

# Effects of Particulate Air Pollution on Children: Potential Impacts of the Proposed New Morro Bay Power Plant

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The adverse health impacts resulting from small increases in concentrations of the worst pollutant, particulate matter (PM), are quite significant. These effects are especially severe in infants and children. Although the proposed new Morro Bay Power Plant (MBPP) will have reduced emissions of some pollutants (specifically those that already have concentrations of only about 1/3 of the state regulatory standard), the PM<sub>10</sub> emissions will increase and PM<sub>10</sub> concentrations are already near or exceed state standards in Morro Bay. Compared to modeled maximum concentrations of pollutants with the existing plant, the modeled maximum concentrations of every key pollutant increase in Morro Bay with the new MBPP. This is because of the dramatically lower stack heights (450 ft. vs. 145 ft.), lower exhaust velocity, and lower exhaust temperature, which more than offset the reduction of some emissions.

Thousands of independent studies by public health professionals have demonstrated that increases of PM<sub>10</sub> cause a variety of adverse health effects. Studies focusing on infants and children suggest the following potential adverse impacts that may result from possible PM<sub>10</sub> concentration increases with the new MBPP: a 5.73% increase in infant respiratory mortality and a 19.94% increase in SIDS deaths; a 12.70% increase in respiratory hospital admissions in children and a 1.50% increase for infants; a 11.30% increase in emergency room visits for asthma by children; a 14.96% increase in acute respiratory symptoms (based on same day PM<sub>1.0</sub> levels) in asthmatic children; a 6.65% to 17.45% increase in lower respiratory illness in young children with mild asthma; a 3.57% increase in restriction of activity of children due to chest illness; and an increase in overall elementary school absences of 9.47%. These are serious potential adverse impacts posing a significant risk to the community, which is directly contrary to the stated goals of the MBPP project and the Memorandum of Understanding between the City of Morro Bay and Duke Energy.

This report addresses the effects of particulate matter generally and, more particularly, the impacts on children, one of many populations which have been found to be especially vulnerable to air pollution.<sup>1</sup> Particulate matter (PM) is the term used to define a complex mixture of naturally occurring airborne particles and those resulting from human activity. The discussion of PM in this report is based on data taken directly from Duke Energy's pending Application for Certification (AFC) filed with the California Energy Commission (CEC) and subsequent filings, and focuses on particulate matter emissions anticipated with the new, larger capacity Morro Bay Power Plant (MBPP).

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<sup>1</sup> Other populations identified by the American Lung Association [2] as especially vulnerable to the adverse health effects of air pollution are the elderly, people who exercise, people with asthma and allergies, the oxygen deprived and minority groups.

**Modeled Increases in PM<sub>10</sub>/PM<sub>2.5</sub> from the New Morro Bay Power Plant Are Associated With Significant Increased Risks Of:**

- Infant mortality from respiratory causes and SIDS (Sudden Infant Death Syndrome)
- Hospital admissions of children for respiratory illness
- Primary care clinic and doctor visits by children
- Medication use in asthmatic children
- Decreased lung function and lung growth in children
- Premature births, lower birth weight and smaller head circumference at birth
- Emergency room visits by children for asthma/pneumonia
  - Cough, phlegm, wheeze
  - breathing difficulties in children
- Altered and reduced immune function in children
- Higher school absenteeism

Some of these increased risks exceed those related to second hand smoke.

## Background on PM

As noted in recent research reports of the Health Effects Institute<sup>2</sup> [47, 70],<sup>3</sup> the size, chemical composition, and other physical and biological properties of PM depend on the sources of the particles and the changes the particles undergo in the atmosphere. In urban environments, fine particles derive primarily from fossil fuel combustion, including mobile sources such as motor vehicles and stationary sources such as power plants (as in the case of MBPP) and wood smoke. Particle size is most commonly described in terms of aerodynamic diameter<sup>4</sup> with surrounding outdoor (i.e., ambient) particles tending to fall into three size classes or modes: ultrafine or nuclei mode (particles less than 0.1 micron in diameter (PM<sub>0.1</sub>); fine or accumulation mode particles between 0.1 and 2.5 microns in diameter (ultrafine and fine particles compose PM<sub>2.5</sub>); and coarse particles which are larger than 2.5 microns in diameter. As described in the American Lung Association's booklet "Health Effects of Outdoor Air Pollution" (1996) [2], larger particles are the least

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<sup>2</sup> This organization, established in 1980, is an independent and unbiased source of information on the health effects of motor vehicle emissions. HEI supports research on all major air pollutants, including particulate matter. Typically, HEI receives half of its funds from the U.S. Environmental Protection Agency and half from 28 manufacturers and marketers of motor vehicles and engines in the U.S.

<sup>3</sup> Bracketed numerals correspond to the studies listed numerically in "References" following Appendix B.

<sup>4</sup> Aerodynamic diameter is defined as the diameter of a unit density sphere that has the same settling velocity in gas as the particle in question. This definition applies not only to spheres but also to the more typical, irregularly shaped particles [4, 112].

threatening, because they are more easily trapped in the nose and throat, where they are blown out, coughed up or swallowed. Smaller particles can be breathed into the lower respiratory tract.

The U.S. Environmental Protection Agency (EPA) has regulated the levels of ambient PM since 1971, when the Clean Air Act was first enacted. This legislation authorized the EPA to set National Ambient Air Quality Standards (NAAQSs) for a number of potentially harmful air pollutants, including PM, in order to protect the public health with standards “allowing an adequate margin of safety” [42 USC §7409(b)(1)]. The original standard was based on controlling total suspended PM (TSP). This standard was revised in 1987 to regulate *inhalable particles*, i.e., particles that can deposit in the respiratory tract and therefore have greater potential for causing adverse health effects. These inhalable particles are those with an aerodynamic diameter of 10 microns or less (PM<sub>10</sub>). For comparison, a human hair is about 100 microns across. More recent epidemiological studies (published in the 1990s) indicate a relatively consistent association between small short-term increases in PM levels and increases in both death (mortality) and illness (morbidity) from respiratory and cardiovascular diseases (see, the 1996 review of these studies by the Committee of the Environmental and Occupational Health Assembly, American Thoracic Society [4]).

Some studies [see, e.g., 31, 37, 100] have also suggested that long-term exposures to low levels of PM are associated with adverse effects, i.e., levels well below existing regulatory standards. These studies also point to a possible role of fine particles (PM<sub>2.5</sub>). In 1997, the EPA considered the evidence for the effects of fine particles sufficient to promulgate a fine particle standard while retaining the PM<sub>10</sub> standard [47, 141]. Implementation of these PM<sub>2.5</sub> standards was delayed by litigation brought by polluters [141], which has been resolved in favor of the EPA by the U.S. Supreme Court.

As noted by the American Lung Association [2], PM<sub>2.5</sub> “represents the most serious threat to our lungs. These particles are classified as fine particles. They can easily penetrate [deeply into the lungs] to the alveoli, the very smallest of the lung’s air sacs where crucial oxygen-carbon dioxide exchange takes place; because this region of the lung has a slow clearance system, the deposits may persist and increase the likelihood of damage.” [2, p. 17] These fine particles also pass readily into the bloodstream from the tiniest compartments in the lungs. [106] The brochure “Particulate Matter Air Pollution: A threat to our health” [7] distributed by the San Luis Obispo County Air Pollution Control District (APCD) notes: “Health problems begin as the body reacts to the foreign particles. PM<sub>10</sub> can increase the number and severity of asthma attacks, cause or aggravate bronchitis and other lung diseases, and reduce the body’s ability to fight infections.”

As set forth in an April 2001 New York Times article [106], the EPA has released a new draft review of the substantial adverse health effects of PM<sub>2.5</sub> based on its consideration of more than 3,000 new health studies published since the EPA first proposed PM<sub>2.5</sub> rules in 1997 [149].<sup>5</sup> In this review the EPA “has concluded that there is a stronger link than ever between the tiniest soot particles [PM<sub>2.5</sub>] and thousands of premature deaths each year.” [106]

California has adopted more stringent PM<sub>10</sub> standards than those required by the EPA, and has recently proposed<sup>6</sup> but not yet adopted a separate PM<sub>2.5</sub> annual standard of

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<sup>5</sup> A copy of the most recent draft EPA research review [149] on the substantial adverse health effects of PM<sub>2.5</sub> is available on the agency’s Web site ([www.epa.gov/ncea/](http://www.epa.gov/ncea/)).

<sup>6</sup> This proposal was part of the Public Review Draft dated November 30, 2001 entitled “Reviews of the California Ambient Air Quality Standards for Particulate Matter and Sulfates. Report to the Air Quality

12  $\mu\text{g}/\text{m}^3$  that is even stricter than the federal standard of 15  $\mu\text{g}/\text{m}^3$ . The current California  $\text{PM}_{10}$  standard is 50 micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ) for 24 hours and a geometric mean of 30  $\mu\text{g}/\text{m}^3$  for the annual standard, with a proposal to lower the annual standard to 20  $\mu\text{g}/\text{m}^3$ . These standards address concentration levels. As described in the AFC, Morro Bay exceeded the state 24 hour standard for  $\text{PM}_{10}$  (see, e.g., AFC, p. 6.2-59) for only one day during the years 1994 through 2000. See also, the Final Staff Assessment (FSA), Part 1, p. 3.1-7 -3.1-8.

