Responses to CASAC Questions on the Ozone PA from Consultant Dr. D. Warner North

My comments on the draft Policy Assessment follow themes from my earlier comments on the draft PM Policy Assessment and the draft Ozone Integrated Science Assessment. I remain concerned about confounding in the interpretation of epidemiological data. I believe modeling of exposure level to health response needs to be done carefully, reflecting biological knowledge and expert judgment. I have substantial concerns about how EPA has modeled both air quality and exercise patterns to predict the health responses that were termed adverse in revising ozone standard in 2015. I remain concerned about wildfires as an important source of ozone exposure at unhealthy levels. Wildfire plumes may have contributed to Sacramento, California being the highest in ozone levels of the eight metropolitan areas that EPA used in its PA analysis.

While the draft PA represents a great deal of work in assembling information, it is disappointing that there is so little new information on human clinical studies or in assessing impacts on asthmatics, as might be measured by hospital admissions or emergency department visits in areas with high ozone levels.

Although I am not trained as a medical professional I have extensive personal experience with the frequency and severity of asthma episodes. The most common triggers for a bronchospasm ("asthma attack") come from pet dander, dust mites, and cockroaches, not air pollution. (https://www.xolair.com/allergic-asthma/what-is-allergic-asthma/allergic-asthma-triggers.html). These common triggers may be more frequent in low socioeconomic status locations. Asthma patients control their airway reactivity with inhalers such as albuterol, and for more severe cases, corticosteroids. These medications may be needed frequently (several times per week, or even daily). Especially for severe asthmatics, activities involving exercise may be moderately to severely curtailed. Yes, air pollution can trigger or aggravate asthma. (https://www.asthma.com/what-is-asthma.html). But the context ought to be considered – for most asthmatics, air pollution at, or near, present standards is a minor contributor to their symptoms and their need for medication. Most asthmatics are not going to do prolonged exercise out of doors, especially on days with an unhealthy air warning. As one who has personal experience over decades with a severely asthmatic patient and also with an adult with cystic fibrosis, I am skeptical about the analysis used in 2014-5 for the previous round of ozone review. The distinction between responses at 60, 70, and 80 ppb exposures is based on only a few studies, most of them not recent. Inflammation is not well defined and measured. Virtually all observed symptoms including lung function decrements (FEV1) and airway reactivity are transient, going away after a few hours to a day. But: exposure to unhealthy air in the form of a wildfire smoke plume persisting over a metropolitan area for many days should be viewed as a serious public health threat to people such as the two I am close to. Exposure or potential exposure to smoke plumes this year and last year have motivated decisions about getting masks, special filters for air conditioning systems, and sending the sensitive person(s) out of the affected area until the air clears.

Nearly four decades ago, at a time when I was first involved in working on air pollution health effects, there were repeated episodes in a European city of asthma attacks in which many people went to hospital emergency departments for treatment. The incident was initially blamed on air pollution from an electric power plant. More careful investigation indicated it came from unloading ships in the harbor, and the main culprit was soybean dust. The reference is Antó et al., *New England Journal of Medicine*, 1989. Yes, air pollution can trigger episodes of asthma attacks across an urban area. But indoor air

pollution from pets, dust mites, and cockroaches is much more likely to be the trigger for asthma attacks in urban areas of the United States.

The studies listed in Appendix 3B on emergency department visits (EDV) and hospital admissions for asthma include numerous ones on New York City and Atlanta, Georgia. I found only one, state-wide study for California, Malig et al., 2016, with data from 2005-2008. This study showed a small increase (1.5 to 3.9% per 10 ppb O_3) for EDV for asthma and upper respiratory infections using warm season data. (Full year results were slightly smaller.) A little less than half of the EDVs were for children 18 and under, rather than adults. I would characterize these results as weak association evidence motivating further studies. The authors' final sentence is, "Studies examining the health benefits of ozone reductions should try to account for ozone-EDV relationships to get a fuller picture of those benefits." I heartily concur.

