



## **EPA’s Proposal to Waive Clean Air Protection for Millions of Americans: The Politics of Coarse Particle Pollution**

### **I. Introduction and Background**

Scientific studies link concentrations of airborne coarse particulate matter (“PM”) with hospitalizations and deaths due to heart and lung disease. Coarse PM comprises particles between 2.5 and 10 microns in diameter—less than 1/7 the diameter of a human hair. Air pollution from coarse PM is currently regulated under EPA’s National Ambient Air Quality Standard (NAAQS) for “PM<sub>10</sub>,” which limits airborne concentrations of particles 10 microns in diameter and smaller. The Clean Air Act requires EPA to adopt NAAQS at levels strong enough to protect the public health, allowing an adequate margin of safety.<sup>1</sup>

Once a NAAQS like that for PM<sub>10</sub> is promulgated, states must employ State Implementation Plans (“SIPs”) that ensure achievement of the standard within specified periods of time defined by the Act.<sup>2</sup> EPA must update the NAAQS every 5 years, based on the most recent scientific studies.<sup>3</sup> EPA is currently undertaking its 5-year review of the PM NAAQS.

In 1997, EPA adopted a separate “PM<sub>2.5</sub>” NAAQS for “fine” particles—those 2.5 microns in diameter and smaller—based on scientific studies showing a need for additional health protection from such particles. At the same time, EPA retained the PM-10 NAAQS to provide continued protection against health threats from coarse particles.

EPA has recently proposed to revise the PM<sub>2.5</sub> standard, abolish the PM<sub>10</sub> standard entirely, and adopt a separate coarse PM standard for particles in the 2.5- to 10-micron size range. Under EPA’s proposal, however, the new coarse particle standard would apply only in areas dominated by “urban” particulate sources, and would not apply at all to mining and agricultural emissions. The result of the proposal would be to substantially weaken the protection provided against coarse particles by current standards. As further discussed below, EPA’s proposed approach is not supported by science and conflicts with basic requirements of the Clean Air Act.

EPA’s proposal irrationally assumes that suspended rural coarse particles, and particles generated by the mining and agriculture industries, are inherently less toxic than those found in urban areas. Based on this assumption, the proposal would arbitrarily regulate and monitor airborne coarse particles only in urban regions—defined as those regions having total populations of 100,000 persons or more, and population densities in excess of 500 persons/mi<sup>2</sup>—leaving residents and visitors to rural areas unprotected. EPA’s proposed NAAQS rule does not stop there, however, but goes one step further, arbitrarily proposing to delineate among various source categories, of which some would be subject to the coarse-particle NAAQS, and some would not. Specifically, EPA’s proposed standard would be “qualified so as to include any

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<sup>1</sup> See 42 U.S.C. § 7409(b).

<sup>2</sup> § 7410.

<sup>3</sup> § 7409(d).

ambient mix of PM<sub>10-2.5</sub> that is dominated by resuspended dust from high-density traffic on paved roads and PM generated by industrial sources and construction sources, and excludes any ambient mix of PM<sub>10-2.5</sub> that is dominated by rural windblown dust and soils and PM generated by agricultural and mining sources.”<sup>4</sup>

Although the “urban/rural” distinction appears most prominently in the currently proposed “Monitoring Rule”,<sup>5</sup> that rule’s clear demarcation of those areas that will be monitored, and those that will not, means that the monitoring rule and the rule proposing to establish air quality standards must be considered as a unit. Under the Monitoring Rule, in order to be eligible for comparison to the proposed air quality standard, a monitoring site must be contained within an urbanized area having a population of at least 100,000 persons, and a population density of at least 500 persons per square mile.<sup>6</sup>

EPA’s rationale for proposing to regulate coarse particulate matter in urban regions, but not rural, agricultural and mining ones, is flawed for a number of reasons. In the first instance, there is no scientific basis to support EPA’s broad generalizations about the nature and composition of rural particles. Moreover, there is ample peer-reviewed scientific evidence showing that airborne coarse particles in rural, agricultural and mining regions pose a threat to human health. The flaws in EPA’s reasoning demonstrate that politics—not science—are the driving forces behind the urban/rural dichotomy that EPA is proposing to recognize.

## **II. EPA Has Ignored Sound Science in its Attempt to Carve Out an Exemption for Rural, Mining and Agricultural Particulates in its Proposed Coarse PM Standard.**

### **1. In proposing to exempt rural coarse particles, EPA ignored epidemiological studies conducted in rural communities showing health effects resulting from coarse particle pollution**

As stated above, EPA’s basis for attempting to establish an urban/rural dichotomy within the realm of regulatory protections against harmful particulate matter emissions is fundamentally flawed, and is clearly being driven by politics rather than science.

The assumption that rural coarse particle pollution is not harmful is a false one that is refuted by some of the very epidemiological studies that EPA relies upon in attempting to formulate a rule that applies only to urban areas. For example, one of those studies was conducted in the town of Steubenville, Ohio. Steubenville is a small steel mill town in eastern Ohio that is inhabited by about 19,000 residents.<sup>7</sup> Studies of particulate matter concentrations in Steubenville have shown a statistically significant increase in death rates that are associated with increases in airborne concentrations of coarse PM.<sup>8</sup>

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<sup>4</sup> See Environmental Protection Agency, Proposed Rule, National Ambient Air Quality Standards for Particulate Matter, 71 Fed. Reg. 2620, 2620 (Jan. 17, 2006) [hereinafter “Proposed Rule”].

<sup>5</sup> See Environmental Protection Agency, Proposed Rule, Revisions to Ambient Air Monitoring Regulations, 71 Fed. Reg. 2710 (Jan. 17, 2006) [hereinafter “Monitoring Rule”].

<sup>6</sup> See *id.* at 2782.

