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Characterizing the Toxicity of Polycyclic Aromatic Hydrocarbons from Light-Duty On-Road Fuel Emissions in the United States

Report Prepared for the
Wisconsin Corn Growers Association and
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Researched and written by

Tracey Holloway, Ph.D. and David Abel

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Nelson Institute for Environmental Studies
& Department of Atmospheric and Oceanic Sciences
University of Wisconsin—Madison
Madison, WI 53726; taholloway@wisc.edu

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Table of Contents

Main Findings

- Health research has found consistent evidence for health impacts from polycyclic aromatic hydrocarbons (PAH), both in terms of ambient concentrations and contribution to the toxicity of particulate matter
- Atmospheric chemistry research has identified PAHs as a potentially important contributor to secondary organic aerosol formation, a component of fine particulate matter ($PM_{2.5}$)
- Ethanol blend fuels have been shown to reduce PAH and PM emissions in some studies, but there remains uncertainty as to the overall impact of fuel blending on PAH emissions as a function of blend level
- Ambient measurements of PAHs are limited, so computer models of emissions and air quality play an important role in public health assessment

List of Figures

List of Tables

List of Acronyms

Term	Meaning
ADHD	Attention Deficit Hyperactivity Disorder
ATSDR	Agency for Toxic Substances and Disease Registry
AURAMS	A Unified Regional Air Quality Modeling System
BaP	Benzo(a)-pyrene
BC	Black carbon
BGP	Benzo[ghi]perylene
BTEX	Benzene, toluene, ethylbenzene, and xylenes
CAA	Clean Air Act
CAP	Criteria Air Pollutant
CH_4	Methane
CI	Confidence Interval
CMAQ	Community Multiscale Air Quality Model
CO	Carbon monoxide
EC	Elemental carbon
EPA	United States Environmental Protection Agency
ESCI	Emerging Sources Citation Index
HAP	Hazardous Air Pollutant
IARC	International Agency for Research on Cancer
IPCS	International Programme on Chemical Safety
IVOC	Intermediate Volatility Compounds
MILAGRO	Megacity Initiative: Local and Global Research Observations
MOVES	EPA Motor Vehicle Emissions Stimulator
NAAQS	National Ambient Air Quality Standards
NATA	National Air Toxics Assessment
NEI	National Emissions Inventory
NMHC	Non-methane hydrocarbons
NO_2	Nitrogen dioxide
NO_x	Nitrogen oxides
OC	Organic carbon
O_3	Ozone

List of Acronyms (Concluded)

Term	Meaning
PAH	Polycyclic aromatic hydrocarbons
Pb	Lead
PM	Particulate matter
$PM_{2.5}$	Fine particulate matter
PM_1	Ultrafine particulate matter
PN	Particle number
POA	Primary organic aerosols
SCI	Science Citation Index
SOA	Secondary organic aerosols
SO_2	Sulfur dioxide
SMOG	Surface Meteorology and Ozone Generation
SMOKE	Sparse Matrix Operating Kernel Emissions
SVOC	Semi-volatile organic compounds
TexAQS	Texas Air Quality Study
URR	Unit relative risk
VOC	Volatile organic compounds

Background

Transportation fuel composition has been recognized as a factor in the chemical speciation of hydrocarbon emissions dating back the mid-1960s [*McReynolds et al.*, 1965; *Ebersole and McReynolds*, 1966], including the contribution of anti-knocking additives [*Gagliardi*, 1967]. Emitted hydrocarbons arise from compounds in the fuel mixture ("petrogenic" emissions), as well as hydrocarbons formed during the combustion process ("pyrogenic" emissions) [*Mishra et al.*, 2016]. These emitted hydrocarbons enter the atmosphere, where they are subject to chemical reactions and atmospheric transport. Human exposure to atmospheric concentrations of select hydrocarbons has been associated with cancer and other adverse health outcomes. In addition, chemical reactions involving hydrocarbons impact near-surface ozone (O_3) and the composition of particulate matter (PM), thus playing a role in a wide range of air quality and public health issues.

In considering the potential benefits or drawbacks of fuel composition and fuel additives, there is value in characterizing how fuel hydrocarbon mixtures impact air quality and health. These health and air quality impacts connect with the Clean Air Act (CAA) of the United States Environmental Protection Agency (US EPA). There are four main issues pertinent to a discussion on hydrocarbons:

1) direct health impacts of hydrocarbon species;
2) ozone formation associated with hydrocarbon speciation and abundance;
3) fine particulate matter ($PM_{2.5}$) formation associated with hydrocarbon speciation and abundance;
4) toxicity of individual PM species and/or the formation of ultrafine PM (PM_1).

The CAA divides atmospheric pollutants into two classes: criteria air pollutants (CAPs) and hazardous air pollutants (HAPs). Criteria pollutants are those regulated under the National Ambient Air Quality Standards (NAAQS) using a threshold approach, and include O_3, nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), PM, and lead (Pb). Hazardous pollutants, also known as air toxics, include 187 chemicals and are regulated from specific sources, often with technology-based regulations.

The health and air quality impacts associated with on-road vehicle hydrocarbon emissions cut across a range of pollutants and regulatory structures:

- The direct health impacts of individual hydrocarbon species are regulated as HAPs;
- The impacts of hydrocarbons on ozone production are regulated through the ozone NAAQS; and
- The impacts of hydrocarbons on the formation of fine particulate matter are regulated through the PM NAAQS.

It is noteworthy, however, that hydrocarbon impacts on PM toxicity are not currently included in the CAA, as PM is regulated based on mass concentration (e.g. micrograms/m^3) and size fraction ($PM_{2.5}$, referring to PM < 2.5 microns or $PM_{10,}$ referring to PM < 10 microns), but not the chemical composition of the PM. Similarly, there is no separate regulation for PM smaller than 2.5 microns, for example PM_1. Research on the health impacts of PM species [*Grahame et al.*, 2014] and ultrafine PM [*Heal et al.*, 2012] is rapidly advancing, and may lead to findings that alter the format in a manner akin to the 1997 addition of the $PM_{2.5}$ NAAQS standard [*Bachmann*, 2007].

Hydrocarbons are a class of chemical defined as containing only hydrogen and carbon, of varying sizes and bond structures. Methane (CH_4) is the simplest hydrocarbon, which exhibits a low level of chemical reactivity in the atmosphere. As a result, the atmospheric chemistry community considers atmospheric hydrocarbons in two general categories: methane, and non-methane hydrocarbons (NMHCs). The NMHCs may be further divided into three broad classes based on their volatility: gas-phase volatile organic compounds (VOCs); semi-volatile organic compounds (SVOCs) that may be emitted in particulate phase, but evaporate into gas phase; and intermediate volatility compounds (IVOCs), with volatility in between VOCs and SVOCs [*Hodzic et al.*, 2010]. These gas-phase or evaporative-particulate emission categories complement the non-reactive particulate organic carbon emissions known as primary organic aerosols (POA, where "primary" is an atmospheric chemistry term meaning "directly emitted").

The specifics of atmospheric hydrocarbon characterization are complex, and most studies treat NMHC emissions in two categories: POA (all particulate) and VOC (all gas phase). From this simpler starting point, VOCs may react in the atmosphere to form other hydrocarbons, or chemically react to form particulates known as secondary organic aerosols (SOA, where "secondary" is an atmospheric chemistry term meaning "chemically produced"). Each of these volatility-based classes includes a mix of specific NMHCs. By way of example, a list of VOCs from the EPA is given in Appendix A of this report.

Compounds in the IVOC range include long-chain hydrocarbons (e.g. > 20 C atoms [*Hodzic et al.,* 2010]), including many polycyclic aromatic hydrocarbons (PAHs) that are known air toxics. As such, a better understanding of atmospheric PAHs offers the potential to support improved management of air pollution health impacts in terms of air toxics, PM formation processes, and the health impacts of PM. In fact, PAHs are considered a strong candidate – along with a small group of other known carcinogens – to explain the carcinogenic impacts of PM [*Heal et al.*, 2012]. Appendix B provides a list of 72 unique PAHs, ranked by molecular weight and including chemical formula and carcinogenic characterization (from a 1983 report from the National Research Council, so results on health impacts are not up to date).

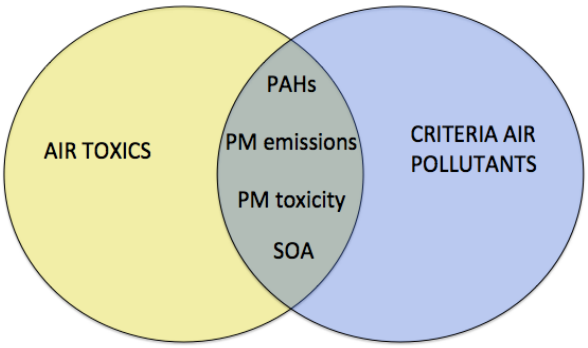

Figure 1: Schematic showing that PAH emissions from gasoline intersects both criteria and air toxic issues, affecting direct exposure to air toxics (PAHs), the emissions of PM, the formation of secondary PM (secondary organic aerosol, SOA), and the toxicity of PM. These impacts are discussed in the text

There has been extensive research on emissions, exposure, and health impacts of monocyclic aromatic hydrocarbons, especially benzene, toluene, ethylbenzene, and xylenes (often referred to as BTEX). These compounds have been associated with cancer and non-cancer health impacts [*Bolden et al.*, 2015]. Fewer studies, however, have focused on PAHs. In fact, in a 2014 review of regulatory issues related gasoline toxicology, no mention is made of particulates or PAHs [*Swick et al.*, 2014].

A number of studies have been published that do not include a discussion of PAHs in considering the air quality and/or health impacts of ethanol relative to other transportation fuels. *Alhajeri et al.* [2011] compare E85 with fleet electrification in Austin, Texas under 2030 conditions with respect to ozone, and find that a 17% assumed adoption of plug-in electric vehicles would yield greater ozone

improvements than a 100% assumed adoption of E85 vehicles. Other studies compare ethanol impacts on air emissions for total hydrocarbons, but not PAHs specifically [*Hubbard et al.*, 2014; *Dardiotis et al.*, 2015], or focus on atmospheric ethanol directly [*Millet et al.*, 2012]. Studies have also considered the ozone formation potential of ethanol relative to gasoline [*Jacobson*, 2007; *Ginnebaugh et al.*, 2010; *Ginnebaugh and Jacobson*, 2012].