Fine and ultrafine particles ( $\text{PM}_{2.5}$ ) are predominantly emitted from combustion processes, such as power plants and motor vehicles, while coarse particles are mostly generated by mechanical processes from a variety of noncombustion sources. As combustion-sourced PM, the PM emissions from the MBPP thus will be entirely  $\text{PM}_{2.5}$ .<sup>7</sup> [145] Generally, the fine and ultrafine particles are composed of carbonaceous material, metals, sulfate, nitrate and ammonium ions, whereas the coarse fraction is composed mostly of mechanically generated particles and consists of insoluble minerals and biologic aerosols, with smaller contributions from primary and secondary aerosols and sea salts [47]. Duke Energy's AFC addresses only  $\text{PM}_{10}$  with respect to the existing power plant and the proposed MBPP. The AFC confirms that the  $\text{PM}_{10}$  emissions from the new plant will be primarily carbon and sulfate.<sup>8</sup>

New emission control technologies, such as catalytic converters in automobiles, have helped to reduce levels of particles and other air pollutants over the years, despite an increased amount of combustion from industrial, commercial and personal activities. Nonetheless, numerous studies have shown that concentrations of ambient air particles, e.g.,  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$ , are associated with a wide range of effects, both short-term (acute) and long-term (chronic), on human health, including increased hospitalization for respiratory disease [68, 97, 98, 115, 116]; increased emergency room visits for respiratory illness [28, 124, 132]; increased severity of asthma episodes [6, 85, 102, 108, 144]; increased incidence and duration of respiratory symptoms, including coughing, wheezing, difficulty breathing, runny nose, and phlegm production [14, 54, 108, 142]; decreased lung function [20, 54, 63, 101, 104]; restricted activity in adult workers, and increases in absences of children from elementary school [44, 63, 81, 86, 105]; potentially life-threatening arrhythmia leading to therapeutic interventions by an implanted cardioverter defibrillator [88] and reduced heart rate variability, which is a predictor of increased risk for cardiovascular mortality and morbidity [45, 67, 99]; and increased mortality [32, 53, 103, 112, 114, 117-119, 122, 125, 130, 131, 137], particularly cardiopulmonary mortality [23, 112, 122].

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Advisory Committee" issued by the California Air Resources Board (CARB) and the Office of Environmental Health Hazard Assessment. This report is available on CARB's web page at [www.arb.ca.gov/research/aaqs/std-rs/pm-draft/pm-draft.htm](http://www.arb.ca.gov/research/aaqs/std-rs/pm-draft/pm-draft.htm).

<sup>7</sup> In its response to Data Request 13 of The Coastal Alliance on Plant Expansion (CAPE) March 9, 2001 Data Requests, Duke Energy confirmed that all of the combustion particulate emissions from the gas turbines are expected to be less than or equal to 2.5 microns in equivalent diameter (i.e., fine particles) and most are expected to be less than 1.0 micron in size.

<sup>8</sup> See, section 2.2.3.10 (p. 2-60) of the AFC. For comparison purposes, elemental carbon represents approximately 60% of the diesel exhaust particles observed in the Los Angeles atmosphere [62]. Elemental carbon is the only significant light absorbing aerosol in the atmosphere and makes a substantial contribution to reduced visibility. [147] Brown et al (2000) [15] recently demonstrated that the increased inflammation and intracellular calcium caused by the ultrafine carbon black component of  $\text{PM}_{10}$  is independent of transition metals or other soluble components.

Some of these adverse effects of  $PM_{10}$  appear to be related to short-term exposures [23, 61, 83], whereas others have to be considered the result of long-term exposure [31, 100]. [64, 96] Using life table methods and reviewing the available evidence of the mortality effects of long-term or chronic exposure to PM pollution, Brunekreef (1997) [16] derived an estimate of the reduction in life expectancy.<sup>9</sup> A difference of 1.11 years was found between the PM exposed and clean air groups' overall life expectancy at age 25 in the Netherlands. A similar calculation for the 1969-71 life table for U.S. males resulted in an even larger reduction of 1.31 years for the entire population's life expectancy at age 25. These calculations imply that relatively small differences in long-term exposure to ambient PM can have substantial effects on life expectancy. In this connection, bear in mind that the new MBPP can be expected to have an operating life of 50 years or even an indefinite life, i.e., it will result in chronic  $PM_{2.5}$  exposure.

In a 1993 article in the New England Journal of Medicine [31], public health researchers reported on a study following more than 8,000 persons in six different locations over a period of 17 years (the Harvard Six Cities study), in areas with varying levels of  $PM_{2.5}$ . After controlling for other factors (smoking status, occupational risks, etc.), they found the risk of early death in high-level areas to be increased by 26% over that in areas with the lower levels. "Building on the results of that study, as well as similar smaller-scale investigations, a 1995 study involving more than 550,000 residents in 151 U.S. metropolitan areas, in which the subjects were followed for more than seven years, found a 17 percent increase in mortality risk in areas with higher concentrations of small particles [100]." [2, p. 1] Using daily mortality data, a  $10 \mu g/m^3$  increase in the two-day  $PM_{2.5}$  level was associated with a 1.5% increase in total daily mortality, as well as a 3.3% increase in deaths for chronic obstructive pulmonary (lung) disease and a 2.1% increase in deaths caused by heart disease. [122]

These studies likewise showed very significant correlations between high  $PM_{2.5}$  levels and increased cardiopulmonary disease.<sup>10</sup> Aside from cardiovascular disease and the increased risk of premature death, PM exposure levels have been correlated in the short term with a number of other effects. All particulates are irritants, and persistent cough with phlegm has been linked to high PM levels, along with wheezing and physical discomfort in breathing. Much of the most worrisome impact of high levels of particulate matter is on sensitive population subgroups. They include individuals suffering from certain chronic respiratory problems likely to be aggravated by irritants in the air, such as asthma. A number of studies have conclusively linked  $PM_{2.5}$  with increased prevalence of coughs and bronchitis, with especially severe effects on children with asthma.

More recently Samet et al. (2000) [112] investigated the association between daily  $PM_{10}$  concentration and the number of deaths reported on the following day in 20 of

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<sup>9</sup> Based on prior studies [31, 100] where the mean fine particle concentrations ranged from 11.0 to  $29.6 \mu g/m^3$  and the  $PM_{10}$  concentrations ranged from 18.2 to  $46.5 \mu g/m^3$ , Brunekreef [16] assumed a relative risk of 1.1 per  $10 \mu g/m^3$  exposure over 15 years for the effect of PM air pollution on men 25-75 years of age. He then developed a life table for men for the 25-75 year old range and calculated life expectancy of a 25 year old which he compared with the calculated life expectancy for the PM exposed case, where death rates were increased in each age group by a factor of 1.1.

<sup>10</sup> A recent review and analysis [66] of 29 estimates of the PM-mortality link from the Harvard School of Public Health found that of "all the variables tested, the  $PM_{2.5}/PM_{10}$  ratio appears to be the strongest predictor of the relationship between  $PM_{10}$  and mortality." [66, p. 113] This is "compelling evidence that the  $PM_{10}$ -mortality relationship is stronger in locations with higher  $PM_{2.5}/PM_{10}$  ratios, supporting the hypothesized role of fine particles." [66, p. 116]

the largest U.S. cities and metropolitan areas, i.e., acute impacts, and found an average increase in the rate of death from all causes of 0.51% for every increase in the PM<sub>10</sub> concentration of 10 µg/m<sup>3</sup>. These correlations were observed in cities with PM<sub>10</sub> concentrations well below the federal standard. These findings “are consistent with those of time-series studies in Europe [61] and cohort studies in the United States [31]. Thus, the evidence in support of an association between the concentration of particulate air pollution and the mortality rate is consistent, is not affected by differences in statistical methods, and can be generalized.” [141, pp. 1798-1799] Correlations between PM<sub>10</sub> and next day deaths from cardiovascular and respiratory causes were even higher (0.68% for each increase of 10 µg/m<sup>3</sup> in PM<sub>10</sub> level) [112]. Ostro et al. (1999) [83] found each increase of 10 µg/m<sup>3</sup> in daily PM<sub>10</sub> concentrations was associated with a 1-2% increase in overall mortality, a 1-2% increase in cardiovascular-related mortality, and a 3-6% increase in respiratory-related mortality in Bangkok, Thailand.

Samet et al. (2000) [112] also analyzed the effects of levels of carbon monoxide (CO), nitrogen dioxide (NO<sub>2</sub>) and sulfur dioxide (SO<sub>2</sub>) in a fashion similar to that of the analysis of PM<sub>10</sub> levels. The study’s authors concluded that “[a]fter adjustment for PM<sub>10</sub> and ozone levels, we found little evidence that these pollutants had a significant effect on the relative rate of death.” [112, p. 1747] The association of PM<sub>10</sub> was not affected by the inclusion of other pollutants in the statistical model or by the time at which data were collected. Epidemiologic studies of both the longer-term effects [16, 31, 100] of air pollution on the risk of death and studies of daily mortality data [113, 152] suggest that exposure to air pollution goes well beyond the simple shortening of life by a few days.

As described by the American Lung Association [2], the levels of particulates need not be extraordinarily high to precipitate these problems. One of the studies confirming the asthma association was conducted in Seattle, where PM concentrations never exceeded 70% of the current permissible standard [124]. There has been evidence in the many PM<sub>10</sub> and PM<sub>2.5</sub> studies that there exists no “safe” threshold below which PM<sub>10</sub> and PM<sub>2.5</sub> will cause no adverse health effects [13, 19, 25, 37, 53, 96].<sup>11</sup> “The epidemiologic evidence suggests that the association between fine-particle concentrations and mortality is linear across the entire range of current concentrations.” [141, p. 1799] In other words, the effects are proportional [25,96]. As Duke Energy’s AFC notes, based on the 1997-1999 ambient air concentrations Morro Bay is not currently in attainment of the California PM<sub>10</sub> 24-hour standard, even before the new plant comes on line. See, section 6.2.1.1 and Table 6.2-1 of the AFC. Based on the 1998-2000 ambient air concentrations, however, Morro Bay is currently in attainment of the 24-hour standard. [FSA, Part 1, p. 3.1-8]

## **Anticipated PM<sub>10</sub> Increases from the New MBPP**

Set forth below is a summary of some of the key data provided by Duke Energy in its AFC and subsequent filings with respect to the existing plant’s actual and the new plant’s modeled PM<sub>10</sub> emissions and, even more importantly, the modeled maximum concentrations of PM<sub>10</sub> including these emissions. Although emission levels clearly contribute to concentration levels of a particular pollutant, it is the concentration level of the pollutant in the air that is most critical in determining adverse health effects. Concentrations may vary with equal emissions depending on dispersion of the pollutants, which in turn depends upon factors such as stack height, exhaust velocity, and exhaust temperature. Note

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<sup>11</sup> See, the more extensive discussion of the absence of thresholds in the authors’ companion report entitled “Effects of Particulate Air Pollution on Susceptible Populations Other than Children, Mortality Displacement and Absence of Threshold: Issues Relating to the Proposed New Morro Bay Power Plant.”

that all of the PM<sub>10</sub> emissions from the MBPP discussed in the AFC are in fact PM<sub>2.5</sub> fine and ultrafine particles.

