**Comments Submitted by the Energy Future Coalition and the Urban Air Initiative**

**Appendix II. Displacing aromatics now used for octane in gasoline would produce substantial benefits for public health.**

1. **Gasoline aromatic compounds are a primary cause of the most dangerous urban pollutants**

Aromatic compounds constitute 20-30% of U.S. gasoline, which means that more than 40 billion gallons are combusted each year in U.S. light-duty vehicles. Their physico-chemical properties make them very difficult to combust efficiently. The higher distillation aromatics (high molecular weight, HMW), have higher double-bond equivalents (DBEs), and are particularly important contributors to urban ultrafine particles (UFPs) and polycyclic aromatic hydrocarbon (PAH) emissions. In the 1990 Clean Air Act Amendments, Congress instructed the EPA to achieve the “greatest reduction possible” in gasoline aromatics and the lethal air toxics they produce.

EPA has called ambient particulate matter (PM) one of the nation’s greatest health threats, but it regulates only particle mass (vs. particle number), in the form of PM2.5 (2.5 to 0.1 micrometers in aerodynamic diameter). Unfortunately, the much smaller UFPs (.1 micrometer, or 100 nanometers and smaller) are the most toxic, most bio-available, and the most effective carriers of the carcinogenic and mutagenic PAHs to the human body. <http://www.particleandfibretoxicology.com/content/6/1/24/ref>

PAHs are semi-volatile organic compounds (SVOCs) found in both gaseous and particle form. They comprise the largest mass fraction of UFPs, the primary urban source of which is also gasoline aromatics. Gasoline PAHs are carcinogenic, mutagenic, and genotoxic. <http://aaqr.org/VOL10_No1_February2010/6_AAQR-09-05-IR-0036_43-58.pdf>

UFPs have orders of magnitude higher number counts, and much larger surface mass with which to attract and carry the PAHs. For example, PM of 2.0 micrometer per cubic meter (2µ/m3) would have 2 particles per ml of air, and a surface area of 30µ/m2 per ml of air. In contrast, a UFP of 0.02 µ/m3 (20 nanometers, or 20 one-billionth of a meter) would have 2,390,000 particles in each ml of air, and a surface area of 3,000 µ/m2 per ml of air. See slide 4 on link below. <https://www.aqmd.gov/tao/ConferencesWorkshops/AircraftForum/FroinesSlides.pdf>

Particle-borne PAHs can persist for days in the environment, and can carry long distances after their emission from the tailpipe. SOAs and UFPs insulate and preserve PAHs, which are able to penetrate indoors, and have been found 1.5 miles from congested roadways. <http://www.ph.ucla..edu/pr/newsitem061009.html>

Gasoline PAHs are high molecular weight (4 – 6 rings), as opposed to diesel PAHs, which are low molecular weight. HMW PAHs are more toxic, and more persistent than LMW PAHs. In the U.S., approximately 250 million light duty vehicles consume more than 130 billion gallons of gasoline each year, and have historically accounted for more than 90% of transportation sector emissions. Thus, contrary to conventional wisdom, since gasoline PAHs are more abundant and ubiquitous, much smaller than diesel PAHs (extremely difficult and costly to trap), and more toxic, gasoline exhaust poses a much greater health threat to humans than does diesel exhaust. For example, a 2012 University of Colorado – Boulder study found that 80+% of PM2.5 secondary organic aerosols in Los Angeles originated from gasoline, as opposed to diesel, exhaust. <http://www.colorado.edu/news/releases/2012/03/02/gasoline-worse-diesel-when-itcomes->

PAHs are considered to be one of the most ubiquitous endocrine disruptor compounds (EDCs) in urban environments. EDCs mimic natural hormones in the body, and experts warn that they are especially damaging to the fetus and young children, and can disrupt genetic structures, causing serious damage that transfers throughout generations. PAHs have been linked to a wide range of disorders, including cancers, heart disease, asthma and other respiratory disorders, premature births, autism, and obesity. [http://ehp03.niehs.nih.gov/article/info%3Adoi%2F10.1289%2Fehp.1104056](http://ehp03.niehs.nih.gov/article/info%3Adoi/10.1289/ehp.1104056)

The California Office of Environmental Health Hazard Assessment has placed PAHs in Tier 1 on its toxic air contaminants list, in part due to the fact prenatal exposure to PAHs results in “serious and irreversible effects in the fetus”. <http://www.oehha.ca.gov/public_info/pdf/GasOEHHA.pdf>

