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National Association of County and City Health Officials •  
Trust for America's Health

August 31, 2012

Lisa P. Jackson, Administrator  
U.S. Environmental Protection Agency  
1200 Pennsylvania Avenue, NW  
Washington, DC 20460

RE: Docket EPA-HQ-OAR-2007-0492: Comments on the Proposed National Ambient Air Quality Standards for Particulate Matter

Dear Administrator Jackson:

As national public health and medical organizations, we call on the U.S. Environmental Protection Agency to significantly strengthen the particulate matter National Ambient Air Quality Standards (NAAQS). Our organizations are keenly aware of the public health and medical threats from particulate matter. Many of our members have conducted the research or treated the patients and know first-hand the dangers from these microscopic particles. We urge the U.S. EPA to strengthen the annual average standard for fine particulate matter (PM<sub>2.5</sub>) to 11 µg/m<sup>3</sup> and the 24-hour standard to 25 µg/m<sup>3</sup>.

Ample scientific evidence supports adopting tighter standards to protect the health of people who are most susceptible to the serious health effects of these pollutants. More than 10,000 peer-reviewed scientific studies have been published since 1997 when EPA adopted the current annual standard. These studies validate and extend earlier epidemiologic research linking both acute and chronic fine particle pollution with serious morbidity and mortality. The newer research has also expanded our understanding of the range of health outcomes associated with PM, and has identified adverse respiratory and cardiovascular health effects at lower exposure levels than previously reported. As discussed and interpreted in the EPA's 2009 *Integrated Science Assessment for Particulate Matter (ISA)*, the new evidence reinforces already strong existing studies and supports the conclusion that PM<sub>2.5</sub> is causally associated with numerous adverse health effects in humans, at exposure

levels far below the current standard. Such a conclusion demands prompt action to protect human health.

Upon completion of a thorough review of the current research, the Clean Air Scientific Advisory Committee and EPA's own staff scientists concluded that PM<sub>2.5</sub> causes or is likely the cause of premature deaths, and significant health problems, such as increased hospital admissions and emergency department visits for cardiovascular and respiratory problems, including heart attacks and strokes. PM<sub>2.5</sub> also is linked to the development of chronic respiratory disease, reproductive and developmental harm, as well as cancer, mutagenicity and genotoxicity.

### **Particulate matter causes cardiovascular harm**

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The American Heart Association updated the AHA Scientific Statement in May 2010 to reflect this growing evidence. An independent team of scientists reviewed research from 2004 through March 2009 for this statement, research all well within the timeframe for this review. They concluded:

Exposure to PM <2.5 µm in diameter (PM<sub>2.5</sub>) over a few hours to weeks can trigger cardiovascular disease-related mortality and nonfatal events; longer-term exposure (eg, a few years) increases the risk for cardiovascular mortality to an even greater extent than exposures over a few days and reduces life expectancy within more highly exposed segments of the population by several months to a few years; reductions in PM levels are associated with decreases in cardiovascular mortality within a time frame as short as a few years; and many credible pathological mechanisms have been elucidated that lend biological plausibility to these findings. It is the opinion of the writing group that the overall evidence is consistent with a causal relationship between PM<sub>2.5</sub> exposure and cardiovascular morbidity and mortality.<sup>1</sup>

The evidence points to increased risk for cardiovascular harm in women.<sup>2</sup> In the Women's Health Initiative study, Miller et al. found that each increase of 10 µg/m<sup>3</sup> in PM<sub>2.5</sub> was associated with a 24 percent increase in risk of cardiovascular events including myocardial infarction, as well as a 35 percent increase in the risk of cerebrovascular events, including stroke.<sup>3</sup> Results from the Adventist Health Study of Smog cohort<sup>4</sup> and the Nurses Health Study<sup>5</sup> found similar higher risks for women from PM<sub>2.5</sub>.

## **Particulate matter causes respiratory harm**

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The evidence in this current review shows the breadth of harm PM poses to respiratory health. As Rom and Samet (2006) explained in an editorial, the evidence shows that “Small Particles Have Big Effects”:

PM has now been linked to a broad range of adverse health effects, both respiratory and cardiovascular, in epidemiologic and toxicologic research. The diversity of effects may reflect the complexity of airborne PM, which is made up of a rich mixture of primary and secondary particles.<sup>6</sup>

As noted in the ISA, increasing evidence strengthens the association with PM<sub>2.5</sub> and hospital admission and emergency department visits for asthma, COPD and respiratory infection.<sup>7</sup> In addition, added evidence confirms the need for greater prevention of pediatric pulmonary harm from particulate matter.