<sup>7</sup> Wikipedia, at [http://en.wikipedia.org/wiki/Steubenville,\\_Ohio](http://en.wikipedia.org/wiki/Steubenville,_Ohio)

<sup>8</sup> 2 Nat’l Center for Env’tl. Assessment, Env’tl. Protection Agency, Pub. No. EPA/600/P-99/002aF, Air Quality Criteria for Particulate Matter § 8.2.2.3.1, at 8-40 (2004) [hereinafter CD] (citing J. Schwartz, *Daily deaths*

Another study showed a statistical association between coarse particles and respiratory symptoms in children in both Steubenville and the distinctly non-urban community of Portage, Wisconsin,<sup>9</sup> whose population in the year 2000 totaled 9,728.<sup>10</sup> However, in the preamble to its proposed rule, EPA tries to discount the results of the study, saying the areas studied are not representative of those areas in which coarse particles are the larger fraction of the PM<sub>10</sub> measurement.<sup>11</sup> In other words, notwithstanding the study's direct correlation of health effects with concentrations of PM<sub>10-2.5</sub>, EPA attempts to discredit the association by once again parsing regions of the country—this time dividing them into those “urban” areas whose PM<sub>10</sub> measurement contains a majority fraction of PM<sub>2.5</sub>, and those with a majority fraction of PM<sub>10-2.5</sub>. According to EPA, because the study was performed in the former type of region, they are not representative of the latter type.<sup>12</sup>

Another series of studies was conducted that focused on the closure of a steel mill in the vicinity of Provo, Utah, during the mid-1980's.<sup>13</sup> The studies observed the number of deaths, lung ailments and hospital admissions for the time periods before, during and after the mill's closure, and related the observances to ambient measurements of PM<sub>10</sub>. The studies found a clear association between PM<sub>10</sub> and the observed health effects.<sup>14</sup> Because the Provo vicinity is an area “where the coarse fraction of PM10 is typically much greater than the fine fraction,” in this instance, EPA has conceded that the studies “provide[] . . . supportive evidence for associations between short-term exposure to thoracic coarse particles and health effects, particularly morbidity effects, generally in areas not meeting the PM10 standards.”<sup>15</sup>

A study conducted by Dr. Bart Ostro in Coachella Valley, California, found statistically significant associations between concentrations of coarse particles in the air and deaths due to heart disease.<sup>16</sup> The Coachella Valley consists principally of the communities of Palm Springs

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*associated with air pollution in six US cities and short-term mortality displacement in Boston, in Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health 219-26 (Health Effects Inst. 2004), available at <http://www.healtheffects.org/Pubs/TimeSeries.pdf>*

<sup>9</sup> Proposed Rule, *supra* note 4, at 2664 (citing Joel Schwartz & Lucas Neas, *Fine Particles are More Strongly Associated than Coarse Particles with Acute Respiratory Health Effects in Schoolchildren*, 11 *Epidemiology* 6 (2000)).

<sup>10</sup> Wikipedia, at [http://en.wikipedia.org/wiki/Portage,\\_Wisconsin](http://en.wikipedia.org/wiki/Portage,_Wisconsin).

<sup>11</sup> Proposed Rule, *supra* note 4, at 2664 (citing Office of Air Quality Planning & Standards, U.S. Env'tl. Protection Agency, Pub. No. EPA-452/D-05-001, *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information* (2005) [hereinafter *Staff Paper*]).

<sup>12</sup> EPA's attempt to discount the latter study appears to be a challenge to *all* multi-pollutant studies that find a statistically significant association with a pollutant of less than majority concentration; in any event, EPA does not attempt to explain why the study should nevertheless *not* be considered representative of other rural areas like Portage and Steubenville themselves, where an association was found between respiratory health effects and PM<sub>10-2.5</sub>, even though concentrations of PM<sub>2.5</sub> comprise a majority fraction of the PM<sub>10</sub> measurement.

<sup>13</sup> C. Arden Pope III, *Respiratory Disease Associated with Community Air Pollution and a Steel Mill, Utah Valley*, 79 *Am. J. Public Health* 623 (1989) [hereinafter “Pope I”]; C. Arden Pope III, *Respiratory Hospital Admissions Associated with PM<sub>10</sub> Pollution in Utah, Salt Lake, and Cache Valleys*, 46 *Arch. Env'tl. Health* 90 (1991); C. Arden Pope et al., *Daily Mortality and PM<sub>10</sub> Pollution in Utah Valley*, 47 *Arch. Env'tl. Health* 211 (1992).

<sup>14</sup> *E.g.*, Pope I, *supra* note 13, at 627.

<sup>15</sup> Proposed Rule, *supra* note 4, at 2664.

<sup>16</sup> Bart D. Ostro et al., *Coarse and Fine Particles and Daily Mortality in the Coachella Valley, California: A Follow-Up Study*, 10 *J. Exposure Analysis & Env'tl. Epidemiology* 412 (2000).

(population 42,800)<sup>17</sup> and Indio (population 49,100).<sup>18</sup> Based on their populations, the two communities would be considered rural under EPA's proposed rule.

In the preamble to its proposed rule, EPA attempts to discredit the Coachella Valley study's finding of an association between cardiac deaths and coarse particle concentrations by attacking the methods of Dr. Ostro's data collection and analysis.<sup>19</sup> Specifically, EPA challenged Dr. Ostro's use of statistical modeling to recreate 10 years of PM<sub>10-2.5</sub> data, using 10 years of PM<sub>10</sub> data but only 2.5 years of PM<sub>2.5</sub> data. According to EPA, because the correlation used by Dr. Ostro to estimate PM<sub>10-2.5</sub> data during the 'missing years' was "effectively linear," the study confirms an association between PM<sub>10</sub> and the observed cardiac deaths, but not necessarily between those effects and PM<sub>10-2.5</sub>.

EPA's criticism of Dr. Ostro's study has been refuted by Dr. Ostro, who has pointed out in his comments to the Clean Air Science Advisory Committee ("CASAC")<sup>20</sup> that because virtually all of the airborne PM in the Coachella Valley region is comprised of particles in the PM<sub>10-2.5</sub> range, it should not be surprising that there is a close correlation between PM<sub>10</sub> and PM<sub>10-2.5</sub>, or that there is a similar close correlation between observed deaths and the statistically derived values of PM<sub>10-2.5</sub>. In other words, because most of the daily variation in PM<sub>10</sub> is due to variation in PM<sub>10-2.5</sub>, one could conceivably relate either measure to cardiac mortality, since in Coachella Valley, the two are so closely related.