Water quality regulators are reporting increasing deposition of PAHs in the nation’s waterways, as gasoline exhaust is washed from roadways into rivers, lake, and estuaries. The PAHs are then ingested by fish and other seafood, and can then enter the human food chain. <http://calcium25.com/PAHs-Water-Air-Pollution-0707.pdf><http://www.greencarcongress.com/2005/08/toxic_metals_de.html>

1. **Gasoline aromatic compounds are the predominant precursors to urban secondary organic aerosols (SOAs)**

The Harvard Center for Risk Analysis found that up to $50 billion per year in social costs are attributable to gasoline aromatics. The Harvard study considered only premature mortalities (as opposed to morbidity) caused by PM2.5 secondary organic aerosols (SOAs). In other words, Harvard did not attempt to quantify the even greater health costs associated with particle-borne PAHs, including the increasing evidence of the damage they do to infants and developing children. <http://www.ehjournal.net/content/12/1/19/abstract>

* Excerpts:

"Modeled aromatic SOA concentrations from CMAQ fall short of ambient measurements by approximately a factor of two nationwide...Assuming that the contribution of SOA precursors originating from aromatic hydrocarbons in gasoline is higher in urban areas increases these estimates to 5100 predicted premature mortalities nationwide...associated with total social costs of $37.9B".

"...particulates from vehicular emissions of aromatic hydrocarbons demonstrate a sizeable public health burden. The results provide a baseline from which to evaluate potential public health impacts of changes in gasoline composition."

"Evidence is growing that aromatics in gasoline exhaust are among the most efficient secondary organic matter precursors. In general, air quality models do not adequately capture these increased yields or potential interactions, although improvements have been made."

"In the United States, gasoline-powered vehicles are the largest source of aromatic hydrocarbons to the atmosphere...Therefore, it has been suggested that removal of aromatics could reduce SOA concentrations and yield a substantial public health benefit...a number of studies have noted that gas-phase vehicle emissions lead to a substantial fraction of observed SOA. For example, a source apportionment study of SOA formation during a severe photochemical smog event in Los Angeles found that gasoline engines represented the single-largest anthropogenic source of SOA."

"Although CMAQv5.0 contains updated...processes for predicting SOA formation, evidence suggests that the model may still underestimate secondary PM2.5 concentrations."

"Source-specific speciation reveals that the U.S. emissions of aromatic hydrocarbons are 3.6 million tons per year, of which 69% are from gasoline-powered vehicles as shown in Table 3."

"In addition to premature mortality, which dominates monetized estimates of total social cost, exposures to SOA from aromatics in gasoline are associated with other health outcomes, including exacerbation of asthma, upper respiratory symptoms, lost work days, and hospital emergency room visits."

"A recent study in Los Angeles found that gasoline emissions dominated SOA formation, accounting for nearly 90% of total aerosol formation, and the ratio of SOA to primary organic aerosol was approximately a factor of three...Anthropogenic SOA have been shown to enhance biogenic SOA formation."

1. **SOAs and PAHs synergistically bind together, enabling long-range transport, increased aging, and greater persistence/penetration indoors**

Zelenyuk et al. contend that conventional predictive models of SOAs and PAHs transport and persistence are fundamentally flawed. In its Tier 3 rule, EPA said that PAHs have a half-life of less than an hour, and that they dissipate within 300 feet of emission source. Here, DOE’s PNNL confirms that the PAHs undergo LRT and persist for weeks or longer due to their insulation from atmospheric evaporation by the SOAs. This LRT and persistence has enormous implications for the much greater magnitude of the human health threat predicted by EPA assumptions vs. reality. <http://www.ncbi.nlm.nih.gov/pubmed/23098132>

"[B]ased on current understanding of gas-particle partitioning and atmospheric degradation of PAHs some species, like benzo[α]pyrene and fluoranthene, should not undergo LRT at all yet are found in the Arctic at concentrations similar to those in Europe. In general, existing gas-particle partitioning models severely underpredict observed LRT of particle-bound PAHs, highlighting large knowledge gaps in kinetic partitioning models."

