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Emission Scenarios and the Health Risks Posed by Priority Mobile Air Toxics in an Urban to Regional Area: An Application in Nashville, Tennessee

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ABSTRACT

Toxic air pollutants, also known as hazardous air pollutants, are those that are known or suspected to cause cancer or other serious health effects, such as birth defects or adverse environmental outcomes. The aim of this research was to predict air toxics related health risks due to different emission scenarios by linking Models-3/CMAQ and cancer risk assessments. To demonstrate the effectiveness of this approach, this study was performed on the priority mobile source air toxics (PMSAT) of benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and diesel particulate matter (DPM), based on data from 2003. The analysis was carried out in the eastern US, and mainly in Nashville, TN. Ten emissions scenarios were examined, including a 2020 scenario with the effects of on-road mobile source regulations. The results show that DPM poses a cancer risk that is 4.2 times higher than the combined total cancer risk from all of four other PMSAT. These high cancer risk levels are mainly due to non-road sources (57.9%). The main cancer risk from acetaldehyde, benzene, formaldehyde, and 1,3-butadiene (4HAPs) is due to biogenic sources, which account for 32.2% of this risk, although these cannot be controlled. Excluding DPM, the main on-road cancer risk contribution was due to the air toxics generated by gasoline light duty vehicles (LDVs), principally benzene and 1,3-butadiene. The scenario for 2020 showed reductions in the adverse health effects related to DPM and 4HAPs of 32.8 and 19.4%, respectively. This research provides strong evidence that reducing ambient DPM concentrations will lead to greater improvements in human health than other air toxics, indicating that better technologies and regulations must be applied to mobile diesel engines, as these have more significant adverse health effects than non-road diesel sources.

Keywords: Diesel particulate matter; CMAQ; Modeling; Health risk assessment; Mobile sources air toxics.

INTRODUCTION

Air toxics, which are also called hazardous air pollutants (HAPs), are those pollutants known or suspected to cause cancer and other serious health or environmental effects. While the harmful effects of HAPs are of particular concern in areas closest to where they are emitted, they can also be transported and affect the health and welfare of populations in other geographic areas. Most HAPs originate from anthropogenic sources, including point, area, and mobile sources. In 1999, the mobile sources contributed approximately 50 percent of the total urban HAPs in the U.S. Some HAPs are released in major amounts from natural sources too, called biogenic sources. The fast growth of the mobile sources indicates that some HAPs would increase if the community does not improve the fuels or does not use cleaner vehicle technologies, mainly those PMSATs, which pose the highest health risk on humans.

Since 1987, USEPA has sponsored the Urban Air Toxics Monitoring Program (UATMP) to characterize the composition and magnitude of urban air pollution (USEPA, 2004). In 2003, there were about 59 HAPs monitoring sites in operation, which include 37 urban locations. These data have been used to assess health risk for particular areas (Pratt *et al.*, 2000). However, this strategy is too expensive to monitor every pollutant everywhere.

Air quality models are valuable air quality management tools. They estimate the HAPs concentrations at many locations and the number of the locations in a model far exceeds the number of monitors in a typical ambient monitoring network. Therefore, the integration of advanced air quality modeling methodologies and health risk assessment techniques is extremely powerful to air quality management, policy, and rulemaking issues, principally to analyze emission scenarios, new strategies, and future growth effects over a wide spatial area. Currently, EPA has used a Gaussian plume model (ASPEN) and an exposure model to estimate the annual HAPs concentrations to assess

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health risk based on the National-Scale Air Toxics Assessment (NATA). This is a state-of-the-science screening tool for State/Local/Tribal Agencies to prioritize pollutants, emission sources and locations of interest for further study in order to gain a better understanding of risks (Linder *et al.*, 2008; Ozkaynak *et al.*, 2008; USEPA, 2012). The model has been performed for local scale effects on ambient concentrations from emitted HAPs that have long atmospheric lifetimes, slow loss rates, without biogenic emissions, and no photochemical production. For HAPs, such as formaldehyde, acetaldehyde, acrolein, 1,3-butadiene, and benzene, several of the assumptions fail.

HAPs in the atmosphere are difficult to model because they have half-lives varying from a few minutes to over two years. They can be produced in the atmosphere from other HAPs and non-HAPs. They are temporally variable having large diurnal variations such as secondary HAPs. Some HAPs are produced and destroyed in a cyclical set of chemical reactions involving VOC, OH, NO_3, O_3, and sunlight. Finally, they exist as gases, particles, both gases and particles, or in aqueous phase. As a result, differences in some HAPs and VOC emissions and weather patterns contribute to seasonal differences in HAPs concentrations from urban to urban areas.

