### Correspondence Between Doug Sombke South Dakota Farmers Union and Chris Grundler, Director, EPA/OTAQ

December 18, 2017

Mr. Christopher Grundler USEPA Headquarters William Jefferson Clinton Building 1200 Pennsylvania Ave. NW MC6401A Washington, DC 20460

Dear Mr. Grundler:

The December 16<sup>th</sup> Wall Street Journal carried an article entitled "Blowing Leaves, and Pollution".<sup>1</sup> It contained a chart—appropriately captioned "Blowing Smoke"—that compared emissions from "small nonroad spark ignition engines" (e.g., leaf blowers) to "onroad light duty vehicles" (e.g., cars and light trucks powered by gasoline). The chart noted that the information was 2014 U.S. EPA data.

Since EPA says that fine particulate matter poses the greatest health threat to Americans, I was stunned to see that EPA believes that PM2.5 (fine particulate) emissions from offroad sources (43,442 tons) were nearly the same as from LDVs ((59,301 tons). In fact, the author stated that "Overall, EPA figures show that the small, nonroad engines contribute...1% of particulate matter, the same amount as passenger cars."

As you must know, Mr. Grundler, that statement is absurd! The U.S. has only 12 million "leaf blowers", compared to 260 million light-duty vehicles. It is ludicrous for EPA to expect Americans to believe that 12 million "leaf blowers" emit nearly as much PM2.5 as do 260 million cars and trucks.

So I did some digging. I discovered that your group plays a very big role in all of this. It turns out that just a handful of technocrats at OTAQ are responsible for the nation's transportation fuels regulatory policy, and that you and your team have been its primary architect for 30 years or more. During that time, OTAQ has failed to update its models to include gasoline exhaust <u>secondary</u> organic aerosols (SOAs), which is what comprises the vast majority of urban PM2.5. (OTAQ only acknowledges <u>primary</u> organic aerosols from gasoline.)

It also just so happens that the predominant source of urban SOAs is the 25 – 30% fraction of gasoline known as aromatic hydrocarbons—benzene-based compound (BTEX) used by refiners to increase gasoline octane ratings.

To make matters even worse, OTAQ's attempt to hide the quantities and source of these dangerous SOAs has also covered up the fact that the SOAs bind, insulate, preserve, and

<sup>&</sup>lt;sup>1</sup> Jo Craven McGinty, "Blowing Leaves, and Pollution", Wall Street Journal, December 16 - 17, 2017, A2

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transport some of the most carcinogenic and mutagenic nano-particles known to manpolycyclic aromatic hydrocarbons. These are mobile source air toxics (MSATs) that Congress mandated EPA to reduce to the "greatest achievable extent" in the 1990 Clean Air Act Amendments. They are lethal in the parts per trillion, and urban Americans cannot escape them.

For more than 25 years, OTAQ has defied the will of Congress, and misled the public, media, and policymakers. You have told us that diesel fuels and vehicles, not gasoline and its BTEX carcinogens, accounted for 90+% of urban fine particulate matter.

Apparently, you do not communicate with your colleagues in the Office of Research and Development (ORD). In June of this year, ORD finally admitted what many experts have been saying for years-gasoline exhaust is the predominant source of urban SOAs. In fact, it accounts for 13 times more SOAs than does diesel exhaust.

Mr. Grundler, I think that you and your colleagues at OTAQ are the ones that are "blowing smoke". My question to you: after all these years of inaction and obfuscation, do you have any plans to "come clean", admit the truth about gasoline BTEX, and discharge your sworn duty to protect the health and welfare of the American people, especially the most vulnerable among us-our children?

Yours is a very serious responsibility. OTAQ is viewed by the media, policymakers, and the public as the "experts" on fuel composition and emissions effects. When you provide misinformation as you have here, bad policy decisions results—either by omission or commission.

I have attached my recent letter to Senator Mike Rounds, a member of the Senate Environment and Public Works Committee. The Senator considers this to be a very important issue, and my organization-and many others-plan to elevate this conversation in 2018.

I look forward to having the opportunity to visit with you about all of this soon.

Respectfully,

Sombly

Doug Sombke President, South Dakota Farmers Union

Cc: Senator Mike Rounds

# Grundler Letter (1) Response to Sombke Letter (1)



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY NATIONAL VEHICLE AND FUEL EMISSIONS LABORATORY 2565 PLYMOUTH ROAD ANN ARBOR, MICHIGAN 48105-2498

JAN 1 9 2018

OFFICE OF AIR AND RADIATION

Mr. Doug Sombke, President South Dakota Farmers Union 1410 Dakota Avenue South P.O. Box 1388 Huron, South Dakota 57350

Dear Mr. Sombke:

Thank you for your letter of December 19, 2017, to U.S. Environmental Protection Agency Office Director Christopher Grundler, in which you questioned EPA's emission estimates for onroad light-duty vehicles and small nonroad spark-ignition engines, which include leaf blowers and other applications. You also pointed to problems that you perceive in EPA's treatment of secondary organic aerosols (SOA) from gasoline exhaust. The Office Director has asked me to respond to you on his behalf.

EPA's National Emission Inventory (NEI), cited in the Wall Street Journal article you referenced in your letter, is the Agency's official estimate of emissions from air pollution sources in the U.S.<sup>1</sup> It is based on data provided by State, local, and tribal agencies as well as data developed by the EPA. Emission standards differ between onroad and nonroad engines. As noted in the Wall Street Journal article, the two-stroke engines in small spark-ignition equipment have far higher emission rates of particulates and gases relative to onroad gasoline-powered vehicles (per unit activity) causing them to have a disproportionate impact on air quality. A number of independent academic studies have also pointed to the higher emissions per unit activity or fuel use when comparing onroad and nonroad spark-ignition engines.<sup>2,3</sup> The EPA seeks continuous improvement of its emissions estimates and is currently working to update its estimates of the population and activity of nonroad engines and equipment.

In your letter, you state that the EPA has failed to update our models to address gasoline SOA. In fact, the EPA has continually been working on improvements to the modeling of SOA, including from gasoline exhaust. Since 2010, the EPA has incorporated new mechanisms in its Community Multiscale Air Quality (CMAQ) model to account for new information on:

- SOA yields from aromatic compounds at different NOx levels<sup>4</sup>;
- new mechanisms for SOA from alkanes and polycyclic aromatic hydrocarbons<sup>5</sup>;
- water solubility of SOA<sup>6</sup>;

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- SOA from plant-generated (biogenic) emissions of organic gases (e.g, isoprene, monoterpenes, sesquiterpenes)<sup>7,8,9</sup>;
- role of semi volatile and intermediate volatility organic compounds<sup>10,11</sup>; and
- numerous other improvements related to meteorology and other inputs.

Furthermore, in collaboration with external groups, the EPA has applied multiple iterations of the CMAQ model to the California Research and the Nexus of Air Quality and Climate (CalNex) field campaign to improve our understanding of sources of SOA<sup>10,11,12,13</sup>.

With respect to the contribution of gasoline and its aromatic hydrocarbons to organic aerosols and ambient fine particulate (PM2.5), independent academic summaries of the available scientific evidence indicate uncertainty about the role of gasoline vs. diesel sources and the potential impact of fuel reformulation in reducing SOA.

