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Environmentally Persistent Free Radicals and Their Lifetimes in PM_{2.5}

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Abstract

For the first time, an expansive study into the concentration and extended decay behavior of environmentally persistent free radicals in PM_{2.5} was performed. Results from this study revealed three types of radical decay—a fast decay, slow decay, and no decay—following one of four decay patterns: a relatively fast decay exhibiting a 1/e lifetime of 1–21 days accompanied by a slow decay with a 1/e lifetime of 21–5028 days (47% of samples); a single slow decay including a 1/e lifetime of 4–2083 days (24% of samples); no decay (18% of samples); and a relatively fast decay displaying an average 1/e lifetime of 0.25–21 days followed by no decay (11% of samples). Phenol correlated well with the initial radical concentration and fast decay rate. Other correlations for common atmospheric pollutants (ozone, NO_x, SO₂, etc.) as well as meteorological conditions suggested photochemical processes impact the initial radical concentration and fast decay rate. The radical signal in PM_{2.5} was remarkably similar to semiquinones in cigarette smoke. Accordingly, radicals inhaled from PM_{2.5} were related to the radicals inhaled from smoking cigarettes, expressed as the number of equivalent cigarettes smoked. This calculated to 0.4–0.9 cigarettes per day for nonextreme air quality in the United States.

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Supporting Information

Additional details as noted in text. This material is available free of charge via the Internet at <http://pubs.acs.org>.

INTRODUCTION

Multiple studies established increases in ambient PM_{2.5} levels promote cardiopulmonary dysfunction and decrease life expectancy.^{1,2} These adverse health effects are induced by oxidative stress triggered when the cell is overwhelmed by reactive oxygen species, ROS.³ However, the exact nature of ROS formation and its source is still debatable. PM_{2.5} is documented to activate a toxic response from ROS generation whether from wood smoke, other biomass burning, or ambient PM_{2.5}.⁴⁻⁷ Recent evidence demonstrates ROS is also generated from the redox cycling of environmentally persistent free radicals (EPFRs) from a model system in addition to EPFRs in PM_{2.5}.⁸⁻¹⁰

EPFRs were identified initially on the surfaces of particles containing redox-active transition metals in the post flame and cool-zone regions of combustion systems.^{4,11,12} In addition to combustion systems, EPFRs were established in ambient PM_{2.5}.^{4,13-15} Formation of an EPFR occurs when an organic precursor chemisorbs onto a redox metal site (e.g., CuO or Fe₂O₃) subsequently reducing the metal via electron transfer.¹¹ This mechanism results in the generation of a persistent surface bound radical.

EPFRs associated with the particle surface impart additional stabilization to these radicals, allowing them to persist in the environment.^{11,16} This behavior was observed in the long lifetimes of radicals from the combustion of charcoal,¹⁷ wood, and coal.¹⁸ In the cases of wood and coal, there were two consecutive decays resulting from reaction with oxygen where the relative intensity but not the H_{p-p} of the radical signal decreased.¹⁸ Stabilized organic radical signals were additionally observed in soot from the combustion of plastics¹⁶ as well as the indefinite persistence of semiquinone radicals from cigarette smoke.¹⁹ In addition to natural samples, investigations on radical decay from a model soot system of 1-methylnaphthalene and Fe₂O₃ were performed.²⁰ These studies demonstrated two subsequent decays, and the presence of Fe₂O₃ generated longer lifetimes than the 1-methylnaphthalene soot alone.²⁰

Previous decays from model EFPR systems demonstrated a range of lifetimes in addition to the presence of multiple decays in some cases.^{12,21-23} For example, organic precursors chemisorbed on CuO and Fe₂O₃ displayed one decay with the longest lifetime on CuO (74 min) resulting from phenol.^{12,21} When phenol was chemisorbed on Ni and Zn, two subsequent decays were observed, and these decays were also the longest for each metal.^{22,23} In the case of Zn, this conveyed 1/e lifetimes of 10 days for the faster decay and 23 days for the slower decay.²³ All decays from the model EPFR system implied phenoxyl radicals are the short-lived species, while semiquinone radicals are the long-lived species.^{12,23} Semiquinone radicals, however, were suggested to decompose into phenoxyl radicals.^{12,23} The differences in the aforementioned decay behavior resulted from metal speciation and, although not specifically mentioned here, the adsorbed organic precursor.

Continuing this line of investigation, we quantified the initial concentration and decay of EPFRs in freshly collected PM_{2.5}. Observed decay rates along with initial radical concentrations were correlated among common atmospheric pollutants (ozone, NO_x, SO₂, etc.), meteorological conditions, and the samples' metal content.

