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High-Priority Compounds Associated with Aircraft Emissions

PARTNER Project 11 final report on subtask:
Health Risk Prioritization of Aircraft Emissions
Related Air Pollutants

prepared by

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High-Priority Compounds Associated with Aircraft Emissions
PARTNER Project 11

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Background and Study Framing

There are numerous criteria pollutants and air toxics emitted by aircrafts and other airport sources, but resources are limited for site-specific characterization, and many of these compounds may not contribute appreciably to population risk. By estimating the approximate magnitude of population risk associated with each compound under study, we can screen out those compounds that do not require further attention, and can therefore focus future resources on the primary risk drivers. Further, by approximating (at least qualitatively) the magnitude of uncertainty associated with these risk estimates, we can make recommendations for high-priority research activities in future years. In other words, a compound with a relatively high health risk but low uncertainties would be important to characterize but may not require further basic research on toxicity and/or exposure, while a compound with a strong probability of high health risks and large uncertainties may require more research.

A few key concepts are critical in framing our analyses and interpreting our findings. First, risk-based prioritization must include three components – emissions, the emissions-to-exposure relationship (including pollutant fate and transport and population patterns), and the toxicity of the compound. Previous prioritization efforts at airports have often been based on either emissions alone or emissions and toxicity, omitting the important influence of fate and transport on population exposure and health risk. While some pollutants are inert and would have similar exposures given the same amount of emissions, others are reactive in the atmosphere (both being formed and depleted over time), and pollutants in the particle phase will have different removal processes and subsequent spatial patterns of exposure. These processes will differ across pollutants and across airport settings. We explicitly consider in this analysis whether ignoring exposure or ignoring toxicity would be consequential from a prioritization perspective, while noting that either approach is theoretically unsupportable.

Second, within the context of this analysis, we primarily focus on total population health risks, rather than considering (for example) the maximum individual health risk found within the population. From a public health perspective, prioritization based on the total risk to the exposed population is a more conventional approach, as this will directly inform benefit-cost analyses and other utilitarian approaches for resource prioritization. The implication is that the exposure aggregated across the population is the relevant measure to consider, as opposed to the peak

exposure found near the fenceline of the airport. While local maximum exposures are clearly important for many applications, our focus herein is on total population exposures, which will have multiple implications for our methods and findings.

Third, given a population risk perspective, there are questions about the ideal spatial domain and resolution for atmospheric dispersion modeling. Although the largest domain and finest resolution would be desired in principle, there are logistical and computational constraints, and it is therefore important to know over what spatial domain most of the population exposure occurs, and whether this conclusion depends on the resolution of the model. This will have multiple implications for how the dispersion model is run, including whether it is viable to simulate the impacts of multiple airports simultaneously. Given the issues mentioned above, the optimal spatial domain and resolution may differ across pollutants as well as across airports.

Finally, given differences in relative emission rates, background concentrations, population patterns, and meteorology, it is possible for the ranking of high-priority compounds to differ across airports. While it is unlikely that the rankings will differ substantially, it is important to consider airports in different parts of the country to determine the robustness of our risk prioritization rankings, and to ensure that important pollutants are not omitted from future investigations.

While emissions and toxicity can be readily described by single values, allowing for quick comparisons across compounds, the atmospheric fate and transport of a pollutant is difficult to summarize in a format that is readily interpretable from a health risk perspective. For comparative purposes and to facilitate extrapolation of dispersion modeling outputs to unstudied settings, researchers have developed the concept of an intake fraction, defined as a unitless measure characterizing the total population exposure to a compound per unit emissions of that compound or its precursor [1]. In spite of its definitional simplicity, it allows for detailed exposure data from previous dispersion modeling or monitoring studies to be quickly incorporated into risk assessments for the purpose of prioritization and future model refinement. In the event that there are no non-linearities in the concentration-response function throughout the range of background exposures, the product of emissions and intake fraction will be linearly proportional to health risk. As described in more detail below, the calculation of intake fractions from complex atmospheric dispersion models allows for enhanced interpretability of our findings with respect to criteria pollutant impacts and cancer effects from air toxics, but does not reasonably inform non-cancer effects from air toxics, where population thresholds are effectively presumed and the linearity assumption does not hold.

In this study, we consider emissions from aircraft and other airport-related sources from three airports in the United States – T.F. Green Airport (Rhode Island), Chicago O'Hare International Airport (Illinois), and Hartsfield-Atlanta International Airport (Georgia). These

three airports were selected based on a prioritization scheme that included the number of aircraft per day, total estimated emissions, fraction of county emissions, whether the climate is conducive to high impacts, and the size of the population within various radii of the airport grounds, as well as logistical considerations (i.e., existence of monitoring data, availability of meteorological data suitable for atmospheric dispersion modeling). The purpose of our selection exercise was to ensure that the airports selected would have a significant enough impact to be detected in modeling and/or monitoring activities in the future, as well as to inform the design of such activities. In particular, O'Hare and Hartsfield-Atlanta were selected based on their size and likely magnitude of impact, while T.F. Green was meant to be representative of meteorology in the Northeast and was selected given the existence of extensive current and planned monitoring data.

As described in more detail below, for each of the three airports, we utilize results of different atmospheric dispersion models with different spatial resolution, and we calculate health risks for a number of air toxics and criteria pollutants. We focus our analyses on the degree to which conclusions about intake fraction and health risk depend on the dispersion modeling assumptions, as well as on the high-priority compounds across airports and dispersion models. We additionally consider the magnitude of various uncertainties and the degree to which they may influence the risk ranking across compounds.

Characterization of Emissions and Exposures

As mentioned above, risk-based prioritization is based on the combination of emissions, the emissions-to-exposure relationship, and toxicity. Within this study, monthly emissions of a suite of pollutants were provided by CSSI under the auspices of PARTNER. Emissions were estimated using a research version of the EDMS model, and more information about the analytical approach utilized by CSSI for emissions characterization is available at http://www.faa.gov/about/office_org/headquarters_offices/aep/models/edms_model/.

An initial decision was required about which pollutants should be modeled, dictated both by logistical considerations and by prior evidence regarding exposure and/or toxicity. The candidate list of compounds included criteria pollutants (CO, VOCs, NOx, SOx, and various PM constituents) and multiple air toxics - formaldehyde, acetaldehyde, benzene, toluene, acrolein, 1,3-butadiene, xylene, naphthalene, propionaldehyde, ethylbenzene, styrene, and a suite of PAHs, including phenanthrene, fluorene, fluoranthene, pyrene, anthracene, acenaphthene, acenaphthylene, benzo[g,h,i]perylene, benzo[b]fluoranthene, benzo[k]fluoranthene, benz[a]anthracene, benzo[a]pyrene, chrysene, and indeno[1,2,3-c,d]pyrene. This list of PAHs includes those generally considered to contribute most to health effects, given their toxicity and levels of exposure. Any compound not on this initial list could not be evaluated formally within

our prioritization analysis, so our efforts focused on the relative priorities among these listed compounds. Previous risk assessments have indicated that the air toxics we included contribute the vast majority of inhalation cancer risks in the United States [2].

To characterize exposures, atmospheric dispersion modeling was conducted by researchers from University of North Carolina under Project 16 of PARTNER using two different models – AERMOD and CMAQ. AERMOD is a near-source dispersion model that can capture impacts at high spatial resolution, but is generally applied at a maximum distance of 50 km from the source and with limited ability to capture chemical reactions (all compounds are effectively assumed to be inert or with a fixed first-order reaction rate). On the other hand, CMAQ includes detailed chemical reactions given the modeling of all sources of emissions, capturing a baseline scenario with no airports and a modified scenario with emissions from specific airports added back in (in this case, T.F. Green, O'Hare, and Hartsfield-Atlanta). However, the model is less spatially resolved – the two sets of model runs available at the time this report was completed utilized 36 x 36 km and 12 x 12 km resolution. Thus, each model has its strengths and limitations with respect to characterizing population risk, and we consider all three (AERMOD, CMAQ with 36 km resolution, CMAQ with 12 km resolution) to determine the robustness of our findings and the potential importance of long-range modeling, high-resolution modeling, and characterizing chemical reactions for future risk assessments. Of note, unlike some of the other model components, this allows us to preliminarily estimate the magnitude of uncertainties associated with the emissions-to-exposure component of the model.

It should be noted that different emissions inputs were used within AERMOD and CMAQ, corresponding to emissions only up to 3,000 feet within AERMOD but up to 10,000 feet within CMAQ. Thus, the outputs would not be anticipated to be identical regardless of dispersion model structure, but we still compare the outputs to get a quantitative sense of the impact of all sources of uncertainty on our health risk estimates. As described in more detail below, the difference in the emissions inventory is relatively minimal for air toxics but somewhat larger for criteria pollutants. We provide some bounding calculations to isolate the dispersion model differences, as emissions at high altitude would have a smaller influence on ground-level concentrations than ground-level emissions, but would certainly have a non-zero influence.

Dispersion modeling outputs were largely characterized in our analysis using intake fractions, to help to elucidate the key differences between compounds in the emissions-to-exposure relationship. The quantitative definition of an intake fraction is

$$iF_j = \Sigma_i(P_i \quad \Delta C_{ij})*BR/Q_j$$

where iF_j is the intake fraction for pollutant j, P_i refers to the population contained in geographic area i, ΔC_{ij} (in $\mu g/m^3$) is the change in ambient concentration at geographic area i related to

emissions Q_j, and BR is a nominal population breathing rate (assumed to be 20 m^3/day in this analysis). Of note, the breathing rate is divided back out in the risk calculation, so this assumption has no impact on the results other than to ensure that intake fractions are unitless measures.

In addition, it should be noted that the pollutant emitted is not necessarily the same as the concentration estimated – for example, intake fractions have been calculated for secondary ammonium sulfate formation associated with sulfur dioxide emissions [3-5]. We consider secondary pollution formation to a limited extent within this report, although certain secondary pollutants (such as ozone) cannot be easily captured within the intake fraction framework given the significant contribution of multiple sources of emissions (in the case of ozone, NOx and VOCs). For ozone, we calculate risk and consider population-weighted concentrations but do not formally estimate intake fractions. In addition, estimation of particulate matter intake fractions is complicated by the primary and secondary contributions to $PM_{2.5}$ concentrations; while these can be separated in principle, the inputs and outputs available do not allow for this to be done within our report. We approximate primary particulate matter intake fractions using the incremental total PM concentration and total PM emissions, but note that this will be an overestimate for the CMAQ outputs. Regardless, this will help to illustrate the magnitude of population exposure and therefore health risk that occurs at various distances from the airports, a conclusion that will not be affected by the computational aspects of intake fraction values.

For AERMOD outputs, receptors were placed at all census tracts (from 2000 Census data) within 50 km of the airport centroid, and exposures resulting from AERMOD runs were assigned to these populations. For CMAQ, calculation of intake fractions was complicated slightly by the spatial disconnect between census boundaries and a fixed-distance grid. To estimate populations of the 36 x 36 km or 12 x 12 km grid cells, we used ArcGIS to create a file geodatabase feature class from provided center point coordinates, and projected this class to an Albers projection. Thiessen polygons were created from projected points, and these were intersected with year 2000 census tracts to estimate populations by grid cell for all cells east of the Mississippi River (given our focus on airports in the eastern half of the United States).

An additional complication of the CMAQ output is the fact that the runs simultaneously added all three airports, making it more difficult to extract the effect of individual airports. As the three airports in question are relatively far apart, this is not likely a significant uncertainty, although issues could be greater for a small airport (i.e., T.F. Green) that is downwind from much larger airports (i.e., Hartsfield-Atlanta). To better understand both the spatial extent of impacts (i.e., how far out the dispersion modeling must extend to capture the majority of the population exposure) and the degree to which model outputs are able to separate the impacts of the individual airports, we quantified intake fractions and health risks at various radii from each airport. We

initially focused on the grid cell in which the airport was located, and then sequentially added other grid cells. By comparing the outputs with the immediately prior intake fractions as well as with the national-scale intake fractions (averaging all three airports), we were able to determine the spatial domain over which most of the intake fraction occurred. We focus many of the results presented below on smaller spatial domains, which should contain relatively minimal contamination from other airports and should provide reasonable rank-ordering across compounds, and we consider the possible downward bias associated with this limited domain within our analysis.

These analytical steps provided the necessary information for criteria pollutant risk calculations and cancer effects from air toxics, but non-cancer risk assessment is conducted differently, as described in more detail below. Rather than estimating intake fractions and quantifying risks, we simply compare the ambient concentration with defined reference concentrations, to determine if the marginal contribution of the airport is likely to contribute appreciably to population risks. We characterize background concentrations both with CMAQ outputs and with monitoring and modeling data used in EPA's National Air Toxics Assessment (http://www.epa.gov/ttn/atw/nata1999/).

Toxicity Information

For this analysis, we are considering two categories of compounds – criteria air pollutants and air toxics. As mentioned above, these categories are generally handled differently in a health risk assessment framework. For criteria air pollutants, concentration-response functions are generally derived, assessing the relationship between changes in ambient concentrations and changes in health outcomes throughout the range of observed concentrations. In the standard risk assessment paradigm, air toxics are treated differently, depending on whether the endpoint of interest involves cancer or other diseases. For cancer risk assessment, in most (but not all) cases, the focus is on deriving a potency per unit exposure, under the presumption of low-dose linearity and no population thresholds. For non-cancer risk assessment, the current approach for inhalation involves comparing estimated exposure levels to reference concentrations (RfC), based on the assumption that a population threshold level exists with no appreciable risk if the exposure level is below that threshold.

This framework has undergone recent scrutiny for both cancer and non-cancer endpoints. Some investigators argue that the uncertainties involved at low doses for non-cancer effects are no greater than those used in cancer risk assessment, and that linear extrapolation of risks to low doses for non-cancer effects could still be very informative for regulatory purposes [6, 7]. While these arguments have merit and may influence the long-term structure of cancer and non-cancer risk assessment, for the purpose of this initial prioritization analysis, we follow the conventional paradigm as generally applied by EPA and most risk assessors at present.

In addition, we note that there is often substantial uncertainty associated with potency values for either cancer or non-cancer endpoints. For the example of cancer, significant uncertainties for extrapolated cancer risk estimates include accuracy of the exposure estimates, the appropriateness of the dose metric for dose-response analysis, addressing issues of model selection and potential modifying or confounding factors for epidemiologic data, and dealing with animal-human extrapolation issues for toxicologic data. In this investigation, we do not engage in a formal uncertainty analysis, but do use our discussion about the evidence base for each compound as a qualitative means of characterizing uncertainty. Compounds for which the evidence base is more robust and with a relatively high risk ranking should be considered as higher priority compounds for future risk assessments, while compounds for which the evidence base is weaker but risk estimates are high may require further basic research.

In the following sections, we describe the evidence base for health effects from selected criteria pollutants and air toxics. For criteria pollutants, given modeling capabilities and the findings of previous regulatory impact analyses [8-10], we focus on fine particulate matter ($PM_{2.5}$) and ozone. While pollutants such as NO_2 may also exhibit direct health effects (beyond the effects of NOx emissions on secondary PM and ozone formation), the literature is insufficient to develop a suite of concentration-response functions, as shown in regulatory impact analyses conducted by EPA [9, 10]. For air toxics, we do not formally consider the full suite of air toxics under the purview of US EPA, but instead consider a list of air toxics described above, which were selected based on their likelihood of being emitted at significant levels at airports and their potential for human toxicity. This therefore represents an initial qualitative screening analysis, in which excluded compounds are presumed to be of significantly lower risk than the included compounds.

Particulate matter

For $PM_{2.5}$, we can consider both mortality and morbidity endpoints. Since the primary aim of this calculation is to determine the magnitude of the PM-related impacts in comparison with the magnitude of the ozone- and air toxics-related impacts, we focus primarily on premature mortality (to compare with cancer risk calculations), but also describe the evidence base for selected morbidity endpoints for general comparison and to emphasize the robustness of the literature.

For premature mortality due to long-term $PM_{2.5}$ exposure, Pope and Dockery [11] recently reviewed and documented the available estimates of concentration-response functions. Out of the studies presented in their review, the Harvard Six Cities study and the American Cancer Society (ACS) study provide the most applicable estimates, given their focus on general populations, aggregate measures of fine particulate matter, and extensive peer review and re-analysis. Both of

these studies were long-term cohort studies, in which a population was enrolled and followed for a number of years to determine the association between mortality risks and air pollution. The Harvard Six Cities study was based in six cities in the eastern US (Watertown, MA; Kingston and Harriman, TN; St. Louis, MO; Steubenville, OH; Portage, Wyocena, and Pardeeville, WI; and Topeka, KS), while the ACS study was based in all 50 states, the District of Columbia, and Puerto Rico. The Harvard Six Cities study yields central estimates on the order of a 1.3% to 1.6% increase in premature mortality per $\mu g/m^3$ increase of long-term $PM_{2.5}$, with the most recent publication [12] yielding a value of 1.6% (95% CI: 0.7%, 2.6%). The ACS study generally yields somewhat lower central estimates, on the order of 0.6% or 0.7%, with the most recent publication [13] yielding a value of 0.6% (95% CI: 0.2%, 1.1%).