The modeled HAPs ambient concentrations and the health risk assessment results can be improved by using a model that better simulates the transport and fate of these compounds, such as the state-of-art Community Multi-scale Air Quality model (Models-3/CMAQ) (Byun and Ching, 1999). Thus, the overall objective of this study was to develop a model protocol to assess the public health risk caused by the chronic exposure to the mobile source air toxics (MSATs) on an urban to regional area, based on different emissions scenarios by linking the annual HAPs concentrations predicted by the advanced air quality model Models-3/CMAQ, with the life time risk factors associated to cancer effects. This study used concentrations as a proxy for exposure to pollutants of ambient origin and considered the differences of the HAPs concentrations among the emission scenarios, instead of absolute values. To demonstrate the system's effectiveness, this study was performed on PMSATs, and was applied to Nashville and projected to the Southeast US as an example, using available 2003 urban HAPs monitoring data and the 2002 NEI grew to 2003.

In Tennessee, Nashville is an urban area included in the UATMP, which measures 33 HAPs in two monitors, East Nashville Health Clinic (EATN) and Lockeland Middle School (LOTN), working since May and April of 2002, respectively. The EATN (47-037-0011) site is located on the roof of East Health Center, which is north (predominately downwind) of downtown Nashville and is a population-oriented site predominantly influenced by primarily commercial and mobile sources. Population residing within 10 miles of the monitoring station is 518,357.

The LOTN (47-037-0023) site is a core site located on the roof of Lockland School, which is in the heart of downtown Nashville. This is also a population-oriented site influenced primarily by commercial and mobile sources. Population residing within 10 miles of the monitoring station is 552,749. These sites were selected for the following reasons: they provided secure locations with the necessary electrical service, represented areas that were not in the immediate vicinity of large air pollution sources, and were in the proximity and downwind from areas with the highest population density in metropolitan area. It is important to note that these sites are near substantial interstate routes and local traffic corridors, such as I-40. Air toxics concentrations at these sites would not be indicative of average concentrations throughout Nashville nor could specific conclusions be drawn from concentrations at these sites concerning concentrations at any other location. Actual concentrations may be higher adjacent to industrial facilities and may be lower in less densely populated areas. However, the results of the ambient monitoring at these sites provide concentrations to which the majority of the Nashville population would be exposed because monitoring occurred near areas with the densest population in Nashville.

As an approach, this study simulated a 4-months period to represent the whole year of 2003. Each month represented a season for the Southeastern US. The year 2003 was chosen because of available HAPs monitoring data for Nashville.

METHODOLOGY

Conceptual Model's Development

The overall approach included running CMAQ version 4.4 and CMAQ-Air Toxics (AT) models with and without the following sources categories: on-road LDVs, heavy-duty vehicles (HDVs), diesel fueled sources (DFS), on-road DFS, and biogenic sources, as well as a future 2020 year with the effects of on-road MSATs regulations. The year 2020 was selected as a future scenario to compare the projections estimated by EPA in its study Analysis of the Impacts of Control Programs on Motor Vehicle Toxic Emissions and Exposure Nationwide (USEPA, 1999a, b). The cancer risk expected in a particular area of the modeling domain can be estimated and compared for different emission scenarios. It may be noted that Models-3/CMAQ was assumed as the inhalation exposure model, in other words, the population is exposed to the outdoor ambient HAPs concentrations without taking into account indoor effects. In addition, the risk assessment was designed to be a picture for measuring progress in reducing risks from exposure to air toxics. It then assumed individuals spend their entire lifetimes (70 years) exposed to these HAPs.

The analysis approach involves considering the difference in mass concentrations and health risk values among the studied emission scenarios as compared to the base case scenario rather than the absolute mass concentration or health risk values. This assumes that the factors that contributed to the under and over prediction of those HAPs concentrations would contribute similarly in all the scenarios considered in the analysis, causing minimal effects on the differences among the scenarios. These area, point, on-road, non-road, and biogenic emissions were temporal and spatially allocated using the advanced emissions processing model Sparse Matrix Operator Kernel Emissions (SMOKE) version 2.0

(UNC, 2005). On-road sources were predicted by using National Mobile Inventory Model (NMIM) (USEPA, 2005), for the whole modeling domain, whereas the 2003 NEI was used for point, area, and non-road sources in the modeling domain. The meteorological variables were generated for March, June, September, and December of 2003 through the mesoscale model (MM5) version 3.7 developed by the National Center for Atmospheric Research (NCAR) at the Pennsylvania State University (PSU) and processed by the meteorology-chemistry interface processor (MCIP) version 2.2. For this study, each month was set to start five days earlier to reduce the initial conditions effects.

Description of the Modeling Domain

The domain selected included most of the central eastern region of the United States, and therefore, the size of the domain was performed with grid cells of 36 × 36-km, consistent with the objective of this study in the extent of human daily activity. Because the results of this study were focused principally in the Nashville metropolitan area, a high diesel mobile sources traffic, the domain was selected such that this city was approximately at the center of the domain surrounded by other 23 states to minimize the effect of boundary conditions and pollution transport (Doraiswamy *et al.*, 2007).