EPA also challenged Dr. Ostro's method of relating deaths occurring in Palm Springs with PM levels in the air derived from monitors roughly 20 miles away in the town of Indio.<sup>21</sup> Yet the climate and topography of the two towns are essentially the same; and as Dr. Ostro has pointed out to the CASAC,<sup>22</sup> if differences in the characteristics of coarse particles in Indio and Palm Springs were so substantial as to break the association among particles and observed deaths, it would be reflected in parameters showing a statistically *non*-significant association between the two. That is, if the PM<sub>10-2.5</sub> for the region is being poorly represented using Indio monitors, it would make it more difficult to find an effect that correlates statistically in the first place. Simply put, EPA's argument is not sufficient to negate a positive association that has been found by the study.

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<sup>17</sup> Wikipedia, at [http://en.wikipedia.org/wiki/Palm\\_Springs,\\_California](http://en.wikipedia.org/wiki/Palm_Springs,_California)

<sup>18</sup> *Id.* at [http://en.wikipedia.org/wiki/Indio%2C\\_California](http://en.wikipedia.org/wiki/Indio%2C_California)

<sup>19</sup> According to EPA:

Ostro et al. (2003) used a one-pollutant model to estimate the association between PM<sub>10-2.5</sub> on mortality using an effectively linear construct of PM<sub>10</sub> (as observed in Indio, CA) to represent PM<sub>10-2.5</sub> for the entire study area. By using such a construct of PM<sub>10</sub>, the estimated associations simply reflect a PM<sub>10</sub> association (i.e., the construct does not provide additional information on the effect of PM<sub>10-2.5</sub>).

Proposed Rule, *supra* note 4, at 2672.

<sup>20</sup> Dr. Bart Ostro, Oral Comments to CASAC, CASAC teleconference, Feb. 3, 2006.

<sup>21</sup> According to EPA,

roughly 75 percent of the cardiovascular mortality in this study occurred in or near Palm Springs, CA and PM characteristics differ significantly between Palm Springs and Indio . . . Thus, the Ostro et al. (2003) study suggests a positive association between PM<sub>10</sub> monitored in Indio and mortality in Palm Springs, but some view this study as offering little basis for attributing significant mortality association to PM<sub>10-2.5</sub> as observed in either city.

*Id.*

<sup>22</sup> Ostro, *supra* note 20.

A study of associations between mortality and airborne coarse particles conducted in Phoenix by Smith, et al. in the year 2000 similarly supports the fact of a relationship between observed deaths and the types of airborne particles that, according to EPA, are typical of rural environments. In that study, the authors separated the observed coarse-particle concentrations into ‘crustal’ and ‘metal-enriched’ components, where the crustal components, according to EPA’s presumptions, would be characteristic of those types of particles found in rural areas. However, as pointed out by CASAC member Mr. Rich Poirot, the Phoenix study “noted strongest mortality associations during the spring and summer months when the metal-enriched particle concentrations were lowest (and the crustal component was the highest).”<sup>23</sup>

Other epidemiological studies support the conclusion that ambient coarse particles found in rural areas pose hazards to human health. For example, a series of studies discussed in EPA’s Criteria Document (“CD”)—a review of scientific information on PM health and welfare impacts—show statistically significant associations between ambient particle concentrations and respiratory symptoms among children living near open-pit coal mines.<sup>24</sup> Although the associations found were related to PM<sub>10</sub> measurements, when read in conjunction with laboratory and occupational studies on the characteristics of mine-related particulates,<sup>25</sup> the studies support the conclusion that communities living near open-pit mines are exposed to health hazards as a result of ambient concentrations of PM<sub>10-2.5</sub> arising from those mines.

Taken together, the foregoing epidemiological studies provide ample evidence that rural coarse particulates, presumed to be benign by EPA, are in fact harmful to human health. The laboratory studies discussed in the section that follows provide substantial additional evidence in support of such a conclusion.

## **2. Laboratory and occupational studies have demonstrated that the types of coarse particles that cause adverse health effects are ubiquitous**

As has been demonstrated, in its result-oriented determination to carve out a rural/urban distinction and provide a benefit to the mining and agribusiness industries, EPA irrationally discounted the findings of epidemiological studies showing statistically significant associations between rural airborne coarse particles and human health effects. The result is a proposed “national” air quality standard that would leave inhabitants of vast regions of the country unprotected.

In addition to the *epidemiological* studies finding associations between health effects and rural coarse particulates, there is ample evidence among *laboratory and occupational* studies of the types of particles found in both urban and rural regions of the country to support regulation of those particulates for the purpose of protecting human health.

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<sup>23</sup> Letter from Dr. Rogene Henderson, CASAC Chair, to Stephen Johnson, EPA Administrator, Sept. 15, 2005, at D-21 (comments of Mr. Rich Poirot).

<sup>24</sup> 2 CD, *supra* note 8, § 8.3.2.5, at 8-192; *id.* tbl. 8B-7 at 85-75 (2004) (citing T. Pless-Mullooli et al., *Living Near Opencast Coal Mining Sites and Children's Respiratory Health*, 57 *Occup. Environ. Med.* 145 (2000)); D. Howel et al., *Children's Respiratory Health and Daily Particulate Levels in 10 Nonurban Communities*, 87 *Environ. Res.* 1 (2001)).

<sup>25</sup> See *infra* text accompanying notes 55-61.

Studies of the effects of endotoxin on respiratory function provide a good starting point for this premise. “Endotoxin” refers to a class of bacteria that includes such harmful varieties as *E-coli* and *Salmonella*.<sup>26</sup> Endotoxins have long been known to cause respiratory symptoms among farmworkers, including pig farmers<sup>27</sup> and chicken farmers.<sup>28</sup> Airborne grain elevator dusts have also been found to contain endotoxins.<sup>29</sup>

In a study of exposures to inhalable dusts among California farmworkers, the authors measured levels of inhalable endotoxins associated with various agricultural activities.<sup>30</sup> While noting that previous studies had suggested a “no-effect” level of 100 Endotoxin Units per cubic meter of air (100 EU/m<sup>3</sup>),<sup>31</sup> the study identified a large number of activities exhibiting average inhalable endotoxin levels that far exceeded this value, including the cleaning of poultry houses (3874 EU/m<sup>3</sup>), weed mowing (1942 EU/m<sup>3</sup>), and the machine harvesting of vegetables (1550 EU/m<sup>3</sup>).<sup>32</sup>