The lower value in the ACS study has been attributed to multiple factors, including the fact that the population was generally of higher socioeconomic status and education than in the Harvard Six Cities study (and than in the US population at large), as well as the fact that air pollution data were gathered retrospectively from central site monitors rather than prospectively in community-specific monitors. This latter fact would tend to increase exposure misclassification and result in downwardly biased estimates. This was illustrated in a recent publication [14], in which more refined exposure estimates were derived for the ACS cohort using geographic information systems (GIS), resulting in a central estimate of 1.7%, nearly triple the original estimate.

Thus, the central estimates from the relevant literature range from 0.6% to 1.7% increases in mortality per $\mu g/m^3$ of long-term $PM_{2.5}$, with the lower values likely biased downward due to exposure misclassification in the ACS study. We would therefore consider a value of a 1% increase in mortality per $\mu g/m^3$ of long-term $PM_{2.5}$ to reasonably represent the current knowledge base, and use this value as our central estimate. Of note, a recent expert elicitation study [15] captured the opinions of 12 experts in the field regarding the appropriate concentration-response function for $PM_{2.5}$ mortality. Across the 12 experts, the median concentration response functions (presented as % decrease in mortality per $\mu g/m^3$ decrease in $PM_{2.5}$) were: 1.6%, 1.2%, 1.2%, 0.9%, 2.0%, 1.1% (above 7 $\mu g/m^3$; 0.9% below 7 $\mu g/m^3$), 1.0%, 0.7%, 1.3%, 0.9%, 0.4% (below 16 $\mu g/m^3$; 0.7% above 16 $\mu g/m^3$), and 1.0%. The functions for which no breakpoint is described were determined to be applicable at all ambient concentration levels. Although formally combining these opinions is not well justified, a simple average of these values yields an estimate of 1%, with a median value of 1.05%. Our best estimate of 1% is therefore well supported by the expert judgments provided in a formal elicitation protocol. We consider the values listed above (0.6% and 1.7%) as reasonable lower and upper bounds, although this does not constitute the full extent of uncertainty and is not formally propagated through our analysis. The medians of the experts' 5^{th} and 95^{th} percentiles would yield slightly larger uncertainty

bounds (approximately 0.3% and 2.0%). All-cause mortality rates by age group by county reported in CDC Wonder were used to provide baseline incidence data, necessary to translate the relative risks from the epidemiological studies into absolute population risks.

As stated above, we do not formally quantify morbidity endpoints associated with $PM_{2.5}$ in this report given the emphasis of our analysis. Given the way that non-cancer risk assessment for air toxics is generally conducted, there is no way to compare the magnitude of non-cancer risks either between individual air toxics or between air toxics in aggregate and particulate matter. The ratio between the concentration and RfC provides a qualitative determination of the likelihood of appreciable population risk, but is not a quantitative value that can be translated into population risks. Below, we provide concentration-response functions for hospital admissions (respiratory and cardiovascular), to give general insight about the magnitude of impacts for a selected morbidity outcome, but do not include the full range of morbidity endpoints or quantify these endpoints in the Results section given the focus of the analysis.

For respiratory hospital admissions (RHA), a concentration-response function can be derived from an inverse-variance weighted meta-analysis of the published literature. From the large number of studies available, we eliminated a subset of studies that could not be statistically pooled with other studies for a variety of reasons. This included the application of statistical methods that were not comparable with other studies, use of a pollutant measure other than $PM_{2.5}$ (i.e., only considering acid aerosols or black smoke), consideration of specific respiratory diseases rather than all-cause respiratory hospital admissions, or evaluation of effects on children only. Using the remaining studies [16-26], the central estimate from this meta-analysis is a 0.2% increase in RHA per $\mu g/m^3$ of $PM_{2.5}$, with a 95% confidence interval of (0.14%, 0.29%).

An estimate of cardiovascular hospital admissions (CHA) associated with $PM_{2.5}$ exposures can be drawn from a recent meta-analysis [27], which reported a 0.9% increase in CHA per 10 $\mu g/m^3$ of PM_{10} (95% CI: 0.7%, 1.0%). As the literature in this case is sufficient to rely solely on estimates from the US, we re-ran the meta-analysis with the restricted set. After converting to $PM_{2.5}$, the result is a 0.16% increase in CHA per $\mu g/m^3$ of $PM_{2.5}$ (95% CI: 0.14%, 0.19%). The literature on both respiratory and cardiovascular hospital admissions is therefore robust and supports the notion that the cardiopulmonary deaths found in the cohort studies are biologically plausible and coherent with other existing evidence.

Ozone

As with $PM_{2.5}$, there are both morbidity and mortality effects associated with ozone exposures at current ambient concentrations. For ozone, however, the evidence base indicates that short-term exposures are associated with premature mortality, with no strong evidence (i.e., from the Six Cities or American Cancer Society studies) of a long-term effect. We develop the

ozone mortality concentration-response function from a recent publication that synthesized the relevant epidemiological literature [28]. This estimate was based on the findings in multiple meta-analyses of the epidemiological literature as well as multi-city studies [29-31], providing the most stable and statistically robust estimates.

The three meta-analyses yielded very similar findings, with central estimates of 0.41%, 0.39%, and 0.34% increases in mortality per 10 ppb increase in 1-hour maximum ozone concentrations, respectively. Slightly lower values were seen in the National Morbidity and Mortality Air Pollution Study (NMMAPS) [31], potentially attributable to the methodology used in NMMAPS to control for weather and other factors, and slightly higher values were seen in studies looking only at summertime conditions [32]. From this literature, it is clear that a central estimate of a 0.4% increase in mortality per 10 ppb increase in 1-hour maximum ozone is well supported, although with some uncertainty related to geographic variability in impacts and personal exposure-ambient concentration relationships. As the outputs from the CMAQ model represent daily average concentrations in $\mu g/m^3$, we approximate this concentration-response function using standard ratios between 1-hour and 24-hour concentrations and converting units, resulting in an approximate 0.4% increase in mortality per 10 $\mu g/m^3$ increase in 24-hour average ozone.

Multiple morbidity endpoints, ranging in severity from hospital admissions to days with minor restricted activities, have also been associated with ozone, but these are not formally quantified within this report given our emphasis on comparisons across compounds.

Air toxics

As described above, we consider a suite of air toxics within our risk-based prioritization calculations. For each pollutant, we provide general background information, discuss the evidence for carcinogenicity and the corresponding potency factor, and discuss the evidence for non-cancer health effects and the corresponding reference concentrations. In each case, we consider the possible magnitude of uncertainty associated with the estimate, and we describe the strength of the underlying evidence.

Benzene

Benzene, also known as benzol, is an organic chemical compound with the formula C_6H_6. It is a colorless and flammable liquid with a sweet smell and a relatively high melting point, and is a natural constituent of crude oil.

Benzene is one of the few "known" human carcinogens (category A according to IRIS), based on convincing epidemiological evidence as well as supporting evidence from animal bioassays [33]. Benzene exposure has been associated with leukemia as well as other neoplastic

conditions. Within EPA's IRIS database, the inhalation unit risk of benzene was reported as a range, with values between 2.2×10^{-6} and 7.8×10^{-6} for lifetime exposure to 1 $\mu g/m^3$ benzene in air. The uncertainty related to the potency of benzene is clearly greater than this range, which reflects some dimensions of uncertainty but not others (i.e., the possibility of sublinear exposure-response models is not considered). In addition, the fact that a range is presented for benzene but not most other air toxics is not an indication that there is greater uncertainty for benzene, but rather that the IRIS entry for benzene has been updated more recently. Regardless, the cancer effects of benzene have been relatively well characterized in both epidemiological and toxicological studies. Given the uncertainties, EPA has used the average of the range of values (5×10^{-6}) for their potency estimate in regulatory decision-making. The California Office of Environmental Health Hazard Assessment (OEHHA) has used a somewhat higher potency value of 2.9×10^{-5}, reflective of a different interpretation of the literature (i.e., the use of the 95th percentile value from the epidemiological study deemed most credible by the US EPA) [34]. For the purpose of this assessment, we utilize the center of the range of values reported by EPA, but acknowledge that the magnitude could differ somewhat in either direction.

In particular, the critical question is whether risk estimates drawn from occupational exposure epidemiological studies can be translated to general population risks. As the exposure levels are higher than general environmental exposures, the cancer risk estimates are assuming linear dose-response functions outside of the range of observed data (paralleling assumptions generally made when extrapolating from animal studies to human populations). Although there is no evidence suggesting that the dose-response functions at both low level exposure and high level exposure are not linear, there is no significant supportive evidence either.

However, more recent studies have demonstrated effects of lower levels of exposure, not on cancer but on intermediate effects at the cellular and molecular level. For example, low level benzene exposure (from urban traffic or gas stations) decreases the methylated cytosine percentage (Cm%) of long interspersed nuclear element-1 and significantly increases the P15 Cm%. Acute myelogenous leukemia tissue shows the same features [35]. Increased DNA adducts have also been reported, also suggesting that these relationships are linear at lower benzene exposures [36]. Similarly, a study found that low level benzene exposures are related to decreased DNA-repair mechanisms [37]. This may contribute to the incidence of cancer by lowering cellular defenses against other DNA targeted toxicants. The hematotoxicity of benzene has been confirmed in other studies [38], suggesting that low level benzene exposure may still affect genetically susceptible subpopulations. While these recent studies do not allow for formally updated potency estimates, they provide a refined understanding of the plausibility of relationships between benzene and health outcomes for low-level environmental exposures.

Turning to non-cancer endpoints, the RfC is similarly derived from occupational

epidemiology [39], with a value of 3 x 10^{-2} mg/m^3 based on a critical effect of decreased lymphocyte counts [33]. Following current practice, this is based on the derivation of a lower confidence limit on the benchmark concentration, with the application of uncertainty factors totaling 300 (representing human heterogeneity, subchronic-to-chronic extrapolation, effect-level extrapolation, and database deficiencies). As for the cancer endpoints, there is extensive supporting evidence of the non-cancer effects of benzene exposure. Total white blood cell (WBC) count, ALC, hematocrit, red blood cell (RBC) count, platelet count, and mean corpuscular volume (MCV) are all significantly related to benzene exposure [40-42]. Chronic exposure to benzene results in progressive deterioration in hematopoietic function. Anemia, leukopenia, lymphocytopenia, thrombocytopenia, pancytopenia, and aplastic anemia have been reported after chronic benzene exposure [33, 43, 44].

In summary, the evidence base for both cancer and non-cancer effects of benzene is strong, and is among the most robust evidence bases for any air toxicant. The inhalation unit risk and RfC are both based on occupational epidemiology, and both use the best methods currently available for estimation within the EPA paradigm (i.e., use of the benchmark concentration rather than a no observed adverse effects level to estimate the reference concentration). We therefore consider the values reported above to have relatively less uncertainty than some of the estimates for other pollutants.

1,3-butadiene

1,3-butadiene is a simple conjugated diene, both flammable and irritative. EPA has assessed both its cancer and non-cancer effects within the IRIS system.

U.S. EPA considers 1,3-butadiene to be carcinogenic to humans, based on sufficient evidence from occupational epidemiological studies. While it was formerly classified as a probable human carcinogen, more recent epidemiologic evidence led to the determination that 1,3-butaidene was a known human carcinogen [45]. Studies show an increase in lymphohematopoietic cancers and a dose-response relationship for leukemia in polymer workers, along with sufficient evidence from animal studies and other experiments. Although studies have found excess mortality from lymphosarcoma, the evidence for 1,3-butadiene causing leukemia is still relatively limited.

Thus, while the original inhalation unit risk for 1,3-butadiene was based on toxicological evidence, the most recent value in IRIS is based on epidemiological evidence. This estimate of 3 x 10^{-5} for lifetime exposure to 1 μg/m^3 in air is therefore based on stronger and more directly applicable evidence, but is an order of magnitude lower than the value previously derived from animal bioassays [45]. Of note, the CA OEHHA utilizes an inhalation unit risk value of 1.7 x 10^{-4} [34], based on the determination that the occupational epidemiology remains inadequate for

quantitative risk assessment, and the subsequent utilization of results from rodent bioassays.

More generally, concerns about the strength of the epidemiological evidence have been raised, in spite of the fact that these effects have been seen in numerous epidemiological studies [46-53]. For example, one study argued that the effect of 1,3-butadiene alone contributing to leukemia is marginal and statistically insignificant unless the dose is above 362 ppm [54]. Interestingly, within this study, the effect of 1,3-butadiene increased and became statistically significant only for individuals with high styrene exposures, and the relative risk for leukemia increased along with the styrene exposure level only in the category with high 1,3-butadiene exposure. It is hard to isolate the effect of 1,3-butadiene and styrene, because in occupational exposures, these are highly correlated. While this contributes uncertainty to the quantitative estimate assigned to 1,3-butadiene, EPA stated that linearity for low-dose extrapolation is reasonable given the clear evidence of genotoxicity by 1,3-butadiene metabolites [45].

Other studies suggest that incorporating more recent exposure estimates for the occupational epidemiological studies will results in a 2.5-fold decrease in estimates of leukemia risks computed by EPA, and a 13-fold decrease when updated epidemiologic data and alternative numbers proposed by EPA's Science Advisory Board are incorporated [55, 56]. Regardless, it appears that the inhalation unit risk currently used for 1,3-butadiene in IRIS represents a reasonable value for prioritization, given somewhat higher values when animal bioassay results are used and somewhat lower values given some proposed adjustments to the epidemiological evidence, but that uncertainties may be substantial.

For non-cancer risk estimates, only an RfC is calculated because 1,3-butadiene is a volatile gas and causes hazard by inhalation only. The current RfC (0.9 ppb, 2×10^{-3} mg/m^3) is developed from a 2-year high quality bioassay using ovarian atrophy as the critical effect [45]. As for benzene, this estimate was updated relatively recently and utilized benchmark dose procedures, providing a more robust and interpretable value (albeit with the necessary caveats about the RfC process in general). The RfC estimation is in line with a number of animal studies, in which a variety of reproductive and developmental effects have been observed [57-60]. EPA currently considers that the dominant effects in humans caused by 1,3-butadiene would likely be manifested as infertility (due to reduced fertility or very early deaths) or spontaneous abortions[45].

Acetaldehyde

Acetaldehyde, sometimes known as ethanal, is an organic chemical compound with the formula CH_3CHO or MeCHO. It is a flammable liquid with a fruity smell. Unlike the previous two air toxics, acetaldehyde is not a known human carcinogen according to the EPA classification system, given inadequate epidemiological evidence [61]. The only epidemiological study under consideration within IRIS showed an increased crude incidence rate of total cancer

in acetaldehyde production workers as compared with the general population [62], but the study had several methodological limitations (unadjusted for age or other exposure sources, with a lack of information on subject selection).

However, acetaldehyde is considered a probable human carcinogen (US EPA category B2, IARC category 2B) [61]. This assessment was based on increased nasal tumors found in male and female rats and laryngeal tumors in male and female hamsters after inhalation exposure. Other studies showed significantly increased incidence of laryngeal tumors in hamsters [63, 64] or elevated squamous cell carcinomas in rats [65, 66].

According to IRIS, the inhalation unit risk for acetaldehyde is 2.2×10^{-6} per $\mu g/m^3$, based on a linearized multistage model and using "extra risk", a conventional calculation approach [61]. The estimate from CA OEHHA is comparable, with a value of 2.7×10^{-6} per $\mu g/m^3$ [34]. Differences between the two estimates are based largely on minor differences in scaling and other steps in the potency calculation, and the values should be considered effectively identical. No alternative values have been put forth by either agency, but this does not imply that the uncertainty for acetaldehyde is less than that for benzene and 1,3-butadiene – it is simply an indication that the latter two compounds have been more intensively studied.

For non-cancer effects, the current RfC in IRIS is 9×10^{-3} mg/m^3, based on degeneration of the olfactory epithelium in short-term rat studies [61]. An uncertainty factor of 1000 was applied to the bioassay results, given the need to account for human heterogeneity, extrapolation from subchronic to chronic effects, interspecies extrapolation, and incompleteness of the dataset. The IRIS database characterizes confidence in this RfC value as low (versus medium for benzene and 1,3-butadiene), indicating that the value and corresponding health risk estimates should be considered more uncertain. This uncertainty is driven in large part by the limited human studies available, along with concerns that acetaldehyde's binding in the respiratory tract and rapid metabolism significantly reduces systemic circulation at steady state [67].

Formaldehyde

The chemical compound formaldehyde (also known as methanal) is a gas with a pungent smell, with the chemical formula H_2CO. Formaldehyde readily results from the incomplete combustion of carbon-containing materials, as well as from off-gassing from particle board and other building materials.

Formaldehyde is considered by IRIS to be a probable human carcinogen (IRIS category B1), based on limited evidence in humans and sufficient evidence in animals [68]. This evidence indicates that respiratory neoplasms are seen among humans exposed to formaldehyde either occupationally or residentially, with the animal evidence finding nasal squamous cell carcinomas supporting these findings. EPA reports an inhalation unit risk of 1.3×10^{-5} per $\mu g/m^3$ [68], based on

rodent bioassays. CA OEHHA estimates a lower unit risk of 6 x 10^{-6} per $\mu g/m^3$, based on the same bioassay but using slightly different estimation procedures [34].