For example, a number of different research groups in December 2016 published an article titled, Review of Urban Secondary Organic Aerosol Formation from Gasoline and Diesel Motor Vehicle Emissions.<sup>14</sup> The article summarizes the available scientific literature this way:

- "Gasoline- and diesel-powered motor vehicles, both on/off-road, are important sources of SOA precursors. They emit complex mixtures of gas-phase organic compounds that vary in volatility and molecular structure – factors that influence their contributions to urban SOA. However, the relative importance of each vehicle type with respect to SOA formation remains unclear due to conflicting evidence from recent laboratory, field, and modeling studies. Both are likely important, with evolving contributions that vary with location and over short time scales."
- "Motor vehicle emissions are an important source for urban SOA in the developed world, but it is possible that it no longer dominates due to the dramatic reduction in emissions over the past few decades in response to strict vehicle emission standards."
- "Fuel reformulation is an option to reduce known SOA precursor components (i.e., aromatics), but existing evidence is unclear on how effective reformulation will be for SOA control..."

The Health Effects Institute also recently published an Executive Summary of its December 2016 workshop on fuel composition and PM.<sup>15</sup>

In your letter, you noted the recent study from EPA's Office of Research and Development.<sup>13</sup> The publication, based on the CalNex study, centered exclusively on Southern California around Los Angeles. While their model does show that gasoline is the single largest source of SOA explicitly identified in that area, the majority of total organic aerosol in their study came from non-mobile sources. Other sources, including cooking, biomass burning, and "other anthropogenic SOA," contributed more than 50% of the organic aerosol in urban areas, and more so in non-urban areas. The study also found that their model significantly underestimated organic aerosol concentrations at some locations (a factor of 3 in Pasadena), highlighting that considerable unknowns remain in attributing SOA to sources in CalNex. A number of other key limitations in the study make it difficult to generalize for other uses. Other publications have also argued that vehicles may not be the dominant source of organic aerosols observed in CalNex.<sup>16</sup>

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The EPA will be continuing to systematically follow the relevant scientific literature; consult with independent organizations such as the Health Effects Institute to summarize available evidence; collaborate across our offices and with other researchers to understand and accomplish key research goals; and actively work to improve our emissions and air quality models.

Again, thank you for your letter. I appreciate the opportunity to be of service and trust the information provided is helpful.

Sincerely,

Kathum A.S.

William J. Charmley, Director Assessment and Standards Division

http://pubs.acs.org/doi/10.1021/es100636q]

<sup>&</sup>lt;sup>1</sup> https://www.epa.gov/air-emissions-inventories/national-emissions-inventory-nei

<sup>&</sup>lt;sup>2</sup> Gordon, T.D.; Tkacik, D.S.; Presto, A.A.; et al. (2013) Primary gas- and particle-phase emissions and secondary organic aerosol production from gasoline and diesel off-road engines. *Environmental Science & Technology* 47(24), pp. 14137-14146. [Online at http://pubs.acs.org/doi/abs/10.1021/es403556e]

<sup>&</sup>lt;sup>3</sup> Zhao, Y.; Nguyen, N.T.; Presto, A.A.; et al. (2016) Intermediate volatility organic compound emissions from onroad gasoline vehicles and small off-road gasoline engines. *Environmental Science & Technology* 50(8), pp. 4554-4563. [Online at http://pubs.acs.org/doi/full/10.1021/acs.est.5b06247]

<sup>&</sup>lt;sup>4</sup> Carlton, A.G.; Bhave, P.V.; Napelenok, S.L.; et al. (2010) Model representation of secondary organic aerosol in CMAQv4.7. Environmental Science & Technology 44(22), pp. 8553-8560. [Online at

<sup>&</sup>lt;sup>5</sup> Pye, H.O.T.; Pouliot, G.A. (2012) Modeling the role of alkanes, polycyclic aromatic hydrocarbons, and their oligomers in secondary organic aerosol formation. *Environmental Science & Technology* 46(11), pp. 6041-6047. [Online at http://pubs.acs.org/doi/abs/10.1021/es300409w]

<sup>&</sup>lt;sup>6</sup> Pye, H.O.T.; Murphy, B.N.; Xu, L.; et al. (2017) On the implications of aerosol liquid water and phase separation for organic aerosol mass. *Atmospheric Chemistry and Physics* 17, pp. 343-369. [Online at https://www.atmoschem-phys.net/17/343/2017/]

<sup>&</sup>lt;sup>7</sup> Pye, H.O.T.; Pinder, R.W.; Piletic, I.R.; et al. (2013) Epoxide pathways improve model predictions of isoprene markers and reveal key role of acidity in aerosol formation. *Environmental Science & Technology* 47(9), pp. 11056-11064. [Online at http://pubs.acs.org/doi/abs/10.1021/es402106h]

<sup>&</sup>lt;sup>8</sup> Pye, H.O.T.; Luecken, D.J.; Xu, L.; et al. (2015) Modeling the current and future roles of particulate organic nitrates in the Southeastern United States. *Environmental Science & Technology* 49(24), pp. 14195-14203. [Online at http://pubs.acs.org/doi/abs/10.1021/acs.est.5b03738]

<sup>&</sup>lt;sup>9</sup> Pye, H.O.T.; Zuend, Fry, J.L.; et al. (2018) Coupling of organic and inorganic aerosol systems and the effect on gasparticle partitioning in the southeastern US. *Atmospheric Chemistry and Physics* 18, pp. 357-370. [Online at https://www.atmos-chem-phys.net/18/357/2018/]

<sup>&</sup>lt;sup>10</sup> Murphy, B.N.; Woody, M.C.; Jimenez, J.L.; et al. (2017) Semivolatile POA and parameterized total combustion SOA in CMAQv5.2: impacts on source strength and partitioning. *Atmospheric Chemistry & Physics* 17, 11107-11133. [Online at https://www.atmos-chem-phys.net/17/11107/2017/]

# Sombke Letter (2) Response to Grundler Letter (1)



1410 Dakota Avenue South PO Box 1388 Huron, SD 57350 605:352.6761 www.sdfu.org

February 5, 2018

Mr. Christopher Grundler USEPA Headquarters William Jefferson Clinton Building 1200 Pennsylvania Ave. NW MC6401A Washington, DC 20460

Dear Mr. Grundler:

Thank you for your letter of January 19, 2018, responding to my letter to you of December 19, 2017. I was a bit confused by your letter's signature block, which suggests it was authored by William J. Charmley, but signed on his behalf by Kathryn A. Sargeant. Nonetheless, I am addressing my response to you.

I appreciate your taking the time to explain why EPA's chart printed in the December 16, 2017 Wall Street Journal wasn't a case of "Blowing Smoke". [The article cites 2014 EPA estimates that PM2.5 (fine particulate) emissions from offroad sources (43,442 tons) are nearly as much as light-duty vehicles (59,301 tons). Relying on EPA's data, the author misled the public and policymakers by saying that: "Overall, EPA figures show that the small, nonroad engines contribute...1 percent of particulate matter, the same amount as passenger cars."] I expressed amazement that EPA could actually expect Americans to believe such a statement.

Unfortunately, even though your letter contained a number of footnotes, you did not answer my question. You did not explain how 12 million "leaf blowers" can emit as much PM2.5 as 260 million light-duty vehicles. It may be true that "two-stroke engines...have far higher emission rates of particulates and gases relative to on-road gasoline powered vehicles". However, this does not account for EPA's enormous under-prediction of LDV particulate matter emissions (and their associated toxics), even though by EPA's own admission such emissions pose the greatest threat to Americans' health.

Many experts warn that EPA's modeling of LDV particulate emissions is seriously flawed. EPA's failure to accurately report the extent of these harmful emissions poses a serious threat to the public health and welfare, because the causes are not being correctly identified and controlled.