Questions from Dr. Corey Masuca

1. I do not have a background in the specifics, but I believe the mechanism has been well established in the scientific literature for many decades. Here is a reference to an article by a Norwegian scientist from 1960: Storebø, Per B., "The exchange of air between stratosphere and troposphere," Journal of Meteorology 17:547-554 (1960). During the 1960s there was much interest in the radioactive isotopes from nuclear testing coming from the stratosphere into the troposphere. There are many other papers you could find with a search on the web.

2. Near Road Monitoring sites are most important for determining how ozone and precursors change between emissions on a road or highway and ozone levels at locations a short distance away. The ambient air monitoring discussed in this section and Section 3 is at the regional and national scale, rather than the local scale. I note for your attention my responses to Dr. James Boylan regarding Section 3 and Appendix 3C. The calculations made by EPA for its Section 3 analysis were done with a national model, CAMx, with a 12 km by 12 km grid. That is the size of the City of San Francisco, 50 square miles! Local peaks and valleys in ozone levels are not predicted with a model of this large grid size. There are comparisons in 3C.4.2 of model predictions at the grid cell level and monitoring station(s) within the grid cells. The figures in this subsection show the relatively large differences between the model predictions and MDA8 observations. Only one year of data is used for the model predictions. I believe use of a model with a much finer grid size would be useful for areas violating the present standard, and data from ***all*** the available monitoring stations should be collected and used. It would be important to examine multiple episodes over many years of prolonged high ozone levels, not just one year, to determine the mix of ozone created out of the local area with ozone from local and nearby NOx and VOC sources. I did not find evidence in Section 3 and Appendix C that EPA has done such local analysis or emphasized prolonged high ozone episodes from multiple years. Section 3C.6 describes a mathematical system ("Voronoi Neighbor Averaging") for scaling the CAMx predictions at the monitoring sites to census tract centroids, and these interpolated predictions of ambient ozone levels are used to predict the incidence of health effects for the people in the census tract. (See page 3C-91 for an illustrative diagram. See Figures3D-2 to 3D-5 for pictures of the census tracts.) Local chemistry of ozone formation and absorption on a scale of less than about 10 miles cannot be done with this EPA modeling system. Ozone peaks produced by, for example, rush hour traffic concentrations in an urban area cannot be examined: the "grain size" of EPA's analysis is too big! Compare to the data in Figure 2-5, page 2-12. In the 2015-2017 data the exceedances of the MDA8 70 ppb are appearing only in one broad region, California's Central Valley plus Los Angeles to San Diego (coastal urban areas with

mountains to the east creating a "mountain bowl"; the Central Valley is a big "mountain bowl") -- and in a small number of urban areas – Phoenix, Seattle, Salt Lake City, Denver, Dallas, Houston, Chicago, and the Boston- Washington corridor. It would be useful to have analysis that focuses on these (non-California) urban areas in finer detail. My concern for the California region is that wildfires may play a critical role in causing a large fraction of the red and orange circles in Figure 2-5, which are exceedances of the current 4th worst 8-hour-average day-in-a-three-year-period (MDA8) standard. We should know more about the exceedances in the urban areas other than in California, which are not due to wildfires but to other sources of NOx and VOCs.

3. I am not expert on the details, but I think there is a well-defined protocol for computing the maximum 8-hour average from hourly concentrations. I believe it is described in one of the Appendices, probably in the ISA if not the PA, but in my available time I did not find a page reference to give you.

4. Sunlight drives the photochemical formation and destruction processes for ozone, so what is being discussed in section 2.4.3 is primarily related to the presence/absence of sunlight, that is, day versus night. The length of the day enters as seasonal variation. Temperature and topography/geographical factors affecting air movement are all important for understanding the processes for formation and destruction of ozone. This complexity should be included in the modeling. As I have described under 2, EPA's system cannot accommodate local detail.

5. My big concern on 2.5.1.3, including wildfires as part of Background, "USB." I think wildfires as well as prescribed fires should be considered as anthropogenic sources and not as background. Wildland fires can lead to levels of both $PM_{2.5}$ and ozone far above current standards. At least in this PA "wildland fires" get called out for a heading in the *Table of Contents* and two paragraphs that make it clear that wildfires fires can be an important contributor to ozone exposure in regions where these fires occur. The high exposures from large wildfires should not be kept out of sight by deleting them from the data as "exceptional exceedance" events. They should be considered as the consequence of human activity – in particular, national, state, and local policies that influence the occurrence and severity of wildfires. The adverse health effects are quite real to those who suffer them. Whether or not a state gets relief from whether these "exceptional exceedances" trigger a finding of non-compliance with a NAAQS may be important because of penalties for non-compliance. That is a separate matter from protecting public health.