In one European laboratory study, samples of coarse (PM<sub>10-2.5</sub>) and fine (PM<sub>2.5</sub>) particles were collected in rural (Borken, population 40,000) and urban (Duisburg, population 500,000) locations in Germany.<sup>33</sup> Each type of sample was instilled into the lungs of laboratory rats, which were then examined for signs of inflammation. The authors of the study found that “[i]rrespective of the sampling location, the coarse fraction of PM<sub>10</sub> but not its fine counterpart caused . . . inflammation in rat lungs,” and hypothesized “that endotoxin or related contaminants may play a role in these in vivo effects.”<sup>34</sup> This observation is consistent with the conclusions of other studies that have found pro-inflammatory responses among human subjects whose lung tissues were exposed to coarse particles containing endotoxins,<sup>35</sup> and that have found such responses to the PM<sub>10-2.5</sub> fraction of such particles in particular.<sup>36</sup>

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<sup>26</sup> See Mechanisms of Bacterial Pathogenicity: Endotoxins, in Todar’s Online Textbook of Bacteriology (2002), available at <http://textbookofbacteriology.net/endotoxin.html>.

<sup>27</sup> E.g., D. Heederik et al., *Relationship of Airborne Endotoxin and Bacteria Levels in Pig Farms with the Lung Function and Respiratory Symptoms of Farmers*, 62 Intl. Archives Occup. Envtl. Health 595 (1991);

<sup>28</sup> E.g., Brigitte Nielsen et al., *Exposure to Air Contaminants in Chicken Catching*, 56 Am. Indus. Hygiene Assoc. J. 804 (1995).

<sup>29</sup> E.g., A.J. DeLucca et al., *Gram-Negative Bacterial Endotoxins in Grain Elevator Dusts*, 45 Am. Indus. Hygiene Assoc. J. 336 (1984).

<sup>30</sup> Mark J. Nieuwenhuijsen et al., *Personal Exposure to Dust, Endotoxin and Crystalline Silica in California Agriculture*, 43 Ann. Occup. Hygiene 35 (1999)

<sup>31</sup> *Id.* at 41 (citing R. Rylander, *Evaluation of the Risks of Endotoxin Exposures*, 3 Int’l. J. Occup. Envtl. Health S32 (1997) (supp.)).

<sup>32</sup> *Id.* at 38 tbl. 3.

<sup>33</sup> Roel P.F. Schins et al., *Inflammatory Effects of Coarse and Fine Particulate Matter in Relation to Chemical and Biological Constituents*, 195 Tox. & Applied Pharm. 1 (2004) (abstract).

<sup>34</sup> *Id.* at 1.

<sup>35</sup> E.g., Joleen M. Soukup & Susanne Becker, *Human Alveolar Macrophage Responses to Air Pollution Particulates are Associated with Insoluble Components of Coarse Material, Including Particulate Endotoxin*, 171 Toxic. & Applied Pharma. 20 (2001).

<sup>36</sup> E.g., Christian Monn & Susanne Becker, *Cytotoxicity and Induction of Proinflammatory Cytokines from Human Monocytes Exposed to Fine (PM<sub>2.5</sub>) and Coarse Particles (PM<sub>10-2.5</sub>) in Outdoor and Indoor Air*, 155 Toxic. & Applied Pharma. 245 (1999).

The Criteria Document recognizes the existence of studies demonstrating associations between inhaled particles containing endotoxin and observed health effects,<sup>37</sup> as well as at least one study in which the coarse fraction of sampled particulates, and not the fine fraction, was found to be responsible for inflammatory responses characteristic of endotoxin activity.<sup>38</sup>

In addition to endotoxins, a number of metals found in airborne coarse particulates have been shown by laboratory studies to cause adverse health effects. These include the so-called “transition metals” iron,<sup>39</sup> copper,<sup>40</sup> nickel,<sup>41</sup> cobalt,<sup>42</sup> vanadium,<sup>43</sup> and cadmium,<sup>44</sup> among others. Emissions of particulates containing a number of these metals are associated with the mining and smelting of ores,<sup>45</sup> and the manufacturing of steel and other metal alloys.<sup>46</sup> Certain airborne metal dusts are particularly associated with the combustion of fossil fuels—principally iron (coal)<sup>47</sup> and vanadium (fuel oil).<sup>48</sup> In the construction industry, airborne cadmium particulates are a known health risk.<sup>49</sup>

The Criteria Document notes that observed responses among human and animal test subjects exposed to filter extracts of air samples collected contemporaneously with the Utah Valley epidemiological studies showed a “remarkable” degree of coherence with expectations derived from those studies, based on the metal content of those extracts.<sup>50</sup> According to the CD, “Studies comparing human and rat exposures to both high and low metal content PM collected near [the] steel plant, showed convincingly that the metal content of the PM, and not the mass, was a major determinant of the toxicity of the PM.”<sup>51</sup>

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<sup>37</sup> 2 CD, *supra* note 8, § 7.3.6, at 7-82; § 7B.2.3, at 7B-25.

<sup>38</sup> *Id.* at 7-83 (citing Monn & Becker, *supra* note 36).

<sup>39</sup> *E.g.*, P.S. Gilmour et al., *Adverse Effects of PM10 Particles: Involvement of Iron in Generation of Hydroxyl Radical*, 53 *Occup. & Environ. Med.* 817 (1996); A. Dusseldorp et al., *Associations of PM<sub>10</sub> and airborne iron with respiratory health of adults living near a steel factory*, 152 *Am. J. Respir. Crit. Care Med.* 1932 (1995).

<sup>40</sup> *E.g.*, Thomas Kennedy et al., *Copper-dependent Inflammation and Nuclear Factor-κB Activation by Particulate Air Pollution*, 19 *Am. J. Respir. Cell Mol. Biol.* 366 (1998).

<sup>41</sup> *E.g.*, M. Goebler et al., *Activation of Nuclear Factor-κB and Gene Expression in Human Endothelial Cells by the Common Haptens Nickel and Cobalt*, 155 *J. Immunology* 2459 (1995).