Writing for the Physicians for Social Responsibility, Dr. Doug Brugge from Tufts University School of Medicine said: "Most Americans are unaware that particulate pollution is the single most deadly pollution they face (and the pollutant of greatest economic consequence). Nor is there much awareness that existing regulations are inadequate...the smaller the tiny airborne particles are the more toxic they become."<sup>1</sup>

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In its 2007 Fine Particle rule, EPA acknowledged that its models are in need of major overhaul. The question is: ten years later, why have the problems not been corrected? We have an extensive research effort underway with regard to toxic aromatics and will release a report in the coming months. Some of the issues we are addressing, and issues raised by your letter include the following:

#### EPA's Reliance upon Health Effects Institute (HEI)

We are familiar with EPA's relationship with HEI. Your letter references HEI's Executive Summary of its December 2016 workshop on fuel composition and PM. We have been in contact with a number of people who attended that workshop, and have reviewed their notes of the proceeding. They report that a majority of presenters and commentators emphasized one recurring theme: EPA's models are flawed, and should be informed by real-world data. The majority of experts stated that to make proper decisions, regulators must utilize real-time measurements of real world fuels combusted under real world driving conditions, as Europe is now doing in the aftermath of the "Dieselgate" scandal.

Our literature search has revealed numerous studies that have used advanced real-time measurement techniques [e.g., aerosol mass spectrometers for SOAs, single-particle soot photometers for black carbon measurements, and gas-phase measurements of VOCs (benzene, toluene, etc.) with proton-transfer-reaction mass spectrometers]. The studies confirm that gasoline exhaust emissions far outweigh diesel exhaust emissions. The overwhelming preponderance of gasoline over diesel emissions makes sense, since the U.S. annually consumes more than 140 billion gallons of gasoline, four times greater than diesel fuel consumption. Gasoline-powered internal combustion engines power 95 percent of the vehicles on U.S. roads.

Dr. Roya Bahreini, University of Colorado – Boulder and colleagues published a 2012 study of real-time measurement of Los Angeles pollution plumes that said: *"The result indicates that the contribution from diesel emissions to SOA formation is zero within our uncertainties. <u>Therefore, substantial reductions of SOA mass on local to global scales will be achieved by reducing gasoline vehicle emissions."</u> Similar results were reported for studies of urban pollution plumes in Paris, France, despite their heavier use of diesel vehicles. In 2013, Nordin et al. published a paper that compared the Bahreini work to a Gerntner study (which you cited) that questioned the Gerntner results, and concluded that: <i>"gasoline exhaust readily forms secondary organic aerosol with a signature aerosol mass spectrum with similarities to the oxidized organic aerosol that commonly dominates the OA mass spectrum in and downwind of urban areas. This substantiates recent claims that gasoline SOA is a dominating source to SOA in and downwind of large metropolitan areas.<sup>2</sup>* 

However, in stark contrast to real-time measurements using sophisticated and proven techniques, your Agency's models predict that gasoline exhaust is only a minor contributor to urban PM2.5 emissions, on par with that of 12 million "leaf blowers". Consequently, EPA has failed to address the most harmful fraction of gasoline—the 25 – 30 percent which is comprised of benzene-based aromatic hydrocarbons (BTEX) added by refiners to boost gasoline octane ratings, and which Harvard School of Public Health and other experts confirm is the predominant source of urban SOA-bound toxics. This oversight is causing substantial harm to the public health and welfare.

Research by Pacific Northwest National Labs and others confirms that BTEX-generated secondary organic aerosols (SOAs) synergistically bond with highly toxic polycyclic aromatic hydrocarbons (PAHs), insulate and preserve them, and thus enable their long-range transport (LRT) for months and miles. EPA's official position is that these toxic particles, to the extent they even exist, have very short half-lives (less than an hour), and a range of only 300 feet. In fact, the vast majority of Americans are exposed to these lethal SOA-borne PAHs on a 24/7 basis, and they penetrate into homes, cars, and schools. Infants and children suffer the most from these carcinogenic, mutagenic, and endocrine-disrupting compounds.

HEI has acknowledged the distinctions between gasoline and diesel exhaust. "Typically, gasoline UFPs [ultrafine particles] contain a higher fraction of heavy polycyclic aromatic hydrocarbons (PAHs) than diesel exhaust (DE)

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which may have implications for the differential toxicity of these particles..." [Health Effects Institute Study on Ultrafine Particulates, Feb. 2013, p. 21.]

Dr. Andre Nel, a highly regarded UCLA researcher, published a May 6, 2005 Science article that underscored the dangers gasoline ultrafine particles pose to human health: "Ultrafines are the major component in vehicle emissions—the largest source of air pollution in urban areas—and they have the largest surface area and high content of potentially toxic hydrocarbons among all PM sources. They can also penetrate deeper into lung tissue than fine or coarse particles."

In 2010, National Institute of Environmental Health Sciences (NIEHS) Director Dr. Linda Birnbaum testified before the Senate Environment and Public Works Committee about the dangers these emissions pose to children, comparing them to leaded gasoline's effects: "In a 2009 study of the effects of PAHs on children's IQ in NYC...the mothers' exposure as measured during their pregnancies by wearing backpack monitors was associated with a decrease in IQ among the more exposed children. The extent of this effect was similar to that of low-level lead exposure."

Southern California Particle Center Researchers Have Warned EPA for Years Experts at the Southern California Particle Center (SCPC), led by respected USC researcher Dr. Constantinos Sioutas, have urged EPA to correct its methods for many years, to no avail. They have been especially critical of EPA's exclusive focus on particle <u>mass</u>, as opposed to particle <u>number</u>. SCPC wrote:

"Advanced vehicle emission control technologies are effective in reducing solid, non-labile PM emissions by means of filtration. However, recent investigations have shown substantial increases (by one order of magnitude and often more) of particle number emissions from retrofitted vehicles...We thus recommend that a better understanding of the linkages between PM size, chemistry and toxicity should be developed in order to adopt regulatory strategies that might be sufficiently protective to the public. Of particular note is the semi-volatile PM from vehicular exhaust because of its unique role in atmospheric processes leading to the formation of secondary aerosols...Given the increased toxicity of these semi-volatile species, efforts should be made to reduce their emissions from newer vehicles, including reductions in their gas-phase precursors formed during the combustion process."

"We also recommend that <u>current emission testing procedures should be modified to reflect real-world</u> <u>conditions in characterizing PM emissions</u> and evaluating their impacts of ambient exposure." [Emphasis supplied.]<sup>4</sup>

#### **Biogenic vs. Anthropogenic SOA Emissions**

EPA has long contended that trees and other biogenic sources are responsible for most of urban SOA emissions; we are familiar with the studies you cited in your letter. However, the literature contains numerous peer-reviewed studies that contradict EPA's view of biogenic source contributions to urban SOA inventories. In fact, a 2010 study by EPA's own Office of Research and Development (which your letter neglected to cite) concluded that carbonaceous material from mobile sources (e.g., gasoline aromatic hydrocarbons/BTEX emissions) substantially enhances biogenic SOA formation by facilitating the transformation of naturally-emitted VOCs to the particle phase. ORD estimated that more than 50 percent of biogenic SOA could be controlled by reducing anthropogenic SOA emissions caused by mobile sources, which are predominantly caused by gasoline-powered vehicles in the U.S.

In its study, ORD acknowledged that CMAQ 4.7 has a "persistent negative bias" (which as mentioned earlier EPA also admitted in its 2007 Fine Particulate rulemaking, promising "timely corrections" that have yet to occur), which "likely arises from a combination of errors and uncertainties, such as <u>inadequate representation of anthropogenic</u> <u>SOA, PCM (primary carbonaceous particulate matter)</u> emissions that do not account for intermediate volatility species (IVOC)...". According to a 2013 Harvard study and other experts—including, interestingly enough, HEI—the predominant urban source of these emissions is gasoline BTEX.