6. Methane can be the VOC that forms ozone in the presence of NOx and sunlight. Usually other VOCs are more important, but if these others are not present then methane contributes to NOx formation. The ISA had extensive discussion on the role of methane. Yes, in is true that in our post-industrial society we have a lot of sources of methane such that there is much more than in pre-industrial times, and we are uncertain on how much more methane we now have. But that uncertainty will be much more important for climate alteration from CH4 as a greenhouse gas than for methane contributing to peak levels of ozone above the primary standard. I would say that extensive discussion should be in the ISA rather than the Policy Assessment, since controlling sources of methane is not a focus for the Policy Assessment document now under review. I do not find the three-paragraph discussion in 2.5.1.6 as inappropriate as I do the huge amount of complex detail elsewhere in this long document.

Questions from Dr. James Boylan

Overall, for the three portions of the PA that you chose to ask about, your question of whether it is accurate and complete has led me to comment on what I think about the whole PA draft document, which I have done in my earlier general comments. By and large, I think EPA staff have worked hard and done a good job in assembling a great deal of relevant material. I don't think any document of this type can ever be judged as complete. I think a major goal of the document should be to focus attention on research needs as well as policy needs. The document should be an evolving guide to both government officials and interested parties on the public. Trying to make it more "complete" might add much more detail that is superfluous to the interests of most readers. It should be accurate in the sense that it does not mislead readers. The material on inflammation at low exposure levels in the ISA draft is, in my judgment, at least borderline misleading in suggesting evidence for inflammation at levels at or below the standard. As I expressed in my ISA comments, I thought the support for these statements in the ISA was weak. The discussion in the PA seems a bit better in citing what was actually written by authors of the studies. Section 3.5, page 3-74 line 29-232 cites the ISA rather than the preceding portions of the PA. I repeat my objection to the words, "respiratory inflammation" in this sentence as inaccurate as stated, without any caveats. Two sentences on page 3-76, lines 9-14, are slightly better, indicating that "inflammatory response and airway responsiveness" are "reported for higher exposure concentrations" rather than at "concentrations slightly above 70 ppb with intermittent exercise." See also footnote 69, page 3-76.

Air Quality: Chapter 2. I am a risk person with a physics background, so I can review this chapter with a modest level of understanding but not a close familiarity with recent literature. I find the chapter generally good and have only one major criticism, which is a policy dissent with EPA that I have raised in my previous two response submissions. I do not think wildfires should be considered as background, but as anthropogenic sources that can be strongly influenced by strategies on management over land areas where wildfires occur.

Chapter 3: Review of the Primary Standard. You focus your questions not on this whole chapter, but on Sections 3.4.1 to 3.4.5, page 3-45 to 3-72, 28 pages. You do not include section 3.5, the evaluation of the available evidence as of 2019 and the recommendation that the primary standard, lowered in 2015 from MDA8 75 ppb to 70 ppb, be maintained, or the discussion of the Administrator's reasoning in revising the standard from MDA8 75 ppb to 70 ppb, described in Section 3.1.1.

There is relatively little that is new in the 28 pages. Almost all of it is very similar to what was provided in the EPA Health Risk and Exposure Assessment (HREA) in 2014. Is the reasoning used by the Administrator (after advice from CASAC and the public) valid today? Is there important new information? Might the standard be viewed as overprotective, compared to the previous standard of 75 ppb? Or is it in need of further tightening? Children including asthmatic ones in the Sacramento area experience MDA8 exposure well over the 75 ppb level. How much of public health problem does this pose? Asthmatics and children in other urban areas are experiencing levels in the high 60 ppb range and above. Is their health being protected with an adequate margin of safety? What strikes me as odd is that there is so little described in Section 3.5 beyond the reasoning of a previous Administrator described in Section 3.1.