Emissions Scenarios

The methodology consisted of running the SMOKE 2.0 and CMAQ models with and without the source emissions scenarios as illustrated in Table 1. The base case was run with all sources included. The scenarios NO DFS, NO ONROAD_DFS, NO LDVs, NO HDVs, were estimated through control matrices for the corresponding source (s). Those source categories were eliminated using the source classification codes (SCC) through a control matrix for each scenario. The scenarios NO ONROAD and NO BIO were estimated running the SMOKE 2.0 and merging all the emissions sources without on-road and biogenic emissions, respectively. Finally, the scenario for the year 2020 was estimated merging the 2003 point, area, non-road, and biogenic emissions with the 2020 on-road emissions estimated by NMIM.

Two extra scenarios were run to consider the DPM effect on heavy-duty vehicles and over the year 2020. Those scenarios included running the base case without heavy-duty diesel vehicles (NO HDDVs) and the year 2020 without DFS from point, area, and non-road 2003 emissions, and without DFS from the 2020 on-road emissions generated by NMIM (YEAR 2020_NODFS). These two scenarios were also estimated through control matrices for the corresponding source (s). The difference between the base case scenario and the NO DFS scenario were the DPM emissions and concentrations for the base case run, whereas the difference between the scenario YEAR 2020 and the YEAR 2020_DFS scenario were the DPM values for the year 2020. Those DPM emissions were estimated considering the addition of the following $PM_{2.5}$ species defined in SMOKE2.0: Elemental carbon (EC), primary fine particulate matter (PMFINE), primary nitrate (PNO3), primary organic aerosols (POA), and primary sulfate (PSO4) (UNC 2005). The difference between the NO ONROAD DFS scenario and the NO DFS were the DPM emissions from the on-road DFS. Whereas, the difference between the NO HDDVs scenario and the NO DFS scenario were the DPM results from the HDDVs. A future 2020 scenario for non-road, area, and point sources were not simulated because no activity growth data were available for the 24 states at the time this study was conducted. The effect of non-road regulations on diesel engines were not available in the model NMIM or NONROAD at the time this analysis was conducted. Because secondary diesel particulate matter is formed when gaseous emissions from diesel engines interact with other compounds in the atmosphere (Ning *et al.*, 2004), then for a health risk point of view, diesel particulate matter can be estimated base on primary diesel $PM_{2.5}$ (Diaz-Robles *et al.*, 2008, 2009). This approach did not account for the PM sources that are apportioned between primary and secondary sulfate and nitrate aerosols, since there currently is no way to determine how much of the sulfate and nitrate are primary and how much secondary in the Aitken and accumulative modes on CMAQ 4.3 (Byun and Ching, 1999). However, the sulfate and nitrate concentrations in the DPM emissions modeled by Diaz-Robles *et al.* (2008) on Atlanta, GA, Birmingham, AL, Nashville, TN, Memphis, TN, and Knoxville, TN for

Table 1. Emission Scenarios on SMOKE 2.0 and CMAQ.

Year	Emission Scenario	Name Scenario	Objective
2003	All Sources Present (Base Case)	BC	Base Case
2003	Base Case Without On-Road Sources	NO ONROAD	Contribution of on-road sources to air toxics
2003	Base Case Without DFS	NO DFS	Contribution of DFS to DPM
2003	Base Case Without On-Road DFS	NO ONROAD_DFS	Contribution of on-road DFS to air toxics and DPM
2003	Base Case Without LDVs	NO LDVs	Contribution of LDVs sources to air toxics
2003	Base Case Without HDVs	NO HDVs	Contribution of HDVs sources to air toxics
2003	Base Case Without Biogenic Emissions	NO BIO	Contribution of biogenic sources to air toxics
2020	In effect MSATs regulation for 2020	YEAR 2020	Contribution of in effect MSATs regulations to air toxics
2003	Base Case Without HDDVs	NO HDDVS	Contribution of HDVs sources to DPM
2020	In effect MSATs regulation for 2020 without DFS	YEAR 2020_NODFS	Contribution of in effect MSATs regulations to DPM

summer 2003 were in average as low as 1.82% and 0.16%, respectively, and neither species are considered carcinogenic.

Health Effects Estimation

With the cancer risk Eqs. (1) and (2), the inhalation unit risk (IUR) values from the Table 2, and the annual acetaldehyde, benzene, 1,3-butadiene, formaldehyde, and DPM concentrations from CMAQ, the individual and cumulative cancer risks were estimated for the base case and for each analyzed scenario described in Table 1. The cumulative cancer risk posed by gaseous air toxics (acetaldehyde, benzene, 1,3-butadiene, and formaldehyde) was called 4HAPs, and the cumulative cancer risk for those 4 air toxics and DPM was called 4HAPs + DPM. The IUR values used in NATA are quite different than used in this current study; in fact NATA is starting to use a much lower value for formaldehyde toxicity than in IRIS, and that benzene toxicity is characterized in IRIS with a range (Apelberg *et al.*, 2005; Marshall *et al.*, 2005; Linder *et al.*, 2008; Ozkaynak *et al.*, 2008). As our study compared different scenarios and its contributions on cancer risk, the 2003 IUR values used by IRIS were reasonable. Those cancer risk difference values for the base case and the reductions due to the emissions scenarios were estimated for Nashville and were plotted to see the spatial distribution in the Eastern U.S.