An article describing the PNNL work noted: “The results also show that the particles that envelop pollutants also benefit from this arrangement. The new study shows that the airborne particles last longer with PAHs packed inside.” <http://www.greencarcongress.com/2012/11/pah-20121117.html#more>

As the study put it: "Perhaps the most surprising finding is the observed synergetic relationship between PAHs and SOA. The presence of even a small amount of hydrophobic organics inside SOA significantly decreases the SOA evaporation rate and ampliﬁes the effect of aging, thus creating conditions that ensure efficient LRT of both SOA particles and PAHs, consistent with observations. This synergy between PAHs and SOA particles has important implications not only for human health but also for climate change.”

Using advanced instrumentation, PNNL scientists found that the potent PAHs are trapped within the semisolid SOAs (secondary organic aerosols) during particle formation and thus shielded from oxidation and preserved for extended periods of time.  This is why CARB and others are now reporting UFP-borne PAHs as far away as 2,500 meters (not 300 feet, as EPA contended in its Tier 3 rule) from their source.

This PNNL research is very important.  It helps explain some of the confusion amongst experts about the differences between concentrations of PAHs in gas-phase partitioning (which can be orders of magnitude lower) compared to the much higher concentrations of particle-bound PAHs.  The PNNL scientists proved that the SOAs synergistically bond with the PAHs and serve as the “insulation and preservative” for the PAHs that enables their perpetuation and LRT (and vice versa).

1. **Gasoline vehicles are the principal source of SOAs and toxics from aromatics**
* Excerpts from Bahreini et al. (2012), “Gasoline emissions dominate over diesel in formation of secondary organic aerosol mass”, Geophysical Research Letters, Vol. 39: <http://onlinelibrary.wiley.com/doi/10.1029/2011GL050718/abstract>

Air-borne and ground-based measurements of OA in Los Angeles Basin indicated that “the contribution from diesel emissions to SOA formation is zero within our certainties. Therefore, substantial reductions of SOA mass on local to global scales will be achieved by reducing gasoline vehicle emissions.”

“Consistent with previous studies, this indicates that gasoline vehicles are the dominant source of CO and light, single-ring aromatic VOCs including benzene and toluene.”

“Because diesel emissions contribute to POA, but not detectably to SOA, as photochemical processing and SOA formation proceeds, the contribution of diesel emissions to total OA decreases.”

“…for more accurate modeling of SOA formation in urban areas, future research should be directed at identifying specific species in the exhaust of gasoline engines that are responsible for SOA formation.”

“Assuming that production of SOA relative to POA from gasoline exhaust follows the same trend as in LA…we estimate that within a day of processing, SOA from gasoline exhaust may reach 4 Tg/yr, which is 16% of recent global estimates of biogenic SOA. Our observations suggest that a decrease in the emission of organic species from gasoline engines may significant reduce SOA concentrations on local and global scales.”

* Excerpts from Nordin et al. (2013), “Secondary organic aerosol formation from idling gasoline passenger vehicle emissions investigated in a smog chamber,” Atmos. Chem. Phys., 13, 1601-6116: <http://www.atmos-chem-phys.net/13/6101/2013/acp-13-6101-2013.html>

“Gasoline vehicles have recently been pointed out as potentially the main source of anthropogenic secondary organic aerosol (SOA) in megacities.”

“Gasoline exhaust readily forms oxidized organic aerosols that commonly dominates the organic aerosol mass spectra downwind of urban areas. … Classical C6 – C9 light aromatic precursors were responsible for up to 60% of the formed SOA, which is significantly higher than for diesel exhaust. Important candidates for additional precursors are higher-order aromatic compounds such as C10 and C11 light aromatics, naphthalene and methyl-napththalenes. We conclude that approaches using only light aromatic precursors given an incomplete picture of the magnitude of SOA formation and the SOA composition from gasoline exhaust.”

“Photo-oxidation of gasoline exhaust forms SOA and ammonium nitrate. At the end of the experiments the formed SOA is 9-500 times higher than the emitted POA, which is in sharp contrast to diesel exhaust where the contribution of primary PM often dominates over secondary PM.”

“The benzene concentration is strongly elevated in these idling experiments compared to the fuel content (benzene is regulated to less than 1% by volume in gasoline in Europe), most likely due to formation of benzene from other light aromatic compounds in the catalyst. … The enrichment of benzene in the exhaust is also found in road tunnel emission measurements.”

 “Since gasoline exhaust SOA is a more complex mixture than SOA from pure precursors, it can be expected that gasoline SOA resembles atmospheric observations better than SOA from pure precursors.”