Although the epidemiological evidence is not considered sufficient for determination of cancer potency, at least 28 relevant epidemiologic studies have been conducted. Two cohort studies [69-71] and one case-control study [72, 73] were well-conducted and specifically designed to detect small to moderate increases in formaldehyde-associated human risks [68].There are 25 other related studies - 6 showed significant results for respiratory tract cancers, and 19 indicate that leukemia and neoplasms of the brain and colon may be associated with formaldehyde exposure. However, these studies were limited primarily because of possible simultaneous exposure to other agents, as well as small sample sizes and insufficient follow-up periods [68].

Uncertainties regarding the cancer potency of formaldehyde are somewhat greater than indicated by the range of values between EPA and OEHHA, given information that addresses how formaldehyde moves through the body. For example, some laboratory studies have indicated that formaldehyde levels are not increased in the blood of the metabolized DNA protein cross-links in exposed rats [74]. Formaldehyde is also highly water soluble and may be most plausibly associated with toxicity in the nasal mucosa and proximal trachea. Within the most recent National Air Toxics Assessment, EPA chose a much lower inhalation unit risk of 5.5 x 10^{-9} per $\mu g/m^3$, based on the evidence described above and other recent mechanistic and dosimetric information. Formaldehyde remains under review at EPA within the IRIS system, and it is not clear the value that will be adopted. Regardless, it is clear that uncertainty related to formaldehyde toxicity at low doses is substantial (3-4 orders of magnitude), and that the relative priority placed on formaldehyde would likely depend greatly on the value chosen. For this analysis, we use the current IRIS value, but our prioritization conclusions would clearly change if the value used in the most recent National Air Toxics Assessment were in fact correct.

Turning to non-cancer effects, the IRIS system has no RfC value for inhalation risks from formaldehyde. ATSDR determined a chronic RfC of 9.8 $\mu g/m^3$ for respiratory effects, which was the value used by EPA in the most recent National Air Toxics Assessment, and which we use going forward in this assessment.

Acrolein

Acrolein, also known as propenal, is the simplest unsaturated aldehyde. There is currently inadequate evidence to determine human carcinogenic potential for acrolein, given limited epidemiological evidence and the fact that animal studies do not provide adequate evidence that acrolein causes cancer in laboratory animals [75]. One possible reason is that acrolein is highly reactive and may not reach potential target sites at a sufficient concentration to initiate a carcinogenic response. This reactivity adds to the uncertainty in exposure assessment for acrolein

as well, as it is difficult to accurately measure acrolein concentrations with many conventional monitoring instruments.

However, non-cancer effects of acrolein are potentially significant. The current RfC developed by US EPA is 2 x 10^{-5} mg/m^3 [75], based on a subchronic rat inhalation study finding nasal lesions [76]. This value was updated in 2003 using more refined methods for development of uncertainty factors (including separate assessment of pharmacokinetic and pharmacodynamic components of interspecies extrapolation, as well as standard factors for human heterogeneity, adjustment from subchronic to chronic, and use of a LOAEL rather than a NOAEL), but the identical RfC value was estimated.

While the RfC is a helpful value for prioritization purposes (and is used in this study for comparability with other air toxics with non-cancer effects), more recent investigations have used available toxicological evidence to quantify the effect of acrolein exposure on defined health outcomes. Woodruff and colleagues used data collected by Costa et al. [7, 77] along with ambient acrolein concentration data from the 1999 National Air Toxics Assessment [78], and used lung function data to translate directly into human health effects. This provides a useful estimate of the potential number of cases (not generally available through standard non-cancer methods), and also helps determine what the possible magnitude of adverse effect might be at different levels of exposure. For example, using a linear model derived from benchmark dose methods, Woodruff et al. estimated a 0.53% decrease in specific compliance (a marker for altered lung function) for a 0.077 μg/m^3 increase in acrolein concentrations (change from the median to a no-exposure scenario). In principle, such a model could be used, along with a functional definition of the degree of lung function impairment that would be deemed adverse, to quantify the non-cancer effects of acrolein exposure. As mentioned above, we do not conduct this form of non-cancer risk assessment for acrolein in this report, given the difficulty of constructing comparable analyses for other compounds as well as the complexity of linking altered lung function with defined health outcomes, but this approach provides guidance on potential future directions for research.

Xylenes

The term "xylenes" refers to a group of 3 benzene derivatives which encompasses ortho-, meta-, and para- isomers of dimethyl benzene. The o-, m- and p- isomers specify to which carbon atoms (of the main benzene ring) the two methyl groups are attached.

Adequate human data on the carcinogenicity of xylenes are not available, and the available animal data are inconclusive as to the ability of xylenes to cause a carcinogenic response [79]. The occupational studies all had potential issues with co-exposures to other chemicals, which may confound the results, although there is a suggestion of elevated non-Hodgkin lymphoma in the

medium/high xylene exposure group [80]. Animal data on the carcinogenicity of xylenes are not available for inhalation, and there is only limited evidence for oral exposure, with generally negative findings and limitations with the studies that found significant effects (i.e., no consideration of target organs or tumor types).

Within IRIS, the currently established RfC is 0.1 mg/m^3, based on a study in male rats that found impaired motor coordination (decreased rotarod performance) [79]. Rats exposed to m-xylene alone exhibited statistically significantly decreased rotarod performance and decreased spontaneous activity. Multiple studies similarly determined effects on sensitivity to pain as well as passive and active avoidance, supportive of neurobehavioral effects [81, 82].

Although human evidence was not used to develop the RfC, the limited evidence available is supportive of these findings. In some studies involving single or multiple 4-hour exposures of human volunteers to 200 ppm of xylenes, reversible effects on balance and reaction times have been reported [79, 83-85]. Recent evidence also suggests that the cell toxicity of xylene might cause mitochondrial uncoupling via ATP depletion. Mitochondrial reactive oxygen species (ROS) generation and mitochondrial permeability transition (MPT) also appear to be involved [86]. Other endpoints discovered in recent animal studies include liver toxicity given xylene inhalation [87].

Styrene

Styrene, also known as vinyl benzene, is an organic compound with the chemical formula $C_6H_5CH=CH_2$. Within IRIS, the carcinogenicity of styrene has not been assessed [88]. However, there are some cancer potency estimates available – for example, for styrene oxide, IARC has classified it as a 2A human carcinogen [88], and CA OEHHA developed a unit risk for styrene oxide of 4.6×10^{-5} per $\mu g/m^3$ [34]. As mentioned above, risk assessment for styrene is potentially complicated by the fact that there are often simultaneous exposures to 1,3-butadiene, with the high correlations making it quite difficult to isolate the effects of either compound [54]. Within this study, we use the CA OEHHA value for a screening-level calculation, but recognize that the evidence is weak and that this may represent an overestimate of risk.

The non-cancer effects of styrene are better established, based on findings of CNS effects in an occupational epidemiology study. The current posted RfC in IRIS is 1 mg/m^3 [88]. Of note, since this study involved an investigation of health effects in humans rather than animals, fewer uncertainty factors were required for this RfC – a partial uncertainty factor of 3 was used for database inadequacy, 3 for intraspecies variability (pharmacodynamics only), and 3 for subchronic/chronic extrapolation, for a total of 30 (given EPA rounding conventions). Other adverse effects have been documented in animal studies, including irritation, reproductive, and respiratory effects, but none of these studies were used to derive the RfC [88].

Toluene

Toluene, known as methylbenzene or phenylmethane, is an aromatic hydrocarbon. According to IRIS, there is inadequate information to assess the carcinogenic potential of toluene because studies of humans chronically exposed to toluene are inconclusive, and because toluene did not exhibit cancer effects in rodent bioassays deemed of adequate quality [89]. No evidence of carcinogenicity was observed in animal studies up to 300 and 1200 ppm [89-91], or in most occupational epidemiological studies [92-94]. One recent study, which also found suggestive evidence of effects of xylenes, did determine elevated non-Hodgkin lymphoma in a medium/high toluene exposure group, but this evidence has not been deemed sufficient to develop a cancer potency factor [80].

For non-cancer effects, there is a substantial database examining the effects of toluene in occupationally exposed humans, with evidence of neurological effects (i.e., impaired color vision, impaired hearing, and decreased performance in neurobehavioral analysis, changes in motor and sensory nerve conduction velocity, headache, and dizziness) as the most sensitive endpoint [89]. The RfC is derived from these occupational epidemiology studies, with a value of 5 mg/m^3 [89]. Limited uncertainty factors were needed given the nature of the health evidence – a factor of 10 for human heterogeneity was incorporated, to address potentially susceptible human subpopulations and lifestages, but no other factors were needed given chronic human evidence and a strong evidentiary base. Indeed, there are many human studies that identified toluene's neurological effects, reporting NOAELs between 25~50 ppm [89, 95-102]. Revilla et al. suggest that the cell toxicity of toluene might cause mitochondrial uncoupling via ATP depletion [86].

PAHs

Numerous polycyclic aromatic hydrocarbons (PAHs) are of potential interest in relation to aviation emissions, and the evidence base varies substantially across PAHs. As mentioned above, PAHs considered in this investigation include naphthalene, phenanthrene, fluorene, fluoranthene, pyrene, anthracene, acenapthylene, acenaphthene, benzo[g,h,i]perylene, benzo[b]fluoranthene, benz[a]anthracene, benzo[k]fluoranthene, benzo[a]pyrene, chrysene, and indeno[1,2,3-c,d]pyrene. This represents a subset of PAHs for which there is a stronger evidence base for health effects, although the nature of the evidence differs by chemical. Rather than describing each compound individually, we first discuss one PAH which is hypothesized to contribute substantially to cancer-related health effects of PAHs – benzo[a]pyrene – and then consider approaches used to determine the relative toxicity of PAHs in a risk assessment context.

Benzo[a]pyrene (BaP) is a five-ring PAH, commonly seen in incomplete combustion at temperatures between 300 and 600 degrees C. While EPA does not assign an inhalation unit risk to BaP in the IRIS database, CA OEHHA has developed such a value, and many investigators at

EPA and elsewhere have used these potency values within risk assessments [2]. OEHHA considered BaP to be genotoxic, and developed a potency value based on a study of respiratory tract tumors in hamsters. The resulting inhalation unit risk was 1.1×10^{-3} per $\mu g/m^3$. Of note, more recent research indicates that BaP may be more of a procarcinogen, suggesting that the carcinogenesis of benzo[a]pyrene depends on enzymatic metabolism of benzo[a]pyrene to benzo[a]pyrene diolepoxide, which may increase mutations specifically in the protective p53 gene. This gene is a transcription factor that regulates the cell cycle and hence functions as a tumor suppressor. By inducing G (guanine) to T (thymidine) transversions in transversion hotspots within p53, benzo[a]pyrene diolepoxide may inactivate the tumor suppression ability in certain cells. This information could lead to long-term changes in the presumed potency values for BaP and other PAHs, especially as alternate paradigms for cancer risk assessment are developed, but we use the OEHHA value for our initial prioritization calculations.

OEHHA and others have used BaP as a primary representative of the class of PAHs, with the potency of other compounds estimated relative to the potency of BaP, using Potency Equivalency Factors (PEFs) based on available studies (including bioassays, mutagenicity tests, and structure-activity relationships). A PEF of 1 would indicate identical toxicity to BaP, while a PEF of 0.1 would indicate that the toxicity is one-tenth of the toxicity of BaP. Among the compounds considered within this analysis, the following values have been derived:

- PEF of 0.1: benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, indeno[1,2,3-c,d]pyrene
- PEF of 0.03: naphthalene
- PEF of 0.01: chrysene

Based on IARC categorizations, phenanthrene, fluorene, fluoranthene, pyrene, anthracene, and benzo[g,h,i]perylene were assigned PEFs of zero. This is based on inadequate or limited animal evidence (IARC Group 3 categorization). However, a recent publication [103] did provide PEF values for some of these PAHs, in spite of their relatively weak evidence. We use these values for the purpose of our screening-level calculation, but acknowledge that they are quite uncertain and likely represent upper-bound values. The values include 0.0005 for anthracene and phenanthrene, 0.001 for pyrene, and 0.05 for fluoranthene. Of note, acenapthylene and acenaphthene were not formally considered within the OEHHA classification scheme. These PAHs were addressed in IRIS and had no evidence of carcinogenicity. We therefore do not treat them further in this analysis.

Given its presence at relatively high concentrations, naphthalene deserves some additional attention. Also known as albocarbon, naphthalene is an organic compound consisting of two fused benzene rings. Within IRIS, the carcinogenicity of naphthalene is classified as group C, due to inadequate human data, and IRIS did not propose a cancer potency value as a result.

However, an inhalation unit risk has been estimated by OEHHA, using the PEF approach described above. More recent studies suggest that naphthalene may be genotoxic [104, 105], but this evidence has not been extended to develop cancer potency factors.

Turning to potential non-cancer effects of PAH exposures, inhalation RfC values are available for some, but not all PAHs. Of the PAHs included within the IRIS database, only naphthalene has an RfC value. This value, 3×10^{-3} mg/m^3, was based on a chronic mouse inhalation study with resulting effects in the respiratory and olfactory epithelium. This estimate has an uncertainty factor of 3000, applied to the animal bioassay (which only observed a LOAEL), and the confidence in this estimate is stated to be low to medium within IRIS [106]. More recent studies, largely related to environmental tobacco smoke and traffic exposures, have characterized exposures to naphthalene [107-109], with proposed potential adverse cardiovascular effects through inflammatory pathways. However, methods for exposure assessment continue to be refined. A recent study suggests that, while modeling human exposure to suspended naphthalene, dermal uptake should be included along with the inhalation route of exposure [110].

Fluoranthene, anthracene, fluorine, and pyrene have reference dose (RfD) values within IRIS based on oral exposures, which could in principle be approximated as RfC values, but given some key differences among these routes of exposure for PAHs, we do not quantify these impacts within this report. While there are studies that have generally linked PAHs, or diesel exhaust in general, with respiratory or developmental endpoints [111, 112], this evidence base is not treated formally within our assessment, other than to qualitatively note that PAHs may be of interest for future risk assessments.

Other Toxicity Estimates

For several compounds, including some mentioned above as well as ethylbenzene and propionaldehyde, there are insufficient data to either qualitatively or quantitatively assess its cancer potency. Therefore, IRIS did not post any proposed cancer risk estimates for these compounds. While it is possible to estimate the risk value of such compounds based on the chemical's structure-activity relationship (i.e., structural similarity to other compounds previously studied), such estimates are highly uncertain, so we do not employ them in this analysis. This effectively presumes that these compounds are known to have zero cancer risk, which is a strong assumption, but is deemed preferable in this context, given that we are not conducting formal uncertainty analyses related to our potency estimates.

Results

To help in the interpretation of our prioritization conclusions, we first present summary information related to emissions, followed by intake fractions across airports using both

AERMOD and CMAQ (with both 36 x 36 km and 12 x 12 km resolution), followed by the risk ranking and related prioritization conclusions.

Emissions

The emissions data derived from EDMS and provided by CSSI are summarized in Tables 1a-1c, which present the emissions per year for all pollutants under study for each of the three airports. Along with the aggregate emissions, we also present the ratio of emissions of key compounds, to help understand both the relative and absolute differences between compounds and airports, and we present emissions as input into both AERMOD and CMAQ.

These tables make it apparent that, for air toxics (Table 1a), the relative emission rates (normalized to total VOCs) are nearly identical across airports. The implication is that the relative prioritization across different airports would not be influenced by differences in relative emission rates. Similarly, the composition of particulate matter emissions (among EC, OC, and SO_4) is relatively consistent across airports (Table 1b). Particulate matter emissions do also roughly scale with VOC emissions, with minor differences across airports (ratio of total PM to total VOC of 0.13 at ORD, 0.11 at ATL, and 0.11 at PVD), and all emissions scale relatively closely with total fuel consumption. Thus, it is unlikely that emissions differentials could explain differences in risk rankings across airports, although the relative emissions will clearly influence the risk rankings within individual airports and the absolute emission rates will influence the population risk estimates. In general, this nearly strict proportionality, especially within the category of air toxics, is potentially an indication of limitations in the data underlying EDMS and similar emissions simulation packages (or, if reflective of the physical reality of emissions, indicates that air toxics emissions can be accurately estimated given fuel consumption data).

Finally, Table 1c demonstrates that the aggregate emissions are systematically larger within the CMAQ runs than in the AERMOD runs, with larger differences for criteria air pollutants. As the additional emissions occur at 3,000-10,000 feet, the concentration impact will not scale linearly with emissions, so some differences would be anticipated for the intake fractions for these two models independent of the dispersion models themselves.