The annual mean HAPs concentration (Ci) from a location is multiplied by its IUR to produce a cancer risk. HAPs with cancer risks greater than 1×10^{-6} are considered a potential human health concern.

$$\text{Cancer Risk} = C_i \left[\frac{\mu g}{m^3}\right] \times \text{IUR}_i \left[\frac{m^3}{\mu g}\right] \quad (1)$$

Assuming an additive air toxics mixture effect, the cumulative cancer risk is defined by the Eq. (2).

$$\text{Cumulative Cancer Risk} = \sum C_i \left[\frac{\mu g}{m^3}\right] \times \text{IUR}_i \left[\frac{m^3}{\mu g}\right] \quad (2)$$

RESULTS AND DISCUSSIONS

The inhalation risk estimate differences in this study were based on annual average ambient exposures for a wide population distribution. Tables 4 and 5 show the maximum contributions on annual concentrations and the inhalation cancer risk performed for each analyzed scenario at Nashville, TN, based on the annual concentrations of Table 3 and the IURs of Table 2. It should be noted that Table 4 shows the maximum contributions on the annual concentrations of air toxics in Nashville due to different emission scenarios, noting that the main contributions of atmospheric acetaldehyde come from light vehicles and biogenic sources, at 52.2 and 49.8%, respectively. This behavior is similar to formaldehyde, but the contribution from biogenic sources is much more significant, 53.9%. In the case of benzene and 1,3-butadiene, the main contribution is due to light gasoline vehicles, at 70.2 and 72.3%, respectively. Finally, the main contribution of DPM is due to the HDV at 49.2%. In 2020, the reduction in the concentrations of these toxic due to mobile sources will not be as significant, especially in diesel vehicles with a maximum of 38.1% in Nashville. Therefore, better regulations and technologies on mobile sources will be needed to reduce public exposure to this contaminant in Nashville. Fortunately, the 2020 concentrations of benzene and 1,3-butadiene will be reduced by 57.0 and 62.9%, respectively, due to the implementation of better standards on light duty vehicles in the U.S.. Finally, acetaldehyde and formaldehyde reductions are not significant in 2020 due to mobile sources in Nashville, because biogenic sources generate a significant contribution to the secondary formation of these toxics. The Table 5 shows that the main contributions on those 4HAPs were from biogenic sources at 32.2%, which generated high secondary acetaldehyde and formaldehyde in the summer season. This condition was followed for the scenario that did not consider on-road sources with a 27.5% contribution, where the main reductions were the result of air toxics contributions generated by gasoline LDVs, mainly

Table 2. Carcinogenicity of PMSATs.

MSATs	IURs $[m^3/\mu g] \times 10^{-6}$	Reference
Acetaldehyde	2.2	EPA/IRIS[a]
Benzene	7.8	EPA/IRIS
Formaldehyde	13.0	EPA/IRIS
1,3-Butadiene	30.0	EPA/IRIS
DPM	300.0	CalEPA[b]

[a] IRIS. EPA`s Integrated Risk Information System.
[b] CalEPA. California Environmental Protection Agency.

Table 3. Modeled PMSATs Annual Concentrations in Nashville, 2003.

Scenario	Annual Concentration $[\mu g/m^3]$				
	Acetaldehyde	Benzene	Butadiene	Formaldehyde	DPM
Base Case	1.109	0.682	0.050	2.248	0.545
Non Biogenic	0.753	0.673	0.052	1.356	0.545
Non On-Road	0.670	0.192	0.010	1.895	0.316
Non DFS	1.104	0.670	0.046	2.137	0.000
Non On-Road DFS	1.111	0.676	0.047	2.197	0.316
Non LDV	0.700	0.225	0.015	1.985	0.545
Non HDV	1.085	0.650	0.044	2.171	0.319
On-Road 2020	0.825	0.311	0.020	2.013	0.367

Table 4. Maximum Contributions on Annual Concentrations in Nashville, 2003.

Scenario	Acetaldehyde	Benzene	Butadiene	Formaldehyde	DPM
No Biogenic	49.8%	4.6%	–10.0%	53.9%	
No On-Road	56.6%	75.2%	82.7%	33.7%	48.3%
No DFS	4.6%	2.8%	16.0%	12.2%	100.0%
No On-Road DFS	1.9%	1.4%	11.7%	6.9%	48.3%
No LDV	52.2%	70.2%	72.3%	24.6%	2.2%
No HDV	4.8%	5.2%	17.0%	9.5%	49.2%
On-Road 2020	37.1%	57.0%	62.9%	23.2%	38.1%

Table 5. Inhalation Cancer Risk by Scenarios for those 4HAPs and DPM at Nashville, TN.