EXPERIMENTAL SECTION

Sampling

PM_{2.5} samples were acquired from the Louisiana Department of Environmental Quality (LDEQ) ambient air monitoring station situated 30 ft away from roadside and 10 ft above the ground. This site is located north of the Louisiana State University campus in Baton Rouge, LA, near heavy interstate traffic along a major industrial corridor of the Mississippi River. Samples were collected using a Thermo Scientific Partisol-Plus (Model 2025) equipped with a PM_{2.5} fractionator. The flow rate was 16.7 L/min, and samples were collected on a Whatman 2 μ m PTFE 46.2 mm diameter filter supported by a polypropylene ring. Samples were cumulated for 24 h within a start/end time of 9:00AM from October to November of 2008, July to September of 2009, March to August of 2010, and May to October of 2011.

Electron Paramagnetic Resonance Characterization

All electron paramagnetic resonance (EPR) spectra for PM_{2.5} loaded filters were measured using a Bruker EMX 10/2.7 EPR spectrometer (X-band) equipped with a dual cavity utilizing modulation and microwave frequencies of 100 kHz and 9.516 GHz, respectively. Parameters for radical signal measurement were as follows: 2.05 mW power; modulation amplitude of 4.0 G; scan range of 100 G; time constant of 40.96 ms corresponding to a conversion of 163.84 ms; sweep time of 167.77 s; receiver gain 3.56×10^4 ; and three scans. Radical concentrations for filter bound PM_{2.5} were calculated by comparing the signal peak area, as calculated from the $(H_{p-p})^2$ multiplied by the relative intensity, to a 2,2-diphenyl-1-picrylhydrazyl standard.²⁴ The g-factors were determined by the WinEPR program.

Decay Study

After obtaining a final weight, the filter was removed from the polypropylene supported ring and analyzed by EPR to ascertain its initial spin concentration. The time from removing the sample to initial analysis was an hour or less from the sample collection end time. The filter was placed in a controlled temperature and humidity incubator for decay studies. Accordingly, two separate temperature and humidity settings were employed for this investigation. One setting at room temperature (23 °C) and humidity (60%) while the other at a temperature of 30 °C and a relative humidity of $50 \pm 5\%$. The lower temperature and humidity setting was chosen for comparison with previous EPFR decays. The higher temperature was performed to observe if temperature would affect the decay in addition to modeling for areas with an average higher temperature. The incubator was maintained under ambient air circulation with the aim of reproducing previous EPFR decay experiments.^{12,21–23} Subsequent analyses were performed intermittently, normalized to the initial spin concentration, and plotted against time from the initial analysis. An exponential regression was performed on the plotted data in order to calculate the decay rate constants and 1/e lifetimes of the radicals.

Metal Analysis

All metal analyses were achieved by extracting the metals from the filters using hot nitric acid and analyzed using an inductively coupled plasma atomic emission (ICP-AE) spectrometer.

Substituted Phenol Analysis

After decay analyses, two samples randomly chosen from each decay category were analyzed for substituted phenols (resorcinol, *p*-cresol, *m*-cresol, *o*-cresol, phenol, hydroquinone, and catechol). To accomplish this task, filters were placed in a 50 mL conical flask, and 10 mL of *tert*-butyl methyl ether (TBME) was added with 0.16 mg of *o*-chlorophenol as an internal standard. This was shaken for 20 min, and 250 μ L of the sample extract was transferred to an amber vial with an additional 500 μ L of TBME as well as 250 μ L of N,O-bis(trimethylsilyl)trifluoroacetamide (BSTFA). The vial was subsequently capped using Teflon/Silicone 11 mm crimp caps and mixed. The vial contents were heated using a preheated block for 30 min at 76 °C. Determination of phenols was executed by Agilent Technologies 6890 gas chromatograph system equipped with a 5973 mass selection detector.

Meteorological Data

All meteorological data was retrieved from the LDEQ ambient monitoring station as sample collection, except for solar and ultraviolet (UV) radiation measurements which were obtained from the capitol monitoring site (less than a mile away from sample collection).

Statistical Correlations

Correlations were calculated using Pearson's correlation coefficient (ρ) where coefficients closer to 1.0 indicate a strong positive relationship; coefficients approaching 0.0 attest a weak or no relationship; and coefficients near -1.0 signify a strong negative relationship.