A challenge to the assessment of PAH impacts on air quality and health is the disciplinary breadth of research pertinent to this issue. While the analysis of engine emissions is conducted within the engineering research community, atmospheric processes are studied by the atmospheric chemistry community, and the health impact analysis of PAHs has emerged from the epidemiological and toxicology communities.

The complexity of the linkages between the emissions, chemistry, and health impacts are discussed below. A few studies also offer insight into the complexities in this coupling. For example, *Geller et al.* [2006] use chemical redox potential as a metric of toxicity, and compare various diesel and gasoline engine configurations in terms of PM mass, particle number (PN, # of particles per m^3), and redox potential. They find that PM emissions with and without a diesel particulate matter filter varied by a factor of 25, but the redox potential only varied by a factor of 8. This same study found a high level of correlation between PAHs and other select PM components and redox potential [*Geller et al.,* 2006].

Here, we provide a review of the literature linking emissions, health impacts, and population exposure to PAHs and PM, drawing from studies most relevant to US air quality and on-road gasoline vehicles. We highlight research that directly compared ethanol and/or ethanol-gasoline blends for PAHs, and conclude with suggestions for future work.

To conduct this assessment, we rely on the ISI Web of Science through the University of Wisconsin Library System, which is the most comprehensive database of peer-reviewed scientific literature. Clarivate Analytics (formerly Thomson Reuters) owns the Web of Science, which includes the Science Citation Index (SCI), a rigorously selected subset of journals subject to the peer-review standards set by Clarivate Analytics. A secondary database launched by Clarivate Analytics called the Emerging Sources Citation Index (ESCI) was launched in 2015, and included in the Web of Science for 2015 onward. Inclusion in the ESCI is a first step for consideration for inclusion in SCI. This distinction is relevant here, as a number of papers pertinent this review have been published in ESCI journals, such as the *SAE International Journal of Fuels and Lubricants*. For transparency and reproducibility in this review, only papers included in the Web of Science were considered, such that in some cases papers from particular journals may only be included if they were published in 2015 or later (i.e. with the newer publications in ESCI).

Two approaches were used to identify relevant studies. First, we identified articles in which the title, abstract, and/or keywords included terms indicative of topical relevance through a series of searches. Studies were omitted that focused on non-gasoline fuels (e.g. diesel, coal, solid biomass) unless the discussion bore direct links to the discussion as a whole (e.g. in considering the impacts of PAH exposure on health, we considered studies regardless of the PAH source of origin). Studies were also typically omitted that characterized ambient PAHs or source attribution in non-US contexts.

Additional papers were included to expand on key themes by examining references in key papers (earlier studies cited by the key papers), as well as citations of the key papers (later studies that cite the key papers). Relevant research linking transportation, fuel choice, air quality and public health spans a wide range of journals and research disciplines.

PAH Emissions from Gasoline and Gasoline-Alternative Fuels

All fuel types contain a mix of hydrocarbons, which are released into the air as emissions through incomplete combustion and evaporation. Emitted PAHs may be in gas or aerosol/particulate form, with shorter-chain hydrocarbons typically in gas phase and longer-chain hydrocarbons typically particulate form. Both from vehicles and other sources, PAHs have been detected worldwide in soil, water, and food, as well as directly in the air [*Srogi*, 2007].

Synthesizing the state of knowledge of PAHs in a 1999 paper in *Environmental Science & Technology*, *Marr et al.* note that on-road motor vehicles account for ~1/3 of emitted PAH in the United States; that less is known about PAH emissions from gasoline vehicles than diesel vehicles, despite the fact that gasoline vehicles emit more of certain PAHs than diesel; and that a linear relationship exists between the concentration of PAHs in the fuel mixture and their concentration in exhaust emissions [*Marr et al.*, 1999]. This linear relationship was found in the literature prior to analysis by *Marr et al.,* and is generally consistent with the results of that study (shown below in Figure 1). *Marr et al.* note that while many studies had examined the exhaust mix of PAHs, few compare emissions to the PAH components of the combusted fuel.

The mix of hydrocarbons in fuel depends on the source of crude oil or biofuel feedstock, the refinery process, and additives to improve engine performance. The combustion of these fuels converts most of the liquid hydrocarbons into carbon dioxide. However, no engine provides perfect fuel combustion,

species name	abbreviation	molecular weight	number of rings	time after injection (min)	ions monitored
naphthalene	NAP	128	2	10.0–16.0	128, 102
acenaphthylene	ACY	152	3	16.0–18.4	152, 126
acenaphthene	ACE	154	3	18.4–19.5	153, 126
fluorene	FLU	166	3	19.5–23.0	166, 83
anthracene	ANT	178	3	23.0–27.0	178, 152
phenanthrene	PHE	178	3	23.0–27.0	178, 152
fluoranthene	FLT	202	4	27.0–32.0	202, 101
pyrene	PYR	202	4	27.0–32.0	202, 101
benz[*a*]anthracene	BAA	228	4	32.0–37.0	228, 113
chrysene	CRY	228	4	32.0–37.0	228, 113
benzo[*b*]fluoranthene	BBF	252	5	37.0–47.0	252, 126
benzo[*k*]fluoranthene	BKF	252	5	37.0–47.0	252, 126
benzo[*a*]pyrene	BAP	252	5	37.0–47.0	252, 126
benzo[*ghi*]perylene	BGP	276	6	51.0–64.0	276, 138
indeno[1,2,3-*cd*]pyrene	IND	276	6	47.0–50.0	276, 138
dibenz[*a,h*]anthracene	DBA	278	5	50.0–51.0	278, 139

Table 1: PAH compounds measured in Marr et al., 1999 (This is Table 1 from paper)

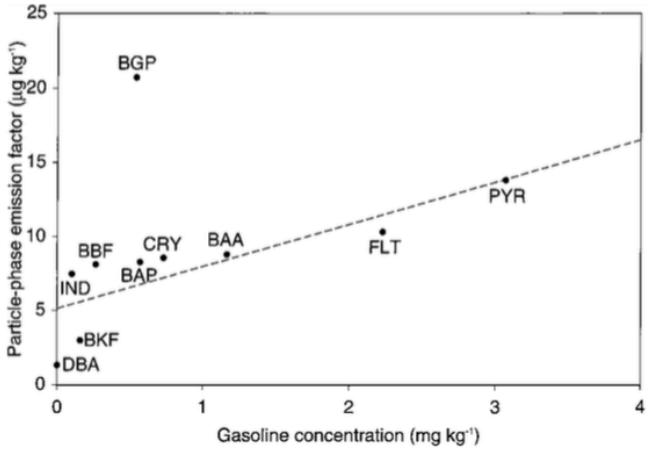

Figure 2 (left): From Marr et al., [1999; Figure 4]. Comparison of PAH emission factor versus PAH gasoline concentration. Line includes all points except BGP (benzo[ghi]perylene).

resulting in a mix of hydrocarbons released through incomplete combustion and through fuel evaporation. Lubricating oils offer an additional source of vehicle hydrocarbon emissions.

Shorter/lighter PAHs are emitted in gas form as VOCs (e.g. Napthalene), whereas longer/heavier PAHs condense quickly into particulate matter and fall into the SVOC or IVOC categories. Diesel vehicles tend to have higher emissions of lighter PAHs relative to gasoline vehicles, but heavy PAH emissions are more comparable between gasoline and diesel vehicles, and long PAHs are exclusively emitted by gasoline vehicles [*Riddle et al.*, 2007]. *Riddle et al.* [2007] attribute the lower emissions of heavy PAHs from diesel vehicles to the breakdown of these compounds into shorter PAHs under the higher engine loads and hotter temperatures typical of the heavy-duty diesel vehicles used in their study.

Even for short PAHs, however, gasoline emissions play a major role. In a 2005 study of Southern California, 44% of naphthalene, the most abundant urban PAH, was attributed to gasoline engines and only 9% to diesel engines [*Lu et al.*, 2005]. In a 2002 review of PAH risk in Sweden, phenanthrene was found to be the most abundant PAH in Stockholm [*Boström et al.*, 2002]. Citing *Nielsen* [1996], a 2010 review attributes 90% of weekday PAH emissions to traffic emissions, of which 1/3 are attributed to gasoline vehicles and 2/3 to diesel vehicles [*Fuglestvedt et al.*, 2010]; however, the original study was conducted in the early 1990s in Copenhagen, and is unlikely to be representative of current US conditions.

A number of studies compare emissions of PAHs and/or health risk across fuels, including a comparison of gasoline and diesel [*Pohjola et al.*, 2004; *Caviedes et al.*, 2016] and liquefied petroleum gas and unleaded petrol [*Lim et al.*, 2007]. For the biofuels target audience of report, we focus on comparative studies specific to ethanol and PAH. We recognize that the specific formulation of the fuel and blending assumptions can affect study results, but the methods or study design were not used as a basis for including or excluding study results here.

- 2016 study measured emissions and cell response to emissions of ethanol-gasoline blends compared with a diesel control. Particle number and the cell response (is an indicator of toxicity expected from PAH emissions) were measured, but PAHs were not directly measured. A non-linear response of particulate number was reported, with the highest PM count from ethanol-gasoline blends observed at E10 blend (higher than E0 and E85, but lower than diesel). The cellular analysis, used as a model for the human lung, showed no response for any of the ethanol-gasoline exhaust mixtures, but did respond to the diesel exhaust mixture (consistent with previous studies). [*Bisig et al.*, 2016]

- 2016 study compared exhaust characteristics of ethanol-gasoline blends in a four-stroke motorcycle. Study found particulate number (PN, a relevant metric for ultrafine particulates) increased moving from gasoline to a 5% ethanol blend, but decreased as the ethanol percentage increased (to 10 and 20%) [*Costagliola et al.*,

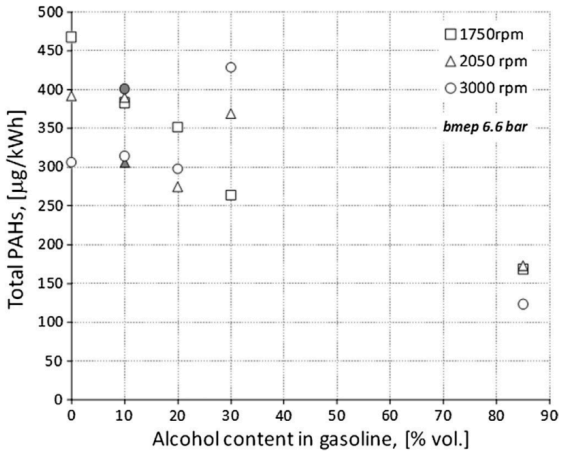

Figure 3: Comparison of PAH emissions with ethanol blend; Figure from [Costagliola et al., 2013]

2016b]. Related work from this same research group focused on conventional and hybrid scooters, finding that PN was reduced in *conventional* scooters under two test conditions using 10%, 20%, and 30% ethanol blends relative to gasoline, but that ethanol blends could increase or decrease PN in *hybrid* scooters depending on the drive cycle [*Costagliola et al.*, 2016a].