Intake fractions and population exposure estimates

For all of the compounds listed in Tables 1a-1c for which cancer or criteria pollutant risk estimates were made, we calculated intake fractions using AERMOD, CMAQ with 36 km resolution, and CMAQ with 12 km resolution. First considering AERMOD outputs, which are summarized in Table 2, a few key insights emerge. The intake fractions are generally highest at ORD, followed by PVD in most cases. This is likely a function of the greater population density surrounding ORD – the number of people living within 50 km of the airport (using 2000 census

data) is approximately 7.2 million for ORD, versus 3.3 million for ATL and 1.7 million for PVD. The intake fraction is not strictly proportional to these population numbers, given non-uniform population density and the relatively greater contribution of populations living proximate to the airport, and also reflects differences in pollutant fate and transport.

In addition, for the gaseous pollutants, the intake fractions are quite similar within individual airports, especially for less reactive compounds. While AERMOD does not include a formal chemical reaction mechanism, first-order decay rates can be included, which could explain why slightly lower values are seen for the aldehydes and acrolein relative to generally non-reactive gases such as benzene. There are generally lower values for particulate matter and particle-bound PAHs, which will have more rapid atmospheric losses and lower intake fractions as a result. For example, at ORD, the intake fraction for non-reactive gaseous pollutants is approximately 2.0×10^{-5}, versus 1.2×10^{-5} for total fine particulate matter.

Using CMAQ, estimates are presented at multiple geographic scales using both 12 km and 36 km model resolution. To maintain concordance between the two CMAQ runs, we present both runs at five different geographic scales: 36 km x 36 km, 108 km x 108 km, 180 km x 180 km, 252 km x 252 km, and 324 km x 324 km. The maximum domain size was selected to avoid areas where it would be difficult to disentangle the relative contributions of multiple airports while capturing a sufficiently expansive domain that would not miss key population centers (e.g., the Boston area with respect to PVD). In addition, a national average intake fraction is calculated, representing the incremental effect of all three airports combined – in theory, this should represent an emission-weighted average of the three individual intake fractions, with any differences corresponding to spatial domain differences. Finally, we present only a subset of air toxics within the comparative results below, given a lack of CMAQ outputs for some air toxics, and we do not include ozone within the intake fraction comparison for reasons described above.

We first consider the intake fractions within the 108 km x 108 km region with both model resolutions, a region that roughly corresponds with the size of the AERMOD domain (with a 50 km radius). When comparing the three model outputs (Table 3), in nearly all cases, the values are within a factor of two of one another, indicating good model concordance. The 12 km resolution CMAQ modeling yielded systematically higher intake fractions than the 36 km resolution CMAQ modeling, indicating that coarse resolution modeling will tend to systematically underestimate population health risks associated with airport emissions. These differences are generally small for most pollutants, with 12 km resolution values approximately 6-60% greater than 36 km resolution values. In most cases, the 12 km resolution outputs are closer to the outputs using AERMOD, which may be an indication that the chemical reaction mechanisms using coarse resolution modeling are sufficiently uncertain that the model underperforms relative to a simpler model (although these comparisons should not be overinterpreted in light of the

numerous model differences). In general, AERMOD and 12 km resolution CMAQ outputs agree quite well with one another across compounds. The intake fraction values are within a factor of two of one another other than for toluene at ORD and ATL and $PM_{2.5}$ at ATL (ratios of 0.45, 0.4, and 2.3, respectively). The particulate matter findings can be explained in part by the greater emissions in CMAQ, which are implicitly assumed by this calculation to have the identical influence on concentrations as emissions at lower altitudes. If we assume as a bounding calculation that emissions above 3,000 feet have a minimal effect on ground-level concentrations, the intake fraction values would be increased within CMAQ and would be similar to those generated with AERMOD (e.g., with 12 km resolution, values of 9E-6, 5E-6, and 6E-6 at ORD, ATL, and PVD respectively). It is not clear why toluene has systematically higher values than other air toxics within CMAQ.

One reason why AERMOD performed similarly as CMAQ from an intake fraction perspective could be the relative insignificance of secondary formation relative to primary emissions and dispersion within this limited modeling domain. For a subset of pollutants (acrolein, acetaldehyde, and formaldehyde), CMAQ provides outputs of both primary and secondary products. The primary contribution (using 12 km resolution and a 108 x 108 km domain) represented 99-100% of the total intake fraction for acrolein, 98-104% for acetaldehyde, and 92-97% for formaldehyde. Of note, a percentage greater than 100% is possible, given secondary processes that can result in removal as well as formation. Regardless, this demonstrates that most of the intake fraction is captured by the primarily emitted products, at least within a relatively small spatial domain. If the domain is extended to 324 x 324 km, the primary contribution decreases slightly but remains dominant (99-100% for acrolein, 98-104% for acetaldehyde, 89-97% for formaldehyde). It should be noted that the dominance of primary products from an intake fraction perspective does not necessarily mean that secondary formation is not important when modeling ambient concentrations; it simply means that the marginal concentration changes associated with airport emissions, considered from a population-weighted perspective, are more affected by primary than secondarily-formed air toxics.

Turning to the spatial extent of the impacts, we examine the percentage of the total intake fraction found at expanding radii away from each of the three airports, using both model resolutions for CMAQ. As indicated in Figure 1, while patterns varied across pollutants and model resolutions, some general conclusions could be drawn. For example, in the majority of cases, a significant portion of the intake fraction was found within the 36 x 36 km grid cell surrounding the airport, with the next largest domain presented (108 km x 108 km) adding appreciably to the total and smaller contributions at longer range. At ORD, at least half of the intake fraction was found within the 36 x 36 km grid cell containing the airport for all pollutants, with higher percentages generally seen with 12 km resolution and for reactive gases (e.g.,

acrolein, formaldehyde). This is largely attributable to the significant population in that grid cell relative to populations elsewhere – over 3 million people are found within the 36 x 36 km grid cell containing ORD. In contrast, PVD has lower percentages of intake fraction contributed by the 36 x 36 km grid cell containing the airport, with contributions of 10-19% for fine particulate matter (and less than 700,000 people within the grid cell). More broadly, while many pollutants appeared to be asymptotically reaching their national intake fraction values within the domain modeled, $PM_{2.5}$ did not seem to have reached its maximum value by 324 x 324 km. While 89-100% of the air toxics population exposure has been obtained by this point, approximately half of the population exposure has been obtained for $PM_{2.5}$. This is consistent with previous studies that showed a significant contribution of long-range transport for PM intake fractions, and is likely a function of emissions at greater altitudes, secondary pollutant formation, and the general fate and transport characteristics of fine particles [4, 113].

While ozone cannot be easily described using intake fractions and was only estimated with CMAQ, we can examine the concentration outputs to determine the likely significance of ozone within our risk prioritization analysis. The marginal concentration changes were generally negative, indicating that addition of the airport emissions resulted in lower ozone concentrations near the airport (likely related to the ability of NOx to scavenge ozone in the near field). The ozone concentration changes remained negative throughout the 324 x 324 km domain surrounding ORD, but some concentration increases were seen at longer range for ATL and PVD. The aggregate impact on population exposures from the emissions from all three airports combined is negative (decreased ozone exposures). Although the direction of the ozone effect is opposite from the direction for the other pollutants, influenced in part by the spatial domain of the analysis, we estimate population health impacts for ozone to determine if the absolute magnitude is significant relative to $PM_{2.5}$ or air toxics.

Risk calculations and risk rankings

For our risk calculations and rankings, we first apply the AERMOD intake fractions to the estimated emissions and use the potency values and concentration-response functions listed above to determine total population risks. To provide comparability between the air toxics and fine particulate matter risk estimates, we divide the lifetime cancer risks by 70 to yield an annualized burden. While this ignores issues of latency, it approximates the long-term impacts for both categories of pollutants. The results of these calculations are presented in Table 4.

First looking within the category of air toxics (Table 4), the top contributor at all three airports is formaldehyde, which contributes approximately 48% of the total air toxics cancer risk at all three airports. In general, the percentage contributions of the various air toxics are almost identical across the airports, given similar relative emissions and intake fractions using

AERMOD. As mentioned above, formaldehyde has somewhat large uncertainties related to its potency, and if the lower inhalation unit risk from the 1999 National Air Toxics Assessment were utilized, the contribution of formaldehyde would be negligible. Other significant contributors to air toxics cancer risk include 1,3-butadiene (21-22%), styrene (11%), naphthalene (8%), and benzene (7%). The remaining PAHs contribute relatively little to the overall cancer risk. In aggregate, using AERMOD, the air toxics are estimated to contribute to approximately 0.09 cancers per year from ORD, 0.07 cancers per year from ATL, and 0.006 cancers per year from PVD.

In contrast (Table 4), using AERMOD, fine particulate matter is estimated to contribute to approximately 15 deaths per year from ORD, 7 deaths per year from ATL, and 0.7 deaths per year from PVD, roughly 100-200 times the impact from air toxics. This is in agreement with most previous risk ranking studies, which consistently show that the cardiopulmonary risks of fine particulate matter greatly exceed the cancer risks from air toxics when considered on a population mortality basis. It should be noted as well that the particulate matter estimates represent deaths, while the air toxics estimates represent all cancers, only a fraction of which will result in premature death, making the difference from a population health burden perspective even greater than presented. In addition, the AERMOD modeling excludes secondary particulate matter formation, which has been shown elsewhere to dominate the population health risks from aircraft emissions, and only considers a 50 km radius around the airport.

For CMAQ, risk estimates are first presented using the 108 x 108 km grid cell outputs using both model resolutions, which will underestimate the impacts of selected pollutants (especially fine particulate matter) but will allow for comparability with the AERMOD outputs. In addition, not all of the air toxics were modeled within CMAQ, although all of the major risk contributors are represented, with the notable omission of styrene. The outputs from CMAQ also include both total PM and the contribution of various primary and secondary constituents, and ozone is listed alongside PM to compare the absolute magnitude of impacts in spite of the difference in directionality.

Running CMAQ, the prioritization conclusions are quite similar to those using AERMOD, as would be expected given the similarity in intake fraction values (Table 5). Within the 108 x 108 km domain, the top-ranking contributor to air toxics cancer risk remains formaldehyde, with approximately 70% of the total cancer risk, with 1,3-butadiene contributing approximately 15% of the total cancer risk. For the five compounds included in both analyses, in spite of the numerous modeling differences, the estimates are reasonably similar across the three model formulations. Total air toxics risk using 12 km resolution CMAQ within the 108 x 108 km domain are 0.09 cancers per year from ORD, 0.06 cancers per year from ATL, and 0.004 cancers per year from PVD (versus 0.09, 0.07, and 0.006 using AERMOD, respectively). Using a

national scope rather than a 108 x 108 km domain increases the total air toxics burden nominally, from 0.15 cancers per year to 0.16 cancers per year.

As with the AERMOD outputs, the fine particulate matter risks are substantially higher than the air toxics risks, even within the 108 x 108 km domain (Table 5). The magnitudes of the particulate matter risks are within a factor of 2 of the estimates using AERMOD, demonstrating good agreement between methods, especially in light of the differences in emissions, model scale, and resolution. The impact from ozone is smaller than that of fine particulate matter but is appreciable and larger than the impact from air toxics, indicating that the effect on ozone should not be discounted. However, if the modeling region were expanded to capture a regional scale, the impacts of fine particulate matter emissions would be greater while the disbenefits associated with control of ozone precursors would reduce given formation at long range. In particular, moving to a national scale using 12 km resolution CMAQ outputs, the PM-related mortality effects increase by about a factor of 2 relative to the 108 x 108 km domain, while the ozone-related mortality effects become less negative (Table 6).

In addition, we can apportion the particulate matter risks across different particle constituents. This should be considered as an illustrative exercise, since (for example) ammonium will generally be bound with sulfate or nitrate, and it was beyond the scope of this analysis to address these dimensions of particle chemistry given the available outputs. In addition, as both primary sulfate emissions and secondary sulfate formation are modeled, this apportionment cannot be directly translated into primary versus secondary formation. Regardless, this apportionment illustrates that, for our national-scale impacts (Table 6), the contributions from sulfate and nitrate are approximately equal and together dominate the health risks. When the combination of sulfate, nitrate, and ammonium is considered, they collectively contribute over 80% of the health risk. In contrast, when looking at only the near-source domain (Table 5), the contribution of EC and OC is greater (approximately 30-40% of the risk), while the impact on nitrate is small for PVD and negative for ORD and ATL. In general, this emphasizes the importance of accurately modeling secondary sulfate and nitrate particles at a regional scale when quantifying the health impacts of aviation.

In terms of the spatial patterns of health risk, Figure 2 demonstrates the grid cells in which the population health risks occur for a few selected pollutants, using 12 km resolution CMAQ. To be clear, these maps are not providing estimates of concentrations or individual health risks, but rather the product of marginal concentrations, potency, population, and baseline mortality incidence. Each map presents the percentiles of the population risk distribution, with the breakpoints for the categories representing the 10^{th}-90^{th} percentiles, 95^{th}, and 99^{th} percentiles. The top percentile is shaded in red and the 95^{th}-99^{th} percentiles are shaded in pink.

For fine particulate matter, of the 35 estimated deaths per year, 21 occur within the grid

cells highlighted in red (the top percentile of cells), with another 7 occurring within the grid cells highlighted in pink (the 95th-99th percentile of cells). This is a function of both proximity to the airports and population size, as some cities with lower marginal concentration impacts (e.g., Detroit) show up on the map because of their larger populations. For reactive pollutants such as formaldehyde (Figure 2), the risk is even more concentrated in a small number of grid cells; of the total population risk, over 96% occurs in the top percentile of grid cells, with 76% occurring in the 10 highest-risk grid cells (versus 34% for $PM_{2.5}$). As indicated in Figure 2, 1,3-butadiene displays a very similar pattern as formaldehyde, as do most of the air toxics. To better understand the near-source spatial patterns of population risk, Figure 3 presents the identical figure as in Figure 2 for fine particulate matter, but zoomed in around the three individual airports of interest.

The above calculations do not take into account non-cancer risks from air toxics. Comparing the reference concentrations listed above with the measured and modeled concentrations, only acrolein is found at concentrations significantly above its RfC (RfC of 0.02 $\mu g/m^3$, ambient concentration of 0.03-0.04 $\mu g/m^3$ from baseline CMAQ outputs in the grid cells housing the three airports, ambient concentrations of 0.1-0.2 $\mu g/m^3$ from baseline data in the National Air Toxics Assessment). This would imply that the marginal contribution of airport emissions of acrolein would have non-zero respiratory effects. In addition, in non-cancer risk assessment, other compounds with similar modes of action would also be considered to have non-zero risk, with the hazard quotients (ratio between concentrations and the RfC) of the individual compounds summed up to give an overall hazard index (which, if greater than 1, would be indicative of population health risks). Following the approach taken within the National Air Toxics Assessment, we assume that any compound with identified respiratory effects could have an additive relationship with acrolein impacts (without delving into the precise mode of action). Based on this assumption, given the high baseline exposures to acrolein, we would also be concerned about the respiratory irritant effects of acetaldehyde, formaldehyde, naphthalene, styrene, and toluene at all three airports. To the extent that the precise mode of action differs between acrolein and these listed compounds, the effects of the listed compounds may not be large, but this is a general indication that respiratory outcomes should be more formally considered for this suite of pollutants.

Alternative approaches for prioritization

Some previous studies have attempted to prioritize across compounds either by simply looking at emissions or by considering the product of emissions and toxicity. It is therefore important to know the degree to which inappropriate prioritization decisions would be made if exposure and/or toxicity were ignored. For this illustrative comparison, we consider only the cancer risks from air toxics using AERMOD (given identical conclusions using CMAQ), and we

initially present the combined effects of all three airports (which allows for a broader spatial domain to be captured). We note that the emissions of fine particulate matter are less than those of formaldehyde and similar to those for other air toxics, but the health effects are much greater, implying that a focus on emissions alone would completely miss the significance of $PM_{2.5}$.

As shown in Table 7, prioritization strictly on emissions would tend to overstate the relative importance of acetaldehyde and benzene and would tend to understate the relative importance of 1,3-butadiene and styrene. Most PAHs are emitted in such small quantities that they do not play a significant role in the rankings regardless of the approach, but given that their potencies are at times 3 orders of magnitude greater than the potencies of other air toxics, focusing on emissions only could lead to drastically wrong conclusions in certain settings. Intake fractions generally differed only by a factor of 2 across compounds, much smaller than the variability in emissions or potency, so ignoring exposure does not lead to appreciably different prioritization conclusions. However, this could be a function in part of the underlying assumptions in the atmospheric dispersion models and the limited spatial domain; other studies [114] have shown intake fractions for these compounds varying by multiple orders of magnitude. Regardless of the empirical findings for these three airports, there is no theoretical basis to conduct risk-based prioritization without exposure and/or toxicity, so all components should be considered going forward.

Discussion and Conclusions

Within the above sections, we have presented first-order population risk calculations to aid in the prioritization of future research, including measurement and modeling campaigns. While there are clearly some significant uncertainties associated with our analyses, as described in more detail below, a number of conclusions appear robust and can help inform future studies.