Increasing evidence suggests that PM<sub>2.5</sub> causes lung cancer mortality. The extended follow-up to the Harvard Six Cities Study by Laden et al. (2006)<sup>8</sup> and two follow-up studies of the American Cancer Society cohort study by Jerrett et al. (2005)<sup>9</sup> and Krewski et al. (2009)<sup>10</sup> showed positive association with lung cancer mortality for PM<sub>2.5</sub>. Given the long latency period for cancer, these findings are significant to the assessment of long-term health effects to be prevented by tighter fine particle standards.

Since the previous review, several new studies have documented that cleaner air is healthier. When Pope, Ezzati and Dockery (2009) followed up on the impact of reduced PM<sub>2.5</sub> in 211 counties in 51 metropolitan areas in the U.S., they found that a decrease of 10 µg/m<sup>3</sup> was associated with added life expectancy of five months on average.<sup>11</sup> Downs et al. (2007) found that lower levels of PM<sub>10</sub> over 11 years in Switzerland had reduced the severity of the decline in age-related lung function in nearly 9,651 adults.<sup>12</sup> In a follow-up to that study, Schindler et al. (2009) found that the drop in PM<sub>10</sub> was also associated with fewer adults suffering chronic cough, wheezing and breathlessness and other respiratory symptoms.<sup>13</sup>

## **Risks to children**

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Children face substantial risks from air pollution—especially particulate matter—as growing evidence confirms. As the American Academy of Pediatrics concluded in 2004 and reaffirmed in 2009:

Children and infants are among the most susceptible to many of the air pollutants. In addition to associations between air pollution and respiratory symptoms, asthma

exacerbations, and asthma hospitalizations, recent studies have found links between air pollution and preterm birth, infant mortality, deficits in lung growth, and possibly, development of asthma.<sup>14</sup>

Strong studies confirm that the standards fail to protect the health of children, from the period of fetal development through adolescence, and provide support for more protective standards than EPA has proposed. Research available to EPA in this review documents those risks. In addition, newer studies add weight to the need for greater protection. We note that many of these endpoints have a stronger association with PM<sub>10</sub> than PM<sub>2.5</sub> including in cases where data on both size particles were available. These studies underline the importance for EPA to review the decision to leave the PM<sub>10</sub> standard unchanged.

### **Prenatal and Neonatal Risks**

Increasing evidence indicates that the risk to children begins before they are born. Not surprisingly, the respiratory system is developing from early in embryonic life, starting as early as 3 weeks and continuing after parturition into adolescence.<sup>15</sup> During this prolonged period of pre- and post-neonatal development, the lungs and other developing organs and systems may face higher risk in their immaturity stemming from cell development and metabolic changes.<sup>16</sup> These developing systems, as well as the exposure of the mother, lead to several possible explanations for these risks, as discussed in the ISA.<sup>17</sup>

Newer research has further analyzed the risk for birth defects and low birth weight associated with particle pollution. Although Šrám et al. (2005) found uncertain evidence for birth defects, evidence is growing that some anomalies may be linked. More recently, a 2011 review by Vrijheid et al found PM<sub>10</sub> linked to an increased risk of atrial septal defects.<sup>18</sup> The preliminary results of an initial, though not full, meta-analysis of research by the International Collaboration on Air Pollution and Pregnancy Outcomes found an increase of PM<sub>10</sub> of 10 µg/m<sup>3</sup> associated with lower birth weight of between 2 and 20 grams.<sup>19</sup>