You asked about Appendix 3C, which presents a great deal of information on population exposure. Appendices 3C and 3D support the Section 3.4 modeling exercise on how many health effects might be expected with reductions in ozone precursors such that the eight metropolitan areas just meet a MDA8

standard, with calculations for 75, 70, and 65 ppb. The Appendix 3C material is extremely detailed, and it does not tell us about the extent of observed or predicted public health effects under current levels of ozone exposure, as opposed to these projected "design values" to just meet a standard.

Here are some brief notes on Sections 3C.2 to 3C.7. In Section 3C.2 EPA describes the eight study areas. The section does not explain in any detail why these eight were selected – seven are holdovers from 2015. The extent of the explanation is one sentence, lines 8 to 10 on page 3C-13. Section 3C.3, Ambient Air Ozone Monitoring Data, describes the procedure for determining the NOx emissions changes needed to meet the three MDA8 standards. The maps show us the location of monitors, including buffer sites used for interpolation for modeling air quality. Section 3C.4.1 tells readers that the CAMx model was used with the Higher Order Direct Decoupled Method (HDDM). The CAMx model covers the lower 48 states and adjacent areas of Canada and Mexico with a 12 by 12 kilometer grid, and it was run for all of 2016 including a startup period in late 2015. (This is one year of weather, with a grid cell size of 50 square miles. Local scale phenomena will not be captured in such a system.) Weather data for 2016 came from a mesoscale numerical weather prediction model, and 36 vertical layers are used for this and for CAMx. (Later in the 3C text 44 levels are indicated.) The alpha version of the Inventory Collaborative 2016 emissions was used. Emissions for wildfires and prescribed burns were included. (If any periods were deleted as exceptions, this is not noted, and ***any such exclusions of emissions should be disclosed***.) 3C.4.1.6 describes initial and lateral boundary conditions. Section 3C.4.2 discusses how well the CAMx model can reproduce the actual measured 2016 O_3 concentrations. We are told the predictions "generally reproduce patterns of observed O_3. The notable exception is a persistent underestimate in winter across almost all regions, particularly at the higher latitude sites." (page 3C-29, lines 2-4.) Then a large amount of statistical data and maps are given in support.

EPA has amassed a huge amount of detail on modeling ozone in ambient air nationally on a large grid. Details about local topography and sources such as concentrations of vehicular traffic and major stationary sources are absent. There is no calculation at this stage on what is in indoor air, as opposed to outdoor air. Does the smoke plume from a large nearby wildfire blow into a metropolitan area, or does the plume bypass the area? This depends on the wind direction. Data from only one year will not reveal patterns that may cause the peak ozone exposures over a period of three years, five years, or longer. For seasonal ozone averages over the lower 48 states I would expect a general match, but I doubt if peak ozone concentrations leading to MDA8 standard exceedances at individual monitors will be well reproduced. Figure 3C-13 to 16 indicate a normalized mean bias of the order of 20 ppb in the northeastern United States, with a larger discrepancy in winter. For concern about peak ozone levels in the Washington to Boston metropolitan corridor, that is not great accuracy. In the west with higher background, the validity of the model prediction may be even more questionable in terms of the frequency of exceedances above the standard at specific monitors. See Figures 3C-40 to 43.

Section 3C.5 discusses air quality adjustments, specifically, reductions in NOx emissions to just meet the standards. We are told that EPA used this approach for the 2015 O_3 NAAQS review. This is an effort (using a very detailed model of only moderate accuracy on a regional basis) to predict ozone levels if NOx reductions were made so as to allow the standards to be just met. The chemistry is non-linear, and so there is an HDDM adjustment process. In some metropolitan areas (e.g., Sacramento) big emissions reductions will be needed to meet standards.) Then in 3C.6 we learn about interpolating from a 12 kilometer grid size to 500 meters and centroids of census tracts. How well does this work in downtown urban areas with street canyons? What is the variability of ozone readings within a few kilometers of a monitor?

In 3C.7 all of this is put together to compute results, design values for patterns of ozone exposure over the urban areas. Perhaps something can be learned from this exercise, but it should be realized that it is an effort to go from a lower 48 scale to predict on a "neighborhood" scale, with assumptions compounded all along the way.