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Another 2015 study put the spotlight directly on gasoline aromatic hydrocarbons/BTEX:

"Aromatic hydrocarbons are important precursors of ground level ozone and play an important role in the formation of ozone and secondary organic aerosols (Carter, 1994; Liu et al., 2008a). Once the compounds are released into the atmosphere, most aromatic compounds are released into the air and react rapidly with OH and can participate in the formation of photochemical smog in the presence of nitrogen oxides. Hence, the abundance and speciation of aromatic hydrocarbons in the atmosphere significantly affects the ambient atmospheric chemistry (Monod et al., 2001). It has also been reported that O3 chemical production can be influenced by the concentrations of volatile organic compounds, and different species (e.g., aromatics) have different contributions to the ozone photochemical formation. Thus, identification of the emission sources of these species and their contributions to ozone formation are a crucial issue for the development of an effective O3 control strategy (Tang et al., 2007, 2008)."

#### CMAQ/CalNex Findings Have Been Contradicted by Numerous Studies

Your letter mentioned the CMAQ model and applications to CalNex field work which you say raised uncertainties about SOA sourcing and fuel reformulation benefits. However, a February 2009 study—which EPA funded—indicates that the science is more certain than your letter suggests. Over a multi-year period, five prestigious academic centers—Harvard School of Public Health; UCLA; USC; NY Univ. School of Medicine; and Univ. of Rochester Med Center)—warned about the "greater toxicity of traffic-related particles". They emphasized mobile sources/traffic as primary culprits, and called UFPs (ultrafine particles, which EPA has yet to regulate, and which HEI has singled out as particularly dangerous to humans) the most pathogenic, "proatherogenic fraction".

The experts warned that "...mobile sources are highly relevant to the public health impacts of PM.", and criticized EPA's regulatory philosophy that has fixated only on "mass": "The PM NAAQS are based on mass concentration...a more sophisticated approach to standards will be needed. Based on findings from the PM Centers and others, the potential efficacy of numbers and component based standards should be assessed. As more data becomes available to link specific PM emissions sources...with quantitative measures of toxicity, the questions of source-specific control strategies to maximize public health protection also need to be considered."

Implicit in these PM experts' recommendation is the need for improvements in gasoline composition, specifically reductions in gasoline BTEX content, as Congress directed in Section 202(I) of the 1990 Clean Air Act Amendments.

#### EPA's 2007 MSAT Rule Acknowledged Linkage of SOAs to Gasoline BTEX

In its 2007 MSAT final rule, EPA acknowledged that "there may be compelling reasons to consider aromatics control in the future, especially regarding reduction in secondary PM2.5 emissions, to the extent that evidence supports a role for aromatics in secondary PM2.5 formation". At the time, EPA cited "limits in the modeling tools that limit our present ability to quantitatively predict what would happen..." However, in its 2010 RFS2 Regulatory Impact Analysis (RIA), EPA acknowledged that "toluene is an important contributor to anthropogenic SOA", and that "mobile sources accounted for 70 percent of the total nationwide ambient concentration of toluene".

How the work of these reputable medical professionals, scientists, and academics has been ignored is difficult to reconcile. The linkage of these harmful particulates to gasoline is not the result of a fringe group or a discredited study but instead what is broad consensus. EPA seems to be the only ones that are "uncertain" or "confused". You should be reminded that Congress in effect made its own endangerment finding by directing EPA to address this issue ("shall") in the CAA of 1990. EPA needs to do its job, and embrace the obvious linkage between gasoline BTEX and urban SOAs. In so doing, by its own admission, the agency would be obliged to revisit Section 202(I) and its directive to reduce MSATs to the "greatest achievable extent".

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Notwithstanding EPA's professed "uncertainty", Congressional intent is clear: EPA's responsibility to substantially reduce air toxics caused by gasoline aromatic hydrocarbons has not been fulfilled, and more than 25 years later, action is long overdue.

Once again, Mr. Grundler, thank you for your response. We would like to find a way to get with you and your staff and resolve some of these issues, perhaps a meeting at a neutral site with our respective experts. As I hope you recognize by now, this is a very important issue to us and we intend to remain actively engaged in it.

Respectfully,

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Doug Sombke President, South Dakota Farmers Union

Cc: Senator Mike Rounds

<sup>1</sup>http://www.psr.org/environment-and-health/environmental-health-policy-institute/responses/particulate-pollution.html <sup>2</sup>http://www.atmos-chem-phys-discuss.net/12/31725/2012/acpd-12-31725-2012-print.pdf <sup>3</sup>http://www.niehs.nih.gov/about/assets/docs/testimonybirnbaumfeb042010\_508.pdf <sup>4</sup>http://aaqr.org/VOL10\_No1\_February2010/6\_AAQR-09-05-IR-0036\_43-58.pdf

# Grundler Letter (2) Response to Sombke Letter (2) – Page 1



Mr. Doug Sombke, President South Dakota Farmers Union 1410 Dakota Avenue South P.O. Box 1388 Huron South Dakota 57350

Dear Mr. Sombke:

Thank you for your letter of February 5, 2018, which continues our correspondence of December 19, 2017 and January 19, 2018. We share your interests in reducing particulate matter (PM) and polyaromatic hydrocarbons (PAHs), and in research on measurement and modeling that could help lead to effective control strategies.

With respect to your concern that the Environmental Protection Agency's (EPA's) models incorrectly predict the contribution of light-duty gasoline vehicles to PM (e.g., as compared to leaf-blowers), it is important to note that the EPA estimates cited in the *Wall Street Journal* refer only to directly emitted PM. We agree that ambient levels of PM are a result of secondarily formed particles in addition to direct PM emissions, and that light-duty gasoline vehicles are important sources of the precursors to PM formation.

With respect to Clean Air Act section 202(l), the EPA has acted twice under this specific authority, including the February 2007 rule that addresses the aromatics content of gasoline through required limits on benzene (72 FR 8428, February 26, 2007). In addition, the EPA promulgated additional "Tier 3" vehicle emission and fuel standards under sections 202(a) and 211(c), as referenced in section 202(l) (79 FR 23414, April 28, 2014). These standards took effect last year and will reduce emissions of air toxics from on-highway vehicles, as well as emissions of volatile organic compounds (VOC), nitrogen oxides (NOx), and fine particulate matter (PM<sub>2.5</sub>).

Since Congress established section 202(1) in the Clean Air Act Amendments of 1990, the net result of the EPA regulations and market shifts has been a reduction in gasoline benzene levels by roughly two thirds and aromatics levels by roughly one third (https://nepis.epa.gov/Exe/ZvPDF.cgi?Dockey=P100T5J6.pdf). In addition, the vehicle emissions standards have been reduced through three separate rounds of rulemaking (Tier 1, Tier 2, and Tier 3) such that the VOC emission rate of today's vehicles is more than 90% lower than in 1990. In combination, these fuel and vehicle standards have already dramatically reduced air toxics emissions. While the EPA continues to look for opportunities to further reduce air toxics, as required by Clean Air Act section 202(1), we must also consider technological feasibility and costs,

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among other factors. We take our regulatory authority very seriously and must ensure the appropriateness of taking further regulatory action before doing so.

Again, thank you for your letter. I appreciate the opportunity to be of service and trust the information provided is helpful.

Sincerely,

Christopher Grundler, Director Office of Transportation and Air Quality



January 15, 2019

Mr. Christopher Grundler USEPA Headquarters William Jefferson Clinton Building 1200 Pennsylvania Ave. NW MC6401A Washington, DC 20460

Dear Mr. Grundler:

I hope you and your family had a wonderful holiday season, and that you are ready for a busy and productive 2019. It has been some time since we corresponded, and I promised that you would be hearing from me again.