- 2016 study considered the emissions impacts of ethanol blends and gasoline in a direct-injection, spark-ignition engine, finding that ethanol increased PM emissions, whereas the impact on PN depended on the particle size [*Fournier et al.*, 2016]. PAHs were not directly measured, but total hydrocarbons were reported.

- 2016 study compared a range of fuel formulations in the China market and found that E10 increased the PN emissions relative to gasoline under low load operating conditions [*Wang et al.*, 2016].

- 2016 study found that fuels with higher levels of aromatics produced higher levels of PM [*Ratcliff et al.*, 2016].

- 2016 study compared E0 with E10 and E85, finding a reduction in PN with the ethanol blends (97% and 96% for E10 and E85, respectively) and a reduction in PAHs (67-96% and 82-96% for E10 and E85, respectively) [*Muñoz et al.*, 2016].

- 2015 laboratory study with a smog chamber compares gasoline and ethanol blends (E75 and E85) both in terms of primary emissions (BC and POA) as well as the formation of SOA. The results do not emphasize the difference between gasoline and ethanol emissions, but the text suggests that gasoline produces more SOA than ethanol vehicles attributable to the higher aromatic content [*Suarez-Bertoa et al.*, 2015].

- 2015 studies review air emissions from biofuels, but limit discussion of PAH emissions to biodiesel and compressed natural gas. [*Anderson*, 2015; *Claxton*, 2015]. Another 2015 review compares studies to date in terms of PM emissions, noting that there is a disagreement among published works in terms of PM emissions of ethanol versus gasoline [*Überall et al.*, 2015]; this same review cites *Karavalakis et al.* [2014] (see below) as evidence that a higher aromatic content of the fuel contributes to higher PM emissions.

- 2015 review of air emissions from ethanol and gasoline blends focuses on PAHs primarily through a citation of *Seggiani et al.,* [2012] (noted below), and emphasizes the potential importance of the carcinogenic products that form when nitrogen compounds react with PAHs [*Manzetti and Andersen*, 2015].

- 2015 study compares gasoline and two different formulations of E10, finding that total hydrocarbons are reduced with both ethanol mixtures, although PAHs are not considered separately [*Wang et al.*, 2015].

- 2014 study compared E10, E51, and E83, and found that PM mass and PN decreased with increasing ethanol blend levels (PAH was not reported, but may affect the PN results) [*Karavalakis et al.*, 2014]. However, 2015 work from this same group comparing E10, E15, and E20 does not show the same decrease in PN seen with higher ethanol blends [*Karavalakis et al.*, 2015].

- 2013 study quantified PAH and PM emissions (including ultrafine) for gasoline, E10, E20, E30, and E85 for various engine load conditions. Findings suggest that higher ethanol percentages generally decrease PAH emissions (as shown in Figure 3); findings on PM vary by particulate size and engine load [*Costagliola et al.*, 2013].

- 2013 study compared gasoline (E0), E10 and E85 under different driving conditions. "Use of E85 significantly reduced the emissions of lower molecular weight PAH. However, a reduction in higher molecular weight PAH entities in PM was not observed." [*Hays et al.*, 2013]

- 2012 study of ethanol-gasoline blends in mopeds found, "No evident effect of ethanol on the particulate mass emissions and associated PAHs emissions was observed." [*Seggiani et al.*, 2012]

- 2011 study of ethanol-gasoline blends found that $PM_{2.5}$ mass decreased with higher ethanol blends, and the molecular weight distribution of PAHs were found to decrease with higher ethanol blends. Both of these results were considered positive in terms of human health impacts. However, the higher ethanol blends contributed higher levels of nitrogenated PAHs, which was considered a negative in terms of human health impacts (citing work of [*Collins et al.*, 1998] on the increased genotoxic, mutagenic and carcinogenic characteristics of these nitro-PAHs). [*Dutcher et al.*, 2011]

- Prior to 2010, few studies had compared emissions of ethanol and gasoline. *Liaquat et al.* [2010] include the following summary of ethanol and gasoline emissions studies. The omission of *de Abrantes et al.* [2009], noted below, appears to be an oversight in the summary table below.

Summary report on exhaust emissions of bioethanol as compared to gasoline fuel.

References	Test parameters	Exhaust emission
Kiani et al., 2010	Under varying engine speed and at constant engine loads of 25, 50, 70% and full load	Lower CO & HC emissions Higher CO_2 and NO_x emissions
Eyidogan et al., 2010	At two different vehicle speeds (80 km h^{-1} and 100 km h^{-1}), and four different wheel powers (5, 10, 15, and 20 kW)	(Only performance and combustion based)
Wen et al., 2010	At constant engine load (3 N m) and different engine speeds (idle speed & 3500 to 8000 rpm with 1500 rpm period)	Lower CO & HC emissions Higher CO_2 and NO_x
Koç et al., 2009	At two compression ratios (10:1 and 11:1) Engine speed from 1500 to 5000 rpm at wide open throttle (WOT)	Lower CO, HC and NO_x emissions
Najafi et al., 2009	At full throttle setting & under varying engine speed conditions (1000–5000 rpm)	Lower CO & HC emissions Higher CO_2 and NO_x emissions
Celik, 2008	At a full throttle opening & constant speed (2000 rpm) C.R from 6/1 to 10/1	Lower CO, CO_2, HC and NO_x emissions

Table 2: Review of studies prior to 2010 that compare air emissions from ethanol and gasoline from [Liaquat et al., 2010]

- 2009 study compared ethanol and gasohol vehicles in Brazil, finding significantly lower PAH emissions from the ethanol vehicle ("41.9 mg km^{-1} to 612 mg km^{-1} in the gasohol vehicle, and from 11.7 mg km^{-1} to 27.4 mg km^{-1} in the ethanol-fueled vehicle") [*de Abrantes et al.*, 2009b]. Related work from this team was also published comparing correlations among emissions [*de Abrantes et al.*, 2009a].

- 2008 study compared emissions based on the aromatic and sulfur content of fuels, concluding that fuels with higher aromatic content emitted higher total hydrocarbons (but typically lower NOx emissions) [*Yao et al.*, 2008]. Ethanol-gasoline blends were not directly tested.

The studies above represent laboratory test studies. To calculate emissions from vehicles operating in real-world conditions, mathematical functions are often fit to empirical results in vehicle emissions models like the EPA Motor Vehicle Emissions Simulator (MOVES). The only peer-reviewed study found to use MOVES for PAH analysis focuses on biodiesel [*Pino-Cortes et al.*, 2015].

Health Risks of PAHs

A variety of adverse health outcomes have been associated with PAHs [*Kim et al.*, 2013], with some compounds identified as carcinogenic, teratogenic, and/or genotoxic causing acute and chronic health effects [*Boström et al.*, 2002]. Humans are exposed to PAHs in ambient air, as well as diet, cigarette smoke, and other sources [*Kim et al.*, 2013; *Abdel-Shafy and Mansour*, 2016].

The majority of health impacts can be grouped into cancer risk, risk of compromised cognitive function, respiratory risk and other risks. *Kim et al.*, [2013] summarized the health effects of PAHs shown below in Figure 4, divided into short- and long-term health effects. Short-term health effects from PAH exposure include 1) eye and skin irritation, 2) nausea and vomiting, and 3) inflammation. Long-term health effects include 1) skin, lung, bladder, and gastrointestinal cancer, 2) DNA, cataracts, kidney, and liver damage, and 3) gene mutation cell damaging and cardiopulmonary mortality. Several studies have addressed the use of toxic equivalency and potency equivalency factors for analysis [*Nisbet and LaGoy*, 1992; *Petry et al.*, 1996; *Collins et al.*, 1998; *Agudelo-Castañeda et al.*, 2017].

Evidence of Cancer Risk

Increased risk of various cancers from exposure to PAHs has been reported, including lung, skin, bladder, and gastrointestinal [*Boffetta et al.*, 1997; *Bach et al.*, 2003; *Olsson et al.*, 2010; *Diggs et al.*, 2011].

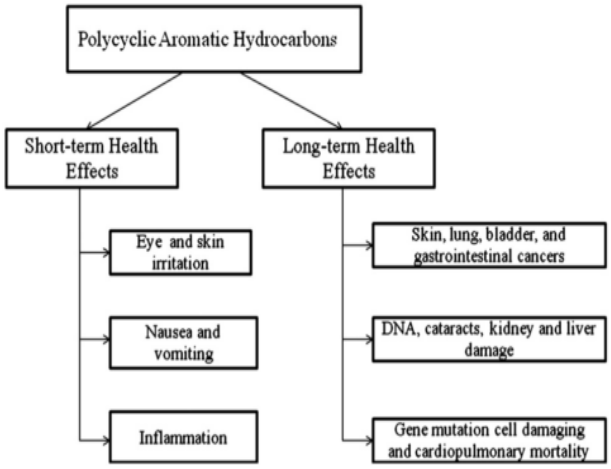

Figure 4: Summary of PAH health impacts from a 2013 review of health effects of PAHs. [*Kim et al.*, 2013]

For humans, most information is based on studies of occupational or environmental exposure through inhalation of mixtures of PAHs and other non-PAH constituents. Thus, assessing the relative risk of other exposures and PAHs alone is difficult. *Armstrong et al.*, [2004] summarize the relative risks from 39 epidemiological studies finding an average unit relative risk (URR) of lung cancer from exposure to 100 $\mu g/m^3$ years benzo(a)-pyrene (BaP) of 1.20 [95% confidence interval (CI), 1.11–1.29].