### **Mortality risks increased during postneonatal period**

Strong evidence warns that particulate matter exposure, especially PM<sub>10</sub>, increases the risk of death in infants. Glinianaia et al. (2004) in their review of research into infant deaths from particulate matter, found the strongest associations for post-neonatal mortality from respiratory causes and sudden infant death syndrome.<sup>20</sup> In a review of research on pregnancy outcomes, Šrám et al (2005) concluded that the evidence was "sufficient to infer a causal relationship between particulate air pollution and respiratory deaths in the post-neonatal period."<sup>21</sup> Looking at infant deaths in Southern California, Ritz et al (2006) found risk of post-neonatal death

increasing by 7 to 12 percent or greater for each increase of  $10 \mu\text{g}/\text{m}^3$  in  $\text{PM}_{10}$  through the first year of life. The risk of death *doubled* after these children had been exposed to at least six months of breathing elevated average levels of  $\text{PM}_{10}$ .<sup>22</sup> Woodruff et al. (2008) found added evidence of increased risk of postneonatal deaths from respiratory-related causes with an increase of  $\text{PM}_{10}$  of  $10 \mu\text{g}/\text{m}^3$ .<sup>23</sup>

### **Childhood and Adolescent Susceptibility to Particulate Matter Pollution**

As with much of their anatomy, the largest portion of a child's lungs will grow after he or she is born. Eighty percent of the air sacs found in the lungs, called alveoli, develop after birth. The lungs and alveoli do not fully develop until ages 18 to 22, when a child physically reaches adulthood.<sup>24 25</sup> In addition, the immune system is still developing in young bodies.<sup>26</sup> Young children have an incomplete respiratory epithelium (a thin protective layer of tissue that lines the respiratory tract) so the same pollution exposure may cause more damage to them.<sup>27</sup> Children contract more respiratory infections than adults, which also seems to increase their susceptibility to air pollution.<sup>28</sup>

Furthermore, children's size and behavior also affect their vulnerability. A resting infant takes in roughly twice the amount of air, proportionate to their body size, as an adult.<sup>29</sup> Children are outside for longer periods and are usually more active when outdoors. Consequently, they inhale more polluted outdoor air than adults typically do.<sup>30</sup>

### **Particulate Matter Associated with Increased Risk of Underdeveloped Lungs**

This period of growth in respiratory capacity means that pollution could impact lung development. Gauderman et al., in the Southern California Children's Health study, looked at the long-term effects of particle pollution on teenagers. Tracking 1,759 children between ages 10 and 18, researchers found that those who grew up in more polluted areas faced an increased risk of having underdeveloped lungs. The average drop in lung function was 20 percent below what was expected for the child's age, similar to the impact of growing up in a home with parents who smoked.<sup>31</sup> Because underdeveloped lungs may never grow or recover to their full capacity, this finding indicates potentially permanent damage with long-term risks.

### **Children suffer significant respiratory effects**

As noted in the ISA, many studies link exposure to PM to new or worsening respiratory symptoms in young children. For example, Peel et al., (2005) found  $\text{PM}_{10}$  associated with increased risk of upper respiratory infection for infants and children in an investigation into emergency department treatment in the extensive

Study of Particles and Health in Atlanta (SOPHIA) investigation.<sup>32</sup> Similarly, Host et al. (2007) found a significant association for childhood respiratory diseases for children up to age 14 with increased coarse particle fractions (PM<sub>10-2.5</sub>), although not for PM<sub>2.5</sub>, in their study in six French cities.<sup>33</sup> Pierse et al., (2006) studied 4,400 children aged 1 to 5 years and found exposure to PM<sub>10</sub> associated with new and returning cough not due to colds.<sup>34</sup>

In some studies the PM<sub>2.5</sub> and PM<sub>10</sub> exposures have had different effects or no effect on children examined, a question that researchers looking at adult exposures have explored and some opponents have cited as negating the entire body of evidence. New research into specific components of PM may further explain the challenges in assessing the impact of such complex pollutants. For example, in the investigation by Patel, et al. (2009) in Dominican and African American New York City neighborhoods, children up to 2 years of age suffered cough and wheezing associated with several PM<sub>2.5</sub> components in the mix, but not PM<sub>2.5</sub> as a whole. The association with nickel and vanadium and (in some seasons) elemental carbon held up even after accounting for potential confounders such as smoking in the home. These components were recognized as common pollutants from urban heating oil combustion and traffic pollution.<sup>35</sup>

Looking at the impact of cleaner, healthier air in the large study of 9 communities in Switzerland, Bayer-Oglesby et al. (2005) found strong evidence that the reduced PM<sub>10</sub> levels over an 11-year period benefited the 9,591 children tracked. The children suffered less from chronic cough, bronchitis, colds and other respiratory symptoms, as well as conjunctivitis, even after controlling for indoor air and other confounders.<sup>36</sup>

