1) *How do doctors measure actual accumulated effects of toxins in the body? (not calculated/ modeled effects but measured effects.) Blood tests? Urine? Hair samples? Other? How accurate are these tests with finding toxins and disease?*

Accumulated toxins are measured either as a native toxin or a metabolite using biomarkers of exposure that may be measured in blood, urine, fat tissue, hair, or exhaled breath. Which sample is used depends on the particular toxin and how it is excreted or metabolized.

For example, exposure to heavy metals, such as lead or mercury, are determined based on blood or urine measurements, and also show up in hair samples. Exposures to solvents may be estimated from measuring them in the blood or by looking for their metabolites in the urine; occasionally they are excreted in the exhaled breath.

Many of these tests involve very sensitive methods that can measure minute levels of the chemical or its metabolites. Some tests, however, are non-specific, especially those that measure metabolites of the toxin because more than one toxin may have a common metabolite.

2) *Particulate matter is measured as PM10, PM2.5 and PM1.0 and .01. What is the difference between the effects of each on our bodies? Do Nanoparticles differ from large particles in our system?*

Particles are routinely measured as PM_{10} or $PM_{2.5}$. Research methods allow other fractions to be measured such as $PM_{1.0}$ or $PM_{0.1}$. As yet, the methods for measuring these finer particle sizes have not been standardized.

In general, particles less than about 12-15 microns in diameter penetrate the lower respiratory tract quite well. Particles 5-15 microns in diameter generally deposit in the larger airways where they can cause irritation (with symptoms of cough, phlegm, and wheezing). Those less than 5 microns can reach the fine airways and alveoli of the lung, where the important gas exchanging functions are performed. Sub-micron particles (that is, less than 1 micron in diameter) or nanoparticles seem to behave more like gases than particles in that the smaller they are the easier it is for them to pass straight through the alveolar walls and into the blood stream. Particles bigger than 1 micron in diameter do not seem capable of doing this.

The consequences of very fine particles passing straight into the blood are not well understood, but it is possible that they can cause direct damage to vital organs such as the heart. Indeed, there is an increased risk of sudden death related to the level of fine particles in the air, and that reducing those exposures to very low levels could reduce overall mortality by as much as 4 percent.

I have attached a list of articles that speak about exposures to particles and the risk of death.

3) *What is the safe level of dioxins/hormone disrupters in the body of a child? An adolescent? Pregnant woman? Elderly?*

Safe levels of exposures to dioxins or endocrine disrupters have not been defined. In general, the developing body of a child or adolescent is often more susceptible to environmental toxins than an adult. This relates to the higher metabolic rate in children (greater ventilation and cardiac output) and the

development of organ systems at young ages. Therefore, dioxin and endocrine disruptors might be expected to have greater effects in young children and adolescents.

In pregnant women the key concern would e the developing fetus, a highly susceptible organism to environmental toxins, especially those that cause genetic damage or interfere with key signals involved with growth and maturation of organ systems. Dioxins and endocrine disruptors would be expected to have substantial effects in the developing human fetus, and post-natally when some of these agents may be present in breast milk.

The elderly would be the least affected by these exposures, although we know that some endocrine disruptors, such as estrogenic compounds, could influence cancers in older subjects.

I have included a list of recent articles about environmental estrogenic compounds. These are highly technical and mainly relate to animal experiments, which reflects the state of knowledge at this time.

4) *If we are talking costs, how can we account for health care costs (due to a technology and fuel choice)?*

Sorry, can't help you with the economics of pollution. This is a specialized area. I would say, however, that the costs of ill health are more than just the costs of health care.

5) *What are the effects of emissions and fallout on our soil and water? If we have home gardens and use collected rainwater, would a vegetarian or homeowner have an increased cancer risk?*

In general, the effects of deposition are to increase soil and water pollution, especially for persistent chemicals such as metals. I can't answer a question about cancer risk because it will vary by the type of pollutant and its amount. Home gardens with substantial consumption of products grown in a polluted area could conceivably increase risks for cancer or non-cancer outcomes. This is in the realm of risk assessment that the MPCA will need to look at. It's a complicated and time consuming process to look at all pathways of exposure from pollutants.

6) *Based on the number of vehicles on I-94 and Hwy 280 (397,000 vehicles per day) how much more of each toxin can our bodies accept without potential cancer, asthma or other health risks? (For this question, I think we are wondering if you can generally speak to bio-accumulation and impact on the body)*

I don't have the data on existing ambient pollution from mobile sources, nor the likely risks from those sources. Again, this is an MPCA issue that would be addressed in a formal risk assessment. I would caution that there is a difference between "cumulative effect" and "bioaccumulation." In this instance, we would be talking about a cumulative effect from mobile/other sources plus the Rock Tenn emissions– these could produce short-term or long-term health effects. In either case, the health effects may or may not be accompanied by bioaccumulation, which is the persistence of chemicals in the body.