Studies of workers exposed to diesel exhaust have suggested a small increased risk of cancer [*Tavares et al.*, 2004; *Boers*, 2005; *Clapp et al.*, 2009]. Exposure to PAHs in ambient air at concentrations near standards specified in regulation is thought to pose a lower risk. *Butterfield and Brown*, [2012] find that of 94,000 in Northern Ireland exposed to BaP concentrations above the 1 ng/m^3 target value, 3 additional incidences would be expected over a 70-year period.

Most information assessing the carcinogenicity of PAHs from human exposure is through occupational exposure in occupation such as coke production, roofing using bituminous products, oil refining, coal gasification, chimney sweeping using tar, and gas workers [*Boffetta et al.*, 1997; *Moolgavkar et al.*, 1998; *Bach et al.*, 2003; *Armstrong et al.*, 2004; *Zhang and Tao*, 2009]. These studies find evidence that inhalation exposure to PAHs increases risk of lung cancer and dermal exposure causes skin cancer.

These findings are supported by studies of laboratory animals, also finding evidence of stomach cancer from ingestion of PAHs [*Hecht*, 2002; *Samanta et al.*, 2002; *Latif et al.*, 2010; *Lynch and Rebbeck*, 2013]. Animal laboratory studies have found long-term exposure to low levels of some specific PAHs, including pyrene and BaP to cause cancer [*Diggs et al.*, 2012].

Several agencies classify specific compounds carcionogenic including the Agency for Toxic Substances and Disease Registry (ATSDR), International Agency for Research on Cancer (IARC) and U.S. Environmental Protection Agency (EPA) [*Kim et al.*, 2013]. For example, the EPA defines benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-c,d)pyrene as probable human carcinogens and acenaphthylene, anthracene, benzo(g,h,i)perylene, and fluoranthene as probable human carcinogens.

Evidence of Impacts on Cognitive Function

Several studies have identified cognitive dysfunction or neurologic and developmental effects associated with PAH exposure in animals and humans [*Kim et al.*, 2013]. For example, *Perera et al.*, [2014a] estimate a value of $43 million - $215 million, for each year's cohort of Medicaid children born in New York City, from a 0.25 ng/m^3 reduction in ambient PAH. This value is based on the lifetime loss of income due to prenatal exposure to PAH and lowered IQ [*Perera et al.*, 2014a]. *Perera et al.*, [2014b] extends this work to show that exposure to PAH may also play a role in childhood Attention Deficit Hyperactivity Disorder (ADHD). Studies of children in Krakow, Poland support these findings with evidence that PAHs harm the fetal brain with effects that extend through childhood and impact academic success [*Edwards et al.*, 2010; *Jedrychowski et al.*, 2015].

Other studies find that traffic-related pollutants are associated with developmental impairment and poorer academic achievement in children as well as cognitive decline in the elderly [*Clifford et al.*, 2016]. However, *Clifford et al.*, [2016] do not find enough evidence to comment on consistency nor specifically exposure to PAHs. *Best et al.*, [2016] supports that PAHs may be responsible for cognitive impairment in the elderly, finding a 1.8% poorer score on the digit symbol substitution test for each 1% increase in the PAH biomarker, urinary 1-hydroxyprene.

Evidence of Respiratory Health Risk

Among the respiratory concerns associated with PAHs are lung cancer, asthma, and infectious disease such as pneumonia [*Braga et al.*, 2001; *Armstrong et al.*, 2004; *Agudelo-Castañeda et al.*, 2017]. The effect of asthma on children has been given particular emphasis [*Jedrychowski et al.*, 2005; *Jung et al.*, 2012, 2014; *Liu et al.*, 2016; *Agudelo-Castañeda et al.*, 2017; *Wang et al.*, 2017], including a recent review of air pollution impacts on children's respiratory health where PAHs are associated with coughing, wheezing, and shortness of breath [*Goldizen et al.*, 2016]. For example, an eight-year study of children directly correlated PAHs with 4, 5, and 6 rings, as well as phenanthrene, with wheezing in children with asthma in Fresno, California [*Gale et al.*, 2012].

Agudelo-Castañeda et al., [2017] find a link between several carcinogenic PAHs (Indeno(1,2,3-c,d)pyrene, benzo(b,k)fluoranthene, dibenzo(a,h)anthracene, BaP, and benzo(g,h,i)perylene) and respiratory diseases such as bronchitis and pneumonia. They also find that these individual PAHs contributed the most to the risk of exposure to ultrafine PM (PM_1). *Jedrychowski et al.*, [2005] find an increased relative risk of various respiratory symptoms in newborns related to prenatal PAH exposure including barking cough, wheezing without cold, sore throat, ear infection, cough irrespective of respiratory infection, and cough without cold. *Jung et al.* [2012] and *Jung et al.* [2014] find that obese

young children and children with non-allergenic asthma may be more susceptible to respiratory consequences of exposure to PAHs or pyrene. *Liu et al.* [2016] find epidemiological evidence that PAHs are associated with asthma in children 6-19 years of age, and *Wang et al.* [2017] identify higher levels of an oxidative stress marker related to exposure to PAHs, suggesting exposure to PAH may enhance oxidative stress and induce asthma, also supported by *Bae et al.* [2010].

Other Health Risks

Many developmental effects have been associated with PAHs, some of which result in impacts on cognitive function or respiratory health described above, but others resulting in outcomes like birth defects and decreased birth weight in humans and animals [*Kristensen et al.*, 1995; *Wassenberg and Di Giulio*, 2004; *Perera et al.*, 2005; *Rice and Baker*, 2007; *Wells et al.*, 2010; *Ren et al.*, 2011; *Langlois et al.*, 2012; *Lupo et al.*, 2012; *Yuan et al.*, 2013]. One study found that PAHs exhibited adverse health outcomes for infants born to African American mothers in New York city, but not for infants born to Dominican mothers [*Choi et al.*, 2008]. A recent study addressing the effects of PAH exposure on birth outcomes finds an impact on several measures of fetal development, especially birth weight, and takes this one step further to show that PAH has a greater impact than $PM_{2.5}$ [*Jedrychowski et al.*, 2017]. Childhood leukemia has also been linked to maternal PAH exposure in the 3rd trimester of pregnancy [*Heck et al.*, 2014]. Developmental impacts of PAHs have also been linked to behavioral issues in children [*Perera et al.*, 2012].

Genotoxicity of PAHs may play a large role in the carcinogenicity and perhaps developmental toxicity of PAHs [*Kim et al.*, 2013]. Some PAHs shown to exhibit high genotoxicity are benzo(ghi)fluoranthene, benzo(j)fluoranthene, benzo(a)pyrene, chrysene, dibenzo(a,l)pyrene, fluoranthene, and triphenylene [*White*, 2002].

Other short-term responses not discussed above associated with exposure to PAH include effects such as eye irritation, nausea, vomiting, diarrhea, skin irritation and allergic reaction [*Unwin et al.*, 2006; *IPCS (International Programme on Chemical Safety)*, 2010], as well as arterial disease [*Xu et al.*, 2013]. Over the long-term effects in addition to those discussed above include cataracts, kidney damage, liver damage, and jaundice [*ATSDR*, 1995].

Population Exposure to PAHs

Human exposure to PAHs depend on the concentrations of each PAH to which humans are exposed. For many air pollutants, especially ozone and $PM_{2.5}$, ground-level monitoring data is often used to estimate community population exposure.

However, in the case of PAHs, there is no monitoring network appropriate for large-scale health-based assessment. In the absence of direct measurements, computer models offer the most rigorous approach to estimating air pollution exposure. The EPA develops and uses the Community Multiscale Air Quality (CMAQ) model, discussed in more detail below, to estimate exposure for the National Air Toxics Assessment (NATA)[1]. In the NATA, individual PAHs are grouped by "unit risk estimates," defined as the concentration that yields 1-in-1 million lifetime risk of cancer.[2]

[1] https://www.epa.gov/national-air-toxics-assessment/2011-nata-assessment-methods
[2] The full table, showing the URE of each PAH included in the NATA is at on pg. 20-22 https://www.epa.gov/sites/production/files/2015-12/documents/2011-nata-tsd.pdf

This approach has also been described in the peer-reviewed literature. An example of this type of study by *Lu et al.* [2005] used an air-shed model for Southern California and a single PAH, naphthalene, which used a 2003 emissions inventory from the South Coast Air Quality Management District and photochemical grid model called the Surface Meteorology and Ozone Generation (SMOG) model to calculate ambient naphthalene and health impacts.

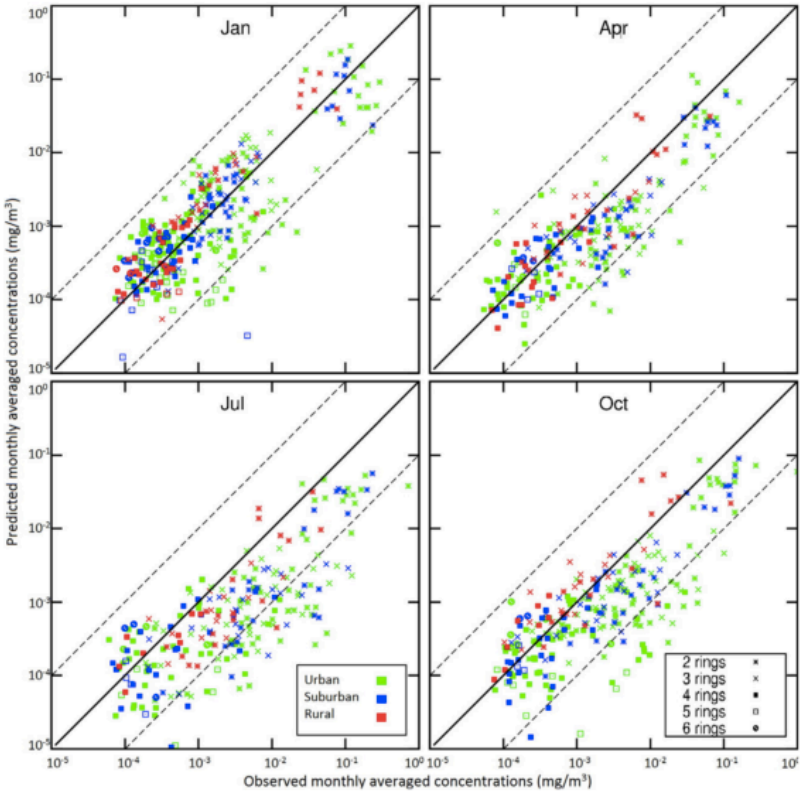

Figure 5: Comparison of CMAQ-modeled (y-axis) PAHs with ground based measurements in the U.S. [Figure 2 in Zhang et al., 2017].