- PM<sub>2.5</sub> emissions increase by 76 tons per year with the new MBPP to 203.2 tons per year from 127.2 tons per year with the existing plant (an approximate 60% increase) [see Table 6.2-4 of the AFC]. When compared to emissions from the existing plant with retrofitting required by 2003, there remains an increase of 60 tons per year (see Duke Energy's Response to California Energy Commission's February 9, 2001 Data Requests, p. B-1).
- Duke Energy erred originally in Section 6.2.6.3.2 and Table 6.2-35 of the AFC in its discussion of modeled air concentrations of four key pollutants, including PM<sub>10</sub>, by using modeled stack heights of only 383 feet for the existing plant, rather than the required actual 450 feet stack height.<sup>12</sup> This factor critically affects the modeled concentrations of all pollutants with the existing plant because of the greater dispersion of the emissions from the taller stacks, higher exhaust temperature and higher velocity of emissions from the existing plant as compared to the new MBPP (see, Tables 6.16-2 and 6.16-8 of the AFC). For example, the modeled PM<sub>10</sub> 24-hour maximum was decreased from 7.82 µg/m<sup>3</sup> (highest second high) for the initial model to 3.11 µg/m<sup>3</sup> (4.28 µg/m<sup>3</sup> high) for the actual 450 feet stacks. In effect, for the existing plant the initial model overstated each of the modeled concentrations for every pollutant.
- A comparison of the maximum modeled concentrations of the key pollutants between the existing plant with the proper modeling (including any retrofitting necessary to meet the new 2003 NOx standard) and the new MBPP results in **increases for every pollutant with the new MBPP** in Morro Bay, including Morro Rock, as set forth in the following Table (based on Table 6.22-36 of the AFC, the Table shown on p. B-3 of Duke Energy's Responses to CEC February 9, 2001 Data Requests and the APCD's consultant's (SAIC) evaluation of the modeled results with revised parameters required by the APCD, the latter of which is attached as Appendix A):

<b>Pollutant</b>	<b>Averaging Time</b>	<b>New MBPP Modeled Concentrations (µg/m<sup>3</sup>) High</b>	<b>Existing Plant Modeled Concentrations (µg/m<sup>3</sup>) High</b>	<b>Increase with New MBPP (µg/m<sup>3</sup>)</b>
NO <sub>x</sub>	1-hour	321.6*	66.4*	<b>255.2</b>
	Annual	2.6	0.35*	<b>2.25</b>
SO <sub>2</sub>	1-hour	17.3	1.97*	<b>15.33</b>
	3-hour	11.9	1.62*	<b>10.28</b>
	24-hour	2.7	0.34*	<b>2.36</b>
	Annual	0.2*	0.01*	<b>0.19</b>
CO	1-hour	326.3	278.1*	<b>48.2</b>

<sup>12</sup> When questioned by the San Luis Obispo County Air Pollution Control District (APCD) through the California Energy Commission's February 9, 2001 Data Requests, after originally stating that "the boiler stacks cannot be modeled at their full physical height" (see, p. 6.2-51 of the AFC), Duke Energy agreed the modeling should have been done based on the actual stack height. Duke still failed to provide modeled high concentrations (as opposed to highest second highs) for all averaging standards other than annual figures, e.g., there is no information on 24-hour PM<sub>10</sub> modeled high concentrations, except from the APCD consultant (SAIC) who analyzed the results of Duke's modeling. Unfortunately, the FSA Part 1 Air Quality Table 7-B (p. 3.1-17) uses the uncorrected "old facility" impact figures with the wrong stack heights.

	8-hour	1,508.3	130.1*	<b>1,378.2</b>
PM <sub>10</sub>	24-hour	24.2	4.28*	<b>19.98</b>
	Annual	2.7	0.14	<b>2.56</b>

\* From SAIC (Appendix A)

- The modeled maximum concentrations of PM<sub>10</sub> in Morro Bay (including Morro Rock) with the existing plant using the actual 450 feet stack heights are 4.28  $\mu\text{g}/\text{m}^3$  for the 24-hour average concentration [see, Appendix A].
- Annual average modeled concentrations of PM<sub>10</sub> increase from 0.14  $\mu\text{g}/\text{m}^3$  with the existing plant to 2.7  $\mu\text{g}/\text{m}^3$  with the new MBPP (an eighteen fold increase) [see Table 6.2-36 of the AFC and Duke Energy's Response to California Energy Commission February 9, 2001 Data Requests, p. B-3]. The modeled maximum concentrations resulting from these increased PM<sub>2.5</sub> emissions in Morro Bay increase at least by 0.66  $\mu\text{g}/\text{m}^3$  (from 0.14  $\mu\text{g}/\text{m}^3$  including Morro Rock for the existing plant to 0.80  $\mu\text{g}/\text{m}^3$  with the new MBPP excluding the uninhabited Morro Rock) or by up to 0.76  $\mu\text{g}/\text{m}^3$  excluding Morro Rock in both cases, based on the annual average PM<sub>2.5</sub>.<sup>13</sup> Using this annual increase in lieu of daily maximums to assess typical daily impacts is much more conservative and would understate the risks to be expected with the higher fluctuation days.
- The modeled maximum project impact 24-hour average concentration of PM<sub>10</sub> with the new plant is 24.2  $\mu\text{g}/\text{m}^3$  [see Table 6.2-6 of the AFC] (an increase of 19.98  $\mu\text{g}/\text{m}^3$  [24.2 - 4.28  $\mu\text{g}/\text{m}^3$  = 19.98  $\mu\text{g}/\text{m}^3$ ] or more than five times the concentrations with the existing plant). This figure includes Morro Rock which is uninhabited. Using overly conservative estimates to exclude Morro Rock, the modeled maximum 24-hour PM<sub>2.5</sub> concentrations increase at least by 4.42  $\mu\text{g}/\text{m}^3$  (from 4.28  $\mu\text{g}/\text{m}^3$  including Morro Rock for the existing plant to 8.7  $\mu\text{g}/\text{m}^3$  with the new MBPP excluding Morro Rock) or by up to 8.31  $\mu\text{g}/\text{m}^3$  excluding Morro Rock in both cases.<sup>14</sup> The latter is a twenty-three fold increase.

<sup>13</sup> This is a very conservative calculation in that it initially compares a low figure which includes Morro Rock to a high figure that excludes Morro Rock. Applying the same ratio as exists between the new MBPP PM<sub>2.5</sub> modeled concentration with and excluding Morro Rock ( $.8/2.7 \mu\text{g}/\text{m}^3 = 29.63\%$ ) to the existing plant maximum modeled annual PM<sub>2.5</sub> average concentrations including Morro Rock ( $0.14 \mu\text{g}/\text{m}^3 \times 29.63\% = .04 \mu\text{g}/\text{m}^3$ ) results in a modeled annual average increase excluding Morro Rock in both cases of  $0.76 \mu\text{g}/\text{m}^3$  ( $.14 - .04 = .10$ ). This is itself a conservative figure because the existing plant operates with stacks that are approximately three times taller than the proposed new MBPP (450 ft. vs. 145 ft.) with both higher exhaust velocity and temperature than with the new MBPP, suggesting that the portion of PM<sub>2.5</sub> remaining in Morro Bay with the existing plant is less than the ratio using the MBPP indicates. "Conservative" in this sense is not most protective of human health interests, but refers to the minimum of what such adverse effects will be.

<sup>14</sup> This is a very conservative calculation in that it likewise initially compares a low figure which includes Morro Rock to a high figure that excludes Morro Rock. Applying the same ratio as exists between the new MBPP modeled maximum 24-hour PM<sub>2.5</sub> concentration including and excluding Morro Rock ( $8.7/.8 \mu\text{g}/\text{m}^3 = 9.2\%$ ) to the existing plant maximum modeled 24-hour PM<sub>2.5</sub> average concentration ( $4.28 \mu\text{g}/\text{m}^3 \times 9.2\% = 0.39 \mu\text{g}/\text{m}^3$ ) to derive the annual PM<sub>10</sub> average for the existing plant excluding Morro Rock results in a daily maximum increase excluding Morro Rock of 8.31  $\mu\text{g}/\text{m}^3$  ( $8.8 - 0.39 = 8.31$ ). As noted above, this in itself is a conservative figure because the existing plant operates with stacks that are approximately three times taller than the proposed new MBPP with both higher exhaust velocity and temperature than the new MBPP.

