient particulate matter and NLRP3, including taking away--every single step along the way and tracking the whole process.

Great paper came out of Mid-west recently. Now there is no question that ambient particulate matter acts through NLRP3 even though there was a disconnect initially. (Aside: the initial disconnect was as big as the disconnect in understanding continental drift.)

?: When you say ambient particulate matter, what size are you talking about?

Wig: Smaller the size, the more of it is in every square centimeter of your body.

?: This would argue for more measurement and ultimately regulation of ultrafines?

Wig: Yes. You can do it here, and in all four of the regions. There are not very many places you can do it. The people who have proven that **this is an unquestionable connection**, they are in specialized labs. When you drill down on little things, that helps with proving that there are causal pathways, but you also have to do population health analysis.

PM2.5 is the easiest thing in the world to measure. All of that stuff is growing.

Wig:

There have been at least four teams of Nobel prize winners working on ion channels. First was in last century. Rod McKinnon got MD at Tufts, then went to Rockefeller in NYC, really detailed the ion channels. Some are small (one atom going through them), others are big. More Potassium ion channels than anything else, second most common is Calcium.

An ion channel is a passageway through a cell membrane or sub-cellular organelle (like mitochondria). Anything with bilayer lipid membrane has ion channel. Everything that is alive has ion channels. There are zillions of them. They control thinking, hearing, heartbeat.

If you look at table of Elements, most prominent elements are CHON--Carbon, Hydrogen, Oxygen, and Nitrogen. Next to those, are Potassium and Calcium. Quite frequent, quite large size (not as big as CHON). Previously were focused on RNA, DNA, but right now, people doing the science of how life originate from inanimate physics, their focus is ion channels. There are 300,000 peer-reviewed NLM papers on ion channels (from last couple of decades).

Three good review papers on ion channels driving NLRP3. Each paper is a few years apart. Also papers, including from the European Consortium on Air pollution and Health. "So" is first author of a good paper that looked at disease and mortality and ion channel association. Found that you cannot activate NLRP3 unless potassium leaves the cells. **Potassium leaving a cell activates NLRP3.** It is supposed to be in the cell, but if it leaves cell, you are in an inflammatory state.

?: What causes Potassium to leave the cell?

Wig: a lot of things. A hole. Viruses have genetic coding that code new channels in cells.

Air pollution particles that have potassium have a high correlation with all the disease categories studied.

Researchers are using the ions as an indicator of source. Researchers aren't trying to understand the cell biology. No one is doing the immune cell biology. But the mortalities are most highly associated with Potassium, and almost highly associated with Calcium. When you concentrate Potassium inside cells, you are also concentrating Calcium outside the cells. Potassium cardioplegia (Del Nido) stops the heart during surgery.⁹

Research is still to be done on ions and whether it is the ions (Potassium, Calcium) in the particles that are driving the causal pathway. Someone needs to test this. Need cell studies, road studies.

What are the take-aways?

You need research in these four regions doing land-based modeling and census block groups; socio-economic modeling; current pollution and noise levels. Each of the four places has great research universities and people.

Mica: We have a new website coming online soon. We will share data and websites to encourage citizen science. How many of these papers are available? Would be cool to be able to share links to these articles and possibly the underlying research. Aaron note: get links to papers for website. Need a research library card to access them.

Wig: The articles are available at National Library of Medicine. May need agreement with people who did initial research. They will share, but they want proof of what you are going to do with it.

Eireen: For MCAC, we need to go back to some of the items where we can set goals. For example, DNL levels and how people can get information about noise in homes. Also, not clear how we deal with confounding variables influencing causal chain of effect of aviation fuel on health outcomes. Presentation was unfocused. Need to come up with a couple of well-defined goals on what we can do for the airport, state of Massachusetts, towns around, and what we can do.

Stephen: Thank you for the incredible wealth of information. Thank you for your work that has informed our work.

This was baseline information. We wanted everyone to have baseline information. The next step is where we go with this information. What are the things to concentrate on, short-term and long-term. We need another meeting to discuss these things.

Populating website with data and studies is important. Next step is for us to have a meeting to follow-up on workshop to set next steps.

Alan: Aaron and I have set up outline of environmental health strategy, talking with expert in environmental justice. We will shape that strategy to incorporate all the implications that Wig has educated us about. Need to think about what we want to accomplish in building a strategy, collaborating with researchers, and bringing in this information. That will involve creating a political/policy/legislative strategy. Once we have evidence to share, what do we do about that? State

⁹ <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6066678/>

can't do anything, so we need to go to federal level. But we can't go to federal level, so how do we start? Do we first go to the State and Massport and others, and talk about the problem? If we need to advance the knowledge so that something can begin to happen at federal level, how do we influence our own state political players, to bring that to the federal government, or get them to promote the multi-city research strategy. We are a small committee, with limited resources, so we need a clear strategy.

Stephen: The way we framed this workshop was Wig helping us by doing a data dump on the basics. Then a second meeting to identify and shape the strategic direction. Smart to have the second meeting to define the strategic direction, "bucketing" ideas, and talk about what should be the bullet points under each bucket.

?: We will have a follow-up meeting that is more strategy and goal focused. Remember, MCAC has no threat power. We can only do advocate. Part of the advocacy will be directed to our federal stakeholders is to convince them to spend political capital to push policies forward that will help our causes. Make it as affordable as possible (in a political capital sense) for them to do that. Our federal delegation is well aware of this issue. It is just how much political capital can they spend to move things forward. That's part of the calculus we have to use. Can talk more about this at the next meeting.

?: Also will need to create a multi-year strategy. Engage with other airports. Figure out what data to share. Do chunks each year.

Officially adjourned the meeting (motioned, seconded, voted). Thank you to Cindy for technical support to manage the meeting.