As you know, I sincerely appreciated your March 15th, 2018 response to my letter of February 5th, 2018. After several unsatisfactory responses from your staff, you answered my question when you wrote "...it is important to note that the EPA estimates cited in the Wall Street Journal refer only to directly emitted PM. We agree that ambient levels of PM are a result of secondarily formed particles in addition to direct PM emissions and that light-duty gasoline vehicles are important sources of the precursors to PM formation".

[You will recall that the WSJ article cited 2014 EPA estimates that PM2.5 (fine particulate) emissions from off-road sources such as leaf blowers (43,442 tons) are nearly as much as light-duty vehicles (59,301 tons). Relying on EPA's data, the author misled the public and policymakers by saying that: "Overall, EPA figures show that the small, non-road engines contribute...1% of particulate matter, the same amount as passenger cars."]

In addition to correcting this misleading data, your letter stated that "We share your interests in reducing particulate matter (PM) and polycyclic aromatic hydrocarbons (PAHs)." That is the focus of my letter to you today.

You also responded to my questions about why EPA/OTAQ has not done its job in enforcing the mandatory Section 202(I) MSAT provision in the Clean Air Act. You said: "With respect to Clean Air Act section 202(I), the EPA has acted twice under this specific authority, including the February 2007 rule that addresses the aromatics content of gasoline through required limits on benzene...". You went on to say "While the EPA continues to look for opportunities to further reduce air toxics, as required by Clean Air Act Section 202(I), we must also consider technological feasibility and costs."

As you know, when Congress enacted the Section 202(I) provision, it made very clear that the nonbenzene aromatic compounds (the "TEX" fraction) are not fully captured by vehicle emissions control systems. Everyone knows that the "TEX" fraction of gasoline BTEX is the primary source of tailpipe benzene emissions. In fact, HEI and other experts have confirmed that 50% or more of today's urban benzene inventories are attributable to the large aromatics fraction that exists in today's gasoline. Unfortunately, as your letter admits, EPA's 2007 MSAT Final Rule focused only on benzene, and it required a miniscule reduction in benzene levels (from 1.0% to 0.6%) and did nothing to address the rest of the aromatic compounds.

That is why we continue to insist that EPA/OTAQ has not "done its jobs" in terms of reducing these lethal emissions, especially in traffic-congested urban areas where most of the nation's people live and work.

EPA Used Obsolete and Wildly Inaccurate Predicates In its 2007 MSAT Rule Cost – Benefit Analysis. EPA justified its decision to restrict its controls only to the benzene molecule by conducting a cost – benefit analysis that relied upon extremely obsolete and, in many cases, just plain wrong factual predicates. These included the use of 1993 EIA projections of \$19 oil, \$.85 gasoline, and perhaps most egregious, the assumption that two gallons of ethanol "octane-equivalent" are required to displace one gallon of toluene's "octane equivalent". As you know, the exact opposite is true.

However, there may a silver lining here. It appears as though you and I agree on one thing: EPA has a continuing obligation to reduce gasoline BTEX levels and MSAT emissions, including SOA-bound PAHs, as technologies present themselves.

EPA's 2007 MSAT Rule Left the Door Open to Revisiting Section 202(I). EPA Must Revisit Aromatics Control If Evidence "Supports a Role for Secondary PM2.5 Formation." We believe that your admission that gasoline exhaust contains "important sources of the precursors to PM formation" is a very big deal. We also believe that it is very timely, since your Agency is in the final stages of considering comments to the SAFE Rule

We commend you and your Agency for opening the door to long overdue enforcement of Section 202(I) in the pending SAFE Rule. For the first time, EPA requested comment on "whether and how it should require higher octane gasoline to be sold, consistent with Title II of the Clean Air Act". As you know, Section 202(I) is part of Title II.

The National Farmers Union, and a number of its state chapters—including mine—signed on to the Clean Fuels Development Coalition (CFDC) SAFE Rule comments, submitted October 26, 2018. I would respectfully direct your attention to p. 10 of those comments, and these words: "Aromatics/BTEX is not only the primary source of the most dangerous urban air toxics, but also the dominant source of PM2.5 secondary organic aerosols (SOAs), which carry the toxics long distances, and are major contributors to ground level ozone. In its 2011 report to Congress, EPA projected that by 2020, 85% of the \$2 trillion in savings from the 1990 CAA will come from reductions in ambient PM, and that its models under predict the amount of the fine and ultra-fine particulates that are caused by gasoline aromatics/BTEX."

On the one hand, your letter appears to have conceded the point about the linkage of ambient PM/SOAs to gasoline aromatics. However, one of your statements is very concerning: "Since Congress established section 202(I) in the Clean Air Act Amendments of 1990, the net result of the EPA regulations and market shifts has been a reduction in gasoline benzene levels by roughly two thirds and aromatics levels by roughly one third...In addition, the vehicle emission standards have been reduced through three separate rounds of rulemaking...such that the VOC emission rate of today's vehicles is more than 90% lower than in 1990. In combination, these fuel and vehicle standards have already dramatically reduced air toxics emissions." (Emphasis supplied)

We strongly disagree with this assertion. In fact, we believe it is a major distortion of the facts. Best available science, and your Agency's own admissions, proves that EPA models are defective, and that they are substantially under predicting the most potent SOA-bound toxics. To make matters worse, best available science confirms that as vehicle advances such as direct injection engines come to dominate the U.S. light-duty fleet, the most dangerous emissions will get worse, not better.

EPA Has Admitted Its Models Are Defective and Fail to Predict SOA/PAH Emissions. The EPA 2015 UFP Workshop—organized by OTAQ and ORD—conceded that "SOAs play an important role in air quality", but that EPA's atmospheric models were "not able to predict SOA formation". [Link to workshop materials [https://sites.google.com/site/2015ufpworkshop/home] EPA's principal PM/SOA model is the Community Multi-Scale Air Quality (CMAQ) model, and the MOVES Model relies heavily on its methodology and findings. EPA has admitted that this model significantly under predicts SOA/fine particulates since its 2011 report to Congress on the Clean Air Act's costs and benefits.

At the 2015 UFP Workshop, EPA stated that the new science represented a breakthrough in the investigation of SOA properties, which "play an important role in air quality but for many years available atmospheric models were not able to predict SOA formation. The main issue was the fact that all models relied on the assumptions that SOA particles were well-mixed low viscosity solutions and maintained equilibrium with the gas-phase by rapid mixing in the condensed phase with evaporation and condensation. Recent studies using the multidimensional characterization approach demonstrated that these assumptions were wrong and that SOA particles must be viscous semi-solid. These studies showed also that there is a synergetic effect between PAHs and SOA since PAHs trapped inside SOA particles slow down SOA evaporation and increase SOA yield and lifetime. This can explain the long-range transport of toxic compounds like PAHs and other persistent pollutants. In conclusion, a new SOA paradigm has been developed: particles are semi-solid, nearly non-volatile and trap organic material during formation. The particles are not in equilibrium with the gas phase." [p. 19 - 20]

It would seem that the "missing SOAs" have been found, and that EPA's models were not reporting them. It is also clear that EPA's working assumption that PAHs dissipate quickly after 300 meters is inoperable—real-time measurements confirm SOA-bound PAHs and BC in urban plumes 30 km from their origination.