- Unlike the historical operations of the existing plant (1997-1999), the new MBPP is expected to operate approximately 90% of the time (up to 24 hours a day, 7 days a week) [see sections 2.1.2 and 2.2.3.5 of the AFC].
- The new plant will result in increased emissions of carcinogenic polycyclic aromatic hydrocarbons (PAHs) compared to the existing plant [compare Table 6.2-32 with Table 6.2-33 of the AFC and p. 6.2-69].
- The new plant will also more than double the sulfur dioxide (SO<sub>2</sub>) emissions from the existing plant (10 tons per year to 23 tons per year with the new plant) [see Table 6.2-4 of the AFC].
- The new MBPP will result in a decrease in nitrogen dioxide (NO<sub>2</sub>) emissions of 66.7 tons per year and a decrease in carbon monoxide (CO) emissions of 830.6 tons per year as compared to emissions from the existing plant with retrofitting required to meet the 2003 NO<sub>x</sub> standards (see Duke Energy's Responses to California Energy Commission's February 9, 2001 Data Requests, p. B-1), although as noted in the above Table, the local concentrations will nonetheless increase with the new plant.
- With the current plant, existing ambient air concentrations in SLO County of NO<sub>2</sub> and CO (based on the maximums from 1997 through 1999 used in the AFC) are only approximately one third (1/3) of, i.e., well below, existing state standards (NO<sub>2</sub> 1 hr 122 μg/m<sup>3</sup> actual vs. state standard of 470 μg/m<sup>3</sup>; CO 1 hr 6,988 μg/m<sup>3</sup> actual vs. state standard of 23,000 μg/m<sup>3</sup> and 8 hr 3,444 μg/m<sup>3</sup> actual vs. state standard of 10,000 μg/m<sup>3</sup>) [see Table 6.2-1 of the AFC].
- With the current plant, existing ambient air concentrations of PM<sub>10</sub> in Morro Bay based on the maximums from 1997 through 1999 already **exceed** the California 24-hour standard for PM<sub>10</sub> and will be further in exceedance of this standard with the new MBPP.<sup>15</sup>

**It is clear then that the new MBPP will reduce emissions (but increase local concentrations) of pollutants that are already well below state and federal standards but will increase PM<sub>10</sub> emissions and concentrations, which as the American Lung Association has stated “represent the most serious threat to our lungs,” and as to which current ambient air concentrations already are near or exceed state standards.**

Duke has argued that pollutant emissions from the new MBPP should only be considered in terms of emissions per kilowatt-hour of electricity produced.<sup>16</sup> However, clearly the amount of overall increased ambient concentrations of pollutants resulting from the MBPP project (i.e., absolute magnitude, not rate) is the only relevant consideration in making assessments of the resulting increased significant adverse health effects.

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<sup>15</sup> Based on Duke Energy's Response to Data Request 26 of CAPE's March 9, 2001 Data Requests, the maximum average 24-hour concentration of PM<sub>10</sub> as measured at the Morro Bay monitoring station was 47 μg/m<sup>3</sup> in 2000. The additional 17.09 μg/m<sup>3</sup> average increased PM<sub>10</sub> concentrations noted above resulting with the new turbines would clearly cause the California 24-hour standard of 50 μg/m<sup>3</sup> to be exceeded, based on the historical levels from 1998-2000.

<sup>16</sup> See, e.g., Testimony of Andrew Trump and Robert Cochran in Duke's Testimony on Group I Issues, p. 28 (1<sup>st</sup> ¶); Duke's Response to Data Request 7 of CAPE's March 9, 2001 Data Requests.

## PM Effects on Children

This section provides a brief summary of some of the recent literature involving studies done by public health professionals regarding the impacts of PM on children's health. In its booklet [2], the American Lung Association specifically identifies the very young as one of the populations especially vulnerable to air pollution effects.

Children playing outdoors, as all children do, are running a special risk simply by being outdoors, in the presence of pollutants, far more than older people who are busy indoors tending to occupational and household chores. Children also inhale more air per pound of body weight, hence proportionately more pollutants as well. And they may be less likely than adults to be conscious of symptoms, such as chest tightness, warning of pollution effects.

Some studies have suggested that episodes of heightened sulfur dioxide levels can impact children especially, causing measurable lung-function declines that may persist for as long as two weeks. Children may also be especially susceptible to a weakening of the immune system encouraged by nitrogen dioxide – specifically, its ability to decrease the body's resistance to lower respiratory infection. And repeated epidemiologic studies have linked ambient air pollution, especially respirable particles (PM<sub>2.5</sub>) and ozone, with increased prevalence of cough, bronchitis, and decrements in lung function – with marked increases among youngsters with a history of asthma. [2, p. 25; citation omitted]

The Association further notes that compounding the threat is the fact that highest levels of various pollutants are likely to prevail in summer, when children spend much more time out of doors (and when the plant can be expected to run at maximum levels). “One study specifically limited to warm months – it was conducted between April and August – found significant associations between lower respiratory symptoms (i.e., involving the lungs and small bronchial tubes) and unusually high concentrations of inhalable particulate matter (PM<sub>10</sub>), ozone, and sulfur dioxide, in that order [123].” [Id.]

The following presents very briefly the highlights of independent public health studies on the adverse health impacts of increasing PM<sub>10</sub> and PM<sub>2.5</sub> levels on children:

- Studies have indicated likely early post-natal PM exposure effects on infant mortality [8, 9, 148]. In one study, high PM exposure in the first two months after birth was associated with increased mortality in the first year for respiratory causes (40%) and sudden infant death syndrome (26%), or 20% and 12%, respectively, for each 10  $\mu\text{g}/\text{m}^3$  [148]. More recent findings [71] point specifically to PM<sub>2.5</sub> impacts on infant mortality with an estimated excess risk for PM<sub>2.5</sub>-related infant mortality of 18.2% per 25  $\mu\text{g}/\text{m}^3$ .
- Hospital admissions of children for respiratory disease in Utah Valley dropped by over 50% during the strike at the local steel mill which led to much lower PM<sub>10</sub> concentrations – a mean of 51  $\mu\text{g}/\text{m}^3$  – with an estimated 4.2% decrease in asthma and bronchitis admissions of children and a 7.1% decrease in all respiratory admissions of children associated with each 10  $\mu\text{g}/\text{m}^3$  decrease in the two-month mean PM<sub>10</sub> concentration. [30, 98] There is a higher excess risk for respiratory-related hospital admissions for infants than other children or adult age groups (roughly double) [18, 49].

- Emergency department visits have also been analyzed by many investigators and several have provided quantitative effect estimates of the effects of particulate pollution [21, 28, 30, 55, 80, 124, 132, 136]. For example, one study of the general population found an increase of 3.4% in emergency room visits for asthma associated with each 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{10}$  in Seattle [30, 124]. A more recent study in Seattle focusing only on children found an increase in emergency room visits for asthma of 13.6% associated with each 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{1.0}$ . [80]
- Panel studies have found 1-4% increases in reports of acute respiratory symptoms associated with each 10  $\mu\text{g}/\text{m}^3$  increase in ambient concentrations of  $\text{PM}_{10}$  [13, 30, 52, 77, 78, 111]. One study [123] found a 30  $\mu\text{g}/\text{m}^3$  increase in daily  $\text{PM}_{10}$  concentration was associated with a 53% increase in incidence of lower respiratory systems, a 22% increase for incidence of coughing, and a 22% increase for incidence of upper respiratory symptoms. Another found an 18% increase in acute respiratory symptoms in asthmatic children with each 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{1.0}$ . [150] Studies have likewise observed increased use of medication for asthma with increases in  $\text{PM}_{10}$ . [27A]
- Recent studies have demonstrated that fine particles ( $\text{PM}_{2.5}$ ) are more strongly associated with acute respiratory health effects in schoolchildren [121, 122, 150] and particles, especially fine particles, readily penetrate indoors [13, 58].
- Relatively low levels of  $\text{PM}_{10}$  (within existing regulatory standards) in ambient air result in increased lower respiratory symptoms and medication use in asthmatic children [43, 63, 72, 82, 90, 108, 138, 140, 150] and increased lower respiratory symptoms in nonasthmatic children [63, 78, 102, 123, 140]. For example, one study demonstrated that an increase of 10  $\mu\text{g}/\text{m}^3$  of  $\text{PM}_{10}$  was related to a 21% increase in lower respiratory illness among children with mild asthma (aged 5 to 7 years) [111]. Another study showed a 26% increase in restriction of activity due to chest illness in children (aged 7 to 11 years) for each 30  $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$ . [79]
- In terms of impacts on the immune system,  $\text{PM}_{10}$  causes the immune system to work “over-time,” resulting in abnormal and reduced function, such as the production of immature cells in the bloodstream [9, 29, 65, 75, 107, 133].
- With respect to lung function, studies have shown that increases in particulate pollution are associated with declines in peak expiratory flow (PEF) and/or lung growth rates in children [20, 33, 41, 52, 54, 63, 78, 102, 138, 140], with somewhat greater deficits in symptomatic children, i.e. those already displaying symptoms [33, 102, 138, 140].
- Numerous studies have demonstrated increased rates of asthma in children despite the overall decreases in air pollution over the last several decades [22, 42, 51, 80].
- The limited experimental and epidemiologic studies [38, 39, 94, 129] currently available identify the early postneonatal period of lung development as a time of high susceptibility for lung damage created by exposure to environmental toxicants, which is likely the reason for high rates of respiratory infectious diseases in young children.<sup>17</sup>

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<sup>17</sup> Infants have diminished immune status and are growing rapidly. There is some evidence supporting the hypothesis that environmental pollution can significantly alter development of the respiratory system at that stage of life. In experimental animals, elevated neonatal susceptibility to lung-targeted toxicants has been reported at doses “well below the no-effects level for adults” [38, 94]. Acute injury to the lung during early postnatal development also causes a failure of normal repair processes [39, 129]. The fact that both the diminished defenses and pollution-induced impairment of repair mechanisms thus can coincide during infancy

- Increases in  $PM_{10}$  have been shown to result in increased absenteeism in elementary school children [44, 90, 105]. For example, in the Utah Valley an increase in average  $PM_{10}$  of  $100 \mu\text{g}/\text{m}^3$  was associated with an increase in overall absences equal to approximately 40% [105]. In Southern California communities (including Atascadero and Santa Maria), a  $10 \mu\text{g}/\text{m}^3$  increase in  $PM_{10}$  resulted in a 22.8% increase in all types of school absences and a 5.7% increase in illness-related absences. [44]
- Recent epidemiological studies have concluded that the risk of premature births and of delivering a growth-retarded and/or low birth weight infant increases (e.g., a 4% increase in premature births for each  $10 \mu\text{g}/\text{m}^3$  increase in  $PM_{10}$ ) with the level of fine particles ( $PM_{10}$ ,  $PM_{2.5}$ ) [9, 26, 27, 107, 109] and with the carcinogenic fraction of polycyclic aromatic hydrocarbons (c-PAHs), which are usually bound to fine particles [26, 87]. Smaller head circumference, associated with lower IQ, was also found with increased PAHs. [87]

These findings of substantial adverse health effects resulting from  $PM_{10}$  and  $PM_{2.5}$  concentrations are discussed in greater detail in Appendix B.