First, it is apparent that the quantified health risks associated with fine particulate matter greatly exceed the quantifiable cancer risks associated with air toxics under study. This is a common finding, exemplified by the fact that the annual cancer risk across the United States from all outdoor hazardous air pollutants is on the order of 200 cancer cases per year (http://www.epa.gov/ttn/atw/nata1999/tables.html), while the benefits of controlling power plant sources of fine particulate matter are on the order of 20,000 fewer deaths per year [115]. Differences of two orders of magnitude, as found within our calculations, are therefore to be expected.

From a risk perspective, uncertainties associated with fine particulate matter risk calculations are therefore paramount. As with any of the above calculations, these uncertainties would be related to uncertainties in the emissions inventory, atmospheric dispersion modeling, and concentration-response functions. Details regarding the robustness of emissions inventory and atmospheric dispersion modeling are available elsewhere (reports prepared by CSSI and

University of North Carolina, respectively), but as a general point, it is unlikely that these are uncertain to a degree that our general conclusions about the importance of fine particulate matter would be affected. In other words, the emissions would need to be overestimated by two orders of magnitude for particulate matter risk to no longer dominate the assessment, which would seem highly unlikely. In addition, although there remain uncertainties with the atmospheric dispersion modeling outputs, the relative concordance between AERMOD and CMAQ results indicates that such large uncertainties would be unlikely.

However, there are two critical assumptions within the concentration-response function for fine particulate matter that could substantially influence our conclusions. First, we have made the assumption that the concentration-response function for $PM_{2.5}$ as a whole is applicable to all constituents under study. To the extent that there is differential toxicity among particle constituents, this assumption may or may not be well supported. There is limited evidence related to primary particles specifically from aircraft, although a broader literature indicates greater toxicity for combustion than non-combustion particles and potentially for traffic sources. Further research would be required to better understand the composition and relative toxicity of aircraft-specific particulate matter, and we recommend that this research be undertaken to increase the accuracy of airport population risk calculations. It is unlikely that such research would determine that particulate matter from aircraft has such low toxicity to make the impact of air toxics greater, but given the dominance of $PM_{2.5}$ in the total risk calculations, relatively small changes in relative toxicity would be influential.

Second, we have made the assumption that the concentration-response function for $PM_{2.5}$ is applicable at all ambient concentrations within our modeling region. If there are non-linearities or population thresholds for this function, our estimates may not be accurate. However, epidemiological evidence to date [12, 13] indicates that the concentration-response function for fine particulate matter is essentially linear down to the lowest concentrations observed in those studies, less than 10 $\mu g/m^3$. As most of the region under study has concentrations greater than this level, especially the highly populated areas close to the airports that contribute a majority of the health impacts, any non-linearities at lower levels would not appreciably influence our findings.

Turning to air toxics, while the contribution to quantified population risk is not as great, some important insights emerged from our analysis. Multiple compounds substantively contributed to either cancer or non-cancer risks, including formaldehyde, acetaldehyde, benzene, 1,3-butadiene, naphthalene, styrene, acrolein, and toluene. Formaldehyde dominates the cancer risks but has a highly uncertain potency value, indicating that research to better understand its low-dose potency would be quite valuable, although complex and time-consuming. In contrast, while the risk from compounds like benzene is not large, the somewhat lesser uncertainty related

to its potency indicates that it is likely to remain a significant contributor even as new research occurs.

In general, it is likely that the cancer risks are small enough to be difficult to observe epidemiologically and to be dominated by cardiopulmonary risks. However, the influence of air toxics on respiratory effects should not be dismissed simply because current risk assessment methods are inadequate to quantify such effects, and future monitoring and modeling studies should continue to characterize those exposures.

Beyond prioritization conclusions, there were some broad-based conclusions related to methods that will be useful to consider in future analyses. First, it is clear that the current emissions estimation methods for air toxics presume nearly strict proportionality among various VOCs, and further research would be needed to determine the degree to which this assumption is justified. Second, the fact that the risks were systematically higher using 12 km rather than 36 km resolution indicates that coarse resolution dispersion modeling will lead to downwardly biased risk estimates. That being said, the fact that the prioritization rankings were largely unchanged across model resolutions (or between AERMOD and CMAQ) emphasizes that large differences in toxicity and emissions will often dominate differences in intake fraction related solely to reaction and deposition rates. In addition, when a larger spatial scale was used to estimate particulate matter health risks, the bias was reduced between the 12 km and 36 km risk estimates.

Given the impact of fine particulate matter and the spatial distribution of that impact, larger spatial domains (regional-scale or national-scale) will likely be needed to capture most of the population risk, and models are needed that can reasonable estimate sulfate and nitrate concentrations. While this would provide more accurate population risk estimates, it complicates the ability to disentangle the effects of individual airports from one another within single CMAQ runs. Future studies should compare the risk estimates when airports are added singly versus in combination, to determine the degree to which this influences quantitative risk estimates.

In conclusion, our risk calculations emphasize the importance of characterizing the contribution of aircraft and airport emissions to local and regional fine particulate matter concentrations, as well as the need to better understand the relative toxicity of different particle constituents to better inform these risk estimates. Although population cancer risks from air toxics are small in comparison to the risks from fine particulate matter, respiratory effects from many of these air toxics may be of concern in light of high baseline exposure to acrolein, and future monitoring and modeling studies should characterize these exposures to provide greater understanding about patterns of respiratory disease. Future monitoring and modeling studies should also attempt to capture small-scale spatial gradients in exposure, which may be significant contributors to population risks and which may be underestimated with current modeling techniques.

Table 1a: Summary of air toxics emissions by compound and airport below 3,000 feet (input to AERMOD).

Pollutant	ORD Emissions (kg/year)	ORD Ratio to total VOC	ATL Emissions (kg/year)	ATL Ratio to total VOC	PVD Emissions (kg/year)	PVD Ratio to total VOC
Formaldehyde	196490.48	2.30E-01	219926.86	2.29E-01	16119.05	2.26E-01
Acetaldehyde	72137.04	8.43E-02	80632.20	8.40E-02	5932.57	8.33E-02
Benzene	32981.94	3.85E-02	36743.82	3.83E-02	2760.46	3.88E-02
Toluene	11280.25	1.32E-02	12552.81	1.31E-02	949.44	1.33E-02
Acrolein	151149.73	1.77E-01	169233.19	1.76E-01	12339.63	1.73E-01
1,3-butadiene	17394.24	2.03E-02	19460.18	2.03E-02	1451.40	2.04E-02
Xylene	12081.97	1.41E-02	13485.31	1.41E-02	1004.67	1.41E-02
Naphthalene	5369.65	6.27E-03	5987.42	6.24E-03	447.78	6.29E-03
Propionaldehyde	1584.77	1.85E-03	1776.04	1.85E-03	140.60	1.98E-03
Ethylbenzene	16173.26	1.89E-02	18106.42	1.89E-02	1323.13	1.86E-02
Styrene	5431.95	6.35E-03	6067.07	6.32E-03	451.58	6.34E-03
Acenaphthylene	133.73	1.56E-04	149.77	1.56E-04	10.89	1.53E-04
Phenanthrene	78.98	9.23E-05	89.98	9.38E-05	6.65	9.35E-05
Fluorene	47.19	5.51E-05	52.85	5.51E-05	3.84	5.40E-05
Fluoranthene	26.29	3.07E-05	29.79	3.10E-05	2.30	3.23E-05
Pyrene	32.41	3.79E-05	36.37	3.79E-05	2.81	3.94E-05
Anthracene	14.52	1.70E-05	16.39	1.71E-05	1.20	1.69E-05
Acenaphthene	27.10	3.17E-05	30.35	3.16E-05	2.21	3.10E-05
Benzo[g,h,i]perylene	26.49	3.09E-05	29.66	3.09E-05	2.16	3.03E-05
Benzo[b]fluoranthene	26.60	3.11E-05	29.80	3.10E-05	2.17	3.04E-05
Benzo[k]fluoranthene	26.60	3.11E-05	29.80	3.10E-05	2.17	3.04E-05
Benz[a]anthracene	6.44	7.52E-06	7.28	7.58E-06	0.53	7.45E-06
Benzo[a]pyrene	6.04	7.06E-06	6.77	7.06E-06	0.49	6.92E-06
Chrysene	7.35	8.59E-06	8.33	8.68E-06	0.61	8.54E-06
Indeno[1,2,3-c,d]pyrene	26.49	3.09E-05	29.66	3.09E-05	2.16	3.03E-05

Table 1b: Summary of criteria pollutant emissions by compound and airport below 3,000 feet (input to AERMOD).

Pollutant	ORD Emissions (kg/year)	Ratio to total PM	ATL Emissions (kg/year)	Ratio to total PM	PVD Emissions (kg/year)	Ratio to total PM
CO	4991292.3		4963365.6		261348.5	
VOC	855893.0		959736.6		71180.9	
NOx	4428921.5		4223206.2		311682.8	
SOx	542216.3		515342.3		34955.2	
Particulate OC	17437.5	1.56E-01	19271.3	1.78E-01	1349.2	1.79E-01
Particulate SO4	41513.4	3.72E-01	39455.9	3.64E-01	2676.3	3.55E-01
Particulate EC	52680.4	4.72E-01	49770.3	4.59E-01	3517.1	4.66E-01
Total fine particulate matter	111631.4		108497.5		7542.5	

Table 1c: Ratio between emissions used in CMAQ (below 10,000 feet) and AERMOD (below 3,000 feet).

	ORD	ATL	PVD
Pollutant	CMAQ/AERMOD	CMAQ/AERMOD	CMAQ/AERMOD
Formaldehyde	1.02	1.01	1.02
Acetaldehyde	1.02	1.01	1.03
Benzene	1.05	1.04	1.07
Toluene	1.06	1.05	1.09
Acrolein	1.01	1.00	1.01
1,3-butadiene	1.04	1.03	1.06
Xylene	1.04	1.03	1.06
Naphthalene	1.05	1.04	1.07
Propionaldehyde	1.19	1.16	1.25
Ethylbenzene	1.01	1.01	1.01
Styrene	1.04	1.03	1.05
Acenaphthylene	1.03	1.03	1.05
Phenanthrene	1.00	1.00	1.00
Fluorene	1.09	1.08	1.11
Fluoranthene	1.00	1.00	1.00
Pyrene	1.18	1.17	1.23
Anthracene	1.16	1.14	1.20
Acenaphthene	1.05	1.05	1.07
Benzo[g,h,i]perylene	1.00	1.00	1.00
Benzo[b]fluoranthene	1.00	1.00	1.00
Benzo[k]fluoranthene	1.00	1.00	1.00
Benz[a]anthracene	1.00	1.00	1.00
Benzo[a]pyrene	1.04	1.04	1.05
Chrysene	1.01	1.01	1.01
Indeno[1,2,3-c,d]pyrene	1.07	1.07	1.08
NOx	1.57	1.58	1.62
SO_2	1.36	1.36	1.44
PM	1.43	1.43	1.50

Table 2: Intake fractions for air toxics and fine particulate matter using AERMOD.

Pollutant	ORD	ATL	PVD
Formaldehyde	8.7E-06	6.0E-06	6.7E-06
Acetaldehyde	1.2E-05	8.2E-06	9.1E-06
Benzene	2.0E-05	1.4E-05	1.5E-05
Toluene	1.9E-05	1.3E-05	1.5E-05
Acrolein	1.4E-05	9.7E-06	1.1E-05
1,3-butadiene	1.9E-05	1.3E-05	1.4E-05
Xylene	1.9E-05	1.4E-05	1.5E-05
Naphthalene	2.0E-05	1.4E-05	1.5E-05
Propionaldehyde	1.2E-05	8.4E-06	8.8E-06
Ethylbenzene	2.0E-05	1.4E-05	1.5E-05
Styrene	2.0E-05	1.4E-05	1.5E-05
Acenaphthylene	2.1E-05	1.4E-05	1.5E-05
Phenanthrene	2.0E-05	1.3E-05	1.3E-05
Fluorene	2.0E-05	1.4E-05	1.3E-05
Fluoranthene	1.7E-05	1.2E-05	8.4E-06
Pyrene	1.8E-05	1.3E-05	1.0E-05
Anthracene	1.6E-05	1.3E-05	1.0E-05
Acenaphthene	1.9E-05	1.4E-05	1.0E-05
Benzo[g,h,i]perylene	1.9E-05	1.4E-05	1.1E-05
Benzo[b]fluoranthene	1.9E-05	1.4E-05	1.0E-05
Benzo[k]fluoranthene	1.9E-05	1.4E-05	1.0E-05
Benz[a]anthracene	1.2E-05	1.1E-05	7.7E-06
Benzo[a]pyrene	1.2E-05	1.1E-05	8.3E-06
Chrysene	1.2E-05	1.1E-05	6.7E-06
Indeno[1,2,3-c,d]pyrene	1.9E-05	1.4E-05	1.1E-05
Particulate OC	1.3E-05	7.8E-06	7.4E-06
Particulate SO4	1.2E-05	7.9E-06	6.6E-06
Particulate EC	1.1E-05	7.0E-06	6.1E-06
Total fine particulate matter	1.2E-05	7.5E-06	6.5E-06

Table 3: Intake fractions for air toxics and fine particulate matter using AERMOD and 12 x 12 km and 36 x 36 km resolution CMAQ runs (with 108 x 108 km domains for CMAQ).

		ORD	ATL	PVD
Acrolein	AERMOD	1.4E-05	9.7E-06	1.1E-05
	CMAQ 12 km	1.4E-05	8.5E-06	7.8E-06
	CMAQ 36 km	8.7E-06	6.6E-06	5.8E-06
Acetaldehyde	AERMOD	1.2E-05	8.2E-06	9.1E-06
	CMAQ 12 km	1.3E-05	8.5E-06	7.2E-06
	CMAQ 36 km	9.0E-06	6.5E-06	5.6E-06
Benzene	AERMOD	2.0E-05	1.4E-05	1.5E-05
	CMAQ 12 km	1.3E-05	9.8E-06	8.2E-06
	CMAQ 36 km	1.0E-05	7.6E-06	6.5E-06
Butadiene	AERMOD	1.9E-05	1.3E-05	1.4E-05
	CMAQ 12 km	1.3E-05	8.4E-06	7.1E-06
	CMAQ 36 km	8.6E-06	6.1E-06	5.4E-06
Formaldehyde	AERMOD	8.7E-06	6.0E-06	6.7E-06
	CMAQ 12 km	1.2E-05	7.5E-06	6.7E-06
	CMAQ 36 km	8.2E-06	6.2E-06	5.3E-06
Xylenes	AERMOD	1.9E-05	1.4E-05	1.5E-05
	CMAQ 12 km	1.3E-05	9.6E-06	7.7E-06
	CMAQ 36 km	1.1E-05	7.6E-06	6.0E-06
Toluene	AERMOD	1.9E-05	1.3E-05	1.5E-05
	CMAQ 12 km	4.2E-05	3.3E-05	2.7E-05
	CMAQ 36 km	3.3E-05	2.5E-05	2.1E-05
Naphthalene	AERMOD	2.0E-05	1.4E-05	1.5E-05
	CMAQ 12 km	1.4E-05	9.2E-06	7.6E-06
	CMAQ 36 km	9.3E-06	7.1E-06	6.2E-06
$PM_{2.5}$	AERMOD	1.2E-05	7.5E-06	6.5E-06
	CMAQ 12 km	6.4E-06	3.3E-06	3.9E-06
	CMAQ 36 km	4.3E-06	3.1E-06	3.2E-06

Table 4: Annual population cancer risks associated with air toxics emissions and mortality risks associated with particulate matter from ORD, ATL, and PVD, using AERMOD (50 km radius).

	ORD		ATL		PVD	
Pollutant		% of air toxics risk		% of air toxics risk		% of air toxics risk
Formaldehyde	4.3E-02	48%	3.4E-02	48%	2.7E-03	48%
Acetaldehyde	3.7E-03	4%	2.9E-03	4%	2.3E-04	4%
Benzene	6.4E-03	7%	4.9E-03	7%	4.0E-04	7%
1,3-butadiene	1.9E-02	22%	1.5E-02	21%	1.2E-03	22%
Naphthalene	6.9E-03	8%	5.4E-03	8%	4.4E-04	8%
Styrene	9.7E-03	11%	7.5E-03	11%	6.1E-04	11%
Phenanthrene	1.7E-06	0%	1.3E-06	0%	9.4E-08	0%
Fluoranthene	4.8E-05	0%	3.9E-05	0%	2.1E-06	0%
Pyrene	1.3E-06	0%	1.0E-06	0%	6.0E-08	0%
Anthracene	2.5E-07	0%	2.3E-07	0%	1.3E-08	0%
Benzo[b]fluoranthene	1.1E-04	0%	9.1E-05	0%	4.9E-06	0%
Benzo[k]fluoranthene	1.1E-04	0%	9.1E-05	0%	4.9E-06	0%
Benz[a]anthracene	1.6E-05	0%	1.6E-05	0%	8.8E-07	0%
Benzo[a]pyrene	1.5E-04	0%	1.6E-04	0%	8.7E-06	0%
Chrysene	2.0E-06	0%	2.0E-06	0%	8.8E-08	0%
Indeno[1,2,3-c,d]pyrene	1.1E-04	0%	9.1E-05	0%	4.9E-06	0%
Total air toxics	9.0E-02		7.0E-02		5.7E-03	
Total fine particulate matter	15.0		7.2		0.65	

Table 5: Population risks associated with airport emissions within a 108 x 108 km region surrounding the airports (using CMAQ with 36 km and 12 km resolution).