Gas-Phase PAHs Preserve Biogenic SOAs and Enable Their Long-Range Transport; Increase Particle Number Concentrations by > Factor of 100. The 2017 Zellenyuk/PNNL et al. study explains why realtime measurement studies have detected large concentrations of SOA-bound PAHs long distances from the point of emission, e.g., up to 30 km, and why they are dominated by gasoline exhaust products. This study (which relies upon the same PNNL multidimensional characterization approach that EPA endorsed at its UFP Workshop) confirms that "when secondary organic aerosol (SOA) particles are formed in the presence of gas-phase polycyclic aromatic hydrocarbons (PAHs), their formation and properties are significantly different from SOA particles formed without PAHs. This is extremely important, because the formation of PAHs and SOAs begins with the incomplete combustion of gasoline aromatics (which modern emission control systems have difficulty in capturing, see Robinson below). SOA particles formed in the presence of PAHs have, as part of their compositions, trapped unreacted PAHs and products of heterogeneous reactions between PAHs and ozone. Compared to 'pure' SOA particles, these particles exhibit slower evaporation kinetics, have higher fractions of non-volatile components, like oligomers, and higher viscosities, assuring their longer atmospheric lifetimes. In turn, the increased viscosity and decreased volatility provide a shield that protects PAHs from chemical degradation and evaporation, allowing for the long-range transport of these toxic pollutants. The magnitude of the effect

of PAHs on SOA formation is surprisingly large. The presence of PAHs during SOA formation increases mass loadings by factors of two to five, and particle number concentrations, in some cases, by more than a factor of 100. Increases in SOA mass, particle number concentrations, and lifetime have important implications to many atmospheric processes related to climate, weather, visibility, and human health, all of which relate to the interactions between biogenic SOA and anthropogenic PAHs". The literature also includes EPA studies (Carlton/Bhave et al.) that confirm this synergy between anthropogenic and biogenic SOA formation in urban areas.

Link to Zelenyuk et al.: [The effect of gas-phase polycyclic aromatic hydrocarbons ...https://pubs.rsc.org/en/content/articlelanding/2017/fd/c7fd00032d

When secondary organic aerosol (SOA) particles are formed by ozonolysis in the presence of gas-phase polycyclic aromatic hydrocarbons (PAHs), their formation and properties are significantly different from SOA particles formed without PAHs.]

Robinson/Carnegie Mellon Faults MOVES 2014 for Unspeciated SOA Under predictions. Dr. Allen Robinson/Carnegie Mellon has frequently criticized EPA's MOVES 2014 model for its gaps between speciated and total emissions (acknowledged by EPA itself in I. above). "I am concerned about the gap between speciated and total emissions. The standard approach (adopted here), assumes that the unspeciated portion of the NMOG behaves the same as the speciated. This likely is not the case when it comes to secondary organic aerosol (SOA) formation. The unspeciated emissions are likely a complex mixture of higher molecular weight species—these species contribute disproportionately to SOA formation relative to lighter species (e.g., propane)." [P. 65 of attached EPA document: "Speciation of Total Organic Gas and Particulate Matter Emissions from On-Road Vehicles in MOVES2014".]

Robinson's blunt criticism of EPA's MOVES Model is consistent with several Robinson et al. studies found in the literature. For example, see excerpts from the August 2014 Robinson – Maricq SOA study (which warns that newer vehicles are likely to produce even MORE SOAs than older vehicles, in part due to direct injection engines' tendency to increase PAH/BC emissions):

"Over the time scale of these experiments, the mixture of organic vapors emitted by newer vehicles appear to be more efficient (higher yielding) in producing SOA than the emissions from older vehicles. About 30% of the non-methane organic gas emissions from the new vehicles could not be speciated, and the majority of the SOA formed from these vehicles appears to be associated with these unspeciated organics." (Emphasis added)

23176: "Numerous reports have shown that the secondary fraction of fine organic PM (secondary organic aerosol, SOA) dominates primary organic aerosol (POA), even in urban areas with substantial fresh PO emissions...However, chemical transport models systematically under predict SOA levels...Motor vehicle emissions contribute to both POA and SOA concentrations."

"Unspeciated NMOG emissions are not typically included in models and inventories."

"Although tightening regulations have significantly reduced emissions of regulated primary pollutants (for example, Fig. 2 highlights the dramatic reductions in NMOG emissions...), the same may not be true for PM. In fact, Fig. 8 suggests that for LDGVs manufactured over the last twenty years...there may not have been much reduction in their contribution to ambient PM. This is not surprising...changes to engine control/after treatment from LEV1 to LEV2 were not aimed at reducing PM (or the non-volatile particles – EC)."

23197: "Catalysts are optimized to reduce emissions of regulated pollutants (NOx, NMOG, and CO), not SOA precursors."

23198: "...in the United States there are substantially more LDGVs than HDDVs."

23199: "...SOA production could not be fully explained by speciated (traditional) SOA precursors. However, about 30% of the NMOG emissions from LEV1 and LEV2 vehicles could not be speciated. These unspeciated emissions appear to be important SOA precursors, likely forming the majority of the SOA in experiments performed with LEV1 and LEV2 vehicles. Given the unexpected finding that the gasphase emissions from newer, LEV2 vehicles are more efficient at producing SOA than emissions from older, pre-LEV vehicles, future studies elucidating the nature of these precursors are needed to advance the development of next-generation SOA models and emission control strategies." (Emphasis added)

http://www.atmos-chem-phys-discuss.net/13/23173/2013/acpd-13-23173-2013-print.pdf

Mr. Grundler, for all these reasons, we strongly disagree with your statements that "...these fuel and vehicle standards have already dramatically reduced air toxics emissions", and that "the VOC emission rate of today's vehicles is more than 90% lower than in 1990". We are confident that EPA's models are simply not reporting enormous quantities of SOA-bound PAHs and BC, which are the most toxic, mutagenic endocrine disruptor compounds in the urban environment.

Even more worrisome: unless EPA does its job under section 202(I), these extremely harmful emissions will only get worse as automakers rapidly transition to direct injection engines.

At some point, we would like to visit with you more about these critically important issues. We also respectfully urge you to give serious consideration to the answer we gave to your SAFE Rule comments question: "We strongly believe that the 'ideal octane level' to optimize LDV performance fuel efficiency and reduce harmful emissions and consumer costs is 98 – 100 RON produced with E30+ "clean octane". In fact, we believe it is the only legally permissible way to make high octane fuels 'consistent with Title II of the Clean Air Act'".

Thank you once again, Mr. Grundler, for your consideration, accessibility, and careful attention to these extremely important issues. You will be hearing from us again soon.

Respectfully,

Day Somble

Doug Sombke President, South Dakota Farmers Union Cc: Senator Mike Rounds, Environment and Public Works Committee



March 11, 2019

Mr. Christopher Grundler USEPA Headquarters William Jefferson Clinton Building 1200 Pennsylvania Ave. NW MC6401A Washington, DC 20460

Dear Mr. Grundler:

I am writing to follow up my January 15, 2019 letter, which focused on the issue of gasoline PAH and toxics emissions, and your assertion that OTAQ actions have dramatically reduced air toxics emissions : "Since Congress established section 202(l) in the Clean Air Act Amendments of 1990, the net result of the EPA regulations and market shifts has been a reduction in gasoline benzene levels by roughly two thirds and aromatics levels by roughly one third...In addition, the vehicle emission standards have been reduced through three separate rounds of rulemaking...such that the VOC emission rate of today's vehicles is more than 90% lower than in 1990. In combination, these fuel and vehicle standards have already dramatically reduced air toxics emissions."

We have uncovered new information that shows your claims to be insupportable. In fact, we believe the opposite to be true, and that PAHs and other MSATs have in fact increased since enactment of the 1990 Clean Air Act Amendments. Of even greater concern, these potent emissions are expected to get worse as gasoline direct injection engines dominate the fleet unless OTAQ does something about truly controlling gasoline aromatics levels as Congress requires under Section 202(l).