Scenario	4HAPS × 10^{-6}	Contribution [%]	DPM × 10^{-6}	Contribution [%]	4HAPS + DPM × 10^{-6}	Contribution [%]
Base Case	38.5		157.8		196.3	
No Biogenic	26.1	32.2%	157.8	0.0%	183.9	6.3%
No On-road	27.9	27.5%	91.4	42.1%	119.3	39.2%
No DFS	36.8	4.4%	0.0	100.0%	36.8	81.2%
No On-road DFS	37.7	2.1%	91.4	42.1%	129.1	34.2%
No LDV	29.5	23.2%	156.8	0.6%	186.3	5.1%
No HDV	37.0	3.9%	93.2	40.9%	130.2	33.7%
On-road 2020	31.0	19.4%	106.2	32.7%	137.2	30.1%

benzene and 1,3-butadiene. Those values were similar with those found in other studies (Luecken *et al.*, 2006; Cook *et al.*, 2007; Isakov *et al.*, 2007, 2009). However, these results are different than those found in the NATA study for 1999, mainly because of the effect of biogenic emissions and secondary formation considered in our CMAQ study. Also we used the 2003 NEI emission inventory with a 36 km domain, which can generates some uncertainties, however, this study was based on emissions scenarios ratios or differences over the same emission inventory and modeling domain.

As the scenario 2020 included the on-road sources regulations only, the 4HAPs cancer risk showed a reduction as low as 19.4%, which were not significant to achieve a strong air quality improvement at Nashville for the year 2020. If there are no on-road sources or DFS in the modeling, the cancer risk reduction is not expected to be reduced significantly for those vapor air toxics. Major sources, like biogenic and point sources, are important sources of acetaldehyde emissions or its precursors, as well as area and non-road sources are important sources of benzene, 1,3-butadiene, and formaldehyde. The rest of the scenarios showed reductions lower than 4.4%, indicating that DFS and HDVs were not important vapor air toxics contributors at Nashville, TN. Our results of those 4HAPs are dependent upon the IUR used. We used the state-of-the-art IURs of the USEPA for 2005, but these could be adjusted for the scientific community in the future due to the weight of evidence. A particular issue is that both formaldehyde and acetaldehyde are treated as genotoxic carcinogens in this study, with a risk increasing linearly with concentration without a threshold. This could generate some uncertainties, because some countries regulate them as having a threshold and therefore not impacting upon cancer at typical ambient concentrations.

DPM posed a cancer risk that was 4.2 times higher than the combined total cancer risk from all other four air toxics simulated on the BC scenario. Those high cancer risk levels were caused mainly by the DMP emitted from goods transportation and construction engines. This higher DPM cancer risk was also estimated by one study (Schneider and Hill, 2005), who reported that DPM posed a cancer risk that was 7.5 times higher than the combined total cancer risk from all those other 33 UATs nationwide. The DPM's IUR was taken uncritically from the California EPA, which could be over-estimated; however, it was used because the USEPA does not have enough evidences for the DPM as carcinogen, generating important uncertainties.

The main reductions in DPM cancer risk were the result of the scenario that did not consider non-road DFS sources with 57.9% contribution, which is the difference between the scenario without DFS and the scenario without on-road DFS. In other words, non-road sources produced the highest contribution on ambient DPM concentrations and its associated cancer risk. The scenario without DFS on-road sources showed a 42.1% contribution, where the main contributions were the DPM generated by HDDVs. For the scenario 2020, the DPM cancer risk showed a 32.7% reduction. If a future 2020 scenario includes the non-road sources with all fuel and technological regulations, like on-road sources, the DPM cancer risk reduction could be important. Therefore, better DPM reduction strategies must be considered on mobile sources to reduce its cancer risk in Nashville, TN. The cancer risk contribution scenarios associated to 4HAPs plus DPM followed similar trends than the contributions that came from DPM. This indicated that DPM generated the highest lifetime cancer risk excess among the other air toxics in Nashville, TN. This results are similar than those found by Cook *et al.* (2007).

The Figs. 1, 2, and 3, show the estimated lifetime inhalation

cancer risk excess from the 4HAPs, DPM, and 4HAPs + DPM, respectively for some scenarios. In general, the plots show that no area fulfilled the EPA's cancer risk rule of one case over a million people, since the 4HAPs, DPM, and 4HAPs + DPM exceeded four, one, and five in a million risk of cancer over a lifetime of exposure respectively. Higher cancer risk occurred on Southeastern urban areas for those 4HAPs and DPM, principally at Atlanta for the 4HAPS, as shown in Fig. 1(a). For DPM, the highest cancer risk occurred mainly in the north east urban areas, mainly at Chicago, IL, Indianapolis, IN, followed by Atlanta, GA, Nashville, TN, Raleigh, NC, Memphis, TN, among others, as shown in Fig. 2(a).

The 4HAPs cancer risk was influenced principally by secondary acetaldehyde and formaldehyde generated in the summer season by the biogenic sources effect, as shown in Fig. 1(b). The scenario for the year 2020 showed higher 4HAPs cancer risk reductions in Atlanta and Birmingham. The area around the Mississippi river showed the impact of diesel marine engines on DPM cancer risk, which produced a cancer risk between 37.5 and 75 per million populations. Finally, it was evident that the population was exposed to greater than five in a million level of cancer risk in the whole domain, especially due to DPM in urban areas.