Calculation for Equivalent Cigarettes Smoked from Inhaling PM_{2.5}

The number of equivalent cigarettes smoked from inhaling PM_{2.5} was calculated first by converting the PM_{2.5} radical concentration to radicals inhaled daily using eq 1

$$RI_{PM} = RC_{PM} FPC_{PM} V \quad (1)$$

where RI_{PM} represents the radicals inhaled from PM_{2.5} (radicals/day);, RC_{PM} is the averaged radical concentration in PM_{2.5} (radicals/g), F is the conversion from g to μ g, PC_{PM} is the particle concentration of PM_{2.5} (μ g/m³), and V is the volume of air breathed daily for an adult male (20 m³/day).²⁵ This was then compared to the number of radicals inhaled from smoking a cigarette using eq 2

$$EQ = \frac{RI_{PM}}{(RC_{cig} C_{tar})} \quad (2)$$

where EQ is the number of equivalent cigarettes smoked, RC_{cig} is the radical concentration in cigarette tar (radicals/g tar),^{26–30} and C_{tar} is the amount of tar per cigarette (g tar/cigarette).

RESULTS AND DISCUSSION

Initial Radical Concentrations

All collected PM_{2.5} samples initially displayed a single, unstructured organic peak exhibiting an average H_{p-p} of 5–8 G. The relatively wide peak in addition to a lack of hyperfine splitting implied multiple organic species of the same radical family present or signal broadening by organic–metal interactions.^{5,11,12,15} These signals displayed initial g -factors of $2.0035(\pm 0.0004)$, suggesting carbon centered radicals from polycyclic aromatic hydrocarbons or a group of semiquinone-type radicals in a complex matrix.^{5,11–14,16,31–33}

In addition to an organic peak, the presence of paramagnetic metals was detected. These metal peaks were persistent throughout the decay and not observed to degrade. The most common peak was Fe^{3+} at an approximate g -factor of 2.1 attributed to Fe^{3+} distributed in clusters.^{34,35} The presence of Mn^{2+} ($I = 5/2$) was also noticed in two samples collected on April 30th and May 1st of 2010, and this is believed to result from the in situ oil burn in the Gulf of Mexico. Aside from the noticeable smell of these fumes in Baton Rouge, the NOAA HYSPLIT model calculated air trajectory during this time showed air from the burns passing over our sampler (data not shown).

The average initial radical concentration along with the number of samples for each decay category (vide infra) are displayed in Table 1, and the fast accompanied by slow decay in addition to the fast followed by no decay categories' designation are established on the presence of multiple decay fits and their slope. The displayed concentrations resulted from an average weight of $512 \pm 300 \mu g$ collected per day. The overall radical concentration is comparable to the same concentration range from cigarette smoke,⁵ corresponding to 69 ppm as a semiquinone. A complete list of samples' initial radical concentration, decay rate constants, and $1/e$ lifetimes are given in the Supporting Information.

Decay of Radical Signal

Decay of a well behaved radical signal is presented in Figure 1. All decays resulted from a diminishing relative intensity; there was no consistent broadening or narrowing of the signal during decay with the H_{p-p} maintaining an average 6.49 ± 1.69 G for all samples. The g -factor slightly increased throughout the decay by an average of 0.0002. Although the change in the g value is small and close to the error limits, only an upward trend in g -factor was observed in all 94 samples indicating a statistically significant increase. A slight increase of g -factor suggests either a higher contribution of oxygen centered radicals from sample oxidation^{11,36–38} or losing more carbon centered radicals, thereby shifting the g -factor toward more oxygenated radicals.

There was no difference in decay behavior between the two temperature–humidity settings chosen. With PM_{2.5} constituents changing daily, any differences in decay rate from temperature are not apparent; however, only 30 samples were analyzed using the lower

parameters, so analyzing more samples at the lower conditions might identify an observable trend.

Categories of Decay

As displayed in Figure 2, four categories of decay were observed. The majority (47%) exhibited two consecutive decays with a relatively fast decay followed by a slower decay, Figure 2A. The fast decay rate constant for this category was $0.050\text{--}0.002\text{ h}^{-1}$ corresponding to a $1/e$ lifetime of 1–21 days, and the slow decay rate constant was $0.002\text{--}8.00 \times 10^{-6}\text{ h}^{-1}$ equivalent to a $1/e$ lifetime of 21–5028 days. The large range for the slow decay results from seven samples decaying extremely slowly yet consistently with $1/e$ lifetimes of 1000–5000 days. The majority of the slow $1/e$ lifetimes was in the range of 21–417 days.

A single slow decay was observed from approximately a quarter (24%) of the samples, Figure 2B. The decay rate constants were $0.010\text{--}2.00 \times 10^{-5}\text{ hr}^{-1}$ indicating a $1/e$ lifetime of 4–2083 days. Comparable to the previous category, there were two samples shifting the range with $1/e$ lifetimes of 833 and 2083 days. The $1/e$ lifetime range for the rest of the samples was 4–595 days. This range is similar to the previous category suggesting these samples may also exhibit a fast decay; however, due to a long atmospheric residence time, they may have decayed before an initial measurement.