In your letter to me of March 15, 2018, you said that "We share your interests in reducing particulate matter (PM) and polycyclic aromatic hydrocarbons (PAHs)."

When I asked why EPA/OTAQ has not done its job in enforcing the mandatory Section 202(l) MSAT provision in the Clean Air Act, you said: "With respect to Clean Air Act section 202(l), the EPA has acted twice under this specific authority, including the February 2007 rule that addresses the aromatics content of gasoline through required limits on benzene..."

Speaking to the future, you also stated "While the EPA continues to look for opportunities to further reduce air toxics, as required by Clean Air Act Section 202(l), we must also consider technological feasibility and costs." This is a bit ironic: if OTAQ actually did an honest job of considering technology, i.e. the influx of DI engines, and costs, i.e. the significantly lower costs



of ethanol in a cost-benefit comparison with expensive and highly toxic aromatics, we would be working together to encourage E30 "clean octane" blends, not exchanging these types of letters.

Unfortunately, disturbing public domain information, and OTAQ's own words, makes us even more convinced that OTAQ has fallen far short of complying with the letter and spirt of Section 202(l).

**EPA Has Admitted Its Models Are Defective and Fail to Predict SOA/PAH Emissions.** The EPA 2015 UFP Workshop—organized by OTAQ and ORD—conceded that "SOAs play an important role in air quality", but that EPA's atmospheric models were "not able to predict SOA formation".

EPA's principal PM/SOA model is the Community Multi-Scale Air Quality (CMAQ) model, and the MOVES Model relies heavily on its methodology and findings. Recently, a University of Wisconsin researcher said that OTAQ's models were "state of the art" and represented "best practice". We respectfully disagree.

In fact, OTAQ has finally admitted that its models significantly under-predict SOA/fine particulates. It has known that since EPA's 2011 report to Congress on the Clean Air Act's costs and benefits yet has done nothing to correct the problem.

At the 2015 UFP Workshop, EPA stated that the new science represented a breakthrough in the investigation of SOA properties, which "play an important role in air quality but for many years available atmospheric models were not able to predict SOA formation. The main issue was the fact that all models relied on the assumptions that SOA particles were well-mixed low viscosity solutions and maintained equilibrium with the gas-phase by rapid mixing in the condensed phase with evaporation and condensation. Recent studies using the multidimensional characterization approach demonstrated that these assumptions were wrong and that SOA particles must be viscous semi-solid. These studies showed also that there is a synergetic effect between PAHs and SOA since PAHs trapped inside SOA particles slow down SOA evaporation and increase SOA yield and lifetime. This can explain the long-range transport of toxic compounds like PAHs and other persistent pollutants. In conclusion, a new SOA paradigm has been developed: particles are semi-solid, nearly non-volatile and trap organic material during formation. The particles are not in equilibrium with the gas phase."<sup>1</sup>

Gas-Phase PAHs Preserve Biogenic SOAs and Enable Their Long-Range Transport; Increase Particle Number Concentrations by > Factor of 100. In my January 15, 2019 letter, which you have not responded to-- I referred you to the 2017 Zelenyuk/PNNL study—which OTAQ endorsed at its UFP Workshop—which confirmed that *"when secondary organic aerosol (SOA)* 

<sup>&</sup>lt;sup>1</sup> See Richard W. Baldauf et al., *Ultrafine Particle Metrics and Research Considerations: Review of the 2015 UFP Workshop*, Int. J. Environ. Res. Public Health 2016, 13, 1054; doi:10.3390/ijerph13111054, p. 19 – 20.



particles are formed in the presence of gas-phase polycyclic aromatic hydrocarbons (PAHs), their formation and properties are significantly different from SOA particles formed without PAHs...allowing for the long-range transport of these toxic pollutants. The magnitude of the effect of PAHs on SOA formation is surprisingly large. The presence of PAHs during SOA formation increases mass loadings by factors of two to five, and particle number concentrations, in some cases, by more than a factor of 100." The literature also includes EPA studies (Carlton/Bhave et al.) that confirm this synergy between anthropogenic and biogenic SOA formation in urban areas.

**The "Missing" Unspeciated SOA and PAHs**. My letter also cited Dr. Allen Robinson's (Carnegie Mellon) stated concerns about OTAQ's MOVES Model's failure to identify the "missing" SOA. In 2014, Robinson objected to OTAQ's inexplicable decision to exclude 86% of known PAHs from its MOVES Model, for which your agency provided the following explanation:

"As mentioned in the Toxics report (Section 2.1.2 Polycyclic Aromatic Hydrocarbons)4, the particulate matter (PM) emission rates are derived from emission test programs, but <u>the gas-particle partitioning is not adjusted to be representative of</u> <u>ambient conditions. We agree that differences between the dilution conditions of the</u> <u>emission test programs and ambient conditions introduce differences in the PM</u> <u>emissions. However, a comprehensive reevaluation of the PM emission rates was not</u> <u>within the scope of the updates for MOVES2014."</u>

Why should including all known PAHs be "outside the scope" of OTAQ's carcinogenicity potency evaluations?

It is frankly inconceivable how OTAQ can attempt to rationalize leaving 86% of known PAHs on the cutting floor, including some of the most potent gasoline PAHs (e.g., benzo(e)pyrene) and the entire inventory of the gas-phase PAHs which are predominant contributors to urban toxic emissions. There is no way OTAQ can claim that it is providing a full and accurate picture of gasoline carcinogenicity potency when it makes arbitrary and capricious decisions like this.

It is no wonder OTAQ claims 90% reduction in mobile source air toxics—you simply refuse to measure it!

OTAQ Contractor Warns of Substantial Understatements in Reporting of Gasoline Carcinogenicity Potency. It's not only Dr. Robinson and Farmers Union that senses something is terribly wrong within your shop—it is OTAQ's own contractors.



On page 41 of EPA's MOVES air toxic emissions report<sup>2</sup>, it referred to its contractor Desert Research Institute (DRI) which was responsible for analyzing the measurement samples from the Kansas City light duty vehicle emissions study on which the data was based.

In 2017, three years later, DRI published a report entitled "Do 16 polycyclic aromatic hydrocarbons (PAHs) represent PAH air toxicity?"<sup>3</sup> The report is a surprisingly blunt and scathing rebuttal of EPA's methodology and the MOVES Model's reliability.

DRI pointed out that estimation of carcinogenic potency based on analysis of 16 polycyclic aromatic hydrocarbons ranked by US Environmental Protection Agency is the most popular approach within scientific and environmental air quality management communities. [A good example of the scientific community's willingness to accept EPA's data as "gospel" can be found in a recent University of Wisconsin study on PAH toxicity, in which the authors endorsed EPA's modeling: "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."]

In other words, EPA's MOVES model is relied upon by researchers, policymakers, and the media. As a result, most PAH monitoring projects have been focused only on particle bound PAHs, ignoring the contribution of the much more important, ubiquitous, and potent gas phase PAHs to the toxicity of PAH mixtures in air samples.

DRI wrote: "The aim was to investigate whether 16 particle-bound U.S. EPA priority PAHs adequately represented health risks of inhalation exposure to atmospheric PAH mixtures. PAH concentrations were converted to benzo(a)pyrene-equivalent (BaPeq) toxicity using the toxic equivalency factor (TEF) approach. TEFs of PAH compounds for which such data is not available were estimated using TEFs of close isomers. Total BaPeq toxicities ( $\sum$ ssBaPeq) of gas- and particle-phase PAHs were compared with BaPeq toxicities calculated for the 16 particle-phase EPA PAH ( $\sum$ \_{16EPA}BaPeq). The results showed that 16 EPA particle-bound PAHs underrepresented the carcinogenic potency on average by 85.6% relative to the total (gas and particle) BaPeq toxicity of 88 PAHs. Gas-phase PAHs, like methylnaphthalenes, may contribute up to 30% of  $\sum$ ssBaPeq."