	ORD		ATL		PVD	
Pollutant	12 km	36 km	12 km	36 km	12 km	36 km
Formaldehyde	5.9E-02	4.2E-02	4.3E-02	3.5E-02	2.8E-03	2.2E-03
Acetaldehyde	4.0E-03	2.8E-03	3.0E-03	2.3E-03	1.9E-04	1.5E-04
Benzene	4.5E-03	3.5E-03	3.7E-03	2.9E-03	2.4E-04	1.9E-04
1,3-butadiene	1.4E-02	9.2E-03	9.9E-03	7.2E-03	6.4E-04	4.9E-04
Naphthalene	4.9E-03	3.4E-03	3.7E-03	2.9E-03	2.3E-04	1.9E-04
Total air toxics	8.6E-02	6.0E-02	6.3E-02	5.0E-02	4.1E-03	3.2E-03
Total fine particulate matter	12	7.9	4.5	4.2	0.57	0.48
% Sulfate	49%	52%	59%	64%	41%	37%
% Nitrate	-2%	-5%	-12%	-8%	13%	21%
% EC	15%	16%	19%	16%	13%	12%
% OC	21%	20%	18%	12%	18%	15%
% Ammonium	17%	17%	15%	16%	15%	16%
% Other	1%	0%	0%	0%	0%	-1%
Ozone	-1.9	-2.3	-2.1	-1.9	-0.2	-0.1

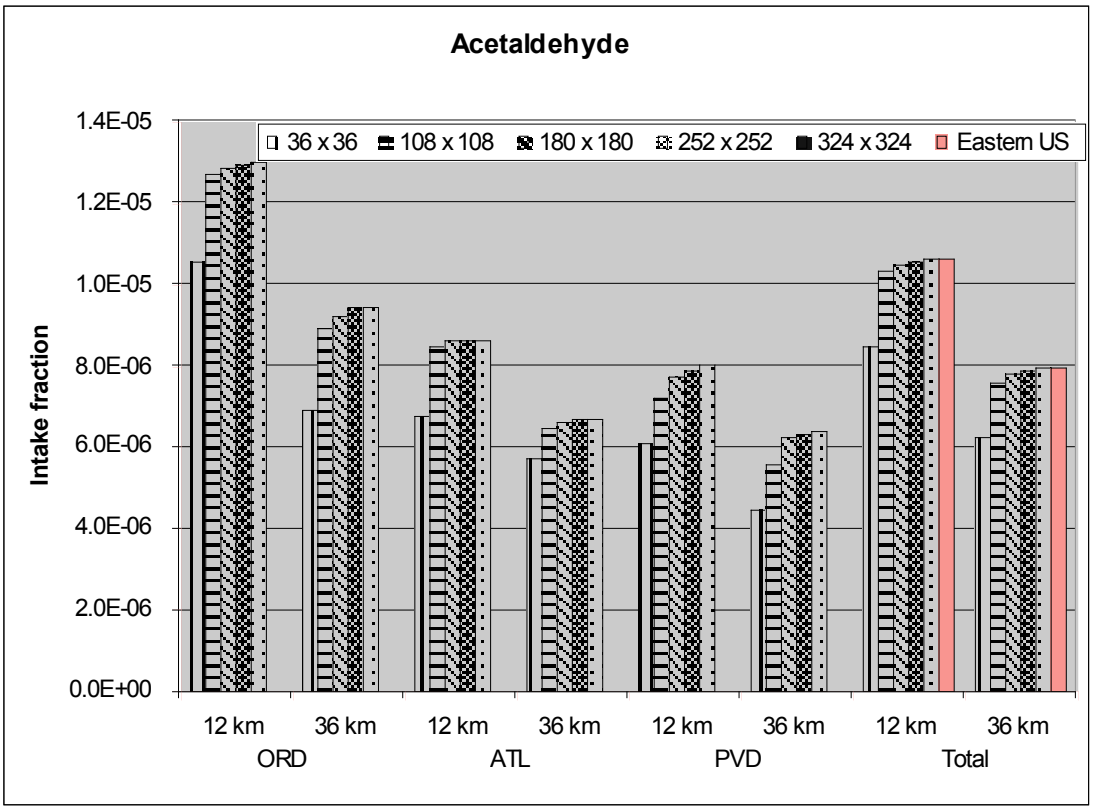
Table 6: Population risks (deaths/year) associated with airport emissions using CMAQ with 12 km and 36 km resolution, all three airports combined (Eastern US domain).

Pollutant	12 km	36 km
Formaldehyde	1.1E-01	8.6E-02
Acetaldehyde	7.4E-03	5.5E-03
Benzene	9.5E-03	7.7E-03
1,3-butadiene	2.6E-02	1.8E-02
Naphthalene	9.6E-03	7.3E-03
Total air toxics	1.6E-01	1.2E-01
Total fine particulate matter	35	34
% Sulfate	35%	31%
% Nitrate	28%	36%
% EC	9%	7%
% OC	10%	7%
% Ammonium	18%	19%
% Other	0%	-1%
Ozone	-3.3	-3.5

Table 7: Alternative prioritization conclusions ignoring exposure and/or toxicity, using AERMOD outputs.

Pollutant	Ranking, emissions only	Ranking, emissions*potency	Ranking, risk
Formaldehyde	1	1	1
Acetaldehyde	2	6	6
Benzene	3	5	5
1,3-butadiene	4	2	2
Naphthalene	6	4	4
Styrene	5	3	3
Phenanthrene	7	14	14
Fluoranthene	9	11	11
Pyrene	8	15	15
Anthracene	13	16	16
Benzo[b]fluoranthene	10	8	9
Benzo[k]fluoranthene	10	8	9
Benz[a]anthracene	15	12	12
Benzo[a]pyrene	16	7	7
Chrysene	14	13	13
Indeno[1,2,3-c,d]pyrene	12	10	8

Figure 1: Intake fractions of selected air toxics and fine particulate matter at three airports with varying geographic scale and resolution.



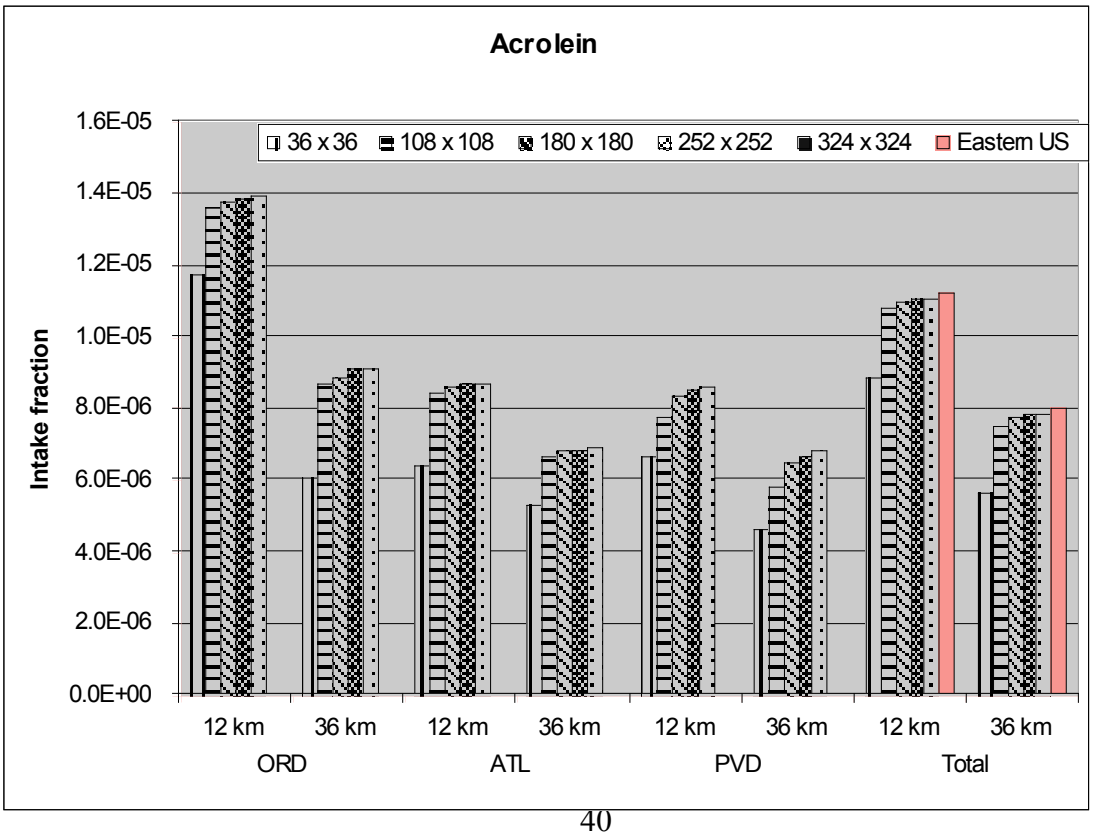
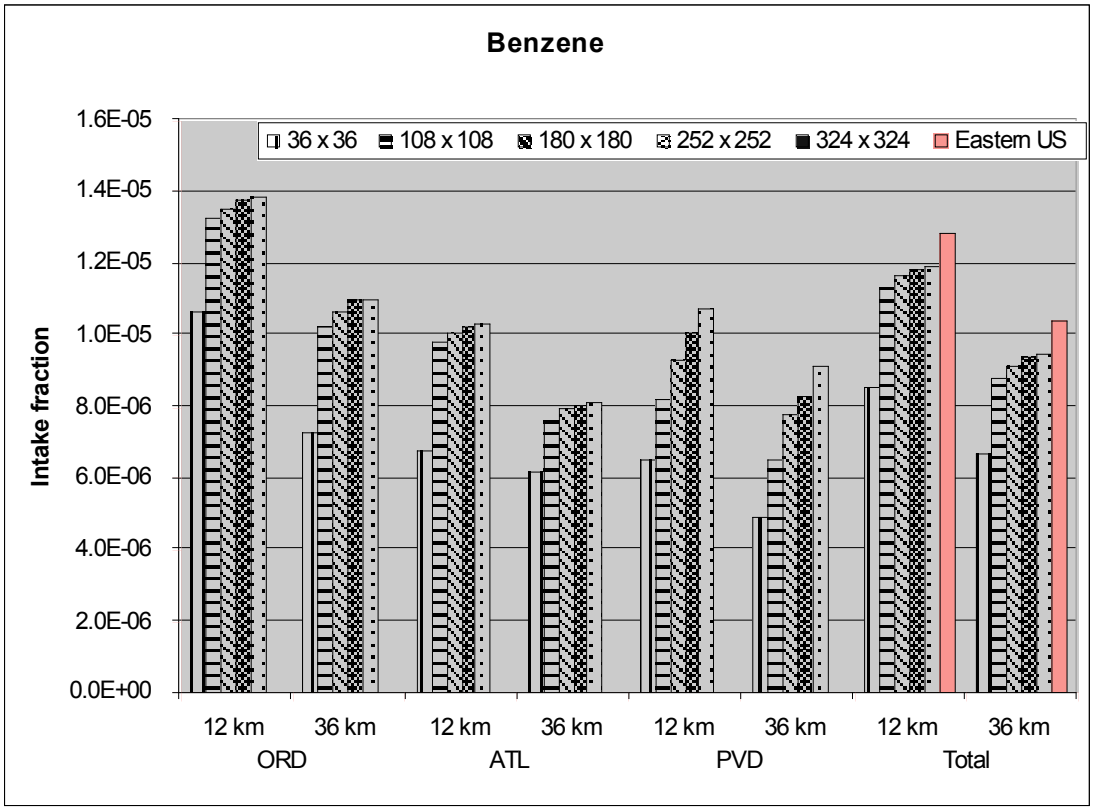


Figure 1, continued



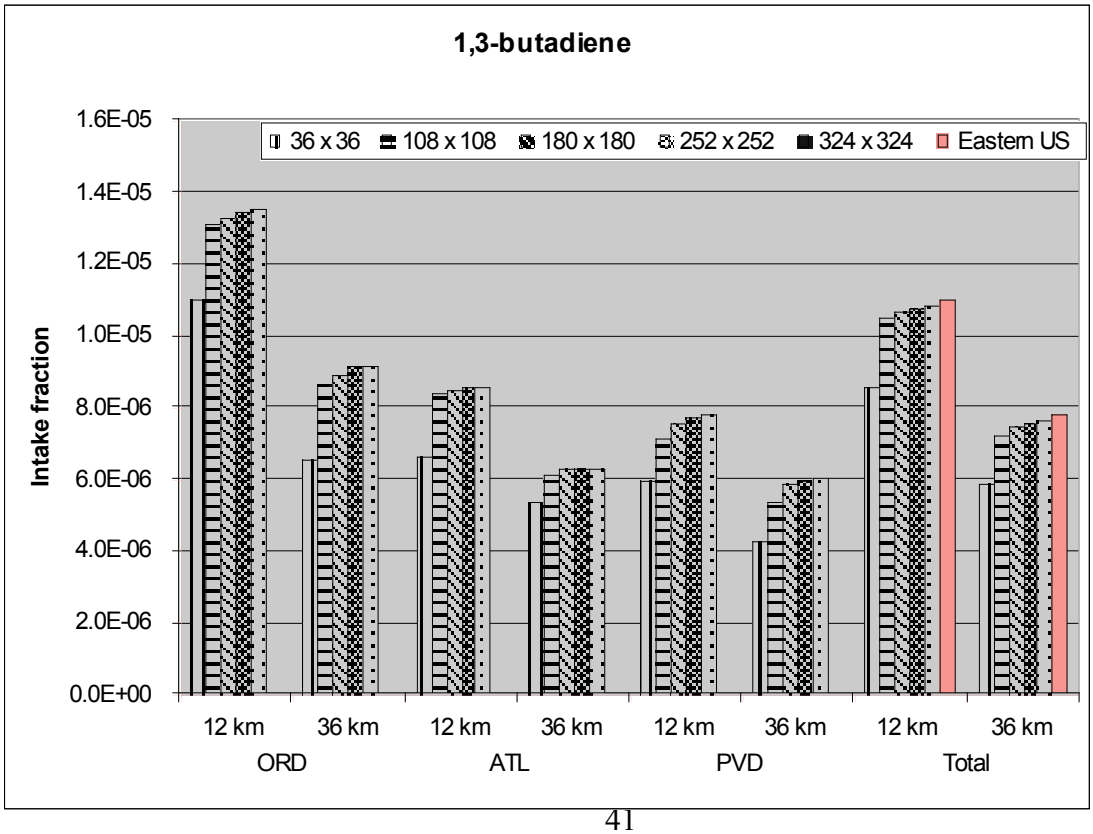
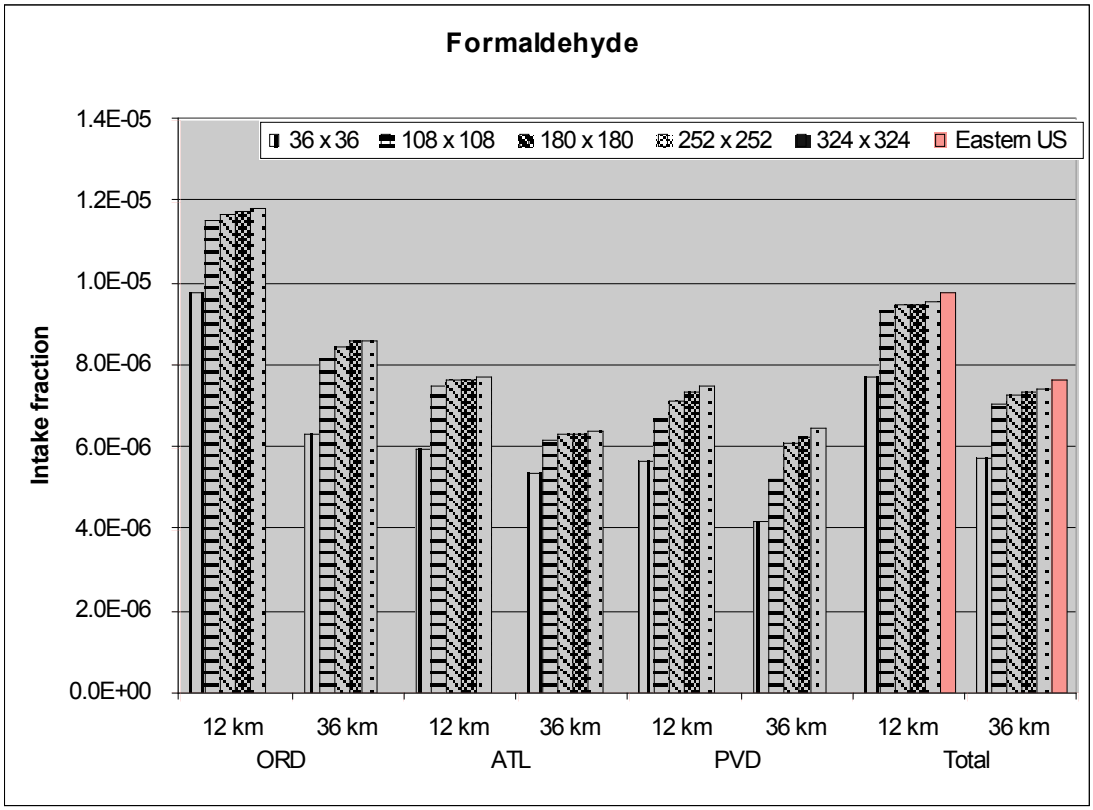
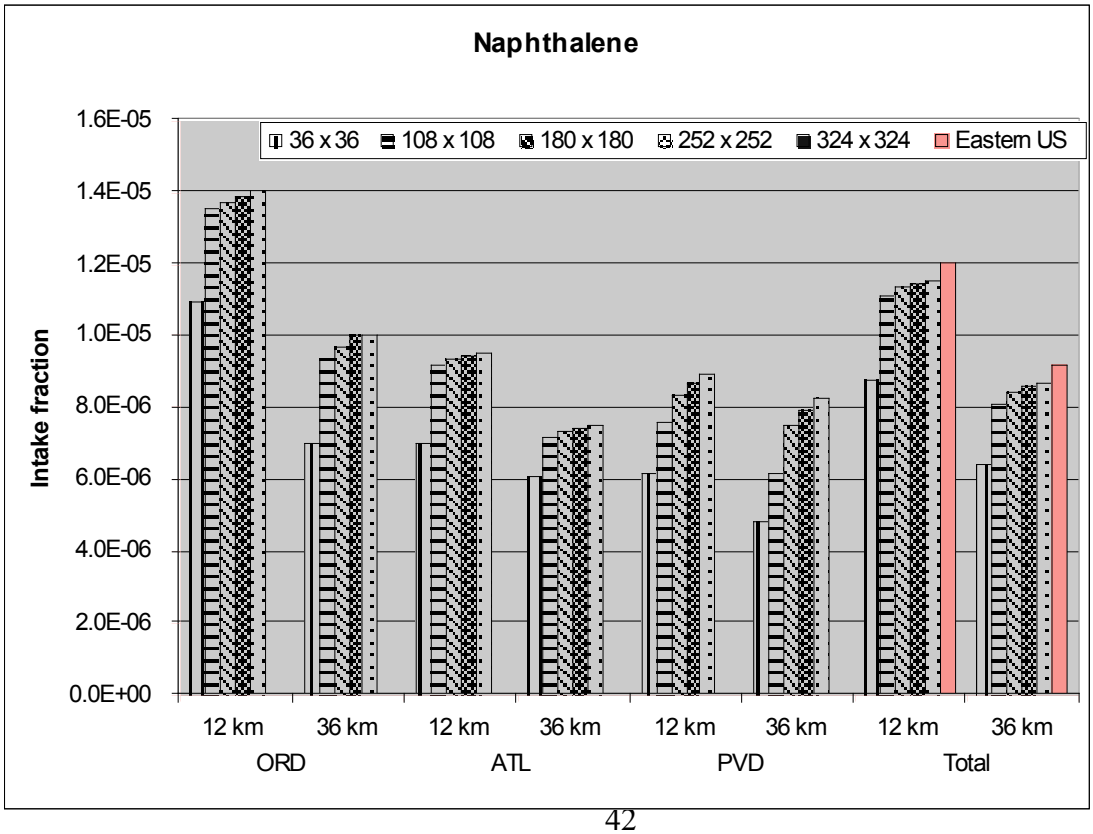