My summary on the completeness and accuracy of 3C2 through 3C7 is as follows. Yes, EPA staff have told us what they have done, and it appears to be little changed from what was done in 2015 to calculate risk numbers for health effects under alternative standards. In several weeks of reading of documentation outside the PA and its Appendices, one might be able to find all the details on what was done. I did not have time or motivation for such investigation. I judge the accuracy of the predictions to be very limited, especially for calculating peaks, the fourth highest 8 hour average over a three year period.

The collection of models and assumptions produces apparently precise numbers about how many health effects might occur under alternative standards. See the discussions supporting the choice of a 70 ppb standard in Section 3.1 and 3.5. The key uncertainties not covered in 3C are the assumptions about human behavior, exercise patterns, and exposures outdoors and indoors where the presence or absence of air conditioning may be important. (The methods are described in Appendix 3D.) I would like to see regional studies using local models, knowledge about socioeconomic status by neighborhoods and involvement of state and local air pollution experts who might know about "hot spots" of peak exposure, and their proximity to schools, playgrounds, sports arenas, and other locations where children and adults might be exercising out of doors for the order of six to eight hours. And do the exposed people learn about unhealthy air conditions and change their behavior so as to avoid exercising at times of high ambient ozone levels? In the unhealthy air from recent wildfires in northern California, schools were closed and sporting events were cancelled. Does an asthmatic want to ride her bike to work on a unhealthy air day, or will she opt for using her car or public transportation?

Section 3.6 of the PA describes key uncertainties and areas for future research. There should be much more attention to "understanding of O3 effects" in the range of 70-120 ppb, and not at "below the lowest concentrations studied," which would mean below 60 ppb (page 3-88). Human behavior is hard to predict. Better understanding is needed on who is at high risk by exercising outdoors under high ambient ozone conditions. Sensitive subgroups such as children and asthmatics need to be protected. How great is the need for protecting them in areas and cities experiencing MDA8 exposures near and above 70 ppb, sometimes above 100 ppb, based on Figure 2-5, page 2-12?

What EPA staff did in examining eight metropolitan areas was to model ozone exposures under a set of assumptions that emissions reductions would occur such that these areas ***would just comply*** with the standards. Were the health impacts predicted at these computed-by-model with assumed patterns of human activity judged to be acceptable for protecting public health with an adequate margin of safety? Yes. that was the claimed goal for the analysis, to enable an evaluation of model predictions of health effects from model predictions of air quality. There is little evidence that EPA worked with its Regional Offices, with state agencies, and the research community ***to ascertain the magnitude of the public health impacts of recorded actual ozone exposures*** in the most recent years, from 2015-2017 to the present, in areas where exposures exceed the standards by a large amount. Why not, as a supplement or a better use of EPA resources, go to Sacramento as the city with the highest ozone exposure (e.g., Figure 3C-81, page 3C-114; Figure 3C-103, page 3C-138) and learn more from the medical professionals in this metropolitan area about the extent to which asthmatic children in that area were suffering health exacerbation, such as inflammation, pain on inspiration, and increased airway responsiveness?

The numbers in Table 3.3 page 3-58 reflect that with air quality just meeting an MDA8 standard of 70 ppb, exposures at 80 ppb and even 70 ppb are quite rare, and exposures above 60 are relatively infrequent, an average of 3 to 9% for children with asthma and slightly less of all children experiencing one day per year of exposure while breathing "at an elevated rate." Compare Table 3-5, with air quality just meeting a higher MDA8 standard of 75 ppb. The numbers for affected children nearly double to about 7 to 16%, because exposures above 60 ppb are projected to increase by that much.

Sacramento is far from meeting a 75 ppb standard. In order ***to bring Sacramento into compliance with the present MDA8 70 ppb, a reduction of 58% in NOx precursor emissions is estimated to be needed.*** A 45% reduction would be needed to meet the old standard of 75 ppb. These are a big numbers! No others of the seven metropolitan areas would need more than a 23% reduction in emissions to meet the old standard of 75 ppb. IF the standard were reduced to 65 ppb, Sacramento would need a reduction of 72% and the need for the other seven areas would be in the range from 38 to 68%. The numbers are large for Sacramento because Sacramento has a high background ozone level, "USB," as EPA uses this term. Phoenix also has high background. Phoenix could meet the 75 ppb standard with an emissions reduction of 14%. But for the 70 ppb standard, Phoenix would need to reduce its emissions by 49%, approaching the high number for Sacramento. Some of that ozone comes from wildfires, now counted as background. Note in Figure 3C-103 the red squares in the observed data, above and below Sacramento in rural adjacent counties. These might have come from the large wildfire plume(s) in the year 2017. (Numbers in this paragraph come from Table 3C-19, page 3C-89.)