**No single study can be used to predict definitive results elsewhere. Nonetheless, using the assumption of linearity [141] and the  $8.31 \mu\text{g}/\text{m}^3$  modeled maximum average daily increase in  $PM_{2.5}$  concentrations with the new Morro Bay Power Plant,<sup>18</sup> these studies suggest, for example, the following potential adverse health impacts:** a 5.73% increase in infant respiratory mortality [71, 148] and a 19.94% increase in SIDS deaths [148]; a 12.70% increase in respiratory hospital admissions in children [30, 98] and a 1.50% increase for infants [49]; a 11.30% increase in emergency room visits for asthma by children [80]; a 14.96% increase in acute respiratory symptoms (based on same day  $PM_{10}$  levels) in asthmatic children [150]; a 6.65 to 17.45% increase in lower respiratory illness in young children with mild asthma [111]; a 3.57% increase in restriction of activity of children due to chest illness [79]; and an increase in overall elementary school illness-related absences of 9.47% [44]. These clearly present serious potential adverse impacts on the community.<sup>19</sup>

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leads to a situation of potentially especially elevated susceptibility to damage by environmental toxicants like PM.

<sup>18</sup> These figures were derived by multiplying the applicable rates per  $10 \mu\text{g}/\text{m}^3$  of  $PM_{10}$  by 83.1%, i.e., the equivalent of a  $8.31 \mu\text{g}/\text{m}^3$  increase in  $PM_{10}$ . Using the even more “conservative” figure of a  $4.42 \mu\text{g}/\text{m}^3$  increase (an “apples and oranges” comparison), these figures would be roughly halved. The increased risk figures that are taken from  $PM_{10}$  studies (as opposed to  $PM_{2.5}$  studies) have been doubled under the assumption that  $PM_{2.5}$  makes up roughly half of the total  $PM_{10}$  in those studies and assuming further that all of the adverse effects are from  $PM_{2.5}$ .

<sup>19</sup> If one is even more “conservative” and uses the annual average  $PM_{2.5}$  increases from the new MBPP excluding Morro Rock of  $0.76 \mu\text{g}/\text{m}^3$  to approximate a “more typical” daily level of  $PM_{2.5}$  concentrations, these studies suggest the following adverse health effects: a 0.52% increase in infant respiratory mortality [71, 148] and a 1.82% increase in SIDS deaths [148]; a 1.16% increase in respiratory hospital admissions in children [30, 98] and a .14% increase for infants [49]; a 1.03% increase in emergency room visits for asthma by children [80]; a 1.37% increase in lower respiratory illness in young children with mild asthma [111]; a .33% increase in restriction of activity of children due to chest illness [79]; and an increase in overall elementary school illness-related absences of 0.87% [44]. Although these percentage increases may appear somewhat small they are all significantly higher than the 10 in 1,000,000 significance criteria used for determining significant cancer risks from hazardous materials.

## Conclusions

The new MBPP as proposed will have significant adverse health impacts on infants and children in Morro Bay, a group making up 15.1% of the population (based on 2000 U.S. Census data). Other costs beyond direct health impacts resulting from the new plant should be considered as well. In addition to the direct adverse health effects resulting from increased PM<sub>10</sub>/PM<sub>2.5</sub> levels, those living in Morro Bay will be burdened by accompanying higher direct medical care costs (increased doctor visits, hospitalization, etc.) and indirect costs (missing work to care for sick children, etc.). For example, increased school absenteeism could have a direct adverse impact on state and federal funding for local schools, as well as result in educational deficits that could arise with poorer school performance and decreased test scores resulting from increased absenteeism. A recent analysis integrating the data from a number of independent studies of air pollution (and specifically PM<sub>10</sub>) impacts on public health states: “It is to be emphasized that the economic loss of the health impact of air pollution goes beyond the direct costs of medical treatment. Loss of production and consumption as well as intangible costs (pain, suffering) of disease and death have to be taken into account.” [64, p. 800] These costs are especially high with infant mortality. [71]

The original Memorandum of Understanding (MOU) between the City of Morro Bay and Duke Energy relating to the new expanded Morro Bay Power Plant specifically addresses and emphasizes the community’s concerns with adverse public health effects. As Duke Energy quotes in Section 2.1 of the AFC, among the goals to be addressed through the technical and design aspects of the project, is the following:

**Protect Public Health and Safety:** To ensure that the modernization plan complies with all Commission and San Luis Obispo County Air Pollution Control District (APCD) requirements to ensure it does not pose any significant risks to public health and safety generally, and specifically, that it does not pose any significant risk to the citizens of Morro Bay resulting from actual air emissions within the City (regardless of any APCD required emission offsets); also, to recognize that with implementation of APCD requirements for the modernization project, the Project is expected to result in overall improvement in air quality over existing conditions. [emphasis added]

Clearly this highlighted language is intended to insure that actual emissions from the new plant will not pose any significant risk to local residents, regardless of what the current regulatory standards may be and regardless of the emission offsets or credits obtained by Duke as required by the APCD. The latter is quite important because mitigation credits only address total emissions and not specific ambient concentration impacts.

In its Response to Data Request 24 of the CEC’s February 9, 2001 Data Requests, which required a clear statement of the project’s objectives, Duke likewise stated it “is seeking a project that **substantially improves the local environment for the residents of Morro Bay** and surrounding areas given the close proximity of the town to the power plant site.” [Emphasis added]. The MBPP as proposed does not meet this basic project objective.

Duke Energy takes the position, however, that it “is responsible for mitigating its contribution to ambient PM<sub>10</sub> levels, and that mitigation is provided, . . . through the provision of emission offsets” (see, Response to CAPE’s March 9, 2001 Data Request 90). Each of the adverse health effects noted in this report are deemed by the authors of the studies cited to be significant by scientific, public health standards. **Clearly, there are**

**significant adverse health effects resulting from the new MBPP increased concentrations of PM<sub>2.5</sub> that require local mitigation beyond the application of emission offsets.**

One issue of particular concern relating to PM<sub>10</sub> and PM<sub>2.5</sub> concentrations is the drastically lowered stack heights of 145 feet with the new MBPP compared to 450 feet with the existing plant. The question is whether these new shorter stacks, although aesthetically more pleasing, can in fact distribute the PM<sub>2.5</sub> emissions as widely as do the old stacks. If not, this results in more of the PM<sub>2.5</sub> emissions remaining staying in Morro Bay than is currently the case. This would further increase the health risk levels to Morro Bay residents vis-a-vis the rest of San Luis Obispo County. Duke Energy has refused to provide any information regarding air quality effects from anything other than the proposed 145 foot stacks.<sup>20</sup> As described above, the fact that the modeled maximum concentrations with the new MBPP increase for every pollutant relative to the modeled concentrations resulting with the actual 450 feet stacks at the existing plant suggests significantly increased adverse air quality impacts in Morro Bay.

This is a far cry from what Duke Energy has advertised in its public brochures and advertising campaign to date or at the time the local non-binding advisory ballot measure, Proposition P, was approved. This measure asked Morro Bay residents whether the City should support a single phase project for the replacement and demolition of the existing plant if the project complies with all regulatory laws, ordinances, regulations and standards. The answer from 36% of the residents was “no.” This election occurred only two weeks after the five-volume AFC was filed with the CEC; it is safe to assume that the contents of the AFC were not read by any Morro Bay residents prior to the vote. Presumably the 64% answering “yes” assumed the new MBPP would in fact meet the promises made by Duke of improved air quality, which are simply not true.<sup>21</sup>

It is abundantly clear that not all pollutants are equal in their adverse health impacts – a ton of NO<sub>x</sub> is not equal to a ton of CO which in turn is not equal to a ton of PM<sub>10</sub> in terms of their health effects. One cannot determine whether there is an “overall improvement in air quality over existing conditions” simply by adding up increased emissions of PM<sub>10</sub> and SO<sub>2</sub> and subtracting decreases in other pollutants by tonnage. **It is the ambient concentrations that are critical and these will be increased for every pollutant.**

Emissions and concentrations of the pollutant with the worst health effects, i.e., PM<sub>10</sub> (which is already near or above the state regulatory standard), will increase significantly compared to actual historical emissions of and resulting concentrations with the existing plant, whereas the largest emission reductions are in pollutants that have lesser effects and are already only about one third (1/3) of the regulatory maximums (i.e., NO<sub>2</sub> and

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<sup>20</sup> See, Duke Energy’s responses 35, 67, 79 and 125 to CAPE’s March 9, 2001 Data Requests.