More recently, *Zhang et al.* [2017] presented an evaluation of 16 PAHs, calculated over the continental United States for four months of 2011 at a resolution of 36 km x 36 km. For grid boxes that contained ground-based measurements, the model and measurements were compared, as shown in Figure 5. They found that CMAQ performed best in rural areas, and in the winter.

The model results from *Zhang et al.* [2017] were used for health assessment in a related study, [*Zhang et al.*, 2016], with example results shown in Figure 6. The health-focused study also attributes health risk of individual PAHs to emission sectors, including motor vehicles. Tables reporting these results are presented in Appendix C of this report.

Figure 6: Excess cancer cases associated with naphthalene, calculated based on atmospheric concentrations and population distribution.
[Figure 1, panel d from *Zhang et al.*, 2016]

PAH contribution to ultrafine particulate matter from on-road vehicles

Background on PM formation

The study of chemical processes controlling the formation of secondary organic aerosols (SOA) is an active area of research. Emitted PAH can form particles in the engine, such that the emissions are carbonaceous aerosols, often referred to as "soot" in the engine research community [*Richter and Howard*, 2000]. The mix of gases and particles released from the engine are considered in emissions inventories like the EPA National Emissions Inventory (NEI). Emitted carbonaceous aerosols are either organic carbon (OC, also known as primary organic aerosol, POA), or elemental carbon (EC). Although these categories are used to track the transformation of chemicals in the atmosphere, and for analysis of most available measurement data, it is recognized that many particles contain a mix of chemicals, and that EC in particular may often be bound with PAHs.

This process-based approach leverages insights from laboratory studies on emission characteristics and atmospheric transformation. The calculation of emissions and atmospheric processes, complements statistical analyses linking sources and pollutants, such as road proximity correlation with PAHs and ultrafine PM [*Levy et al.*, 2003]), and chemical mass balance modeling approaches to interpret source apportionment of observations [e.g. *Hasheminassab et al.*, 2013].

Extending Laboratory Insights with Atmospheric Models

Scientific understanding of PAH chemistry and PM formation has followed a common trajectory of atmospheric chemistry advancements, wherein laboratory studies build understanding, which is then mathematically expressed and evaluated in atmospheric models. In the case of PAH chemical processes, characteristics and chemical processes are still an active area of laboratory research [*Shakya and Griffin*, 2010; *Riva et al.*, 2015, 2016a, 2016b, 2017; *Chen et al.*, 2016]. For example, a 2004 study found that byproducts of aromatic hydrocarbon degradation reacted with sulfur compounds to promote PM formation [*Zhang et al.*, 2004]. More recently, *Suarez-Bertoa et al.*, [2015] analyzed vehicle exhaust and perform experiments with a smog chamber to calculate the formation of SOA (from both gasoline and ethanol vehicles), and find SOA formed from vehicle emission averaged 3 x larger than the primary emissions from the vehicles (including BC and POA). Increasingly, computational simulations at the molecular level complement these laboratory studies to provide foundational understanding of chemical processes. For example, a 2016 study considered the transformation of phenanthrene (a PAH) and acetaldehyde (a VOC) characteristic of an ethanol-gasoline blend. They simulated the mechanisms by which these gases transform into nanoparticles and the interaction of the formed nanoparticles with select other gases [*Manzetti and Andersen*, 2016].

Reaction rates and other parameters describing the chemical transformation process are generalized, and expressed mathematically in various forms. Typically, the mathematical representation of a series of chemical reactions is referred to as a "chemical mechanism." These chemical mechanisms may be tested in chemical box models ("zero-dimensional") and/or column models ("one-dimensional"). Model results are compared with measurements, with the goal of creating a chemical mechanism that captures the relationships among different chemical compounds under a range of atmospheric conditions (e.g. varying temperature, pressure, humidity, etc.).

In some cases, these chemical mechanisms are used to update three-dimensional models that may be used to assess real-world conditions, including providing input to health assessments and regulatory

decision-making. These three-dimensional atmospheric chemistry models come in two basic categories: Lagrangian (e.g. plume, puff, or trajectory models) and Eulerian (grid models). Both may be used to examine pollution events, quantify the sensitivity to emissions or atmospheric processes, quantify ambient concentrations in the absence of measurements, and/or characterize pollution response to a change in emissions.

Nopmongcol et al. [2011] serves as an example of this type of study, which includes the response of PM and ozone over the U.S. to various scenarios of ethanol used for vehicle use, and considers assumptions about associated land use for feedstock production. In that case, study authors assumed E85 exhibited the same emissions of NMHCs as gasoline, so the modeled changes do not include the SOA changes associated with VOC/SVOC/IVOC emission changes associated with ethanol versus gasoline [*Nopmongcol et al.*, 2011]. Similarly, *Tessum et al.*, [2014] use this approach to conclude that corn grain ethanol and coal-produced electric vehicles yield the highest levels of particulate matter across the U.S. (relative to gasoline and a range of other light-duty vehicle alternatives). Although the authors provide information on the chemical mechanisms used in their model, it would require additional investigation to assess the assumptions about hydrocarbon emissions in each scenario, and/or whether the SOA algorithm includes PAHs and other SVOCs/IVOCs and/or the degree to which reported results depend on fuel combustion versus life-cycle production emissions (also included in this study). A study focused on vehicle emissions for Europe used this approach to compare gasoline and E85 vehicle fleet impacts in a region of Sweden, finding air quality benefits to PM and other pollutants using two air quality models at different scales [*Fridell et al.*, 2014]

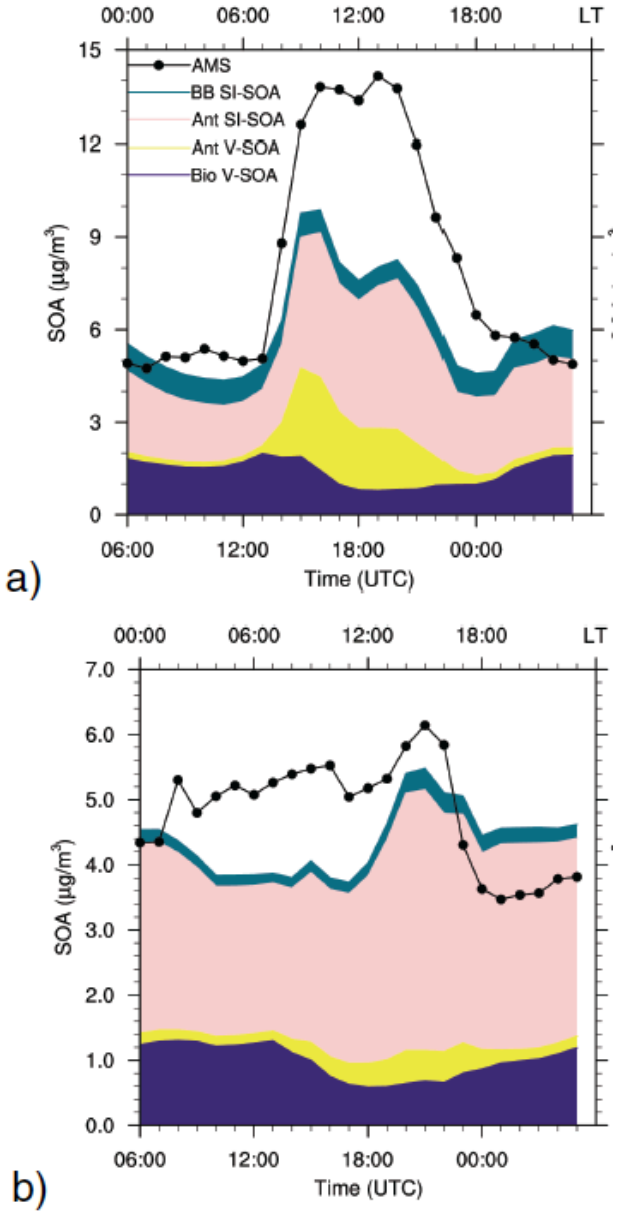

Figure 7: The importance of SVOC and IVOC compounds in SOA formation, based on model simulations and observations in Mexico City during the MILAGRO campaign. Each line represents a different measurement site. Black line shows calculate SOA from observations ("AMS" on plot). Area total represents modeled SOA. Pink area represents SOA formed from longer chain SVOC and IVOC anthropogenic hydrocarbons including PAHs. From Figure 4 of Hodzic et al. [2010]

This type of model-based approach, where pollution is calculated based on sources, meteorology, and chemistry, complements an observational analysis where measurements of PAHs (or other compounds) are analyzed to assess the likely

contributor sources (for example, a study in Brisbane, Australia which attributed 56% of measured PAHs to vehicular emissions [*Mishra et al.*, 2016]).

Advanced Modeling of Ambient PAHs and SOA

A process-based understanding of SOA bears direct relevance to the study of PAHs, which are important precursors to SOA (this is in addition to the role of PAHs in determining the toxicity of POA). On-road vehicle emissions are considered the dominant source of anthropogenic SOA and a limited understanding of vehicular PAH emissions on SOA formation limits current ability to assess the impact of vehicular emissions on secondary PM [*Gentner et al.*, 2012]. Evidence for the potentially important role of SOA from vehicle emissions includes a 2011 study in the San Joaquin Valley of California, where 68-85% of the organic carbon particles < 1.8 microns ($PM_{1.8}$) were found to be SOA or chemically altered POA [*Ham and Kleeman*, 2011].

Although a number of chemical mechanisms representing SOA formation have been developed for atmospheric models, routine monitoring data is insufficient to evaluate these mechanisms. While there are many ground-based $PM_{2.5}$ monitors in the U.S., measurements of air toxics (including PAHs) are sparse and not generally co-located with PM monitors. Further, while some PM monitors report the chemical species of PM (such that OC may be attributed in part to SOA), these speciation measurements are typically available every 3 to 6 days, rather than hourly, as is typical for $PM_{2.5}$ monitors.