CONCLUSIONS

Considering that this research on air toxics emission scenarios was based on relative analysis rather than estimates of absolute exposure concentrations and health risk values, the following conclusions were reached. The health risk assessment associated with the priority MSATs control will continue be one of the most important issues in the EPA's air quality rulemaking, principally for DPM. In this context, the proposed protocol through Models-3/CMAQ was demonstrated and can be used for decision makers in

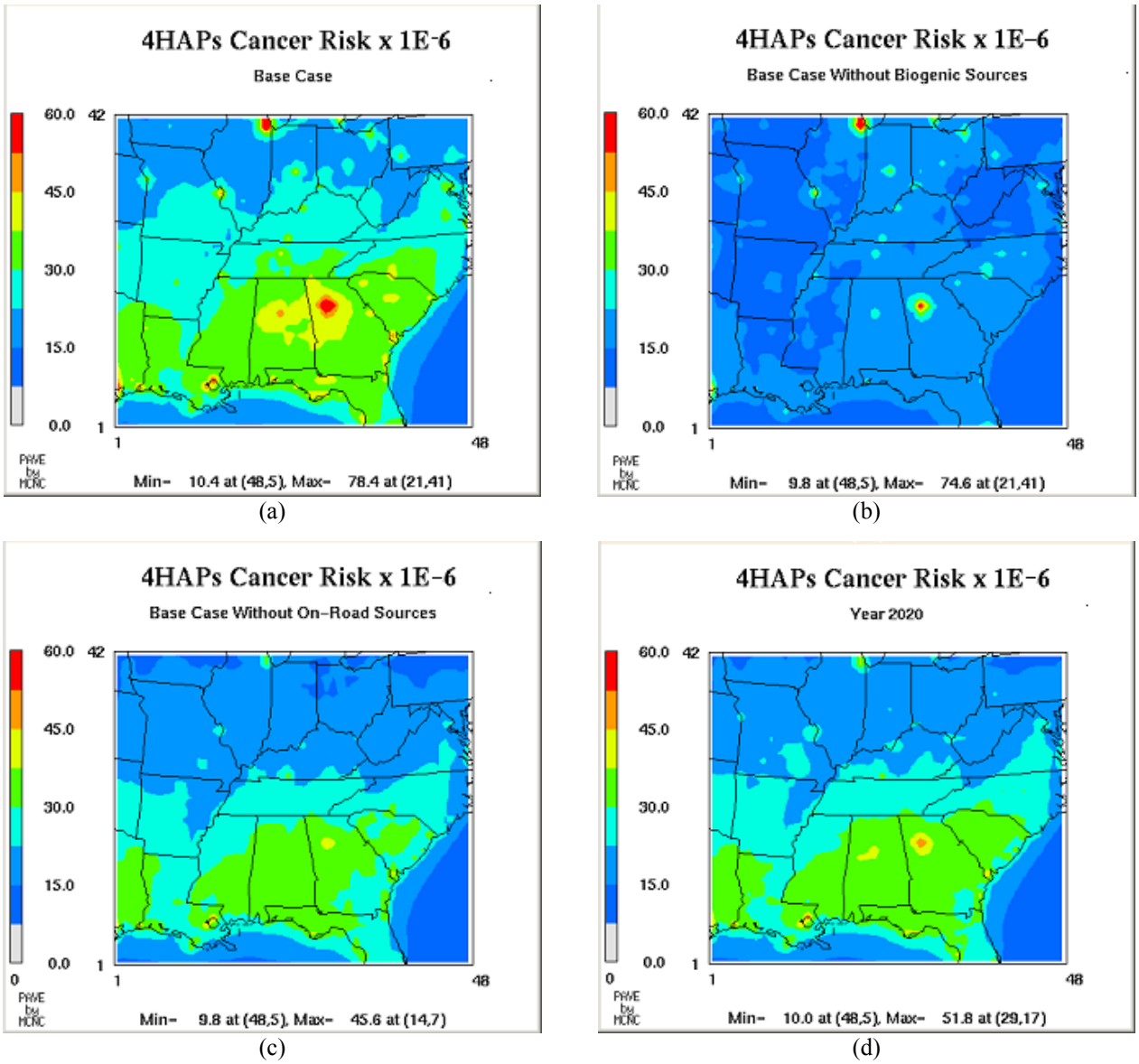

Fig. 1. Spatial Variation of the 4HAPs Cancer Risk by Scenarios.