The last two decay types were no decay (18%), Figure 2C, and a relatively fast decay followed by no decay (11%), Figure 2D. Similar to the first category, the average fast decay rate constants were $0.159\text{--}0.002\text{ h}^{-1}$ analogous to a $1/e$ lifetime of 0.25–21 days. Due to the unknown range of residence times in the atmosphere, decay in Figure 2C may just be after the fast decay in Figure 2D was completed.

In all cases, we attribute the faster decay (displayed as $\tau_{1/e} \sim 21$ days) to decomposition of a phenoxyl-type radical.^{12,23} This is further supported from correlations of phenol with the initial radical concentration in addition to the fast decay rate (vide infra). The slow decay is attributed to decomposition of a semiquinone-type radical²¹ (displayed as $\tau_{1/e} \sim 208$ and 417 days). The no decay pattern is explained by radicals entrapped in the bulk of $\text{PM}_{2.5}$ or restricted in a solid matrix (i.e., internal radicals) where the unpaired electron is delocalized over many conjugated or aromatic bonds.^{12,16,18,39–41} These radicals remain internal cannot undergo oxidation in air and therefore persisting indefinitely.

Substituted Phenol Analysis

Of all the analyzed substituted phenols, only phenol was above the detection limit in 6 out of 12 analyzed samples and in a range of 40–55 ppm. The phenol concentration was correlated to the initial $\text{H}_{\text{p-p}}$, initial radical concentration, initial g-factor, and the fast/slow decay. As observed in Figure 3A, the phenol data exhibited a strong correlation with the initial $\text{H}_{\text{p-p}}$ where the presence of more phenol increased the initial $\text{H}_{\text{p-p}}$. This suggested agreement with the concept of concentration broadening,^{42,43} where an increase in radical constituent will increase the $\text{H}_{\text{p-p}}$.

There was a strong association from the initial radical concentration where an increase in phenol resulted in an increased radical concentration, Figure 3B. This does not suggest only

phenoxyl radicals are present in PM_{2.5}. This is supported from the g-factor correlation, Figure 3C. A shift in g-factor occurs when there is a change in radical species. For example, existence of a semiquinone radical, one of the persistent radicals, was considered present in tobacco tar²⁷ as well as PM,^{11,12} and more recently, semiquinone redox cycling was demonstrated in the oxidative capacity of PM_{2.5}.¹⁰ The increased presence of a semiquinone radical, more oxygen centered in nature when compared to the phenoxyl radical,⁴⁴ will increase the g-factor. This is corroborated by the radical signal, because the organic radical signal is a single, broad, unstructured peak in all the samples studied; therefore, multiple superimposed radical signals may be present.^{12,16,18}

Correlations of the fast decay conveyed a very significant correlation of $\rho = 0.61$ ($n = 6$) with phenol concentration. In contrast to this, the slow decay exhibited a weak, negative correlation of $\rho = -0.20$ ($n = 3$). These associations further implicate the fast decay to occur from phenoxyl radical decomposition.

Metals Analysis and Correlation

Weakly significant or no correlations were observed with metals. This data is presented in the Supporting Information.

Meteorological and Atmospheric Pollutant Correlations

In general, meteorological correlations were not strong. The highest positive association for initial radical concentration resulted from ozone ($\rho = 0.28$) implying the importance of photochemical processes for EPFR formation. This is supported by positive relationships with both solar ($\rho = 0.14$) and UV radiation ($\rho = 0.12$). Correlations for the fast decay indicated the presence of ozone ($\rho = -0.10$) as well as solar ($\rho = -0.45$) and UV radiation ($\rho = -0.42$) decrease the fast decay rate. The previous sets of relationships demonstrate the ability of all three to increase the radical concentration; so consequently, their presence will decrease the fast decay rate due to new radical formation. Correlations for the slow decay were the weakest overall and less clear. Detailed data for meteorological conditions and atmospheric pollutants used in addition to their respective associations are presented in the Supporting Information.

Comparison of Radicals Inhaled in PM_{2.5} to Cigarettes

Our research demonstrated EPFRs induce various types of heart and respiratory dysfunction in rats and mice similar to those observed from smoking cigarettes.^{45–47} While direct comparison of EPFR effects in PM_{2.5} and cigarette smoke were not performed, the data suggests common EPFRs in cigarette tar and PM_{2.5} result in very similar human diseases.