Critical commentaries on EPA's modeling. The PA and the ISA do not acknowledge published criticisms of the methodology used in 2014-15, and used again in this 2019 PA with only minor changes. I have not have the time to find more than a few examples of such criticism, but in reviewing three EPA draft documents I have found little evidence that EPA has included criticisms in peer-reviewed journals, presentations at EPA public meetings, and written comments from members of the public.

In my comments on the Ozone ISA I discussed the Belzer–Lewis paper recently published (2019) in *Risk Analysis*. I will not repeat these comments, which CASAC members should have in my earlier submission on the ISA. Another paper pointing out the uncertainty in estimating FEV1 decrements is Glasgow and Smith (2017). Modeling uncertainty on the concentration response relationship should be considered as well as statistical uncertainty. Neither of these papers is referenced or discussed in the ISA or PA. The Glasgow-Smith paper discusses the methodology used in EPA's Health Risk and Exposure Assessment (HREA) from 2014. Essentially the same methods appear to have been used in generating Table 3.3, based on the McConnell et al. papers from *Inhalation Toxicology*, 2012 and 2013. (See footnote 64, page 3-57, which explains that there are "a number of differences between the 2014 HREA and the quantitative modeling and analysis" in the PA, with these details discussed in Appendix 3D. The HREA and the two McDonnell et al. papers are referenced in the PA Section 3.

Another missing reference from Section 3 (and not in the Jaffe et al., 2018 reference in the PA) is the work on wildfire plume exposures by Larsen et al. (2017). Here is a quote from the *Science Digest* summary: "While plumes had occurred only on 6-7 percent of days, these plumes accounted for 16 percent of unhealthy days due to small particles and 27 percent of unhealthy days due to ozone." A direct quote from Larsen et al. follows: "Smoke-plume days accounted for a disproportionate number of days with elevated air quality index levels, indicating that moderate increases in regional air pollution due to large fires and long-distance transport of smoke can tip the air quality to unhealthy levels." (The data in Larsen et al. are from 2006-2013. The numbers could be much higher for 2017-2019, when

Northern California has had large wildfires affecting air quality in the Central Valley (including Sacramento) and the San Francisco Bay area.)

Questions from Dr. Sabine Lange

1. I fully agree that the decrease in annual average ozone exposure is significant. I continue to have concerns on whether the epidemiological results imply manipulative causality as opposed to association, and I am pleased to read that EPA is not using these epidemiological results but rather basing its recommendations (for the last round and the present one) mainly on human clinical studies. There are still areas of the US, such as the Sacramento area, that have MDA8 levels well above the current standard of 70 ppb. I would like to see CASAC focus on the public health risk in these areas. See my general comments above regarding asthma. There ought to be more research to see if high ozone episodes in Sacramento (and elsewhere in the Central Valley and the Los Angeles to San Diego area) have led to increases in hospital admissions and emergency department visits.
2. No. I responded to a similar question in the O_3 ISA. Statistical power comes from having a large sample size, and NOT from having resolved issues of confounding, error, and bias. Consider we have a study of 10 million children showing that shoe size predicts reading ability. Because data were obtained from 10 million children, a very large number, the confidence interval is quite narrow. Does this apparently accurate prediction imply that getting children larger shoes will improve their reading ability? No way!
3. I am inclined to think that the problem is a general one that will only be resolved by getting data on potential confounders such as income (more generally, socioeconomic status), and extremes of temperature, which have large impacts on mortality and morbidity via mechanisms independent of air pollutants. However, we should understand that at VERY high exposure levels, air pollutants such as ozone and fine particulate matter (e.g., smoke) can cause illness and death. The shape of the exposure-response relationship is critical for assessing the risks. Extrapolation over orders of magnitude is readily done with available mathematics. But how this extrapolation is done should reflect judgment on the biological mechanisms underlying damage to health.
4. Yes, assuming a lack of threshold has become a standard method in many areas of EPA's risk assessment practice. Many of us old-timers believe this practice is questionable, because absence of evidence is not evidence of absence. The biological mechanisms underlying the adverse health response should be assessed based on available information including judgment. Traditional toxicology has used a sigmoid shaped exposure-response function, on the basis that very small exposures (episodic or cumulative) are unlikely to trigger an adverse response but as the exposure increases, the body's defenses and repair mechanisms can become inadequate, so the adverse effect becomes common in an exposed population. And the response may saturate with most or all of those who are susceptible to it having the adverse response – e.g., given enough bacteria in the spoiled food, nearly everyone gets sick from eating it. But linearity to zero became common in cancer risk assessment. This assumption was originated as a health-protective default assumption for screening: a plausible upper bound for identifying chemicals deserving more detailed risk analysis, and not for estimating the incidence of human cancer. But linear to zero is often used for the latter purpose.
5. I am concerned that FEV1 decrements are not a good indicator for adverse health impacts in sensitive populations. (See my general comments at the beginning of this response. FEV1 measurements vary a good deal. The Belzer-Lewis paper mentioned in my O3 ISA response has