<sup>42</sup> *Id.*

<sup>43</sup> *E.g.*, Jacqueline D. Carter et al., *Cytokine Production by Human Airway Epithelial Cells after Exposure to an Air Pollution Particle is Metal-Dependent*, 146 *Toxic. & Applied Pharma.* 180 (1997).

<sup>44</sup> *E.g.*, Occupational Safety & Health Administration [OSHA], *Occupational Exposure to Cadmium in the Construction Industry* (1996), available at <http://www.osha.gov/doc/outreachtraining/htmlfiles/cadmium.html> [hereinafter OSHA on Cadmium].

<sup>45</sup> *E.g.*, Kennedy et al., *supra* note 40, at 376 (citing M.E. Mattson & T.L. Giodotti, *Health Risks Associated with Residence near a Primary Copper Smelter: A Preliminary Report*, 1980 *Am. J. Ind. Med.* 365).

<sup>46</sup> *See generally*, *Understanding the Health Effects of Components of the Particulate Matter Mix: Progress and Next Steps*, in *HEI Perspectives*, Apr. 2002, at 11, available at <http://www.healtheffects.org/Pubs/Perspectives-2.pdf>.

<sup>47</sup> *E.g.*, A.E. Aust et al., *Particle Characteristics Responsible for Effects on Human Lung Epithelial Cells*, 110 *Res. Rep. Health Effects Inst.* 1 (2002).

<sup>48</sup> *E.g.*, Carter et al., *supra* note 43.

<sup>49</sup> *E.g.*, OSHA on Cadmium, *supra* note 44.

<sup>50</sup> 2 CD, *supra* note 8, § 7.10.2.3, at 7-217, -18.

<sup>51</sup> *Id.* at 7-218 (citations omitted) (citing A.J. Ghio & R.B. Devlin, *Inflammatory Lung Injury after Bronchial Instillation of Air Pollution Particles*, 164 *Am. J. Respir. Crit. Care Med.* 704 (2000); J.A. Dye et al., *Acute Pulmonary Toxicity of Particulate Matter Filter Extracts in Rats: Coherence with Epidemiological Studies in Utah Valley Residents*, 109 *Environ. Health Perspect.* 395 (2001) (supp.))

Resuspended road dusts are also known to contain a variety of metals. According to the CD:

The chemical composition of paved road dust consists of a complex mixture of PM from a wide variety of sources.... Automobile contributions arose from: exhaust emissions enriched in Pb; Fe as rust; tire wear particles enriched in Zn; brake linings enriched in Cr, Ba, and Mn; and cement particles derived from roadways by abrasion. In addition to organic compounds from combustion and secondary sources, road dust also contains biological material such as pollen and fungal spores.<sup>52</sup>

Arsenic is another toxic metal that is commonly associated with agricultural and construction wastes. Arsenic is a common constituent of pesticides; and sawdust derived from pressure-treated wood has been shown to contain hazardous levels of arsenic.<sup>53</sup>

Silica particles have also long been associated with respiratory health effects, as revealed by occupational studies of ‘black lung’, or silicosis, among miners, foundry workers and others who process mineral ores. According to the Mine Safety and Health Administration:

Silicosis is a disabling, nonreversible, and sometimes fatal lung disease caused by overexposure to respirable crystalline silica. . . . Silica is the second most common mineral in the earth’s crust and is a major component of sand, rock, and mineral ores. . . . Nearly all metal and nonmetal miners may be exposed to silica dust.<sup>54</sup>

The National Institute for Occupational Safety and Health (“NIOSH”) has published information and materials designed to warn mineworkers about the dangers of inhaling silica dusts and inform them of precautions they should take. According to one such publication:

[T]he generation of respirable silica dust during mining is [a] primary concern. Inhalation of excessive levels of silica dust can lead to silicosis, [a] disabling and potentially fatal lung disease. With continued improvements in production, the potential to generate larger quantities of respirable coal and silica dust increases. As a result, new or improved control technologies are needed to protect the health of mine workers.<sup>55</sup>

The proliferation of open-pit surface mining and mountaintop-removal coal mining, and of similarly widespread and destructive methods of mineral extraction, exacerbates these risks and extends them to members of the general public. A study conducted by the National Institute of Occupational Safety and Health (NIOSH) measured average respirable particle concentrations

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<sup>52</sup> 1 CD, *supra* note 8, app. 3D, at 3D-3.

<sup>53</sup> Agency for Toxic Substances and Disease Registry, Toxicological Profile for Arsenic (draft for public comment), Sept. 2005, available at <http://www.atsdr.cdc.gov/toxprofiles/tps2.html> .

<sup>54</sup> Mine Safety & Health Administration, *Silica Exposures of Metal and Non-Metal Miners: Health Hazard Information Card*, available at <http://www.msha.gov/S&HINFO/BLUNG/SILIMNM.PDF> .

<sup>55</sup> NIOSH Mining and Health Topic: Dusts, at <http://www.cdc.gov/niosh/mining/topics/dusts/> .

of 7690  $\mu\text{g}/\text{m}^3$  outside a surface mining drill,<sup>56</sup> of which 12% consisted of silica.<sup>57</sup> Obviously, explosive blasting at surface mines would generate even higher concentrations of particulates.

Another NIOSH study conducted at two Portland cement limestone quarries measured concentrations of respirable particulates containing quartz silica at the perimeter of the facilities, near residential areas, in the range of 30 to 190  $\mu\text{g}/\text{m}^3$ .<sup>58</sup> One in-town monitor located a half-mile from the facility measured a one-month average concentration of respirable dust of 90  $\mu\text{g}/\text{m}^3$ .<sup>59</sup> Concentrations of respirable dusts within the facilities ranged as high as 25,530  $\mu\text{g}/\text{m}^3$ .<sup>60</sup>

Construction activities are also known to generate respirable silica dusts. As one study noted: “Dust is omnipresent at construction sites. Exposure to dust can occur during almost all activities, from excavation for the foundations up until the final sweeping before the completion of the building. . . . [T]his dust can contain a considerable amount of silica.”<sup>61</sup> Another study measured occupational exposures among a number of trades within the construction industry, and observed that some job-types carried probabilities of overexposure to occupational silica-dust limits as high as 100%.<sup>62</sup> After observing a large number of such overexposure situations within the industry, the study concluded that “such grossly unacceptable exposures across many US construction sites portend a serious health threat requiring determined action. . . . Since construction work involves diverse, primarily outdoor sites and is largely intermittent in nature, this presents an important challenge for the industry.”<sup>63</sup>