In order to assess the potentially negative health consequences of PM_{2.5}, the overall average concentration of radicals from PM_{2.5} in Baton Rouge was compared to the average concentration of radicals in cigarette smoke.^{26–30} The outcome is expressed as the equivalent number of cigarettes a person smokes in a day from exposure to the same number of EPFRs inhaled from polluted air, Table 2. An example calculation using the U.S. 24 h PM_{2.5} concentration average is given below.

$$\begin{aligned}
 RI_{PM} &= RC_{PM} FPC_{PM} V \\
 RI_{PM} &= (3.84 \times 10^{17} \text{ radicals/g}) (1 \times 10^{-6} \text{ g/}\mu\text{g}) (26.9 \text{ }\mu\text{g/m}^3) (20 \text{ m}^3/\text{day}) \\
 RI_{PM} &= 2.07 \times 10^{14} \text{ radicals/day} \\
 EQ &= \frac{RI_{PM}}{RC_{cig} C_{tar}} \\
 EQ &= \frac{2.07 \times 10^{14} \frac{\text{radicals}}{\text{day}}}{(1.75 \times 10^{16} \frac{\text{radicals}}{\text{g tar}}) (0.013 \frac{\text{g tar}}{\text{cigarette}})} \\
 EQ &= 0.3 \text{ cigarettes}
 \end{aligned}$$

On the basis of the initial radical concentration and the U.S. 24 h air quality data, each person in the United States smokes the equivalent of 0.3 cigarettes per day from PM_{2.5} inhalation. The same calculation using the U.S. yearly average results in 47 cigarettes per year. In the more polluted areas (based on air quality exceedances), such as San Joaquin Valley, each person smokes nearly a full cigarette per day and as high as 101 cigarettes per year.

Overall, the decay of EPFRs in PM_{2.5} was observed to have three decays: a fast decay, slow decay, and no decay. These three decays were all filed into four distinct categories without any exception: a fast decay followed by a slow decay, a single slow decay, a fast decay accompanied by a slow decay, and a fast decay followed by no decay. Correlations with phenol suggested the fast decay to result from the phenoxyl radical decay. The slow decay was suggested to result from the semiquinone radical, and no decay is attributed to internal radicals restricted in a solid matrix. Due to the complexity of the PM_{2.5}, only simple meteorological correlations could hint at the underlying cause of these decays as well as the initial radical concentration. In addition to the decay of EPFRs, a quick and novel way to assess adverse health effects was presented suggesting inhaling polluted air could be the equivalent of smoking 0.3–0.9 cigarettes a day in the United States depending on residence.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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REFERENCES

1. Pope CA, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ. Cardiovascular mortality and long-term exposure to particulate air pollution. *Circulation*. 2004; 109(1):71–77. [PubMed: 14676145]
2. Pope CA III, Ezzati M, Dockery DW. Fine-particulate air pollution and life expectancy in the United States. *N. Engl. J. Med*. 2009; 360(4):376–86. [PubMed: 19164188]
3. Ayres JG, Borm P, Cassee FR, Castranova V, Donaldson K, Ghio A, Harrison RM, Hider R, Kelly F, Kooter IM, Marano F, Maynard RL, Mudway I, Nel A, Sioutas C, Smith S, Baeza-Squiban A, Cho A, Duggan S, Froines J. Evaluating the toxicity of airborne particulate matter and nanoparticles