“This implies that relatively low concentrations of PAHs can give a significant contribution to SOA formation.”

“As shown in this study, 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 spectra 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.”

* Excerpts from Delfino et al., "Association of biomarkers of systemic inflammation with organic components and source tracers in quasi-ultrafine particles," 2010. Environ Health Perspect 118:756–762: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2898850/>

“Indoor and outdoor PAHs (low-, medium-, and high-molecular-weight PAHs), followed by hopanes (vehicle emissions tracer), were positively associated with biomarkers, but other organic components and transition metals were not. Vehicular emission sources estimated from chemical mass balance models were strongly correlated with PAHs (R = 0.71)”.

“Traffic emission sources of organic chemicals represented by PAHs are associated with increased systemic inflammation and explain associations with quasi-ultrafine particle mass.”

“To our knowledge, this is the first report from a panel cohort study to show associations of circulating biomarkers of response in human subjects to specific PM organic compound classes. The measured chemicals serve as indicators and tracers for air pollutant sources and for classes of chemicals with the potential for redox activity in the body. In the present analysis, we found the strongest biomarker associations with air pollutant variables for all molecular weight classes of PAHs and specific source markers of vehicular emissions (hopanes) measured in PM0.25 with GC/MS. Furthermore, two-pollutant models of the relation between the biomarkers of systemic inflammation and both total PAHs and PM0.25 mass showed that mass associations were completely explained by PAHs.”

“In the Los Angeles Basin, most outdoor PAHs in PM0.25 are expected to be from mobile sources ([Schauer et al](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2898850/%22%20%5Cl%20%22b37-ehp-118-756). 1996), and the CMB exposure correlations are consistent with this expectation. PAHs were also correlated with source markers of vehicular emissions (hopanes). Hopanes are the most unambiguous source marker of traffic emissions.”

“Overall, the associations of biomarkers with PAHs and hopanes suggest that our previous findings of positive associations of biomarkers with PM2.5, EC, and primary OC ([Delfino et al](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2898850/%22%20%5Cl%20%22b9-ehp-118-756). 2009) were due to PM of mobile-source origin. PAHs are found in greater concentrations in the quasi-UFP range compared with larger particles ([Ntziachristos et al](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2898850/%22%20%5Cl%20%22b26-ehp-118-756). 2007), and this has been hypothesized to explain enhanced prooxidative and proinflammatory effects of urban UFPs in the lungs and peripheral target organs of rodents ([Araujo et al](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2898850/#b2-ehp-118-756). 2008). The increased biological potency of UFPs may be related to the content of organic chemicals that have the capacity to reduce oxygen, such as quinones and nitro-PAHs, for which PAHs may act, in part, as a surrogate ([Ntziachristos et al](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2898850/%22%20%5Cl%20%22b26-ehp-118-756). 2007) or as a source after biotransformation. From the present results we infer that, although PAHs may have an effect by themselves, they are also likely surrogates for other causal species we did not measure that are emitted from the same (traffic) sources.”

“Finding positive associations of biomarkers with both indoor and outdoor PAHs and hopanes along with the indoor/outdoor ratios of these organic components being close to 1.0 suggests that, even though people spend most of their time indoors, indoor air quality and PM exposures are strongly influenced by PM of outdoor origin. These findings are consistent with our previous analysis for the first half of this panel showing that CMB-estimated indoor PM of outdoor origin (particle number, EC, and primary OC) were associated with the biomarkers to a similar degree as outdoor PM ([Delfino et al](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2898850/%22%20%5Cl%20%22b10-ehp-118-756). 2008).”

1. **Heavy molecular weight (HMW) PAHs from gasoline are far more potent than LMW PAHs from diesel**
* California Office of Environmental Health Hazards Assessment report: Gasoline PAHs are heavy molecular weight (HMW), bear many similarities to cigarette smoke PAHs, and are ubiquitous in urban environments, especially adjacent to roadways. <http://www.oehha.ca.gov/public_info/pdf/GasOEHHA.pdf>.
* Prioritization of Toxic Air Contaminants – Children’s Environmental Health Protection Act”, October 2001: <http://www.oehha.ca.gov/air/toxic_contaminants/pdf_zip/PAHs_Final.pdf>

*“*Prenatal exposure to PAHs results in serious or irreversible effects in the fetus…For instance, PAHs are transplacental carcinogens…There is greater exposure of children to environmental PAHs compared to adults…Biomarkers for direct impacts associated with adverse health outcomes, such as DNA adducts, are increased in children exposed to environmental pollution by PAHs and related POM components. In view of this range of evidence for differential sensitivity of the fetus, infants, and children to health effects induced by POM components such as PAHs, and for greater exposure of children to POM, OEHHA has placed POM in Tier 1 of the priority list.”