Yet another study of silica exposures in the construction industry measured dust concentrations in the vicinity of 113 dust-producing construction tasks. The study found that “[c]onstruction masons and laborers are frequently overexposed to silica,” and that concrete demolition and surface grinding were among the tasks with the highest exposures.<sup>64</sup>

The Criteria Document recognizes the existence of laboratory studies confirming the toxicity of silica particles,<sup>65</sup> and identifies at least one publication—the California farmworker

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<sup>56</sup> A.B. Cecala et al., *Reducing Enclosed Cab Drill Operator’s Respirable Dust Exposure at Surface Coal Operation with a Retrofitted Filtration and Pressurization System*, tbl. 1 at 3, available at <http://www.cdc.gov/niosh/mining/pubs/pdfs/recco.pdf>

<sup>57</sup> *Id.* at 3.

<sup>58</sup> Andrew B. Cecala et al., *Respirable Dust Evaluation of Two Portland Cement Operations*, Part 2, at 3 (2000), available at <http://www.cdc.gov/niosh/mining/pubs/pdfs/rd2.pdf>.

<sup>59</sup> *Id.* Part 1, at 3, available at <http://www.cdc.gov/niosh/mining/pubs/pdfs/rd1.pdf>.

<sup>60</sup> *Id.* Part 1, at 4.

<sup>61</sup> Mieke E.G.L. Lumens & Ton Spee, *Determinants of Exposure to Respirable Quartz Dust in the Construction Industry*, 45 Ann. Occup. Hygiene 585, 585 (2001).

<sup>62</sup> S.M. Rappaport et al., *Excessive Exposure to Silica in the US Construction Industry*, 47 Ann. Occup. Hygiene 111, 118 & tbl. 5 (2003).

<sup>63</sup> *Id.* at 120.

<sup>64</sup> M.E. Flanagan et al., *Silica Dust Exposures During Selected Construction Activities*, 64 AIHA J. 319 (2003) (abstract).

<sup>65</sup> *E.g.*, 2 CD, *supra* note 8, § 6.3.4.4., at 6-60 (citing I.Y.R. Adamson & H. Preidontis, *Silica Deposition in the Lung During Epithelial Injury Potentiates Fibrosis and Increases Particle Translocation to Lymph Nodes*, 24 Exp. Lung Res. 293 (1998)).

study discussed earlier<sup>66</sup>—that identifies the potential for health risks resulting from occupational exposure to agricultural silica.<sup>67</sup> In one operation observed by the California study—the machine harvesting of tree crops (nuts)—mean respirable dust levels of 4,470 µg/m<sup>3</sup> were measured, of which 23% (1,028 µg/m<sup>3</sup>) was comprised of respirable crystalline silica.<sup>68</sup>

In spite of the clearly documented dangers of silica particles, the “Toxicology” section of the Criteria Document completely evades any discussion of the hazards of exposures to silica as a result of ambient coarse-particle concentrations. According to that document, “Individual particle species with high inherent toxicity that are of concern mostly because of occupational exposure (e.g., silica) that are discussed in detail in other documents and reports (e.g., U.S. Environmental Protection Agency, 1996b; Gift and Faust, 1997 for silica) are ... not assessed in detail in this chapter.”<sup>69</sup>

Radioactive materials are another component of respirable dusts that are byproducts of various mining, combustion and agricultural activities. Fly ash produced from the combustion of coal contains large quantities of radioactive uranium and thorium; a study conducted by Oak Ridge National Laboratory’s Alex Gabbard estimated that coal-burning power plants released 801 tons of uranium and 1971 tons of thorium in the year 1982, of which 99% was retained in captured fly ash.<sup>70</sup> About two-thirds of all coal utility-generated fly ash, or about 70 million tons per year, is disposed in landfills.<sup>71</sup> A particle-size distribution study showed that for one type of coal-fired power plant, about 35% of fly ash particles were in the PM<sub>10-2.5</sub> fraction.<sup>72</sup> With regard to estimated radiation exposures from the ash, a U.S. Geological Survey review of pre-1985 literature points out that individuals living within 1 kilometer of a coal-burning power plant receive as much as 5% more radiation than they do from natural background, in part because of inhalation of radioactive fly ash.<sup>73</sup>

Various mining activities also produce radioactive tailings that have historically been routinely dumped on open land. For example, radioactive uranium mine- and mill-tailings sites containing vast quantities of radioactive radium and thorium can be found in numerous arid regions of the American West.<sup>74</sup> A study commissioned by EPA estimated that in 1993, there

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<sup>66</sup> See *supra* note 30 and accompanying text.

<sup>67</sup> *E.g.*, 2 CD, *supra* note 8, tbl. 5-5, at 5-39 (citing Mark J. Nieuwenhuijsen et al., *supra* note 30).

<sup>68</sup> Nieuwenhuijsen et al., *supra* note 30, tbls. 4 & 6, at 39, 40.

<sup>69</sup> 2 CD, *supra* note 8 § 7.1.2, at 7-6.

<sup>70</sup> Alex Gabbard, Oak Ridge National Laboratory, *Coal Combustion: Nuclear Resource or Danger*, available at <http://www.ornl.gov/info/ornlreview/rev26-34/text/colmain.html>

<sup>71</sup> Department of Energy, Coal Utilization Byproduct Research, available at [http://www.fossil.energy.gov/programs/powersystems/pollutioncontrols/overview\\_coalbyproducts.html](http://www.fossil.energy.gov/programs/powersystems/pollutioncontrols/overview_coalbyproducts.html)

<sup>72</sup> Francois Botha, Illinois Clean Coal Institute, *Overview of the Fluidized Bed Combustion Process and Material, Particle Size Distribution Curve for FBC Fly Ash*, at 3 (200?), available at <http://www.mcrcc.osmre.gov/PDF/Forums/CCB5/1.1.pdf>

<sup>73</sup> U.S. Geological Survey, *Radioactive Elements in Coal and Fly Ash: Abundance, Forms, and Environmental Significance* [Fact Sheet No. FS-163-97], available at <http://www.aaa-usa.org/PDF/FS-163-97.pdf>.