- by measuring oxidative stress potential—a workshop report and consensus statement. *Inhalation Toxicol.* 2008; 20(1):75–99.
4. Dellinger B, Pryor WA, Cueto R, Squadrito GL, Hegde V, Deutsch WA. Role of free radicals in the toxicity of airborne fine particulate matter. *Chem. Res. Toxicol.* 2001; 14(10):1371–1377. [PubMed: 11599928]
 5. Squadrito GL, Cueto R, Dellinger B, Pryor WA. Quinoid redox cycling as a mechanism for sustained free radical generation by inhaled airborne particulate matter. *Free Radicals Biol. Med.* 2001; 31(9):1132–1138.
 6. de Kok T, Driessche HAL, Hogervorst JGF, Briede JJ. Toxicological assessment of ambient and traffic-related particulate matter: A review of recent studies. *Mutat. Res., Rev. Mutat. Res.* 2006; 613(2–3):103–122.
 7. Dabuelson PH, Møller P, Jensen KA, Sharma AK, Wallin H. k. Bossi R, Autrup H, Mølhav L, Ravanat J-L, Briedé JJ, de Kok TM, Loft S. Oxidative Stress, dna damage, and inflammation induced by ambient air and wood smoke particulate matter in human A549 and THP-1 cell lines. *Chem. Res. Toxicol.* 2011; 24(2):168–184. [PubMed: 21235221]
 8. Khachatryan L, Vejerano E, Lomnicki S, Dellinger B. Environmentally persistent free radicals (EPFRs). 1. Generation of reactive oxygen species in aqueous solutions. *Environ. Sci. Technol.* 2011; 45(19):8559–66. [PubMed: 21823585]
 9. Khachatryan L, Dellinger B. Environmentally persistent free radicals (EPFRs). 2. Are free hydroxyl radicals generated in aqueous solutions? *Environ. Sci. Technol.* 2011; 45(21):9232–9239. [PubMed: 21942783]
 10. Gehling W, Khachatryan L, Dellinger B. Hydroxyl radical generation from environmental persistent free radicals (EPFRs) in PM_{2.5}. *Environ. Sci. Technol.* 2013
 11. Dellinger B, Lomnicki S, Khachatryan L, Maskos Z, Hall RW, Adoukpe J, McFerrin C, Truong H. Formation and stabilization of persistent free radicals. *Proc. Combust. Inst.* 2007; 31(1):521–528.
 12. Lomnicki S, Truong H, Vejerano E, Dellinger B. Copper oxide-based model of persistent free radical formation on combustion-derived particulate matter. *Environ. Sci. Technol.* 2008; 42:4982–4988. [PubMed: 18678037]
 13. Valavanidis A, Fiotakis K, Bakeas E, Vlahogianni T. Electron paramagnetic resonance study of the generation of reactive oxygen species catalysed by transition metals and quinoid redox cycling by inhalable ambient particulate matter. *Redox Rep.* 2005; 10(1):37–51. [PubMed: 15829110]
 14. Valavanidis A, Fiotakis K, Vlahogianni T, Papadimitriou V, Pantikaki V. Determination of selective quinones and quinoid radicals in airborne particulate matter and vehicular exhaust particles. *Environ. Chem.* 2006; 3(2):118–123.
 15. Valavanidis A, Fiotakis K, Vlahogianni T. The Role of Stable Free Radicals, Metals and PAHs of Airborne Particulate Matter in Mechanisms of Oxidative Stress and Carcinogenicity.. In: Zereini, F.; Wiseman, CLS., editors. *Urban Airborne Particulate Matter*. Springer; Berlin, Heidelberg: 2011. p. 411–426.
 16. Valavanidis A, Iliopoulos N, Gotsis G, Fiotakis K. Persistent free radicals, heavy metals and PAHs generated in particulate soot emissions and residue ash from controlled combustion of common types of plastic. *J. Hazard. Mater.* 2008; 156:277–284. [PubMed: 18249066]
 17. Yordanov, ND. Introduction to the theory of electron paramagnetic resonance and its application to the study of aerosols.. In: Spurny, KR., editor. *Analytical Chemistry of Aerosols*. Lewis; Boca Raton, FL: 1999. p. 197–213.
 18. Tian L, Koshland CP, Yano J, Yachandra VK, Yu ITS, Lee SC, Lucas D. Carbon-centered free radicals in particulate matter emissions from wood and coal combustion. *Energy Fuels.* 2009; 23(5):2523–2526. [PubMed: 19551161]
 19. Pryor WA. Biological effects of cigarette smoke, wood smoke, and the smoke from plastics: the use of electron spin resonance. *Free Radicals Biol. Med.* 1992; 13(6):659–76.
 20. Herring P, Potter P, Wu H, Lomnicki S, Dellinger B. Fe₂O₃ nanoparticle mediated molecular growth and soot inception from the oxidative pyrolysis of 1-methylnaphthalene. *Proc. Combust. Inst.* 2013; 34:1749–1757. [PubMed: 25530732]