perceptive criticism about using FEV1 data in research.) It seems to me that lack of information, referring to the words you use in your first sentence, (1) should motivate detailed studies of the people that are judged to be at highest risk, and (2) leaders of agencies such as EPA should think beyond legally required standard setting to the bigger issue of how to protect public health with an adequate margin of safety. If adverse health effects are judged to be essentially absent for much of the United States (a reasonable inference from Figure ES-1 in the ISA and Figure 2-5, page 2-12 in the PA), then attention should be focused on the remaining areas where such adverse health effects may still be occurring. Are these adverse health impacts really there in these remaining areas, or are our government officials being overly precautionary and protective in setting standards, but ignoring major public health protection needs by assuming that some causes, such as wildfires, are "natural background?" ***EPA should be using common sense and not be trapped in traditions that violate common sense.*** The levels of ozone and fine particulate matters that millions of people in California have experienced from wildfire smoke plumes in 2017, 2018, and 2019 are far above the NAAQS standards and pose serious health effects, especially to members of sensitive subgroups. Some of these people are among my family, my friends, and my neighbors. The costs involved in reducing these risks to health from wildfire plumes are very large. So are the costs of bringing ozone levels in Sacramento into compliance with a 70 ppb MDA8 standard, even if with wildfire periods are exempted. (In my humble judgment, the former activity makes much more sense than the latter.) EPA staff and CASAC should acknowledge these facts in their written documents, as part of advising the EPA Administrator on strategy with respect to criteria air pollutants. I believe giving such advice is within the legal mandate of CASAC under the Clean Air Act.

Comment: I appreciate and endorse the message in the graph you show in your questions for the St. Louis area comparing 2001 to 2003 with 2013 to 2015. Please consider also a graph of the same sort of data for the Sacramento area for 2017-2019, compared to earlier years with no big wildfires, such as 2013 to 2015. The message will be almost opposite. Compliance is not nearly achieved, but a distant and receding goal, especially if wildfires are not exempted.

References not in the PA

Garrett Glasgow and Anne E. Smith, "Uncertainty in the Estimated Risk of Lung Function Decrements Due to Ozone Exposure," *Journal of Exposure Science and Environmental Epidemiology* 27:535-538, 2017.

Alexandra E. Larsen, Brian J. Reich, Mark Ruminski, Ana G. Rappold, "Impacts of fire smoke plumes on regional air quality, 2006–2013." *Journal of Exposure Science & Environmental Epidemiology*, 2017; DOI: 10.1038/s41370-017-0013-x. Summary in *Science Daily*: https://www.sciencedaily.com/releases/2018/01/180109112415.htm.

J.M. Antó, J. Sunyer, R. Rodriguez-Roisin, M. Suarez-Cervera, and L. Vazquez, "Community outbreaks of asthma associated with inhalation of soybean dust," *New England Journal of Medicine*, 320(17):1097-1102, 1989.