As a result, major advances in understanding SOA formation processes and precursors have occurred through "field campaigns," wherein multiple instruments are deployed over a short period of time to measure a more comprehensive portfolio of chemical species. As one example of a field campaign, the Megacity Initiative: Local and Global Research Observations (MILAGRO) project took place in Mexico City in 2006, providing an opportunity to evaluate two mechanisms of SOA formation from larger hydrocarbons by *Hodzic et al.* [2010]. The study finds that inclusion of SVOCs and IVOCs (including PAHs) increases model calculated SOA formation by 3-6 times, improving agreement with measured OC.

Advances in PAH chemistry and SOA formation have been included in the Community Multiscale Air Quality (CMAQ) model. CMAQ was developed by the U.S. EPA to support decision-making, and is widely used by the scientific community around the world as a state-of-the-art photochemical grid model.

The earliest version of CMAQ used to evaluate SOA formation from PAHs was v. 4.7, used by *Zhang and Ying* [2012] for a two-week model simulation period over Southeast Texas in support of the 2006 TexAQS (Texas Air Quality Study) field campaign. Of particular interest from this study is the poor level of agreement between model-simulated PAH concentrations and observations, with the exception of naphthalene. Modeled concentrations were found to be significantly lower than observations, suggesting an error in the model emissions inventory. It may be notable that *Bieser et al.* [2012] also used a modified version of CMAQ to simulate ambient BaP, which compares well with observations over Europe. However, in that case, the BaP emissions inventory appears to have been explicitly developed based on the agreement between CMAQ simulations and ambient BaP measurements [*Bewersdorff et al.*, 2009], which would be expected to produce a higher level of agreement in subsequent model simulations.

Pye and Pouliot [2012] present an updated version of CMAQ (v. 5.0) with a focus on SOA formed from PAHs and alkanes. They find alkanes and PAHs together contribute 20-30% of anthropogenic SOA in the summer, although the contribution from PAHs may be largest in the summer. The authors note that the mix of hydrocarbons in the model emissions inventory introduces uncertainty into these results, and that the results are sensitive to vehicle fuel mix change [*Pye and Pouliot*, 2012].

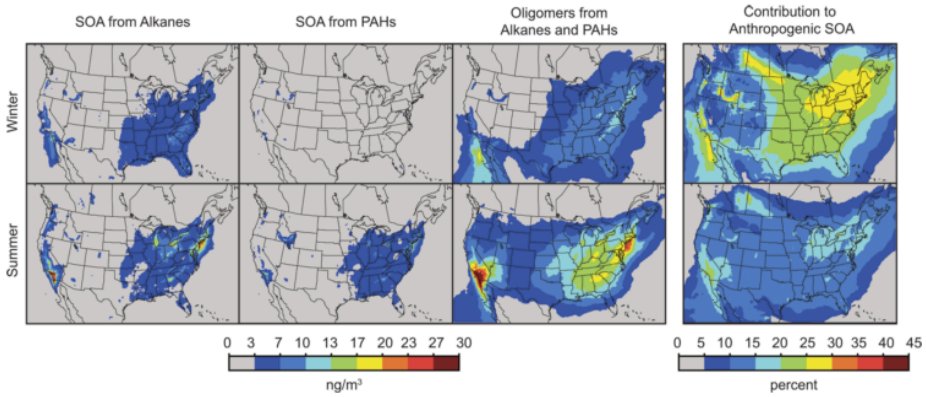

Figure 8 (Figure 4 from Pye and Pouliot [2012]): "Predicted surface level SOA from alkanes, PAHs, and their oligomers as well as their contribution to surface level SOA from anthropogenic hydrocarbons. The top row is an average for January 11 through February 10 and the bottom row for July 1−31, 2006." Additional SOA (not shown here) arise from biogenic SOA, which are discussed in the paper.

Another regional photochemical grid model used to assess PAHs over North America has been the Eulerian AURAMS (A Unified Regional Air quality Modeling System) chemical transport model, which was simulated for 2002 conditions and compared with measurements across Canada and the U.S. [*Galarneau et al.*, 2014].

To date, most efforts at regional modeling of PAH and SOA have focused on chemical processes and evaluation. Once developed and validated, these models may be used to assess technology and policy scenarios (by calculating "what if?" scenarios in the model), support public health assessment (by calculating ambient concentrations at unmonitored locations), and inform geographic and sectoral source attribution (by simulating the contribution of individual regions and/or individual sources to total concentrations).

Conclusion: Research to Better Connect Fuel Mix and Human Health and Toxicity

This review was motivated by the overall question of how changes in the utilization of ethanol could affect public health impacts associated with PAH emissions in the US. As discussed, these health effects span the category of pollutants categorized as hazardous air pollutants (or air toxics) by the US EPA, as well as the category of criteria air pollutants due to the contribution of PAHs to ambient particulates. The health impacts of ethanol use with respect to PAH emissions are determined by a number of factors:

1. The change in PAH emissions associated with fuel mix
2. The impact of changing emissions on ambient PAH concentrations
3. The impact of changing emissions on ambient PM (including ultrafine PM) concentrations
4. The exposure of populations to changing concentrations of PAH and/or PM
5. The toxicity of individual PAH compounds and the toxicity of PM associated with PAH (potentially by chemical compounds and/or particle size)
6. The summative change in health outcomes associated with changes in emissions, ambient concentrations, population exposure, and toxicity

Each of these impacts will be addressed in the context of next-stage research.

1. The change in PAH emissions associated with fuel mix

The majority of research specific to ethanol impacts on PAHs has focused on the changes in emissions associated with fuel blends. These studies are generally laboratory measurements of engine combustion products under different fuel blends and operating conditions. Results are difficult to compare in terms of fuel mixes, engines, and measured outcomes.

It would be valuable to compare results from the literature in a detailed way with current generation emissions models, especially the Motor Vehicle Emissions Simulator (MOVES). The process of fitting a mathematical relationship to a diverse collection of laboratory study results is not straightforward, and often alternate mathematical relationships could be presented that reasonably capture observed behavior. The challenge of modeling fuel-dependent emissions is particularly complex in the case of PAH from ethanol, where there is a large number of chemical compounds in both gas and particle form, where engine conditions and additives affect emissions, and where are alternate methods may be used to develop a particular blend level (e.g. "splash blending" versus "match blending"[3]).

Research Question #1: What is the correct function to represent ethanol impacts on PAH emissions be modeled?

Past studies could be fit with alternate mathematical functions, assessing the sensitivity to key parameters (e.g. engine type, speed, blend level, etc.). These functions could be compared with existing model formulations used in MOVES, with uncertainty metrics derived from studies to date. Based on results from this modeling analysis, new laboratory studies could be designed to target points of disagreement or high uncertainty. The only peer-reviewed study we found that uses MOVES for PAH analysis focuses on biodiesel [*Pino-Cortes et al.*, 2015]. More research on MOVES overall

[3] http://www.eesi.org/articles/view/how-is-gasoline-blended-two-groups-ask-epa-to-consider-this-important-detai

performance for on-road vehicle PAH emissions, and response to ethanol in particular, would be helpful to advance understanding on health impacts of ethanol fuel mixes.

The National Emissions Inventory (NEI) and the MOVES model for vehicle emissions, both developed from the U.S. EPA, are state-of-the-art, and considered best practice for emissions calculation. Like any emissions inventory, the NEI and MOVES have known uncertainties and unknown errors. Increasing their use for studies related to ethanol and/or PAH more broadly will aid in characterizing and reducing uncertainties and errors. The most recent year of the NEI is 2014, which includes a wide range of PAH compounds, based on the EPA Toxic Release Inventory and information from State, Local, and Tribal governments. NEI emissions are provided on a county level, annual total, which may be spatially and temporally disaggregated to support input into air quality models using the Sparse Matrix Operating Kernel Emissions (SMOKE) model.

2. The impact of changing emissions on ambient PAH concentrations

In the past few years, a series of studies have developed, applied, and evaluated CMAQ to quantify exposure and health impacts of PAHs over the United States. This framework would be well suited to modify emissions and assess the changes in fuel composition, or other technology and/or policy scenarios.

Research Question #2: How would ambient PAH change in response to ethanol fuel mix?

Calculating ambient concentrations under "what if?" scenarios requires the use of chemical transport models, like the EPA CMAQ model. These models take into account the emissions of PAHs to the atmosphere, their chemical transformations, removal, and dispersion or build-up in the atmosphere. This methodology is widely used from urban to global scales for many air pollutants, but less frequently for PAHs.

Conducting model simulations with current generation MOVES, CMAQ, and other simulations would allow calculated PAH fields to be compared with ambient measurements, and provide estimates on the response of PAH and health outcomes to fuel mix scenarios. CMAQ is not the only choice of model, but it is well suited to this type of scenario, given recent work in calculating ambient PAH concentrations and associated SOA formation. CMAQ is commonly run for month- or multi-month-long simulations at scales of 12 km x 12 km over the continental United States, and may be run down to 4 km x 4 km over limited areas and time frames.

3. The impact of changing emissions on ambient PM (including ultrafine PM) concentrations

The formation of particles, especially complex SOA and semi-volatile species, is an active area of research in atmospheric chemistry. The gaps in knowledge in this area are vast. As with ambient PAHs, the CMAQ model could be used to calculate SOA associated with PAH emission changes, and the newer versions of CMAQ have been tested in this area. A more comprehensive approach could involve the design of a field campaign to measure PAHs and PM metrics, and use these to evaluate existing models and chemical mechanisms. A field campaign of this type is usually a month or so coordinated research activities and measurements focused in a single area. TexAQS and MILAGRO were two such campaigns for Texas (2000 and 2006) and Mexico City (2006), respectively.

Research Question #3: How do PAH emissions from liquid fuels and fuel blends affect SOA?

This question could be addressed with model simulations, field measurements, and/or laboratory experiments. A dedicated field campaign would be an ambitious but high-impact approach to advancing this area of research.

4. The exposure of populations to changing concentrations of PAH and/or PM

Population exposure to air pollution is a challenge for all pollutants, given the expense of ground-based monitoring and interactions between emission sources (roadways, point sources) and communities (in terms of population density, at-risk populations, etc.).