21. Vejerano E, Lomnicki S, Dellinger B. Formation and stabilization of combustion-generated environmentally persistent free radicals on an Fe(III)₂O₃/silica surface. *Environ. Sci. Technol.* 2011; 45:589–594. [PubMed: 21138295]
22. Vejerano E, Lomnicki SM, Dellinger B. Formation and stabilization of combustion-generated, environmentally persistent radicals on Ni(II)O supported on a silica surface. *Environ. Sci. Technol.* 2012; 46:9406–9411. [PubMed: 22831558]
23. Vejerano E, Lomnicki S, Dellinger B. Lifetime of combustion-generated environmentally persistent free radicals on Zn(II)O and other transition metal oxides. *J. Environ. Monit.* 2012; 14:2803–2806. [PubMed: 22990982]
24. Chesnut DB. On the use of AW2 method for integrated line intensities from first-derivative presentations. *J. Magn. Reson.* 1977; 25:373–4.
25. Recommendations for and documentation of biological values for use in risk assessment. Environmental Protection Agency; Washington, DC: 1988.
26. Pryor WA, Prier DG, Church DF. Electron-spin resonance study of mainstream and sidestream cigarette smoke: Nature of the free radicals in gas-phase smoke and in cigarette tar. *Environ. Health Perspect.* 1983; 47:345–355. [PubMed: 6297881]
27. Pryor W, Hales B, Premovic P, Church D. The radicals in cigarette tar: their nature and suggested physiological implications. *Science.* 1983; 220(4595):425–427. [PubMed: 6301009]
28. Blakley RL, Henry DD, Smith CJ. Lack of correlation between cigarette mainstream smoke particulate phase radicals and hydroquinone yield. *Food Chem. Toxicol.* 2001; 39(4):401–406. [PubMed: 11295487]
29. Valavanidis A, Haralambous E. A comparative study by electron paramagnetic resonance of free radical species in the mainstream and sidestream smoke of cigarettes with conventional acetate filters and “bio-filters”. *Redox Rep.* 2001; 6(3):161–171. [PubMed: 11523591]
30. Baum SL, Anderson IGM, Baker RR, Murphy DM, Rowlands CC. Electron spin resonance and spin trap investigation of free radicals in cigarette smoke: development of a quantification procedure. *Anal. Chim. Acta.* 2003; 481(1):1–13.
31. Lachocki TM, Church DF, Pryor WA. Persistent free radicals in woodsmoke: An ESR spin trapping study. *Free Radicals Biol. Med.* 1989; 7(1):17–21.
32. Pryor WA. Cigarette smoke radicals and the role of free radicals in chemical carcinogenicity. *Environ. Health Perspect.* 1997; 105(Suppl 4):875–82. [PubMed: 9255574]
33. Valavanidis A, Salika A, Theodoropoulou A. Generation of hydroxyl radicals by urban suspended particulate air matter. The role of iron ions. *Atmos. Environ.* 2000; 34(15):2379–2386.
34. Kumar B, Chen C, Lin S. Effects of melting atmosphere on properties of a glass in the calcia-phosphorus pentoxide-iron sesquioxide system. *Phys. Chem. Glasses.* 1992; 33(5):204–6.
35. Ardelean I, Peteanu M, Filip S, Simon V, Györfy G. EPR and magnetic susceptibility studies of iron ions in 70TeO₂-25-B₂O₃-5PbO glass matrix. *Solid State Commun.* 1997; 102(4):341–346.
36. Barckholtz C, Fadden MJ, Hadad CM. Computational study of the mechanisms for the reaction of O (³ 2 Σ_g) with aromatic radicals. *J. Phys. Chem. A.* 1999; 103(40):8108–8117.
37. Korkmaz M, Polat M. Free radical kinetics of irradiated durum wheat. *Radiat. Phys. Chem.* 2000; 58(2):169–179.
38. Tonokura K, Norikane Y, Koshi M, Nakano Y, Nakamichi S, Goto M, Hashimoto S, Kawasaki M, Sulbaek Andersen MP, Hurley MD, Wallington TJ. Cavity ring-down study of the visible absorption spectrum of the phenyl radical and kinetics of its reactions with Cl, Br, Cl₂, and O₂. *J. Phys. Chem. A.* 2002; 106(24):5908–5917.
39. Borg, DC. In *Oxygen free radicals and tissue injury: a reference outline*. Birkhaeuser; Boston: 1993. p. 12-53.
40. Jankovic J, Jones W, Castranova V, Dalal N. Measurement of short-lived reactive species and long-lived free radicals in air samples from structural fires. *Appl. Occup. Environ. Hyg.* 1993; 8:650–4.
41. Yordanov ND, Mladenova R. EPR study of thermally generated free radicals in nuts. *Int. J. Food Sci. Technol.* 2007; 42:1384–1389.
42. Eaton SS, Eaton GR. Interaction of spin labels with transition metals. *Coord. Chem. Rev.* 1978; 26(3):207–262.