### **Increased Risk of Asthma and Allergies in Children**

Beyond the question of worsening respiratory symptoms, some studies provide evidence that PM can increase the risk of new onset asthma or allergies. Nordling et al. (2008) found that in a study of 4,089 Swedish children up to age 4, traffic-related PM<sub>10</sub> was linked to increased sensitization to inhaled allergens, such as pollen.<sup>37</sup>

Clearly, research has not yet resolved the question of whether air pollution—especially particulate matter--can cause new onset asthma. However, the research available in the ISA and published since then continues to add to the likelihood that the answer to that question is yes. Islam et al., (2007) found that children in the California Children's Health Study who were more likely to have developed asthma were those who lived in areas with higher PM<sub>2.5</sub> where their lung function was lowered. The higher PM<sub>2.5</sub> levels reduced the "protective effect of better lung

function.”<sup>38</sup> Morgenstern et al. (2008) found German six-year olds had an increased risk for asthmatic bronchitis or asthma diagnosis with each  $1.0 \mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$ .<sup>39</sup> Wilhelm et al. (2009) found that children living in some Los Angeles neighborhoods with higher levels of  $\text{PM}_{10}$  were more likely to have doctor-diagnosed asthma, though not asthma attacks in the previous year.<sup>40</sup> Clark et al. (2010) assessed 37,401 children born in British Columbia in 1999 and 2000, finding a significantly increased risk of asthma diagnosis associated with higher exposures to  $\text{PM}_{10}$ , though not to  $\text{PM}_{2.5}$ .<sup>41</sup>

Newer studies show strong evidence as well. Penard-Morand et al. (2010) found children who had lived in one of two French communities for at least three years in areas with higher modeled  $\text{PM}_{10}$  had significant higher risk of suffering from asthma, eczema, allergic rhinitis and sensitivity to pollens. Children who had lived there throughout their lives had a strong association with lifetime asthma.<sup>42</sup> Gehrig et al. (2010) also found positive association for  $\text{PM}_{2.5}$  with a significant increase in incidence of asthma, prevalence of asthma and symptoms in a follow up with their study of a cohort of children at age 8 in the Netherlands. As with the French study, these Dutch children who lived in the same place for their entire lives had an even stronger likelihood of asthma.<sup>43</sup>

### **Worsening Pediatric Asthma**

As noted in the ISA, many studies have linked PM with worsened asthma. In addition, newer studies have also found that association, particularly with  $\text{PM}_{10}$ . In their extensive review and meta-analysis published in 2010, Weinmayr et al. (2010) concluded “clear evidence” that  $\text{PM}_{10}$  triggered asthma episodes in children, though they did not include an analysis of the impact of  $\text{PM}_{2.5}$  due to less extensive data. This review examined 36 studies from around the world published between 1992 and 2006, including 14 from Europe.<sup>44</sup>

Lung function in children with asthma decreased, even when breathing very low concentrations of  $\text{PM}_{2.5}$  over the course of a day, according to the findings of a study of urban air pollution in Windsor, Ontario, by Dales et al (2009). Monitoring 182 elementary school children for 28 days, they found that the lung function declined during the day, even though the daily mean was  $7.8 \mu\text{g}/\text{m}^3$ , well below both the current U.S.  $\text{PM}_{2.5}$  NAAQS, and adjusted for potential confounders.<sup>45</sup>

In a newer follow up to the SOPHIA study, Strickland et al. (2010) found that data on over 10 million Atlanta emergency department visits gave it strong statistical power to identify impacts of  $\text{PM}_{2.5}$  and its components on pediatric asthma. They found that even at “relatively low levels” a strong association with emergency department visits for asthma with  $\text{PM}_{2.5}$ ,  $\text{PM}_{10}$  and  $\text{PM}_{10-2.5}$ . The mean 24-hour  $\text{PM}_{2.5}$

was  $16.4 \mu\text{g}/\text{m}^3$  with a 7.4 standard deviation. That low level of  $\text{PM}_{2.5}$  reinforces “the need for the continued evaluation” of the NAAQS to “ensure that the standards are sufficient to protect susceptible individuals.”<sup>46</sup>

### **Disparities in the Impact of Air Pollution Calls for More Protection**

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The burden of air pollution is not evenly shared. Poorer people and some racial and ethnic groups are among those who often face higher exposure to pollutants and who may experience greater responses to such pollution. The nation has long recognized the differences in exposure and impact. As the 1994 Executive Order 12898 required, the EPA must:

“make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations in the United States.”<sup>47</sup>

EPA has pledged to work to reduce these disparities, most recently, in the Plan EJ 2014 issued in September 2011 that set as the Agency’s goal to “protect the environment and health in overburdened communities.”<sup>48</sup> The particulate matter standards are a powerful opportunity to advance those goals. The evidence is strong and growing that particulate matter disproportionately exposes and harms many groups.