<sup>21</sup> See, Duke’s Response No. 283 (and attached literature of Duke’s statements regarding air quality) to CAPE’s March 9, 2001 Data Requests. For example, Duke representatives in various public workshops and other forums indicated the new MBPP would result in “reduced air emissions” [which is not true for SO<sub>2</sub> or PM<sub>10</sub>], or obfuscated the increase in the latter emissions by referring only to reductions in “PM<sub>10</sub> Precursor Emissions,” and “total emissions,” without ever mentioning the increased concentrations in all pollutants regardless of emission reductions in NO<sub>2</sub> and CO, until the authors raised this issue in an earlier version of this report (May 2001). In literature mailed to all Morro Bay residents on October 2, 2001, Duke claimed “air improvements” with the MBPP and repeatedly advertised Duke’s support for Proposition P, likewise claiming “improved air quality” as a promise kept to Morro Bay residents. That simply is not true.

CO). This cannot possibly be deemed to be an “overall improvement in our air quality over existing conditions” as required by the MOU, as stated as a project objective and as assumed by the residents who supported the ballot measure in favor of the project. Based on the issues addressed in this report, it is clear that the new plant cannot meet these agreed upon goals as the project is currently proposed.

Perhaps the most obvious way to eliminate the increased pollutant concentrations is to reduce the total MW output size of the new MBPP, for example by the elimination of all proposed duct firing, which would otherwise occur up to 16 hours a day and up to 4,000 hours per year per turbine (see section 6.2.1.2 of the AFC). This is the most fuel inefficient portion of the operation. Elimination of duct firing would result in a 1,032 MW new plant (which would still be 30 MW larger than the existing plant). See, sections 2.1 (p. 2-9), 2.1.1.2 (p. 2-21) and 8.3.1 (p. 8-18) and Tables 6.2-25 and 6.2-26 of the AFC. Duct firing clearly increases the level of  $PM_{10}$  emissions disproportionately to operations without duct firing and adds another 2.3 pounds of  $PM_{2.5}$  emissions per hour. Likewise permit conditions for operations could further limit the operations of even a 1,032 MW plant to require even less  $PM_{2.5}$  emissions, thereby reducing the health risks to Morro Bay residents.

Other creative ongoing mitigation measures by Duke Energy should be considered. For example, Duke Energy could commit to provide the funding or seed money for a low cost 24-hour medical clinic in Morro Bay and for mobile asthma clinics (as a smaller plant in Glendale is doing [50]), continued subsidization of such clinics, increased funding for school nurses, installation of advanced air filtration systems at all Morro Bay schools and subsidization for residents who wish to acquire advanced home air filtration systems, etc.

## APPENDIX B

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## I. Increases in Infant Mortality with Increased PM<sub>2.5</sub> Levels

A study of neonatal (ages less than one month) and post-neonatal mortality (ages 1-12 months) in the Czech Republic by Bobak and Leon (1992) [8] reported significant and robust associations between post-neonatal mortality and PM<sub>10</sub>, even after taking other pollutants into consideration. “At an absolute level, the estimated effect of a 25  $\mu\text{g}/\text{m}^3$  increase in [PM<sub>10</sub>] concentration would be to increase postneonatal respiratory mortality by a factor of 1.58.” [8, p. 1012]

More recently consistent results indicating likely early post-natal PM exposure effects on neonatal infant mortality have emerged. Woodruff et al. (1997) [148] studied the possible association of post-neonatal (over 27 days of age) mortality from ambient PM<sub>10</sub> pollution analyzing a cohort of about 4 million infants born during 1989-1991 in 86 U.S. metropolitan statistical areas. The infants were categorized as having high ( $> 40 \mu\text{g}/\text{m}^3$ ), medium (28.1 - 40.0  $\mu\text{g}/\text{m}^3$ ) or low ( $< 28.0 \mu\text{g}/\text{m}^3$ ) exposures based on tertiles of PM<sub>10</sub> averaged over the first two postnatal months.

Using logistic regression analysis adjusted for demographic and environmental factors, the authors examined the relationship between early neonatal PM<sub>10</sub> exposure and total and cause-specific post-neonatal mortality (from 1 month to 1 year of age). The odds ratio for the high versus the low exposure groups was 1.10 (i.e., a 10% excess risk). In normal birth weight infants, high PM<sub>10</sub> exposure was associated with increased mortality for respiratory causes (40% excess risk) and sudden infant death syndrome (26% excess risk). “Normal birth weight infants with high PM<sub>10</sub> exposure were 45% more likely to die of respiratory causes than normal birth weight infants with low exposure.” [148, p. 610]

The relative risk of death associated with each 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> level after adjustment for confounding variables was 4% for overall postneonatal mortality. Postneonatal respiratory mortality among normal birth weight infants increased 20% per 10  $\mu\text{g}/\text{m}^3$ , whereas postneonatal respiratory mortality among low birth weight infants increased by 5%. SIDS among normal birth weight infants increased by 12% for each 10  $\mu\text{g}/\text{m}^3$  increase in PM10. This study, however, did not consider other pollutants as possible confounders. “This ... study is important because it is the first to show that infant mortality may be associated with present-day air pollution in an affluent country with an established air pollution control program.” [71, p. 122]

The basic findings of Woodruff et al. [148] were replicated by Lipfert et al. (2000) [69] using a similar modeling approach but annual PM<sub>10</sub> air quality data for one year instead of the first two post natal months. The results of Bobak and Leon (1999) [10] likewise bolster the results of Woodruff et al. [148]. The authors conducted a matched population-based case-control study covering all births registered in the Czech Republic from 1989 to 1991 that were linked to death records. They used conditional logistic regression to estimate the effects of suspended particles and nitrogen oxides on risk of death in the neonatal and early post-neonatal period after controlling for maternal socioeconomic status and birth weight, birth length and gestational age. The effects of all pollutants were strongest in the post-neonatal period and specific for respiratory causes. The odds ratio for a 50  $\mu\text{g}/\text{m}^3$  increase in TSP was 1.95 (i.e., almost double) after controlling for all covariates for respiratory causes. When all pollutants were entered into the model, only PM showed a consistent association, suggesting that long-term exposure to PM is the pollutant most strongly associated with excess post-neonatal deaths.

Loomis et al. (1999) [71] studied infant mortality in Mexico City during 1993-1995 with additional interesting results pointing towards fine particle impacts on mortality. In Mexico City (where mean 24-hour  $PM_{2.5} = 27.4 \mu g/m^3$ ), infant mortality was associated with  $PM_{2.5}$ ,  $NO_2$  and  $O_3$  in single pollutant models, but much less consistently with  $NO_2$  and  $O_3$  than  $PM_{2.5}$  in multipollutant models. The estimated excess risk for  $PM_{2.5}$ -related infant mortality lagged 3-5 days was 18.2% per  $25 \mu g/m^3$  of  $PM_{2.5}$  or 6.9% excess infant deaths per  $10 \mu g/m^3$ . For comparison, the new MBPP will result in increased modeled maximum average daily  $PM_{2.5}$  concentrations of  $17.09 \mu g/m^3$ . The authors note: "To put these observations in context, the pollution-related excess mortality we observed among infants in Mexico City is greater in relative risk terms than the excess mortality among the elderly estimated to be associated with exposure to particulate pollutants in a recent review conducted for the US [EPA]. Moreover, the number of lost life years associated with infant deaths is large, so their epidemiologic, social, and economic impact can be far greater than that of deaths among the elderly." [71, p. 122; citations omitted]

## **II. Increases in Hospital Admissions and Emergency Room Visits with Increased $PM_{10}$ Levels**

In one of the various studies investigating the impacts of  $PM_{10}$  air pollution in the Utah Valley,<sup>22</sup> Pope (1991) [97] focused on respiratory hospital admissions associated with  $PM_{10}$  pollution in the Utah, Salt Lake and Cache Valleys during the period April 1985 through March 1989. Utah and Salt Lake Valleys had high levels of  $PM_{10}$  pollution that exceeded EPA annual and 24-hour standards; much lower  $PM_{10}$  levels occurred in the Cache valley. Pope found that bronchitis and asthma admissions for preschool-age children were approximately twice as frequent in the Utah Valley when the steel mill was operating versus when it was not. "Furthermore, per capita bronchitis and asthma admissions for all ages were approximately twice as high in Utah Valley compared to the less polluted Cache valley. During the 13-[month] period when the steel mill was closed, the differences in per capita admissions between Utah and Cache valleys narrowed considerably. Also, remarkable consistency existed in the regression results for Utah and Salt Lake valleys." [97, p. 96] In other words, the same basic associations between respiratory admissions,  $PM_{10}$  and temperature were observed in both the Utah and Salt Lake valleys.

Earlier Pope (1989) [98] had assessed the association between hospital admissions and  $PM_{10}$  in the Utah Valley during the period April 1985 through February 1988, which likewise included the closure and reopening of the local steel mill.

An association between elevated  $PM_{10}$  levels and hospital admissions for pneumonia, pleurisy, bronchitis, and asthma was observed. During months when 24-hour  $PM_{10}$  levels exceeded  $150 \mu g/m^3$  [the federal standard], average admissions for children nearly tripled; in adults, the increase in admissions was 44 percent. During months with mean  $PM_{10}$  levels greater than or equal to  $50 \mu g/m^3$  average admissions for children [aged 0 to 17 years] and adults increased by 89 and 47 percent, respectively. [98, p. 623]

During the worst winter air months, when the mill was open,  $PM_{10}$  levels were almost double the levels observed during the winter when the mill was closed, even with relatively stagnant air. Children's hospital admissions were two to three times higher during the years the mill

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<sup>22</sup> The Utah Valley during this period provides a somewhat unique, almost experimental context for these studies in that the principal source of  $PM_{10}$  pollution in the valley was an integrated steel mill built during World War II. On August 1, 1986 the mill shut down as a result of a labor strike and reopened 13 months later on September 1, 1987. [98]

was open compared to when it was closed. Using regression analyses, Pope found that  $PM_{10}$  levels were strongly correlated with hospital admissions. “They were more strongly correlated with children’s admissions than with adult admissions and were more strongly correlated with admissions for bronchitis and asthma than with admissions for pneumonia and pleurisy.” [Id.]