DRI concludes: *"Accounting for other individual non-EPA PAHs (i.e., benzo(e)pyrene) and gas-phase PAHs (i.e., naphthalene, 1- and 2-methylnaphthalene) will make the risk assessment of PAH-containing air samples significantly more accurate.* 

<sup>&</sup>lt;sup>2</sup> Air Toxic Emissions from On-road Vehicles in MOVES2014, Assessment and Standards Division, Office of Transportation and Air Quality, U.S. EPA, EPA-420-R-14-021, December 2014

<sup>&</sup>lt;sup>3</sup> See Vera Samburova et al., *Do 16 Polycyclic Aromatic Hydrocarbons Represent PAH Air Toxicity*?, Toxics 2017, 5(3), 17, doi.org/10.3390/toxics5030017



It is clear that the "PM-only analysis" approach could significantly underestimate the carcinogenic potency of PAH mixtures. Similar conclusions have been presented by Ramirez et al. [60]. In their work, 18 gas and particle-phase PAHs have been analyzed in air samples collected near industrial cities, and the contribution of gas-phase PAHs to the total BaPeq was found to be significant (34–86%)."

This DRI critique is consistent with EPA's own confession at its 2015 UFP workshop where it admitted that its atmospheric models do not properly capture gas phase partitioning and the full range of SOA PAH mixtures. In fact, EPA has admitted that PAHs when they are present during the formation of SOAs (which is all the time in the case of the incomplete combustion of gasoline aromatics) have a powerful synergistic effect on particle number amplification, preservation, and transport.

Scientists now know that the gas-phase PAHs that OTAQ methodically excluded from its MOVES model are responsible for "weaponizing" urban SOA and their related toxics. In my humble opinion, to exclude them from your MOVES Model is nothing short of professional malpractice. It most certainly makes the MOVES Model unusable and unworthy of serious consideration for purposes of protecting the public health and welfare.

There should be no mistake: <u>the benzo(e)pyrene and gas-phase PAHs that OTAQ deceptively</u> (some experts suggest illegally) excluded from its MOVES Model are primarily combustion products of gasoline aromatics. This sleight of hand helps to explain how/why EPA/OTAQ have insisted that gasoline exhaust is only a minor contributor to urban SOA/PM and that mobile source air toxics emissions have been dramatically reduced in recent years.

**Stakes are Enormous Especially for our Children.** As you know, you have an enormous responsibility. OTAQ has a duty to protect the public from the most potent mobile source air toxics that Americans cannot escape like they can with secondhand smoke. Infants and children are particularly vulnerable, as Dr. Frederica Perera and her colleagues reminded us in a December 2018 meta-analysis of 205 peer-reviewed studies of children's adverse health end points caused by PM and PAHs.<sup>4</sup>

**Perera** reviewed thousands of studies from 2000 – 2015 that considered fossil fuel combustion byproducts including particulate matter (PM), polycyclic aromatic hydrocarbons (PAH) on adverse children's health endpoints. They warn that only a few adverse outcomes in children have been considered resulting in an undercounting of the benefits to this vulnerable

<sup>&</sup>lt;sup>4</sup> F. Perera et al., *Towards a fuller assessment of benefits to children's health of reducing air pollution and mitigating climate change due to fossil fuel combustion*, Environmental Research 172 (2019) 55-72, doi.org/10.1016/j.envres.2018.12.016



population. The authors conducted a systematic review of the literature published between January 1, 2000 and April 30, 2018 to identify relevant peer reviewed case control and cohort studies and meta-analyses. The authors reviewed 205 peer reviewed studies and posted results for each pollutant and health outcome. The health endpoints that were analyzed included preterm birth (PTB), low birth weight (LBW), autism, attention deficit hyperactivity disorder (ADHD), IQ reduction, and the development of childhood asthma.

This review presents compelling evidence that exposures to PM and PAH adversely affect birth outcomes and neural behavioral development and contribute to risk of asthma development in childhood. Fossil fuel combustion byproducts including toxic air pollutants such as PM and PAH are a significant threat to children's health and future well-being. The developing fetus and young child are disproportionately affected by outdoor air pollution because of their biological vulnerability and rapid development.

Perera et al. cite PAH as a toxicologically important component of PM. Significant correlations have been reported between PAH and PM. On page 65 the authors note that "multiple cognitive and behavioral effects including ADHD have been observed in association with PAH exposure. Given the significant correlation between PM and PAH and the importance of PAH in their own right this body of evidence indicates substantial benefits for early brain development from lowered exposure to fossil fuel combustion byproducts."

As you know, there is no way to separate PM, SOA, PAHs, and UFPs. They are all part of a noxious toxic "stew" that permeates the urban environment and their predominant urban source are gasoline aromatic compounds. Your manipulated models hide that fact, but real-time measurement studies prove it.

PM and PAH effects are particularly linked with adverse health outcomes in PTB, neural developmental disorders, and childhood asthma. The observed loss in IQ with PAH is in the range seen with low level lead exposure. In addition, the literature review found significant associations between prenatal PAH exposure and attention problems and ADHD. A recent review concluded that PAH exposure is associated with onset of childhood asthma. There is evidence that the PAH component of PM may contribute to asthma development in concert with ETS exposure or childhood obesity.

Bottom line: America's children suffer from unnecessary mortalities and morbidities caused by gasoline aromatics that Congress has charged OTAQ with reducing to the greatest achievable degree.

Regrettably, the authors note that compared to PM, PAH have been less-studied and no metaanalyses have been possible. OTAQ must accept responsibility for this gap in the health science.



However, the authors point to an extensive body of literature, especially that done by Perera and Columbia University that has over the years established linkages between PAH and childhood adverse health outcomes. This and other work helped to inform this analysis of 205 peer-reviewed studies.

EPA/OTAQ can no longer assert that "there is no science", or that "uncertainties" prevent you from acting. OTAQ has had 30 years since the 1990 CAAA debates—that should be enough time for you to do your jobs. Furthermore, Congress made a "legislative finding of harm" in Section 202(l) of the Clean Air Act—which means you are legally obligated to act.

Mr. Grundler, I farm for a living. I am a grandfather, and I care deeply about my family and my country. From where I sit, the facts really are quite simple: OTAQ has admitted that its models are broken. OTAQ's contractors have criticized the MOVES Model toxics methodology for major shortcomings, and warned it is likely "significantly underestimating the carcinogenic potency of PAH mixtures". SOA bound toxics, especially associated PAHs from incomplete combustion of gasoline aromatics, are the predominant source of the most dangerous pollutants in urban environments. They pose great danger to the fetus, infants, and children, as well as other vulnerable populations.

For all these reasons, we strongly dispute your statements that "...these fuel and vehicle standards have already dramatically reduced air toxics emissions", and that "the VOC emission rate of today's vehicles is more than 90% lower than in 1990". We believe your models have been manipulated and are seriously deficient, not worth the paper they are written on.

Even worse: unless OTAQ does its job under section 202(l), these extremely harmful emissions will only get worse as automakers rapidly transition to direct injection engines.

As OTAQ Director, you have a very important job. You and most of your colleagues have been in your positions for 30 or more years. It is time for you to do the right thing and do what Congress has demanded: clean up our gasoline and protect our kids.

Day Somble

Doug Sombke President, South Dakota Farmers Union

Cc: Senator Mike Rounds, Environment and Public Works Committee