In the ISA, EPA concluded appropriately that people with low socioeconomic status should be included in the groups facing higher vulnerability to particulate matter. While the evidence did not clearly indicate higher susceptibility based solely on racial or ethnic groups, some evidence warns that some groups may face higher risks.

Many studies have looked at differences in the impact of air pollution on premature death. Results have varied widely, particularly for effects between racial groups. Some studies have found no differences among races,<sup>49</sup> while others found greater responsiveness for Whites and Hispanics, but not African-Americans,<sup>50</sup> or for African-Americans but not other races or ethnic groups.<sup>51</sup> Other researchers have found greater risk for African-Americans from air toxics, including those pollutants that also come from traffic sources.<sup>52</sup>

In a 2003 review article, O’Neill et al. concluded that there are three broad reasons why disparities may exist.<sup>53</sup> First, groups may face greater exposure to pollution because of factors ranging from racism to class bias to housing market dynamics and land costs. For example, pollution sources may be located near disadvantaged

communities, increasing exposure to harmful pollutants. Second, low social position may make some groups more susceptible to health threats because of factors related to their disadvantage. Lack of access to health care, grocery stores and good jobs, poorer job opportunities, dirtier workplaces or higher traffic exposure are among the factors that could handicap groups and increase the risk of harm. Finally, existing health conditions, behaviors, or traits may predispose some groups to greater risk. For example, diabetics were among the groups most at risk from air pollutants, and the elderly, African-Americans, Mexican-Americans and people living near a central city had higher incidence of diabetes.<sup>54</sup>

Socioeconomic position has been more consistently associated with greater harm from air pollution. Lower education levels were associated with the increased risk of premature death from PM in evidence from the large American Cancer Society database<sup>55</sup> and the Harvard Six Cities<sup>56</sup> database, as recognized in EPA's *Policy Assessment*.<sup>57</sup>

Recent studies show increased evidence of that link. Low socioeconomic status consistently increased the risk of premature death from fine particle pollution among 13.2 million Medicare recipients Zeger et al. (2008) studied in the largest examination of particle pollution mortality nationwide.<sup>58</sup> In the Bell et al. (2008) study that found greater risk for premature death for African-Americans, researchers also found greater risk for people living in areas with higher unemployment or higher use of public transportation.<sup>59</sup> Babin et al, in their 2008 study of Washington, DC, found that while poor air quality and worsened asthma went hand-in-hand in areas where Medicaid enrollment was high, the areas with the highest Medicaid enrollment did not always have the strongest association of high air pollution and asthma attacks.<sup>60</sup> Looking at Toronto, Canada, Burra et al. (2009) found that higher PM<sub>2.5</sub> levels were associated with increased physician visits for asthma, especially for those in the low socioeconomic group.<sup>61</sup>

Newer research published since the ISA shows that EPA is correct to include people in low socioeconomic groups in the list of susceptible populations. Exposure to particulate matter appears to be significantly greater for lower income communities. Brochu et al. (2011) looked at census tracts in six states in the Northeast and found that places with lower socioeconomic populations were exposure to higher levels of particulate matter.<sup>62</sup> Examining all counties with PM<sub>2.5</sub> monitors, Miranda et al. (2011) found that counties with a higher percentage of poverty had a higher likelihood that these counties would have the worst air quality for both annual and daily PM<sub>2.5</sub>, a relationship that did not hold for ozone<sup>63</sup>. Bell et al. reviewed exposure by census tracts and found, "the highest PM<sub>2.5</sub> exposures for non-Hispanic blacks, the least educated, the unemployed, and those in poverty,"

although the differences between those groups and whites were “small in magnitude.”<sup>64</sup>

New evidence published since the ISA adds to the need for improved near-road monitoring of PM. Jephcote and Chen (2011) found that inner city children in Leicester, England faced a “double burden” of roadway emissions of PM<sub>10</sub> and low income, increasing their risk of hospitalization for respiratory conditions.