These Utah Valley studies are especially valuable because there was little pollution in the valley except particles, and the particles were not acid [85, 104]. Similarly, Bates et al. (1990) [6] found a significant association between asthma emergency visits in Vancouver in the summer and the previous day’s sulfate concentration in both the 0- to 14-year and 15- to 60-years age group ( $PM_{10}$  was not directly assessed there). As set forth in the AFC, SLO County has very low levels of sulfates and the  $NO_2$  and CO levels, even with the existing plant, are only about 1/3 of the state standards. PM, which currently exceeds the state standard and will increase significantly with the new plant, can itself cause significant increases in hospital admissions in children as well as adults.

More recently Gouveia and Fletcher (2000) [49] examined daily public hospital records in Sao Paulo, Brazil for children under 5 years of age for pneumonia and asthma. Children’s hospital admissions for total respiratory and pneumonia were positively associated with  $O_3$ ,  $NO_2$  and  $PM_{10}$ , with larger effects for pneumonia than for all respiratory diseases. Effects on infants (less than 1 year) gave even higher estimates. For example, pneumonia admissions for children less than 5 years old were increased by 2.5%, whereas those for infants were increased 4.7%. This confirms the excess risk pattern found earlier by Burnett et al. (1994) [18] in Ontario, Canada. They reported that the largest percentage increase in admissions was found among infants (neonatal and post-neonatal, one year or less in age).

As noted in the summary report, Schwartz et al. (1993) [124] studied the association between particulate air pollution and hospital emergency room visits for asthma in Seattle, Washington. Seattle is a hilly coastal city with a moderate climate (similar to Morro Bay) with sources of PM including wood smoke, gasoline and diesel vehicles, resuspended road dust, and industry. The authors found an increase in hospital admissions of 3.4% associated with each  $10 \mu g/m^3$  increase in  $PM_{10}$  [30, 124].  $PM_{10}$  concentrations were quite low during the study period, with a maximum 24-hour concentration of  $103 \mu g/m^3$  and a mean of  $29.6 \mu g/m^3$  (which is considerably lower than the California standard already being exceeded in SLO County). There was no significant associations between  $SO_2$  levels and asthma emergency visits.

Schwartz et al. (1993) [124] emphasize that the highly significant relationship they observed between asthma admissions and  $PM_{10}$  concentrations showed clear evidence of a dose-dependent increase and no evidence of a threshold (i.e., there is no “safe” threshold for  $PM_{10}$ ). At the mean concentration (i.e.,  $30 \mu g/m^3$ ) of inhalable particles observed in the Seattle area during the study period,  $PM_{10}$  exposure appears to have been responsible for approximately 12% of the asthma emergency visits as calculated by the authors, i.e., a “nontrivial concern. Moreover, this association occurred in a community where 24-[hour]  $PM_{10}$  concentrations never exceeded 70% of the current [federal] ambient air quality standard.” [124, p. 830] The authors further noted that “39% of the asthma visits occurred in people aged 20 or under, a population that spends more time outdoors than the average adult.” [Id.]

In a more recent Seattle study, Norris et al. (1999) [80] likewise studied the association between fine particles ( $PM_{2.5}$ ) and asthma emergency department visits for children. Asthma is the most common chronic illness of childhood and the cause of most school absences, and its prevalence is increasing [22, 42, 51, 80]. In a previous study

conducted by the authors in Seattle [128], they found a relationship between asthma visits to emergency departments in all persons under 65 years of age and air pollution levels, with the most frequent zip codes of the visits being in the inner city.

In addition, the Seattle-King County Department of Public Health (1998) published a report showing that the hospitalization rate for children in the inner city was over 600/100,000 whereas it was less than 100/100,000 for children (under 18 years of age) living in the suburbs. As a result, Norris et al. [80] evaluated whether asthma visits to six hospital emergency departments (ED) in the inner city of Seattle were associated with daily outdoor air pollution levels over a 15 month period during 1995 and 1996. “Significant associations were found between ED visits for asthma in children and fine PM and CO. A change of  $11 \mu\text{g}/\text{m}^3$  in fine PM was associated with a relative rate of 1.15 ...[i.e. a 15% increase].” [80, p. 489] The majority (54-55%) of the ED visits were for children younger than 5 years of age.

In their discussion of these results, Norris et al. (1999) [80] emphasized that they found significant associations between ED visits for asthma in children and  $\text{PM}_{10}$ , light scattering measurement of fine PM (effectively  $\text{PM}_{1.0}$ ) and CO.<sup>23</sup> They did not find a significant association between  $\text{NO}_2$ ,  $\text{SO}_2$  or ozone and increased ED asthma visits. One would thus not expect asthma ED visits to decrease as a result of reduced  $\text{NO}_2$  or CO emissions sufficiently enough to offset the effects of increased  $\text{PM}_{10/2.5}$  levels in Morro Bay. “The higher relative risk in this study as compared to the earlier results of Schwartz and colleagues [124] may be due to the fact that our population was restricted to individuals under the age of 18, a more susceptible group than the population at large.” [80, p. 492]

Addressing the fine PM levels in greater detail, Norris et al. report: “The average concentration for the 15-month period of this study was approximately  $12 \mu\text{g}/\text{m}^3$   $\text{PM}_{2.5}$ , a concentration below the new EPA annual standard ( $15 \mu\text{g}/\text{m}^3$ ).  $\text{PM}_{10}$  was associated with a 14% increase in ED asthma visits for an increase of  $12 \mu\text{g}/\text{m}^3$   $\text{PM}_{10}$ .” [Id.] This suggests that even the 1997 EPA  $\text{PM}_{2.5}$  standards are inadequate to protect the public health (see also [37, 106]).

Tolbert et al. (2000) [136] evaluated the association between  $\text{PM}_{10}$  air quality and pediatric emergency room visits for asthma in the major emergency center in Atlanta, Georgia. The estimated relative risk for  $\text{PM}_{10}$  was 1.042 per  $15 \mu\text{g}/\text{m}^3$  increase in 24-hour  $\text{PM}_{10}$ . In the authors’ view, this study supports accumulating evidence regarding the relation of  $\text{PM}_{10}$  pollution to childhood asthma exacerbation.

Using data for ambient TSP air pollutant levels, meteorologic factors, and hospitalization or ER visits for acute asthma in Singapore children over a 5-year period (1990-1994), Chew et al. (1999) [21] found positive correlations between TSP levels (which were within regulatory guidelines) and daily ER visits for asthma in children aged 3 to 12 years, but not among adolescents and young adults (aged 13 to 21 years). An adjusted increase of 5.8 ER visits for every  $20 \mu\text{g}/\text{m}^3$  increase in TSP daily atmospheric levels, lagged by one day, was observed on days with levels above  $73 \mu\text{g}/\text{m}^3$ .

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<sup>23</sup> The authors state with respect to CO: “Because CO has no biologically plausible mechanism for the exacerbation of asthma [4] we interpret it as a general indicator of air pollution.” [80, p. 492]. The authors conducted a further factor analysis and derived three factors which accounted for 95% of the variance observed. They showed “that the variability in  $\text{PM}_{2.5}$  composition is influenced by three factors: a) incomplete combustion products consisting of CO, elemental carbon, organic carbon, and soluble potassium (wood smoke marker); b) secondary aerosols consisting of ammonium and sulfate; and c) fine and coarse soil.” [Id.]

A recent study by Ilabaca et al. (1999) [55] investigated the association between levels of fine particulates and emergency room visits for pneumonia and other respiratory illnesses (REVs) among children under 15 years of age in Santiago, Chile, where there are very high levels of  $PM_{10}$  and  $PM_{2.5}$  ( $PM_{2.5}$  ranged from 10 to  $156 \mu\text{g}/\text{m}^3$  in winter, with a high in the summer of  $111 \mu\text{g}/\text{m}^3$ ). “As health end points, [hospital emergency room visits] have the advantage of reflecting adverse health events of clear clinical significance, which at the same time is more frequent in occurrence than deaths or hospital admissions and may more easily relate to recent exposure.” [55, p. PM154] The number of REVs were strongly associated with particulate levels, with stronger associations for  $PM_{2.5}$  than  $PM_{10}$  or coarse particles. “More specifically, a  $45\text{-}\mu\text{g}/\text{m}^3$  increase in the  $PM_{2.5}$  24-hr average was related to a 2.7% increase in REVs ... With a  $30.6 \mu\text{g}/\text{m}^3$  increase in the weakly mean  $PM_{2.5}$ , the increase in REVs was 3.5% ... The major effect was observed for the number of cases of pneumonia, which increased 6.7% ... with an increase of  $45 \mu\text{g}/\text{m}^3$  in  $PM_{2.5}$  24-hr average with a three-day lag.” [55, p. PM159]

$PM_{10}$  levels were also related to the risk for pneumonia. The authors observed an increase of 1.4% in ER visits for pneumonia for every increase of  $10 \mu\text{g}/\text{m}^3$  in the  $PM_{2.5}$  daily mean levels. They note that diesel exhaust has been related to a decrease in immune response, which may explain the increase in pneumonia observed among the Santiago children. “Particulate air pollution could increase the incidence of REVs by adversely affecting specific and nonspecific host defenses of the respiratory tract against pathogens in particular, by adversely affecting mucociliary clearance and macrophages [4].” [55, p., PM160; other citation omitted]

In the greater Paris, France area, where doctors still made house calls during the period 1991 to 1995 as part of the national health system, Medina et al. (1997) [73] reported that the relationship between asthma visits and air pollution was stronger for children (aged 0 to 14 years). Specifically, they observed a relative risk of 1.32 (i.e., a 32% increase) for an increase from the 5<sup>th</sup> to 95<sup>th</sup> percentile ( $5\text{-}51 \mu\text{g}/\text{m}^3$ ) in daily concentrations of black smoke, a fine particle measurement used mainly in Europe (somewhat akin to  $PM_{2.5}$ , see Dockery & Pope, 1994 [30]). Potential shortcomings of house call studies exist, e.g., home visits are determined more often by the patient’s perceived needs rather than by physician diagnoses and their validity may vary depending on the nature of the medical problem. The authors observe, however, that an asthma attack is less subject to misinterpretation than many other symptoms.