In the case of PAHs, there are few ground-based monitors in the US, which are insufficient to address exposure issues. The EPA uses modeled data to support exposure assessment for the National Air Toxics Assessment (NATA). In the case of PM, existing monitors measure mass concentration (e.g. micrograms/m^3) and (to a more limited extent) chemical speciation. Based on laboratory studies discussed above, the impacts of emitted PAH on ambient PM are more likely apparent in PM number concentration (# of particles/m^3) which is infrequently monitored. PAHs may also be detected in a chemical analysis of PM, but this is not a standard part of EPA chemical speciation reporting. Monitors located near roadways may be relevant to the question of fuel mix and PAH emissions.

Research Question #4: How do PAH emissions from liquid fuels and fuel blends contribute to ambient PAH and PM?

The establishment of a near-road monitoring site – either for a short-term field study, noted above, or for longer-term tracking – would be useful to address local community exposure. A single community analysis could be appropriate to extrapolate results to other locations. To estimate exposure over a wider area without measurements, model data could be used (as in the EPA NATA), but model skill would affect the reliability of results.

5. The toxicity of individual PAH compounds and the toxicity of PM associated with PAH (chemical compounds and/or particle size)

Health risk of air pollution exposure is determined by epidemiological studies (population based) or toxicological studies (laboratory control studies). Both of these approaches have been used with PAH as well as ultrafine PM. In the experience of the authors, epidemiological studies are more commonly discussed to support the criteria air pollutant levels, whereas toxicological studies are more commonly discussed to support hazardous air pollutants – most likely because acute health affects from criteria pollutants are more easily observed in the population (e.g. more hospital visits on high PM days) as opposed to cancer and other effects that are more difficult to link with exposure.

Research Question #5: What are the health risks associated with PAHs, PAHs in PM, and total fine PM exposure?

This question could be addressed with epidemiological studies (especially in concert with increased ambient monitoring) or laboratory studies akin to those discussed earlier in this report. The research needs in this area are vast, but existing research offers examples of studies that could be advanced in new locations with additional data.

6. The summative change in health outcomes associated with changes in emissions, ambient concentrations, population exposure, and toxicity

Linking vehicle emissions with public health is a challenge. The wide range of disciplinary expertise, methodological approaches, and spatial scales appropriate to each component require integration across individuals and expertise to tackle this policy-relevant problem. Whereas vehicle emissions are typically evaluated by engineers in a laboratory setting, ambient concentration are assessment by atmospheric chemists through field studies and models, and health impacts are address by public health and medical researchers at the individual or community scale.

Research Question #6: What is the net impact of on-road PAH emissions on public health?

Models offer a unifying framework to represent emissions changes (e.g. with the MOVES model), calculate ambient concentrations and population exposure (e.g. with the CMAQ model), and from this calculate health impacts (by applying concentration-response functions from epidemiological or toxicological studies). Each component in this type of modeling system depends on laboratory and field measurements, and could be advanced in coordination with empirical vehicle, atmospheric chemistry, and/or public health monitoring and tracking. Alternatively, an effective step forward may be the convening of a scientific meeting specific to vehicular PAH and PM health effects.

Much is known about on-road emissions of PAHs, and options for reducing these emissions and associated air and health impacts. However, results do not always point in the same direction, and current generation models may be not yet be suited for policy-relevant applications. Still, rapid advances in PAH-related research offer promise to create links between fuel mix and public health impacts.

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Appendix A: List of Volatile Organic Compounds created by the EPA[4]

Volatile Organic Compounds (VOCs)		
1,1,1,2-Tetrachloro-2, 2-difluoroethane	1,1,2-TCA	1,1-Dichloro-1-nitroethane
1,1-Dimethylcyclohexane	1,2-Dichloroethylene	1,2-Dimethylbenzene
1,2,2,2-Tetrachlorethane	1,2,2,2-Tetrachlorethane	1,2-Dichloroacetate
1,2-Dichlorobenzene	1,2-Dichloropropylene	1,2-Epoxybutane
1,2,3-Trichloropropane	1,3-Butadiene	1,3-Dichlorobenzene
1,3-Dichloropropene	1,3-Dimethybutyl acetate	1,4-Dichloro-2-butene
1-Chloro-1-nitropropane	1-Nitropropane	2,2,4-Trimethylpentane
2,3-Dichloropropene	2-Chloro-1, 1,1-trifluorethane	2-Chloroethyl vinyl ether
2-Nitropropane	2-Propyn-1-ol	3-Chloro-2-methylpropene
4-Methyl-2-pentanone	Acetaldehyde	Acetylene
Acetylene dichloride	Acetic acid	Acetic acid, propyl ester
Acetic anhydride	Acetone	Acetyl bromide
Acetyl chloride	Acetylene tetrabromide	Acrolein
Acrylyl chloride	Aminoethane	Aziridine
Benzene	Bromochlorodifluoromethane	Bromoform
Bromomethane	Chlorinated fluorocarbon	Chlorodibromomethane
Chloroethane	Chloroethanol	Chloroform
Chloromethane	Chloromethyl methyl ether	CIPC
Diacetone alcohol	Diazomethane	DIBK
Dibromodifluoromethane	Dibutyl phosphate	Dichloroacetylene
Dichlorobromomethane	Dichlorodifluoromethane	Dichloromethane
Dichloromethylphenylsilane	Dichloromonofluoromethane	Dichloropropanes
Dichlorosilane	Diethyl ketone	Diethylamine
Diisopropylamine	Dimethoxane	Dimethyldichlorosilane
Dimethylhydrazine	Dimethylvinyl chloride	Dipropyl ketone
Ethane	Ethanol	Ethanolamine
Ethyl acetate	Ethyl acrylate	Ethyl alcohol
Ethyl bromide	Ethyl butyl ketone	Ethyl chloroformate
Ethyl ether	Ethyl isocyanate	Ethyl sec-amyl ketone
Ethyl sulphide	Ethylbenzene	Ethylene
Ethylene dichloride	Ethylene fluorohydrin	Ethylenediamine
Ethylidene chloride	Ethyltoluene	Ethylxylene
Formaldehyde	Formamide	Formic acid
Freon 11	Freon 112	Freon 114
Glycidaldehyde	Halon 1301	Heptane
Hexafluoroacetone	Hexane	Hydrazine
Isoamyl acetate	Isobutyl acetate	Isobutyl alcohol
Isobutyl chloroformate	Isobutylamine	Isobutylraldehyde

[4]https://iaspub.epa.gov/sor_internet/registry/substreg/searchandretrieve/advancedsearch/search.do?details=displayDetails&selectedSubstanceId=83723

Isobutyronitrile	Isooctyl alcohol	Isopropene
Isopropyl acetate	Isopropyl alcohol	Isopropyl benzene
Isopropyl chloroformate	Isopropyl ether	Isopropylamine
Ketene	Lead acetate	MBK
Mercuric acetate	Mesitylene	methanamine
Methane	Methyl 2-chloroacrylate	Methyl acetate
Methyl acrylate	Methyl alcohol	Methyl chloroform
Methyl cyanide	Methyl ethyl ketone	Methyl ethyl ketone peroxide
Methyl formate	Methyl iodide	Methyl isoamyl ketone
Methyl isopropyl ketone	Methyl mercury	Methyl methacrylate
Methyl n-amyl ketone	Methyl propyl ketone	Methyl tert-butyl ether
Methyl vinyl ketone	methylchlorosilane	Methyldichlorosilane
Methylamine	Methylcylohexane	Methylhydrazine
Methylene bromide	Methylene chloride	Methyltrichlorosilane
Monochloroacetone	Monochlorobenzene	Morpholine
n-Butyl alcohol	n-Butyl ether	n-Propyl nitrate
Naphthalene	Nickel acetate	Nitroethane
Nitromethane	Nitropropane	Nonane
Octane	ortho-Chlorotoluene	Oxirane
p-Dioxane	Paraformaldehyde	PCE
Pentachloroethane	Pentane	Perchloroethane
Perchloroethylene	Phenyl ether, vapor	Phenylhydrazine
Phenylhydrazine hydrochloride	Propane	Propanol
Proparagyl bromide	Propenylbenzene	Propionaldehyde
Propionic acid	Propionitrile	Propyl chloroformate
Propylene	Propylene dichloride	Propylene oxide
Propyleneimine	Propyne	sec-Amyl acetrate
sec-Butyl acetate	sec-Butyl alcohol	Stoddard solvent
Styrene	T-1,2-Dichloroethylene	TCA
tert-Butanol	tert-Butyl acetate	tert-Butyl chromate
Tetrachloroethane	Tetrachloroethanol	Tetraethyllead
Tetraethyltin	Tetramethyl lead	Toluene
Toluol	trans-1,4-Dichlorobutene	Trichloroacetic acid
Trichloroacetyl chloride	Trichloroethylene	Trichloroethylsilane
Triethanolamine	Triethoxysilane	Triethylamine
Triiodomethane	Trimethyl phosphite	Trimethylamine
Trimethylchlorosilane	Vinyl acetate	Vinyl bromide
Vinyl chloride	Vinyl cyanide	Vinylbenzene
Vinyltoluene	M-Xylene	O-Xylene
P-Xylene	Xylxyl bromide	

Appendix B: PAHs and Related Compounds

Adapted from the National Research Council (US) Committee on Pyrene and Selected Analogues. Polycyclic Aromatic Hydrocarbons: Evaluation of Sources and Effects. Washington (DC): National Academies Press (US); 1983. APPENDIX A, LISTS OF POLYCYCLIC AROMATIC HYDROCARBONS.[5]

Name	Molecular Formula	Other names	Molecular Weight	Carcinogenic Activity (NA = not available)
1H-Indole	C8H7N		117	0
Naphthalene	C10H8		128	0
Isoquinoline	C9H7N		129	0
Quinoline	C9H7N		129	+
Acenaphthylene	C12H8		152	0
Biphenylene	C12H8		152	NA
9H-Fluorene	C13H10		166	0
1H-Phenalene	C13H10		166	NA
9H-Carbazole	C12H9		167	0
Anthracene	C14H10		178	0
Phenanthrene	C14H10		178	0
Acridine	C13H9N		179	0
Benzo[f]quinoline	C13H9		179	0
Benzo[h]quinoline	C13H9		179	0
Phenanthridine	C13H9N		179	+
9H-Fluoren-9-one	C13H8O		180	NA
Benzo[c]cinnoline	C12H8N2	(Phenazone)	180	NA
1,10-Phenanthroline	C12H8N2		180	NA
Phenazine	C12H8N2		180	NA
9H-Xanthene	C13H10O		182	NA
Dibenzothiophene	C12H8S		184	0
4H-Cyclopenta[def]phenanthrene	C15H10		190	NA
9(10H)-Anthracenone	C14H10O	(Anthrone)	194	0
Acephenanthrylene	C16H10		202	NA
Fluoranthene	C16H10		202	+
Pyrene	C16H10		202	0