Figure 1, continued





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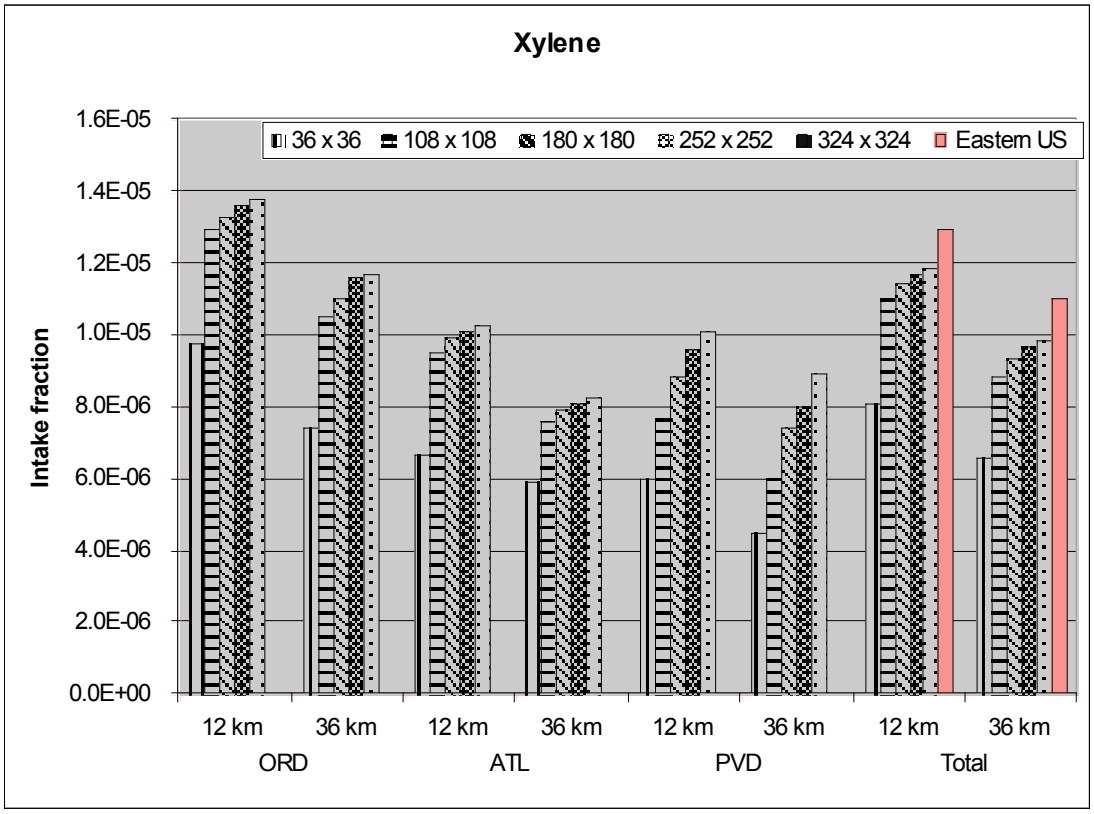


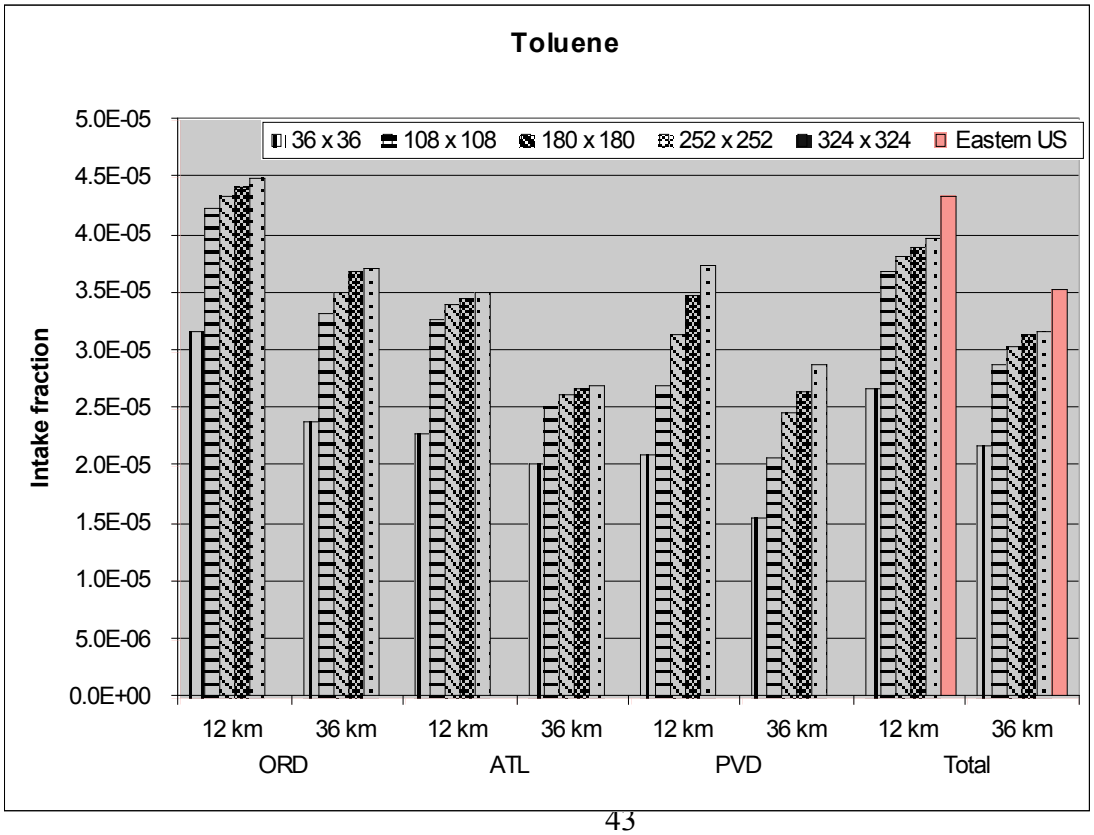


Figure 1, continued

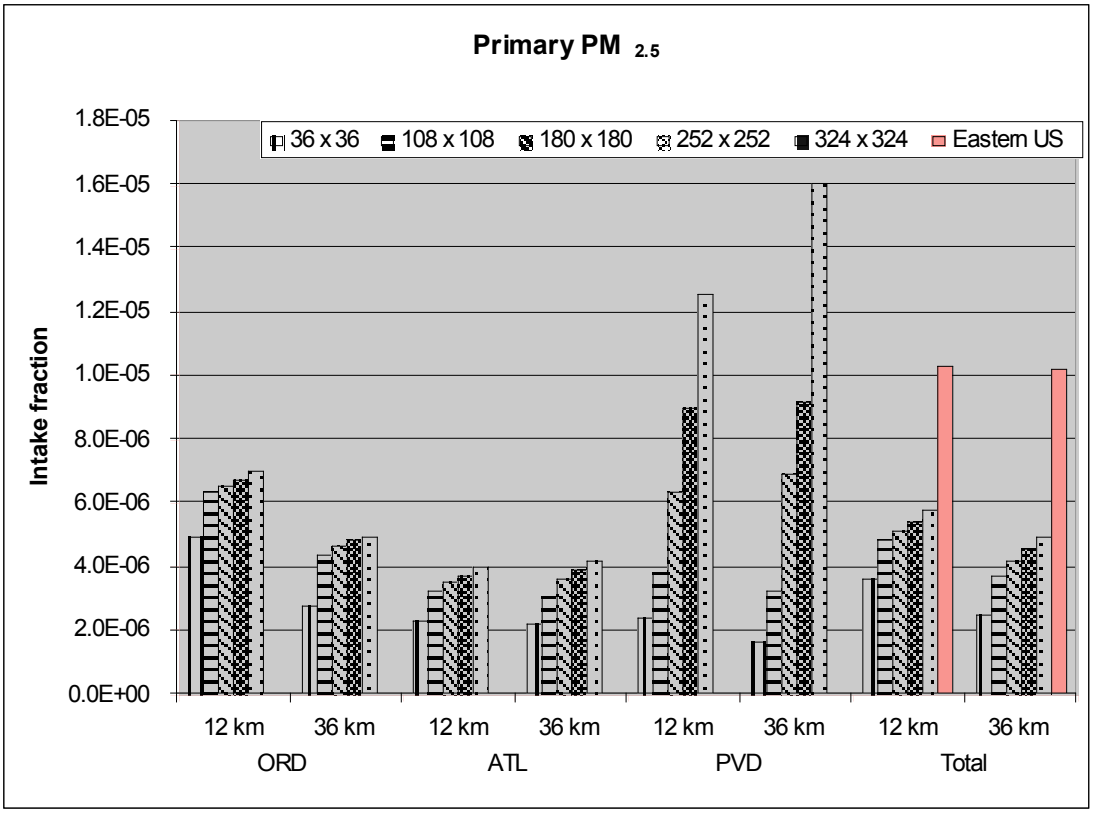


Figure 2: Maps of total population risk by grid cell using 12 km resolution CMAQ runs. The values in each grid cell represent number of deaths per year multiplied by 10^6, and the breakpoints for the categories represent the 10^{th}-90^{th} percentiles, 95^{th}, and 99^{th} percentiles (red = 99^{th}-100^{th} percentiles, pink = 95^{th}-99^{th} percentiles).