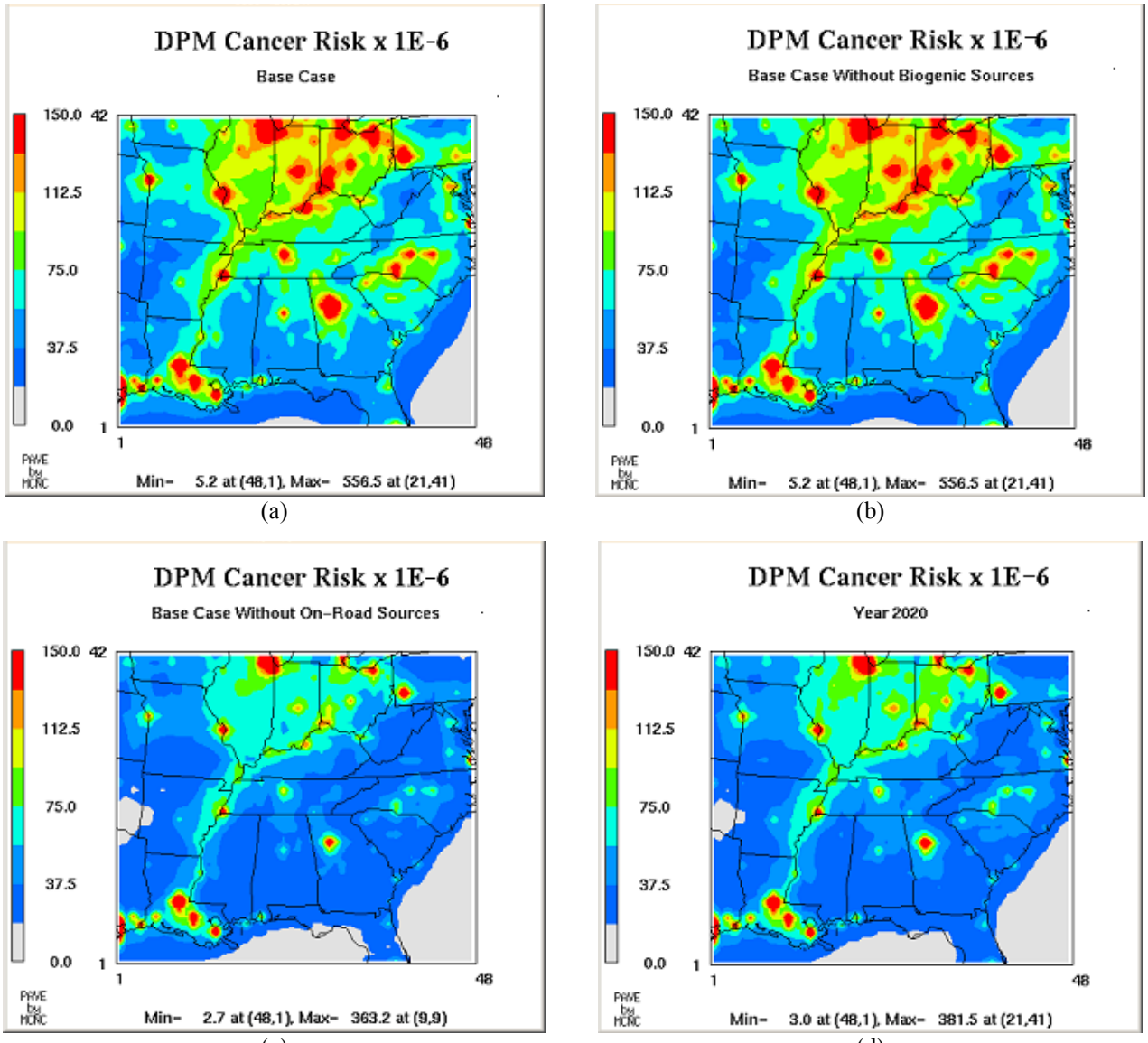

Fig. 2. Spatial Variation of the DPM Cancer Risk by Scenarios.

the quantitative assessment of new policies that will affect the public health and the air quality by air toxics; however, it is important to reduce the uncertainty of using better IURs. Eliminating emission source categories is clearly not a policy option, but rather was used to help to gain a better understanding of the total magnitude of the health effects associated with these major sources of HAPs, principally of DPM.

Higher formaldehyde and acetaldehyde exposure occurred in the summer season, while higher benzene and 1,3-butadiene concentrations occurred in the winter season. DPM did not show a strong seasonality exposure during the year 2003 in Nashville but its cancer risk was higher than those 4HAPs (Diaz-Robles *et al.*, 2009). In fact, DPM posed a cancer risk that was 4.2 times higher than the combined total cancer risk from all other air toxics simulated in the base case scenario for Nashville. Those high cancer risk levels were due mainly to the DPM emitted from goods transportation and construction engines. The highest cancer risk from DPM occurred in Chicago, Indianapolis, and Atlanta followed by Nashville, Birmingham, Raleigh, and Memphis. The cancer risk from those 4HAPs was not only higher in urban areas, but also was high over rural areas of the Southeastern U.S., mainly the result of secondary formation of acetaldehyde and formaldehyde.