This echoes the findings of the research by O’Connor et al. (2008) that found that near-roadway concentrations of PM<sub>2.5</sub> were associated with significantly lower pulmonary function in the low income children with asthma included in the Inner City Asthma Study.<sup>65</sup> Although the discussion in the ISA reported that this study found that “PM<sub>2.5</sub> concentration was not statistically associated with respiratory symptoms in this study” (p. 6-85), that conclusion is not consistent with the findings actually reported in the study which found “a significant association between decrements in lung function and increments in PM<sub>2.5</sub>,” particularly in the 5-day average readings.<sup>66</sup>

To reduce the burden on the people who have long suffered more than others in society from the harms of particulate matter, EPA should set a much more protective standard and expand the network of near-road monitoring above the levels currently proposed.

### **Both standards must be strengthened to save lives**

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Evidence during the last review showed clearly that the annual average standard needed to be much lower than the standard of 15 µg/m<sup>3</sup> that was first set in 1997.<sup>67</sup> The evidence has only grown since then. Multiple, multi-city studies over long periods of time have shown clear evidence of premature death, cardiovascular and respiratory harm as well as reproductive and developmental harm at contemporary concentrations far below the level of the current standard.

Some of the most extensive studies have confirmed that particulate matter causes premature death. Extended analyses of the cohorts in the landmark American Cancer Society study<sup>68</sup> and the Harvard Six Cities study<sup>69</sup> have been published since the last review. These studies, Laden et al. (2006)<sup>70</sup> and Krewski et al. (2009),<sup>71</sup> not only confirmed the findings of the original major studies, but added to the evidence. Laden et al. found statistically significant evidence that during this extended period, as the air quality improved, premature mortality dropped as well. A further follow-up extended the Six Cities study by 11 additional years, during which air quality was well below the level of the current annual average standard.<sup>72</sup>

Investigators reported an association between PM<sub>2.5</sub> and increased risk of premature all-cause, cardiovascular, and lung cancer deaths. The concentration-response relationship was linear down to 8 µg/m<sup>3</sup>, with no evidence of a threshold.

These findings were consistent with a large Canadian cohort study that reported that long-term exposure to PM<sub>2.5</sub> (mean, 8.7 µg/m<sup>3</sup>; interquartile range, 6.2 µg/m<sup>3</sup>) was associated with increased risk of cardiovascular mortality in adults.<sup>73</sup>

Newer large, long-term studies also added to the evidence of premature death from PM<sub>2.5</sub> at levels well below the current standard. The Women's Health Initiative studied over 65,800 women who had no history of cardiovascular disease in 36 U.S. cities.<sup>74</sup> Each increase of PM<sub>2.5</sub> of 10 µg/m<sup>3</sup> was associated with a 76 percent increase in premature deaths from cardiovascular causes.

Evidence from multiple studies that examined short-term exposures found harm at levels well below the current annual standard. The health harms found in these studies range across those found in the ISA to be causally and likely causally associated with PM exposure. Below are just a few of the studies that looked at short-term exposures and had mean annual PM<sub>2.5</sub> concentrations well below 15 µg/m<sup>3</sup>.

- Dominici et al (2006) examined hospital admissions for 11.5 million Medicare enrollees. The researchers found that hospital admissions for cardiovascular and respiratory causes increased significantly with every 10 µg/m<sup>3</sup>. There were clear regional differences, but the researchers concluded the PM<sub>2.5</sub> levels indicated "an ongoing threat to the health of the elderly population from airborne particles" and strong evidence that the NAAQS should be "as protective of their health as possible."<sup>75</sup> Bell et al. (2008) followed up on these Medicare enrollees in 202 counties looking at regional and seasonal differences. This analysis found continued strong associations with increased hospital admissions for both cardiovascular and respiratory harm, particularly in the Northeast, for increases of 10 µg/m<sup>3</sup>.<sup>76</sup>
- Bell et al (2007) reviewed the data for 358,504 births in Connecticut and Massachusetts and found an increased risk for low birth weight with higher levels of PM<sub>2.5</sub>.<sup>77</sup> The researchers noted that average concentrations were all below the 2006 PM<sub>2.5</sub> NAAQS.
- Zanobetti and Schwartz (2009) studied daily PM<sub>2.5</sub> and PM<sub>10-2.5</sub> in 112 cities in the United States between 1999-2005. They found an increased risk of premature death from all causes, myocardial infarction, cardiovascular disease, stroke and respiratory disease. The researchers concluded that this

study suggested that “tens of thousands of early deaths” could be prevented each year, especially since  $PM_{10-2.5}$  was not regulated.<sup>78</sup>