Particulate matter

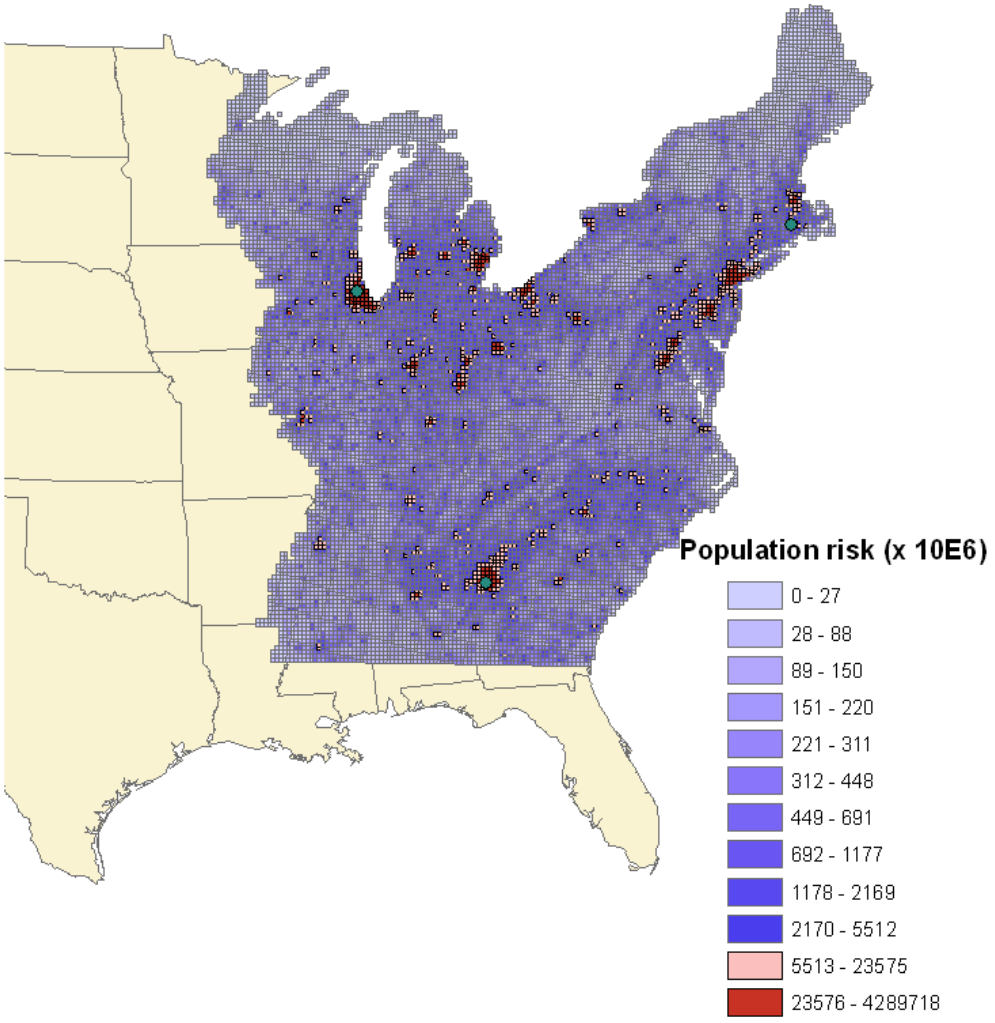


Figure 2, continued

Formaldehyde

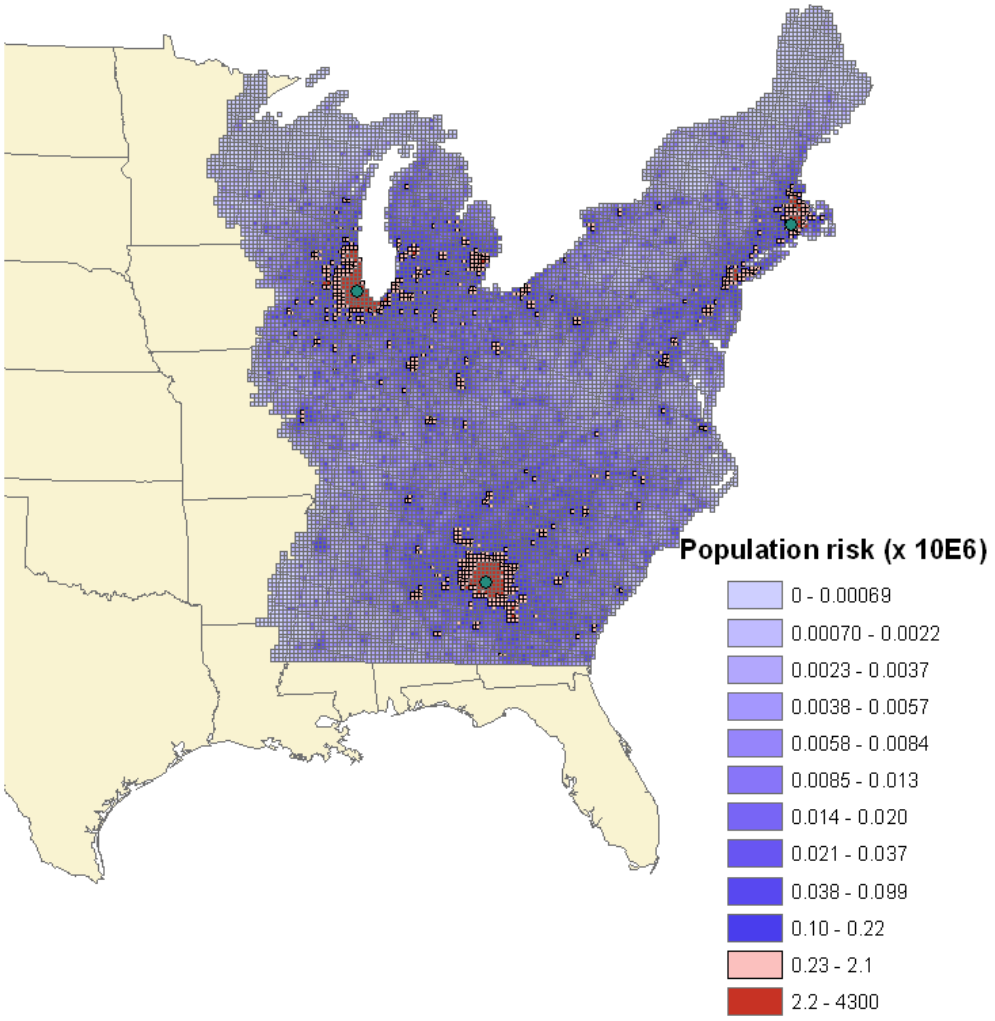


Figure 2, continued

1,3-butadiene

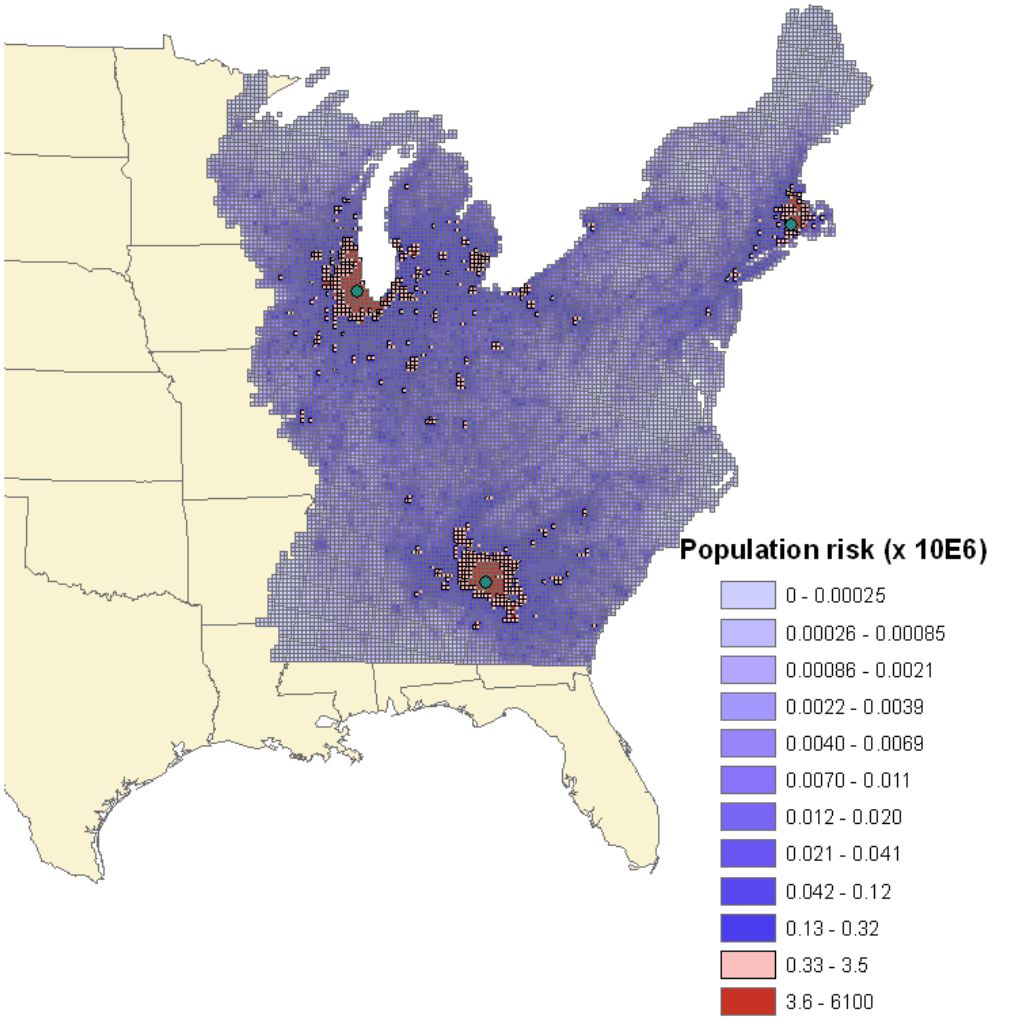


Figure 3: Maps of total population risk for fine particulate matter by grid cell using 12 km resolution CMAQ runs, focused on areas near the three airports. The values in each grid cell represent number of deaths per year multiplied by 10^6, and the breakpoints for the categories represent the 10^{th}-90^{th} percentiles, 95^{th}, and 99^{th} percentiles (red = 99^{th}-100^{th} percentiles, pink = 95^{th}-99^{th} percentiles).

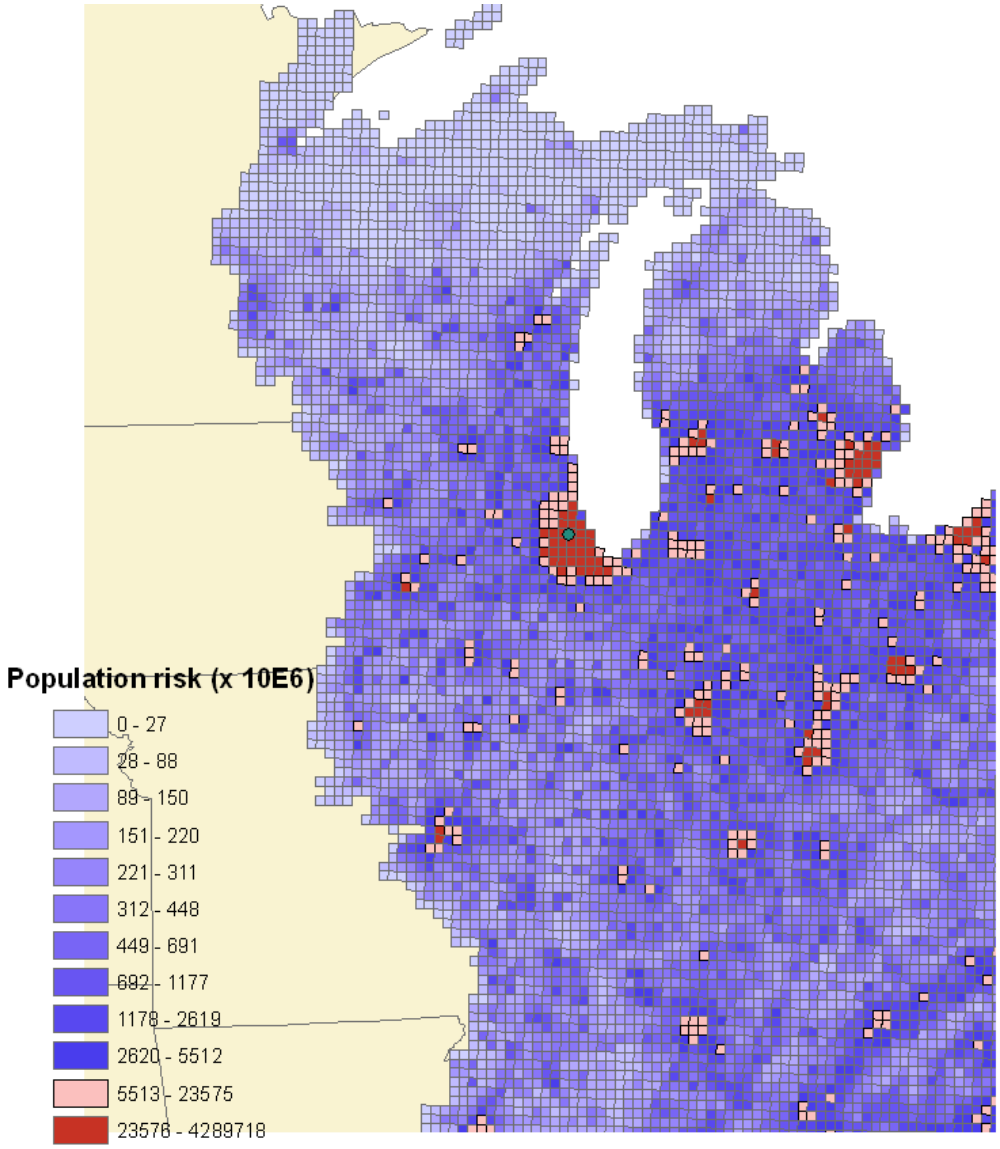


Figure 3, continued

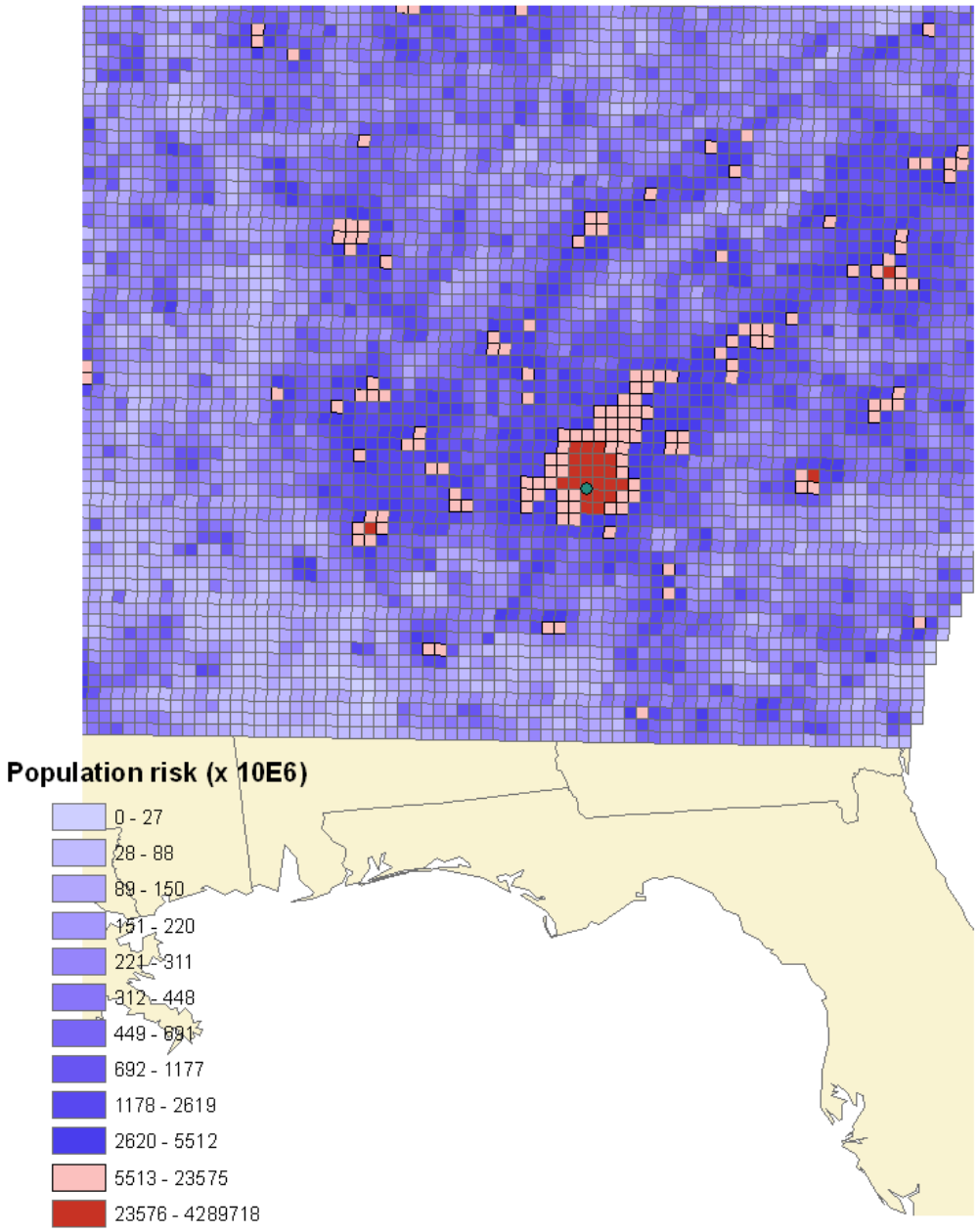
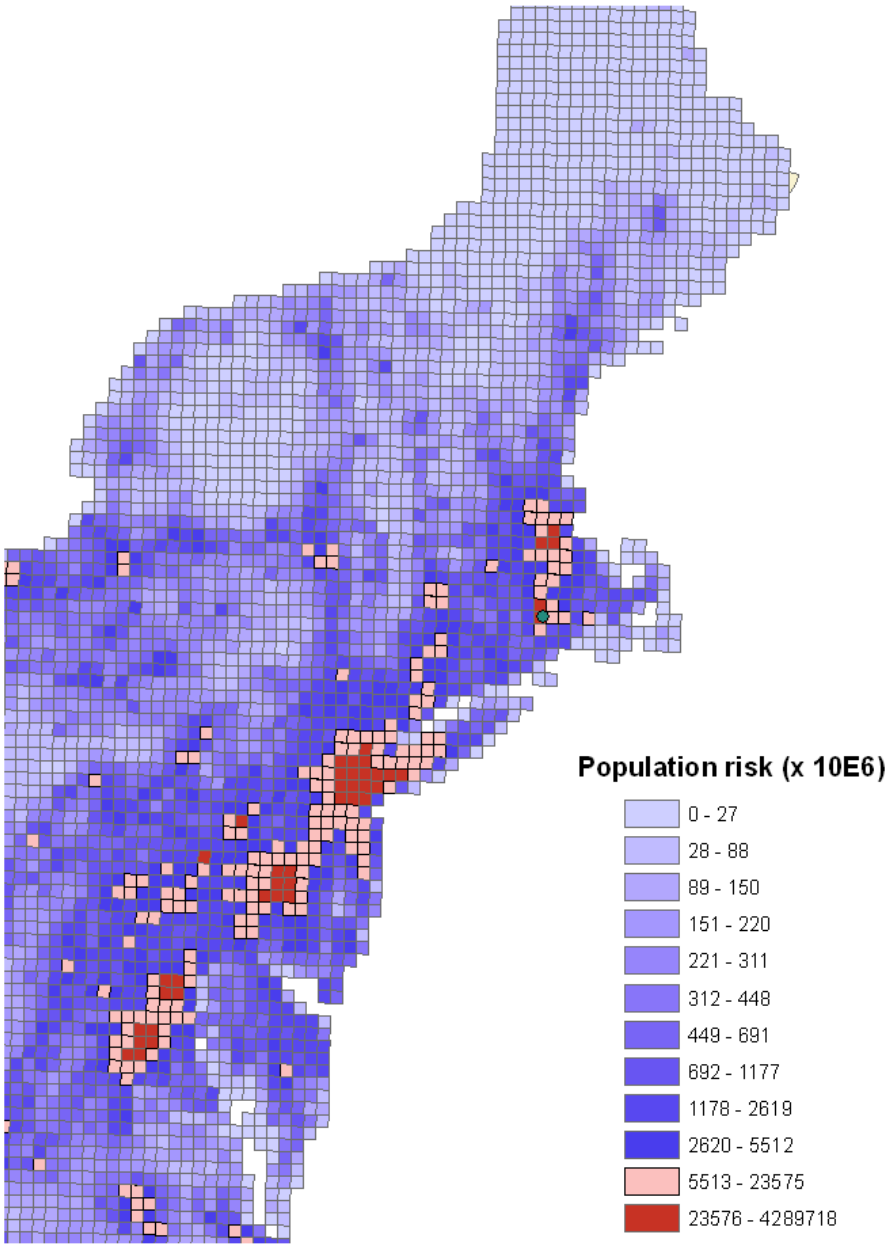


Figure 3, continued



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