November 30, 2022

Attention: Dr. David Holstius

Senior Advanced Projects Advisor
BAAQMD

Re: Comments on "Modelling Local Sources of Fine Particulate Matter (PM2.5) for Risk Management"

We have read and reviewed your "Modeling Local Sources of Fine Particulate Matter (PM2.5) for Risk Management" model description. Thank you for your interest in improving the air quality in the Bay Area and developing this model to estimate the effect on premature mortality and asthma incidence of changes in PM2.5. In the past, one of the authors (Dr. Walsh) has participated in discussions organized by BAAQMD concerning the health effects of PM2.5. An increasing number of scientific studies demonstrate that life expectancy, premature mortality, disease incidence all improve when the PM2.5 level in the air is less. There is no threshold for PM2.5 because mortality and morbidity continue to decrease as PM2.5 declines.

We understand that this Model has been developed as part of a regulatory process to decrease PM2.5 emissions from local sources and thereby decrease the PM2.5 levels in the Bay Area. We just have a few comments and questions that we found could use further clarification or development. We wish that our contributions will further reinforce the arguments and findings of your article.

Our comments address the following topics:

1. Use of the model in the regulatory process
2. Underestimation of PM2.5 caused mortality and morbidity
3. How much PM2.5 is emitted and its location
4. Factors used in the model
5. Vulnerable populations

1. **Use of the model in the regulatory process**

It is unclear from the description in this Draft Model under what circumstances this Model will be applied: how it will be used for policy and regulation, and how the process of regulating PM2.5 emissions from local sources will use this model. Located in the Bay Area are a number of stationary sources that produce large amounts of PM2.5, among them are: several refineries, cement plants, and other industries. The PM2.5 is emitted from the refinery smokestacks, vehicles on the roads entering and exiting within the properties of the refineries, from the ships docking outside of the refineries, and from other processes associated with its production. Each of these refineries are among the very largest single emitters of PM2.5 in the Bay Area. How will this model be used to ensure the decrease in PM2.5 emitted into the air? What will be the criteria for PM2.5, premature mortality, asthma incidence or other estimates from the model that will result in BAAQMD requiring that the stationary source must decrease its emissions? How will that be determined?

Please include a clear outline of the steps in the process of regulating PM2.5 emitted from a local source, the role of the model and timeline for implementation.

2. Underestimation of PM2.5 caused mortality and morbidity

Your model substantially underestimates morbidity and mortality from PM2.5 since it only includes premature mortality and asthma incidence. It ignores the many other disabilities and illnesses that scientific studies have demonstrated result from exposure to PM2.5 such as low birth weight, preterm birth, chronic obstructive pulmonary disease, dementia, cardiovascular disease and cancer among other illnesses. **How will these be considered and included?** The studies relating PM2.5 levels to premature mortality are clear (e.g., Di and Yazdi). But, the Winer 2012 paper referred to in your draft does not clearly demonstrate how the asthma incidence changes with PM2.5 levels, and how incidence would decline with lower levels. **We suggest you include a more comprehensive reference and explanation**

3. **How much PM2.5 is emitted and its location**

The model appears to rely on PM2.5 emissions reporting from the local source. These reported emissions that are then entered into the model to estimate the health risks. **How accurate are these emissions? Is there a way to use monitoring of the local source for PM2.5 levels and emissions to corroborate the reported emissions in order to increase reliability of the emissions reporting?** The emissions are reported as those released into the external air. Workers are likely also exposed to PM2.5 inside the work place (and also in their homes as some homes have very high levels). **Will only including the external emissions outside the workplace accurately measure the PM2.5 that the worker is exposed to from the emitting source? Please comment.**

We are concerned about how this Model converts the epidemiologic studies that clearly demonstrate the increasing incidence and prevalence of premature mortality and many other disabilities such as cardiovascular disease, lung disease, cancer, low birth weight and preterm birth and dementia into this model. The epidemiologic studies average results of both monitoring and health data over large areas and with widely spaced monitors while this model seems to focus on nearby exposures that occur over part of the day. The epidemiologic studies do not estimate breathing rate and proportion of the day exposed, but average over years of exposure. It is unclear how these broad epi studies translate into breathing studies. Please explain and provide references.

Short term, large increases in PM 2.5 appear to affect health and this is not included. For example, in the areas where air pollution from California wildfires was great, the incidence of COVID increased (ref on req). **How are these short term, large increases incorporated into the model?** If these occur, they will worsen the effects of the local PM2.5 sources .

4. **Factors used in the model**

The paper mentions a number of factors that are incorporated into the model; however, there is little explanation of how they were chosen and how adjustment factors are incorporated into models as they can greatly modify the results. These include:

Both Tables 8 & 12 refer to adjustment factors of 4.2 and 3.36. The 'factor of 4.2 applied to account for potential overlap in the schedules of the source and receptor when modeling is used to calculate and annual average concentration increment (OEHHA 2015).' For school and daycare, the adjustment factor is 3.36. The reference seems to be from Tables 5.7 and 5.8 in the OEHHA 2015 report. I have looked at these tables and several pages surrounding these tables without finding a clear explanation of their derivation and use. Please consider listing the location of these factors in more detail. Further discussion of reasoning and methodology could also be beneficial in accepting your reasoning. We would like to be able to more easily recreate your methodology and factor use for ourselves, expanding our understanding of your data analysis.

Please explain the use of these factors and derivation

5. Vulnerable populations

We understand that environmental justice and protecting sensitive populations that have the highest risk of poor health from PM2.5 exposure are priorities for BAAQMD. These sensitive populations include seniors, pregnant women, infants and young children, those with chronic diseases, lower socioeconomic status, those living in areas where exposures are greatest, among others. Pages 12 & 13 address these issues; page 12 states" we can adjust the estimates of relative risk (as represented by the term β) to compensate for individuals who exhibit a larger or more severe dose-response relationship. We can also do this to account for data deficiencies" What are these data deficiencies? Are these the same data deficiencies mentioned in the subsection "Data deficiencies" on page 13 where the Advisory Council suggests an additional factor of 3 to compensate?

The spreadsheet tables estimate both a 'statistically average individual' and 'sensitive individual'. The draft mentions a factor of 3 to account for the increase in premature mortality among vulnerable seniors (as demonstrated in the Di and Yazdi papers), and also an adjustment factor of 'three to account for data deficiencies.' In the spreadsheet, the factor 3 for Relative Risk is included, for example, in the seniors calculation, but not the factor of 3 for data deficiencies. I concluded from your explanation on pages 12 and 13 that both factors should be included in estimating risk in sensitive and vulnerable populations. That would substantially increase the health risks from PM2.5 exposure for sensitive populations. Please explain.

Please demonstrate clearly the results with the derivation and use of these factors in the vulnerable populations, and explain data deficient populations. Please explain what are the data deficiencies and the size and distribution of the vulnerable populations in the Bay Area.

Again, we wish to commend you on a successful and thought-provoking study. Looking into the breathing rates as a means to measure the intake of PM2.5 is an innovative approach that reaped compelling data and findings.

Best Regards,

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