Furthermore, multiple epidemiological studies have found significant evidence of harm with strong confidence well below EPA’s proposed annual standard range of 12-13  $\mu\text{g}/\text{m}^3$ . As EPA noted in Figure 2-8 of the Policy Assessment,<sup>79</sup> many of these studies had long-term mean  $PM_{2.5}$  concentrations below 13, including the Women’s Health Initiative study and the Medicare study by Bell et al (2008) discussed earlier. Bell et al (2007) showed that low birth weights were associated with long-term mean concentrations below 12  $\mu\text{g}/\text{m}^3$ .

Earlier this year in the journal *Risk Analysis* EPA staff published another analysis that estimates the annual toll from  $PM_{2.5}$  at 130,000 premature deaths each year, based on 2005 air quality levels. This same analysis estimated a staggering 1.1 million life-years lost among people over age 65, accounting for 7 percent of life-years lost in 2005 in this population of elderly Americans. Looking at it another way, this translates into an average shortened lifespan of 8.5 months per individual affected. Further, the analysis estimates 1,800 deaths among babies and infants attributable to PM air pollution. This same analysis gauged the annual morbidity impacts of  $PM_{2.5}$  pollution at tens of thousands of hospital and emergency department visits for cardiac and respiratory causes and millions of asthma exacerbations, bronchitis, and other respiratory symptoms in children.<sup>80</sup>

These are preventable deaths. These are avoidable sicknesses and suffering. We have the means to control manmade air pollution to end this unnecessary toll on human life. Air pollution control efforts driven by more protective air quality standards for PM can give us the relief so sorely needed.

## **Conclusion**

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The new EPA standards must follow the requirements of the Clean Air Act and be set at levels that will protect the public with an adequate margin of safety, including children, the elderly, people with respiratory or cardiovascular disease or diabetes and people already disproportionately burdened. The evidence shows that strong confidence exists for harm occurring well below not only the existing annual standard, but below both the 13  $\mu\text{g}/\text{m}^3$  and 12  $\mu\text{g}/\text{m}^3$  annual standards proposed by EPA.

We urge you to set the following primary NAAQS for particulate matter to protect public health:

- **Annual average PM<sub>2.5</sub> standard of 11 µg/m<sup>3</sup>**
- **24-hour average PM<sub>2.5</sub> standard of 25 µg/m<sup>3</sup>**

We support a stronger 24-hour standard as well as a stronger annual standard. Lowering the annual standard will reduce chronic exposures, but it cannot protect against peak daily concentrations that have also been linked to serious harms to human health. Studies of short-term exposure demonstrate that PM<sub>2.5</sub> air pollution increases the risk of hospital admissions for heart and lung problems even when excluding days with pollution concentrations at or above the current daily standard of 35 µg/m<sup>3</sup>.

Daily concentrations must be capped at lower levels to protect against peak exposure days that occur due to local and seasonal sources of emissions. Strengthening both the annual and daily standards is necessary to provide healthier air to breathe for people all across the nation.

Numerous scientific studies have now identified increased health risks in association with traffic-generated air pollution, including fine particulate matter. With more than 45 million Americans living less than 300 feet from a highway, there is growing concern about the health impacts of living near heavily traveled roads."<sup>81</sup> Yet there are currently no requirements to monitor particulate air pollution. One of the most critical aspects of this proposal is the requirement to begin a program of roadside monitoring of PM<sub>2.5</sub> pollution, but the extent of the roadside monitoring proposed is far too limited. We urge EPA to accelerate and expand the proposed roadside PM monitoring network to better protect the health of millions of Americans who live in high traffic areas.

To help us protect the health of our patients and our nation, we call on EPA to set much more protective fine particulate standards than proposed.

Thank you for considering our views.

Sincerely,

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Trust for America's Health

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