[5] https://www.ncbi.nlm.nih.gov/books/NBK217760/

Phenanthro[4,5-bcd]thiophene	C14H8S		208	NA
9,10-Anthracenedione	C14H8O2	(Anthraquinone)	208	NA
9,10-Phenanthrenedione	C14H8O2	(Phenanthraquinone)	208	NA
11H-Benzo[a]fluorene	C17H12		216	0
11H-Benzo[b]fluorene	C17H12		216	NA
7H-Benzo[c]fluorene	C17H12		216	0
Benzo[ghi]fluoranthene	C18H10		226	0
Cyclopenta[cd]pyrene	C18H10		226	+
Benz[a]anthracene	C18H12		228	+
Benzo[c]phenanthrene	C18H12		228	+
Chrysene	C18H12		228	0/+
Naphthacene	C18H12		228	0
Triphenylene	C18H12		228	0
Benz[c]acridine	C17H11		229	+
Naphtho[2,3-f]quinoline	C17H11		229	0/+
7H-Benz[de]anthracen-7-one	C17H10O	(Benzanthrone)	230	NA
Benzo[b]naphtho[2, 1-d]thiophene	C16H10	(Benzo[a]dibenzothiophene)	234	0
Benzo[b]fluoranthene	C20H12	(Benz[e]acephenanthrylene)	252	++
Benzo[j]fluoranthene	C20H12		252	++
Benzo[k]fluoranthene	C20H12		252	++
Benzo[a]pyrene	C20H12		252	++
Benzo[e]pyrene	C20H12		252	0/+
Perylene	C20H12		252	0
7H-Dibenzo[a,g]carbazole	C20H13		267	+
13H-Dibenzo[a,i]carbazole	C20H13		267	+
7H-Dibenzo[c,g]carbazole	C20H13		267	++
Anthanthrene	C22H12	(Dibenzo[def,mno]chrysene)	276	0
Benzo[ghi]perylene	C22H12		276	+
Indeno[1,2,3-cd]pyrene	C22H12		276	+
Benzo[b]chrysene	C22H14		278	0
Benzo[c]chrysene	C22H14		278	+
Benzo[g]chrysene	C22H14		278	+

Benzo[b]triphenylene	C22H14	(Dibenz[a,c]anthracene)	278	+
Dibenz[a,h]anthracene	C22H14		278	+
Dibenz[a,j]anthracene	C22H14		278	+
Pentaphene	C22H14	(Dibenzo[b,h]phenanthrene)	278	0
Picene	C22H14		278	0
Benzo[h]naphtho [1,2-f]quinolene	C21H13		279	+
Dibenz[a,h]acridine	C21H13		279	+
Dibenz[a,j]acridine	C21H13		279	+
Dibenz[c,h]acridine	C21H13		279	+
Coronene	C24H12		300	0/+
Benzo[rst]pentaphene	C24H14	(Dibenzo[a,i]pyrene)	302	++
Dibenzo[b,def]chrysene	C24H14	(Dibenzo[a,h]pyrene)	302	++
Dibenzo[def,p]chrysene	C24H14	(Dibenzo[a,l]pyrene)	302	++
Naphtho[1,2,3,4-def] chrysene	C24H14	(Dibenzo[a,e]pyrene)	302	++

Appendix C: Population-weighted source contributions to PAHs in the Continental United States

For January (Table 1) and July (Table 2) from *Zhang et al.* [2016].

Table 1
Population-weighted source contributions to PAHs in the continental United States for January 2011.

	C123	EGU	MVH	NPT	NRD	OGS	OPT	RWC	OTH
NAPH	1.1%	0.3%	12.9%	7.0%	4.1%	0.1%	5.3%	60.1%	9.1%
ACE	1.3%	0.8%	17.2%	2.0%	8.4%	1.7%	27.3%	34.6%	6.8%
ACY	0.4%	0.0%	10.9%	0.2%	4.2%	0.3%	5.7%	75.8%	2.4%
FLU	1.5%	0.1%	19.8%	0.3%	9.9%	0.2%	5.4%	51.1%	11.7%
PHE	1.3%	0.1%	15.1%	0.6%	5.9%	1.1%	11.4%	55.2%	9.4%
ANT	1.6%	0.1%	14.3%	0.8%	6.9%	0.2%	8.7%	64.2%	3.2%
FTH	2.2%	0.2%	15.7%	2.0%	8.1%	0.2%	12.9%	53.5%	5.2%
PYR	3.0%	0.1%	20.9%	1.3%	10.7%	2.0%	12.2%	44.9%	4.8%
CHRY	2.0%	0.2%	15.6%	3.0%	8.8%	0.5%	14.1%	52.0%	3.8%
BaA	2.5%	0.1%	16.0%	1.5%	9.2%	2.4%	30.5%	35.1%	2.6%
BbF	0.6%	0.4%	16.5%	3.4%	9.5%	0.0%	14.2%	52.0%	3.5%
BkF	0.1%	0.3%	17.2%	1.7%	9.5%	0.0%	17.9%	49.0%	4.3%
BghiP	0.0%	0.1%	27.1%	0.5%	16.6%	13.0%	26.4%	14.3%	2.0%
BaP	1.2%	0.1%	19.4%	1.4%	11.3%	7.7%	31.0%	25.9%	2.0%
IcdP	0.2%	0.2%	30.6%	1.1%	18.1%	0.4%	9.1%	37.4%	3.0%
DahA	1.4%	1.3%	14.6%	25.9%	9.6%	0.0%	39.6%	0.0%	7.5%
15PAH	1.5%	0.1%	16.7%	0.9%	7.9%	1.3%	12.3%	52.1%	7.2%
$\sum$ cPAH	1.4%	0.2%	18.1%	2.3%	10.4%	2.0%	21.2%	41.3%	3.2%
TEC_{cPAH}(low)	1.3%	0.2%	19.1%	2.5%	11.1%	5.8%	29.0%	28.5%	2.5%
TEC_{cPAH}(high)	1.3%	0.4%	17.6%	5.3%	10.3%	3.6%	27.1%	31.0%	3.3%

C123: (locomotives and class 1–3 marine vessels), EGU (point sources of electric generation units), MVH (motor vehicles), NPT (non-point sources), NRD (non-road engines), OGS (oil and gas processes), OPT (other industrial point sources), RWC (residential wood combustion) and OTH (non-US sources; also includes all non-US C3 CMV and offshore oil production processes). 15PAH is the sum of the concentrations of all the PAHs except NAPH. $\sum$ cPAH is the sum of the concentrations of the seven carcinogenic PAHs (cPAHs).

Table 2
Population-weighted source contributions to PAHs in the continental United States for July 2011.

	C123	EGU	MVH	ARE	NRD	OGS	OPT	RWC	OTH
NAPH	3.0%	0.9%	23.9%	15.1%	13.9%	0.2%	13.5%	3.7%	25.8%
ACE	1.9%	1.0%	14.2%	1.9%	15.5%	1.6%	43.1%	1.2%	19.5%
ACY	2.0%	0.1%	24.1%	0.5%	19.8%	1.2%	25.1%	5.9%	21.2%
FLU	2.7%	0.2%	20.3%	0.3%	23.3%	0.2%	9.2%	2.6%	41.2%
PHE	2.8%	0.1%	20.1%	0.7%	15.5%	1.4%	22.5%	2.7%	34.2%
ANT	4.7%	0.2%	25.7%	1.4%	26.0%	0.4%	22.8%	4.6%	14.1%
FTH	4.6%	0.3%	22.2%	1.9%	24.6%	0.3%	24.9%	3.3%	17.8%
PYR	5.2%	0.2%	23.4%	1.3%	26.0%	2.8%	23.0%	2.4%	15.6%
CHRY	4.7%	0.4%	19.4%	3.2%	25.9%	0.7%	28.6%	4.0%	13.1%
BaA	4.1%	0.2%	15.2%	1.3%	20.1%	3.0%	46.3%	2.1%	7.7%
BbF	1.5%	0.8%	14.8%	3.4%	26.7%	0.0%	31.4%	4.6%	16.8%
BkF	0.2%	0.7%	11.5%	1.7%	22.0%	0.0%	36.6%	3.8%	23.4%
BghiP	0.1%	0.1%	9.7%	0.4%	24.9%	10.5%	49.7%	0.6%	4.0%
BaP	1.7%	0.2%	10.3%	1.0%	18.0%	7.6%	53.9%	1.2%	6.1%
IcdP	0.4%	0.3%	18.6%	1.4%	43.5%	0.6%	19.1%	3.0%	13.2%
DahA	1.8%	1.3%	8.0%	8.8%	14.5%	0.0%	45.5%	0.0%	20.2%
15PAH	3.1%	0.3%	19.5%	1.1%	20.7%	1.8%	25.4%	2.7%	25.5%
$\sum$ cPAH	2.8%	0.4%	14.9%	2.0%	23.4%	2.6%	39.8%	2.8%	11.4%
TEQ_{cPAH}(low)	2.0%	0.3%	11.3%	1.6%	19.3%	5.9%	50.2%	1.5%	7.8%
TEQ_{cPAH}(high)	2.2%	0.5%	11.8%	3.0%	19.6%	3.8%	46.2%	1.8%	11.1%

C123: (locomotives and class 1–3 marine vessels), EGU (point sources of electric generation units), MVH (motor vehicles), NPT (non-point sources), NRD (non-road engines), OGS (oil and gas processes), OPT (other industrial point sources), RWC (residential wood combustion) and OTH (non-US sources; also includes all non-US C3 CMV and offshore oil production processes). 15PAH is the sum of the concentrations of all the PAHs except NAPH. $\sum$ cPAH is the sum of the concentrations of the seven carcinogenic PAHs (cPAHs).