<sup>74</sup> See Nuclear Regulatory Commission, *Radioactive Waste: Production, Storage and Disposal*, Pub. No. NUREG BR-0216, Rev. 2, available at [http://www.nrc.gov/reading-rm/doc-collections/nuregs/brochures/br0216/#mill\\_tailings](http://www.nrc.gov/reading-rm/doc-collections/nuregs/brochures/br0216/#mill_tailings).

were 3.1 billion metric tons of unreclaimed uranium-mining overburden in the U.S.,<sup>75</sup> with concentrations of radioactive radium-226 ranging from about 3 picoCuries per gram (pCi/g) to several hundred pCi/g.<sup>76</sup> Normal soil concentrations of radium-226 are about 1 pCi/g.<sup>77</sup>

Phosphate fertilizer also contains appreciable quantities of radioactive elements. The U.S. uses about 7 million metric tons of phosphate fertilizer annually.<sup>78</sup> Phosphate fertilizers contain average concentrations of 20 to 60 pCi/g of uranium (compared to typical soil concentrations of about 0.7 pCi/g)<sup>79</sup>, and 5 to 20 pCi/g of radium.<sup>80</sup> An estimated additional 221,000 metric tons of phosphogypsum—a waste product generated by the phosphate production process, with typical radium-226 concentrations in the range of 11 to 35 pCi/g—are used each year in place of processed fertilizer.<sup>81</sup>

Large quantities of radioactive phosphate slag are also extensively used in the construction industry. In 1993, it was estimated that of the 1.6 million metric tons of phosphate slag produced each year, roughly 40%, or 640,000 metric tons, were used to build roads and building blocks for residential and other buildings.<sup>82</sup> High radiation levels measured in homes and other buildings constructed with materials made from phosphate slag have caused some states to ban its use in residential structures.<sup>83</sup>

Clearly there are a number of activities that occur in non-urban regions of the U.S. that generate immense quantities of materials that are likely components of rural particulate matter. Even undisturbed soils and crustal materials contain background concentrations of harmful silica, metals, biological contaminants and radioactive materials. In contrast, there are no studies from which it could be concluded that rural coarse particulates are completely benign, or less harmful than urban coarse particles. Thus, as will be argued more fully below, EPA's attempt to exempt rural, mining and agricultural coarse particles from the rule establishing a PM NAAQS is groundless.

### **III. EPA's proposal to set air quality standards for coarse particulate matter in urban areas, but not rural ones, is driven by politics—not science.**

Considering together EPA's proposed exemptions of rural, agricultural and mining particulates from the coarse particle standard, it takes little deductive reasoning to surmise that those who would benefit most from EPA's proposed coarse particle rule would be mining and agricultural operations in arid rural regions of the American West. Based on the above review of

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<sup>75</sup> 1 Rogers & Assoc., U.S. Evtl. Protection Agency, Diffuse NORM: Waste Characterization and Preliminary Risk Assessment, Pub. No. RAE-9232/1-2, at B-1-11 (1993)

<sup>76</sup> *Id.* at B-1-15.

<sup>77</sup> *Id.* at ES-1, -2.

<sup>78</sup> See Maurice Korol & Éric Larivière, Fertilizer Pricing in Canada tbl. 3-17 (1998), available at [http://www.agr.gc.ca/spb/fiap-dpraa/publications/fertil/fertil\\_toc\\_e.php](http://www.agr.gc.ca/spb/fiap-dpraa/publications/fertil/fertil_toc_e.php).

<sup>79</sup> U. Mich., Health Physics Soc., Radioactivity in Nature, available at <http://www.umich.edu/~radinfo/introduction/natural.htm>

<sup>80</sup> 1 Rogers & Assoc., *supra* note 75, at B-3-7.

<sup>81</sup> *Id.* at B-2-24.

<sup>82</sup> *Id.* at B-2-41 to -45.

<sup>83</sup> *Id.* at B-2-19 to -22.

epidemiological, occupational and laboratory studies, it takes little more deductive power to conclude that EPA’s proposed exemption draws an arbitrary line between scientific considerations, while drawing a far brighter line between political ones.

A simple chart identifying the primary harmful components of some typical particulates generated by various industries that are relevant to the proposed standard serves to illustrate the degree to which EPA’s line drawing is scientifically arbitrary:

**Table 1: Principal Harmful PM Components vs. Industry**

	<b>Construction</b>	<b>Mining</b>	<b>Agriculture</b>	<b>Road Dust</b>	<b>Other Industrial</b>
<b>Silica</b>	X	X	X	X	X
<b>Metals</b>	X	X		X	X
<b>Radionuclides</b>	X	X	X		X
<b>Biogenics</b>			X	X	

Clearly, Table 1 reveals that insofar as the primary harmful components of PM are concerned, there is no basis for distinguishing among the principal industrial categories mentioned in the preamble to the proposed rule and supporting documents.

A similar comparison between harmful PM components and EPA’s proposed “urban/rural” distinction reveals line-drawing that is even *more* arbitrary:

**Table 2: Principal Harmful PM Components vs. Urban/Rural Distinction**

	<b>Urban</b>	<b>Rural</b>
<b>Silica</b>	X	X
<b>Metals</b>	X	X
<b>Radionuclides</b>	X	X
<b>Endotoxins</b>	X	X

The Provo example highlights the fallacy of any attempt to delineate a distinction between urban and rural coarse particles, insofar as their relative health effects are concerned. According to census reports from 1990 and 2000, Provo’s population increased to more than 100,000 sometime during the 1990s.<sup>84</sup> If EPA’s current proposal had been in effect then, at some point in the 1990s, the addition of one new member of the Provo community would have caused the coarse particles being emitted from the nearby steel mill to switch from being ‘rural’ to ‘urban’ under EPA’s definitions of those areas, and suddenly cause them to be subject to regulation. The same protection still would not be provided to Steubenville, Ohio, whose residents would have the misfortune of living in a town with a population below the magic 100,000 threshold.