The main cancer risk contributions from those 4HAPs were caused by biogenic sources at 32.2%. This condition was followed for the scenario that did not consider on-road sources with a 27.5% contribution. The main contributions were from the HAPs generated by gasoline LDVs, principally benzene and 1,3-butadiene. The 4HAPs cancer risk showed a reduction as low as 19.4% in 2020. The rest of the scenarios showed contributions lower than 4.4%, indicating that DFS and HDVs were not important vapor HAPs contributors in Nashville, TN. The main contributions in DPM cancer risk were the non-road DFS sources with a 57.9% contribution,

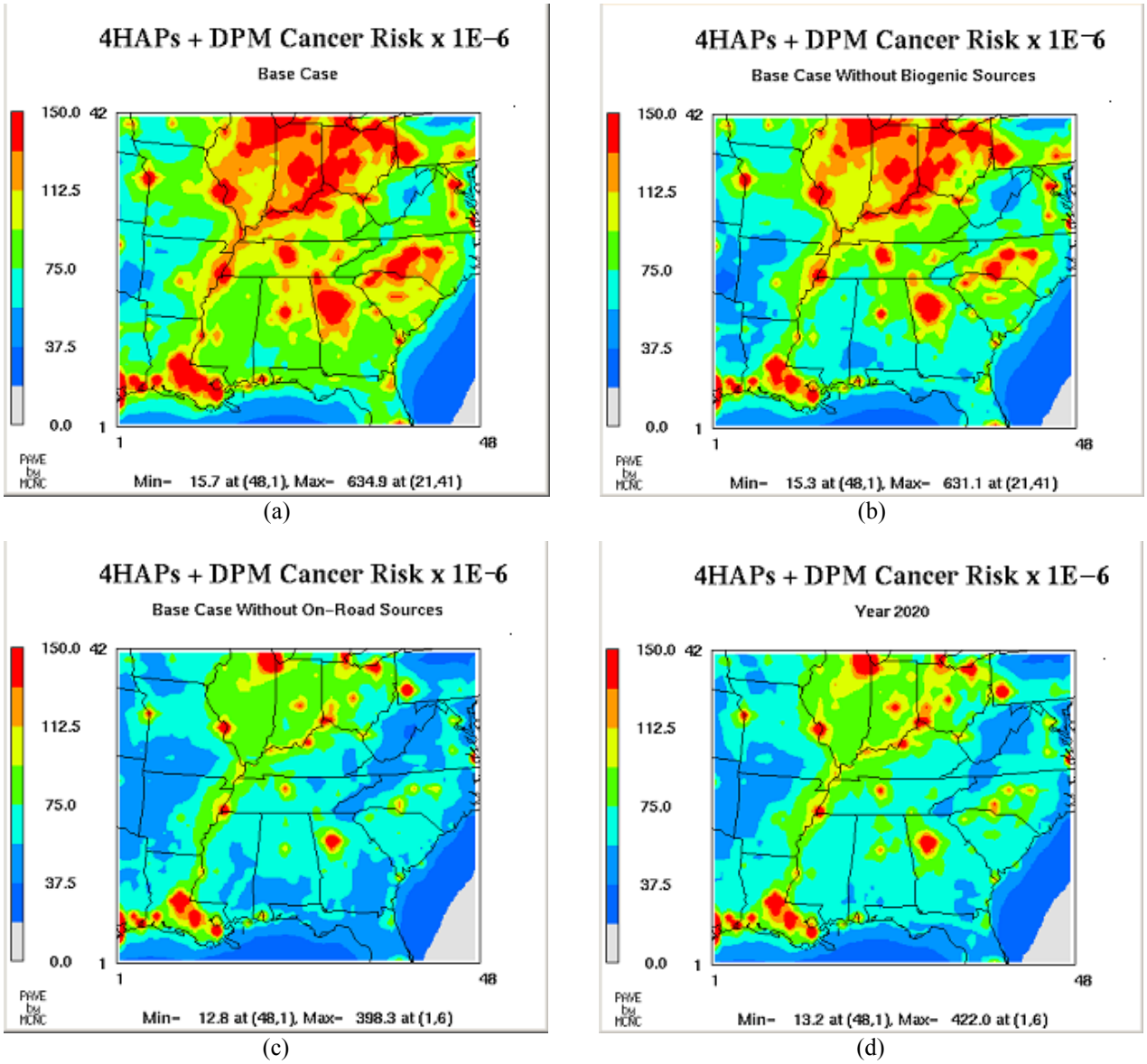

Fig. 3. Spatial Variation of the DPM + 4HAPs Cancer Risk by Scenarios.

followed by the scenario without on-road DFS with a 42.1% contribution. For the on-road DFS, the principal contributions were the DPM generated by HDDVs rather than LDDVs. This research provided strong evidence that reducing ambient DPM concentrations will lead to improvement in human health more than other HAPs in Nashville and the Eastern US, indicating that better technologies and regulations must be applied to the mobile diesel engines, principally, over non-road diesel sources. This approach has inherent limitations because of inability to simulate some primary DPM species, such as sulfate and nitrate. However, their contribution on DPM is negligible and did not alter the relative analysis of this research, and neither of those species is considered carcinogenic.

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