43. Filippov AI. Nature of the concentration contributions to the EPR line widths of solutions of copper(II) diethyldithiocarbamate. *Theor. Exp. Chem.* 1984; 20(1):101–104.
44. McFerrin CA, Hall RW, Dellinger B. Ab initio study of the formation and degradation reactions of semiquinone and phenoxyl radicals. *J. Mol. Struct.: THEOCHEM.* 2008; 848(1–3):16–23.
45. Lord K, Moll D, Lindsey JK, Mahne S, Raman G, Dugas T, Cormier S, Troxclair D, Lomnicki S, Dellinger B, Varner K. Environmentally persistent free radicals decrease cardiac function before and after ischemia/reperfusion injury in vivo. *J. Recept. Signal Transduction Res.* 2011; 31(2): 157–67.
46. Fahmy B, Ding L, You D, Lomnicki S, Dellinger B, Cormier SA. In vitro and in vivo assessment of pulmonary risk associated with exposure to combustion generated fine particles. *Environ. Toxicol. Pharmacol.* 2010; 29(2):173–82. [PubMed: 20369027]
47. Balakrishna S, Lomnicki S, McAvey KM, Cole RB, Dellinger B, Cormier SA. Environmentally persistent free radicals amplify ultrafine particle mediated cellular oxidative stress and cytotoxicity. *Part. Fibre Toxicol.* 2009; 6:11. [PubMed: 19374750]
48. [1/31/11] 2007–2009 PM_{2.5} Design Value Report.. <http://www.epa.gov/airtrends/values.html>

Figure 1.

Decay of organic radical signal over 2 months as observed by EPR spectra. The g -factors are included to indicate oxidation of the radicals as the signal decays. This sample was collected on 08/28/ 2009.

Figure 2.

Representation of the four categories of decay observed. All represented decays occurred at 30 °C and 50% RH. All displayed lifetimes are 1/e lifetimes. (A) Representation of samples exhibiting two consecutive decays, a relatively fast decay followed by a slower decay (47% of samples). (B) Representation of samples exhibiting one slow decay (23%). (C) Representation of samples exhibiting no decay (18%). (D) Representation of samples exhibiting a relatively fast decay followed by no decay (11%).

Figure 3.

Plots of phenol correlations. (A) Initial H_{p-p} vs phenol concentration with a correlation value of $\rho = 0.95$. (B) Initial spins/g vs phenol concentration with a correlation value of $\rho = 0.61$. (C) Initial g-factor vs phenol concentration with a correlation value of $\rho = 0.97$.

Table 1

Average Radical Concentration and the Number of Samples for Each Decay Category

decay category	avg initial radical concn (radicals/g)	# samples
fast decay followed by slow decay	2.32×10^{16} – 3.48×10^{18}	54
slow decay	2.02×10^{16} – 1.34×10^{18}	27
no decay	2.65×10^{16} – 1.17×10^{18}	21
fast decay followed by no decay	5.92×10^{16} – 1.99×10^{18}	12
all samples	2.02×10^{16} – 3.48×10^{18}	94

Table 2Number of Equivalent Cigarettes Smoked from Inhaling PM_{2.5} with 95% Confidence Interval^a

average concentrations and regulatory standards	concentration of PM _{2.5} (µg/m ³)	# of equivalent cigarettes
U.S. 24 h average	26.9	0.3 ± 0.1 per day
U.S. yearly average	10.6	47 ± 16 per year
EPA 24 h standard (2011)	35	0.4 ± 0.1 per day
EPA yearly standard (2011)	15	67 ± 23 per year

24 h PM _{2.5} non-attainment locations			
region	state	PM _{2.5} 24 h design value (µg/m ³) ^b	# of equivalent cigarettes
Chico	CA	59	0.7 ± 0.2
Cleveland–Akron–Lorain	OH	36	0.4 ± 0.1
Fairbanks	AK	57	0.7 ± 0.2
Klamath Falls	OR	47	0.6 ± 0.2
Liberty–Clairton	PA	50	0.6 ± 0.2
Logan	UT–ID	40	0.5 ± 0.2
Los Angeles–South Coast Air Basin	CA	49	0.6 ± 0.2
Milwaukee–Racine	WI	37	0.5 ± 0.2
Oakridge	OR	41	0.5 ± 0.2
Pittsburgh–Beaver Valley	PA	37	0.5 ± 0.2
Provo	UT	50	0.6 ± 0.2
Sacramento	CA	51	0.6 ± 0.2
Salt Lake City	UT	48	0.6 ± 0.2
San Francisco Bay Area	CA	36	0.4 ± 0.1
San Joaquin Valley	CA	70	0.9 ± 0.3
Seattle–Tacoma	WA	46	0.6 ± 0.2
Steubenville–Weirton	OH–WV	37	0.5 ± 0.2
Yuba City–Marysville	CA	42	0.5 ± 0.2

^a All PM_{2.5} data is for 2007–2009 designation values from ref 48.^b Design values are computed from PM_{2.5} monitoring data reported to the EPA's Air Quality System from the local agencies. Exceptional events (wildfires, construction, volcanic eruption) are not included in the calculation.