<sup>84</sup> Provo City Community Development Department, Provo Demographic Profile, *available at* [http://www.provo.org/econdev.Provo\\_Profile\\_main.html](http://www.provo.org/econdev.Provo_Profile_main.html)

EPA has had to engage in strained reasoning in order to support its decision to propose to exclude mining and agricultural particulates from regulation as primary criteria pollutants under the Clean Air Act. In a classic *non sequitur*, EPA’s Staff Paper—a supporting document prepared by EPA in advance of the proposed rule—found that

under some conditions, crustal particles may become sufficiently toxic to cause human health effects. For example, resuspended crustal particles may be contaminated with toxic trace elements and other components from previously deposited fine PM, e.g., metals from smelters (Phoenix) or steel mills (Steubenville, Utah Valley), PAHs from automobile exhaust, or pesticides from agricultural lands.

***This is consistent with CASAC’s conclusion . . . that the focus of an indicator for thoracic coarse particles should be on such particles found in urban, not rural environments.***<sup>85</sup>

The preamble to the proposed rule expands the *non sequitur*, finding that “[t]he limited evidence does not support either the existence or the lack of causative associations for community exposures to thoracic coarse particles from agricultural or mining industries.”<sup>86</sup>

The Criteria Document evades any discussion of the potential for non-occupational exposures to endotoxins in the agricultural context, or of the difficulty in distinguishing between occupational and community exposures in that context in the first instance. According to the CD:

[Studies have] noted that animal feces and plant materials contaminated with bacteria contribute most to organic dust-related endotoxin exposure. Although there is strong evidence that inhaled endotoxin plays a major role in the toxic effects of bioaerosols encountered in the work place, it is not clear as to what extent typical ambient concentrations of endotoxin are sufficient to produce toxic pulmonary or systemic effects in healthy or compromised individuals.<sup>87</sup>

Rather than address this issue and recognize the simple fact that in agricultural regions, the residence and the workplace are often one-and-the-same, and assume that “ambient” exposures in this context pose the same risks as workplace ones, EPA simply states that the answer “is not clear,” and otherwise ignores the issue.

The preamble to the proposed rule similarly flatly dismisses any concerns regarding the toxic character of silica particles in mining particulates:

[I]n the last review, EPA considered health evidence related to long-term silica exposures from mining activities, but found that there was a lack of evidence that such emissions

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<sup>85</sup> Staff Paper, *supra* note 11, at 5-57 (emphasis added) (citing 2 CD, *supra* note 8, at 8-344.)

<sup>86</sup> Proposed Rule, *supra* note 4, at 2667.

<sup>87</sup> 2 CD, *supra* note 8, § 7.3.6., at 7-82 (citations omitted).

contribute to effects linked with ambient PM exposures. Similarly in this review, there is an absence of evidence related to such community exposures.<sup>88</sup>

Perhaps nowhere is EPA's determination to protect mining interests over the health of rural citizens more evident than in the observations of the CASAC's own chair, Dr. Rogene Henderson, as reported by National Public Radio's Jeff Young in a recent broadcast of the show Living on Earth:

YOUNG: EPA's proposal also would not regulate or even monitor coarse particle dust in rural areas. It would exempt from regulation dusty activities like mining and agriculture. Henderson says the proposal makes it sound as if that idea came from her committee.

HENDERSON: The CASAC committee did not say that. We recommended monitoring in both urban and rural areas and *at no time did we ever mention the mining industry.*<sup>89</sup>

With regard to biological agricultural particle wastes like endotoxins, EPA used similiary strained reasoning, concluding that

[w]hile crustal and organic dusts generated from agricultural activity can include a variety of biological materials, and some occupational studies discussed in the Criteria Document report effects at occupational exposure levels, such studies do not provide relevant evidence for effects at much lower levels of community exposures. Further, it is unlikely that such sources contribute to the effects that have been observed in the recent urban epidemiologic studies.<sup>90</sup>

Clearly, EPA has concocted an arbitrary 'urban/rural' distinction that doesn't exist in the scientific world in an attempt to justify giving an exemption from the coarse-particle NAAQS to the mining and agribusiness industries. Using that contrived distinction, the agency seeks to advance its political agenda that would protect the profits of mining and agribusiness industries rather than the health of ordinary people.

#### IV. Conclusion

In the review of national air quality standards under consideration, EPA has identified health risks for a pollutant—ambient coarse particles—that is ubiquitous. EPA identified those risks based on epidemiological studies conducted mostly in cities, but also in rural areas as well. EPA could easily have “filled the gaps” by giving credit and attention to numerous occupational and laboratory studies showing that the same species of particulates, routes of exposure and toxicological mechanisms apply to coarse particle pollution *regardless* where it is found.

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<sup>88</sup> Proposed Rule, *supra* note 4, at 2666-67.

<sup>89</sup> National Public Radio, *Particulate Particulars*, on Living on Earth, Feb. 10, 2006 (emphasis added), available at <http://www.loe.org/shows/shows.htm?programID=06-P13-00006#feature1>

<sup>90</sup> *Id.* at 2666.

Instead, EPA chose politics over science, and decided to attack those rural studies that have found associations between coarse particles and health risks, and to ignore those occupational and laboratory studies that collided with its agenda.

The very name of the air quality standard being proposed in EPA's proposed rule—the *National Ambient Air Quality Standard*—shows the illegality of EPA's attempt to set a *local* standard for coarse particulate matter, applicable only to urban areas and not to mining or agribusiness pollutants. There is no statutory authority under the Act, and no other legal precedent, for setting such a standard.

The National Academy of Sciences has articulated the so-called “Precautionary Principle” in this context: “Even great uncertainty does not imply that action to promote or protect public health should be delayed. . . . The potential for improving decisions through research must be balanced against the public health costs incurred because of a delay in the implementation of controls. Complete certainty is an unattainable ideal.”<sup>91</sup> EPA must follow this sound advice.

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<sup>91</sup> Committee on Estimating the Health-Risk-Reduction, National Research Council, *Benefits of Proposed Air Pollution Regulations: Estimating the Public Health Benefits of Proposed Air Pollution Regulations*, No. ISBN: 0-309-08609-4, at 126-27 (2002).