* Excerpt from Riddle et al., “Large PAHs detected in fine particulate matter emitted from light-duty gasoline vehicles,” [Atmospheric Environment](http://www.sciencedirect.com/science/journal/13522310), [Volume 41, Issue 38](http://www.sciencedirect.com/science/journal/13522310/41/38), December 2007, Pages 8658–8668: <http://www.sciencedirect.com/science/article/pii/S1352231007006553>

“Emission factors of large PAHs with 6–8 aromatic rings with molecular weights (MW) of 300–374 were measured from 16 light-duty gasoline-powered vehicles (LDGV) and one heavy-duty diesel-powered vehicle (HDDV) operated under realistic driving conditions. LDGVs emitted PAH isomers of MW 302, 326, 350, and 374, while the HDDV did not emit these compounds. This suggests that large PAHs may be useful tracers for the source apportionment of gasoline-powered motor vehicle exhaust in the atmosphere. Large PAHs made up 24% of the total LEV PAH emissions and 39% of the TWC PAH emissions released from gasoline-powered motor vehicles. Recent studies have shown certain large PAH isomers have greater toxicity than benzo[a]pyrene. Even though the specific toxicity measurements on PAHs with MW >302 have yet to be performed, the detection of significant amounts of MW 326 and 350 PAHs in motor vehicle exhaust in the current study suggests that these compounds may pose a significant public health risk.”

* MW 302+ PAHs are especially toxic, see slides 16 and 17 on Relative Potency Factors, 2012 Simonich/OSU deck. <http://www.niehs.nih.gov/research/supported/assets/docs/r_s/what_goes_around_comes_around_chasing_polycyclic_aromatic_hydrocarbons_from_the_beijing_olympics_to_the_us_west_coast.pdf>
* Abstract of a study by Y. Jia, “Estimated Reduction in Cancer Risk due to PAH Exposures If Source Control Measures during the 2008 Beijing Olympics Were Sustained,” *Environ Health Perspect*. 2011 Jun; 119(6): 815–820: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3114816/>

“The 2008 Beijing Olympic Games provided a unique case study to investigate the effect of source control measures on the reduction in air pollution, and associated inhalation cancer risk, in a Chinese megacity.

“We measured 17 carcinogenic polycyclic aromatic hydrocarbons (PAHs) and estimated the lifetime excess inhalation cancer risk during different periods of the Beijing Olympic Games, to assess the effectiveness of source control measures in reducing PAH-induced inhalation cancer risks.

“We estimated the number of lifetime excess cancer cases due to exposure to the 17 carcinogenic PAHs [12 priority pollutant PAHs and five high-molecular-weight (302 Da) PAHs (MW 302 PAHs)] to range from 6.5 to 518 per million people for the source control period concentrations and from 12.2 to 964 per million people for the nonsource control period concentrations. This would correspond to a 46% reduction in estimated inhalation cancer risk due to source control measures, if these measures were sustained over time. Benzo[b]fluoranthene, dibenz[a,h]anthracene, benzo[a]pyrene, and dibenzo[a,l]pyrene were the most carcinogenic PAH species evaluated. Total excess inhalation cancer risk would be underestimated by 23% if we did not include the five MW 302 PAHs in the risk calculation.

“Source control measures, such as those imposed during the 2008 Beijing Olympics, can significantly reduce the inhalation cancer risk associated with PAH exposure in Chinese megacities similar to Beijing. MW 302 PAHs are a significant contributor to the estimated overall inhalation cancer risk.”

1. **Particle filters are not an adequate solution**

Advanced GDI (gasoline direct injection) systems could make particle number (PN) emissions worse unless fuel composition is improved by reducing aromatic content. Mid-level ethanol blends have been shown to reduce particulate and black carbon emissions by 45 to 80% in direct injection and port fuel injection engines, respectively. Some have argued for the use of particulate filters on gasoline engines; however, the much smaller particles in gasoline exhaust (compared to diesel exhaust) elude capture by such filters, which also will interfere with, possibly even reverse, important fuel efficiency and carbon reduction gains.