If the French national health care system home visits can be likened to an emergency service, “... Bates (1992) [5] reports that the greatest changes in health indicators, when studied in relation to air pollution, occur for self-reported symptoms, the second greatest for emergency room visits, and the smallest changes for hospital admissions. Despite their limits, emergency room visits, self-reporting, and [general practitioner] GP activity may represent less severe incidents than hospital admissions, may be more sensitive to the effect of air pollution, and may concern large numbers of patients (Dab et al., 1996 [24]).” [73, p. 81; other citations omitted] There are very few studies, however, that have related GP activity or house calls to air pollution, given the methodological difficulties involved.

Ostro et al. (1999) [84] investigated the association between  $PM_{10}$  levels in Santiago, Chile and the number of medical visits among children to primary health clinics. In Chile, almost 75% of the population are members of the public health care system, which serves primarily the lower 70% of the population income distribution. The authors conducted time-series analyses of two years of daily visits to the clinics, where counts were computed for either upper or lower respiratory symptoms and for groups of children 3 to 5 years of age and below age 2. Their multiple regression analysis indicates a statistically

significant association between  $PM_{10}$  and medical visits for lower respiratory symptoms in children aged 3 to 15 and in children under age 2.  $PM_{10}$  was also associated with visits related to both upper and lower respiratory symptoms in the older group. For children under 2, a  $50 \mu\text{g}/\text{m}^3$  increase in  $PM_{10}$  was associated with a 4 to 12% increase in lower respiratory visits. For children aged 3 to 15, the increase in lower respiratory visits ranged from 3 to 9% for a  $50 \mu\text{g}/\text{m}^3$  increase in  $PM_{10}$  and the increase in upper respiratory visits associated with a  $50 \mu\text{g}/\text{m}^3$  increase in  $PM_{10}$  was 7%. “These magnitudes are within the range of effects reported in studies undertaken in Western industrialized nations. For example, these studies suggest that a similar change in  $PM_{10}$  is associated with a 4% increase in hospital admissions, a 5% increase in emergency room visits, and a 15% increase in lower respiratory symptoms [30].” [84, p. 73]

### III. Increases in Acute Respiratory Symptoms with Increased $PM_{10}$ Levels

Panel studies have found 1-4% increases in reports of acute respiratory symptoms associated with each  $10 \mu\text{g}/\text{m}^3$  increase in ambient concentration of  $PM_{10}$  [52, 111]. In a panel study of 71 children (aged 5 to 7 years) with mild asthma who resided in the northern part of Mexico City, Romieu et al. (1996) [111] found that respiratory symptoms (coughing, phlegm production, wheezing, and difficulty breathing) were associated with  $PM_{10}$ . “An increase of  $20 \mu\text{g}/\text{m}^3$  of  $PM_{10}$  was related to an 8% increase in lower respiratory illness (LRI) among children on the same day ..., and an increase of  $10 \mu\text{g}/\text{m}^3$  in the weekly mean of  $[PM_{2.5}]$  was related to a 21% increase in LRI ...” [111, p. 300] Similarly, Romieu et al. (1997) [110] found a 2% increase in the occurrence of cough and a 5% increase in difficulty breathing with an increase of  $20 \mu\text{g}/\text{m}^3$  of  $PM_{10}$  in children (aged 5 to 13 years) with mild asthma.

Braun-Fahrländer et al. (1997) [13] assessed the impact of long-term exposure to air pollution on respiratory symptoms and illnesses in a cross-sectional study of 4,470 schoolchildren (aged 6 to 15 years) living in 10 different communities in Switzerland. “Within the range of observed  $PM_{10}$  concentrations ( $10$  to  $33 \mu\text{g}/\text{m}^3$ ), the odds of chronic cough were estimated to increase by a factor of 3.1... Similar associations with  $PM_{10}$  were observed for nocturnal dry cough apart from colds, conjunctivitis symptoms, and bronchitis.” [13, p. 1044] These results are in close agreement with those reported by the Harvard Six Cities study (Dockery et al., 1993 [31]). “Pollution levels in Switzerland at the time of our study were comparable to or even lower than those observed for the less polluted U.S. cities in the Six Cities study (i.e., some 15 to 20 yr ago) [and much less than the California 24-hour standard]. The similarities of the effect size estimated in both studies with overlapping exposure ranges might indicate that there is no threshold level for effects of air pollution on respiratory health.” [13, p. 1046]

Summer occurrences of excess particulate pollution were found to be associated with increased respiratory symptom episodes by Neas et al. (1995) [78] in a stratified sample of 83 schoolchildren living in Uniontown, Pennsylvania, and by Neas et al. (1996) [77] in a sample of 137 fourth and fifth graders living in State College, Pennsylvania. The latter study concluded that daytime exposures to summer episodes of excessive particulate pollution “are acutely associated with declines in peak respiratory flow rates and increased incidence of cough and cold episodes in children.” [77, p. 806]

Acute effects of summer air pollution on respiratory symptom reporting in schoolchildren was the focus of Schwartz et al. (1994) [123] using data from the Harvard Six Cities diary study. A change in 24-hour  $PM_{10}$  concentration from  $20$  to  $50 \mu\text{g}/\text{m}^3$  was associated with a relative odds of 1.53 for the incidence of lower respiratory symptoms (at least two of cough, chest pain, phlegm, and wheeze), a relative odds of 1.22 for the incidence of coughing, and a relative odds of 1.22 for the incidence of upper respiratory symptoms (at

least two of hoarseness, sore throat, and fever). The highest daily  $PM_{10}$  concentration was  $117 \mu\text{g}/\text{m}^3$  during the study, indicating that these relationships occurred at concentrations below the federal ambient air quality standard.

The acute effects of summer air pollution on the respiratory health of 61 asthmatic children (aged 7 to 13 years) in Amsterdam were investigated by Gielen et al. (1997) [43].  $PM_{10}$  concentrations during the study period never exceeded  $60 \mu\text{g}/\text{m}^3$ . Both black smoke and  $PM_{10}$  were associated with acute respiratory symptoms and with medication (bronchodilator) use. "These results suggest that in this panel of children, most of whom had asthma, relatively low levels of particulate matter ... in ambient air are able to increase symptoms and medication use." [43, p. 2105] Similarly TSP was associated with increased croup reports in children in an analysis of acute respiratory illness in five German cities [126].

#### **IV. Fine Particles ( $PM_{2.5}$ ) Have Been Found to Have Greater Adverse Health Effects Than $PM_{10}$**

In the Romieu et al. (1996) [111] study described above, the researchers observed similar acute respiratory health effects associated with  $PM_{10}$  and  $PM_{2.5}$  exposure. On further examination of the health effects of a comparable increase in the weekly mean of  $PM_{10}$  and  $PM_{2.5}$ , however,  $PM_{2.5}$  appeared to have a larger effect. "This suggests that cumulative exposure to fine particles ( $\#2.5\mu\text{m}$ ), which are more likely than larger ones to reach the small airways, may subchronically alter respiratory health and that  $PM_{2.5}$  levels may be a better indicator than  $PM_{10}$  levels of the potential adverse effects on particulate exposure." [111, p. 305] Schwartz et al. (1996) [122] likewise demonstrated greater effects on daily mortality from  $PM_{2.5}$  than coarse particles using the Harvard Six Cities study data.

More recently, Schwartz and Neas (2000) [121] concluded that fine particles ( $PM_{2.5}$ ) have "much stronger acute respiratory effects than coarse particles" of between 2.5 and  $10 \mu\text{m}$  in diameter (CM) in their reanalysis of data from previous studies of respiratory health among schoolchildren. Similar findings have been made in studies involving adults [31, 100] and in infants [71]. In the Harvard Six Cities diary study [31],  $PM_{2.5}$  measures were much more strongly associated with asthma-related responses (increased lower respiratory symptoms) than coarse particle mass (relative odds ratios in a two-pollutant [ $PM_{2.5}$  and CM] model were 1.29 for  $PM_{2.5}$  and 1.05 for CM).

The association between fine and ultrafine particles (ranging 0.01 to  $2.5 \mu\text{m}$  and measured with an aerosol spectrometer) and respiratory health (peak expiratory flow (PEF) and respiratory symptoms) was studied in 27 nonsmoking asthmatic adults in Erfurt, Eastern Germany (Peters et al., 1997 [89]). "Most of the particles (73%) were in the ultrafine fraction (smaller than  $0.1 \mu\text{m}$  in diameter), whereas most of the mass (82%) was in the size range of 0.1 to  $0.5 \mu\text{m}$ . ... Both fractions were associated with a decrease of PEF and an increase in cough and feeling ill during the day." [89, p. 1376] Health effects of the 5-day mean of the number of ultrafine particles were larger than those of the mass of the fine particles. Likewise, the effects of the number of ultrafine particles on PEF were stronger than those of  $PM_{10}$ .

A large number of studies of  $PM_{10}$  from different sources have found adverse respiratory effects of similar sizes [30]. Such sources have ranged from steel mills in the Utah Valley [102, 104] and in the Netherlands [35, 108] or produced by local combustion of brown coal in Eastern Europe [91] or transported to Steubenville, Ohio [78]. It has been suggested that  $PM_{10}$  might serve as a surrogate for the properties of particles or responsible for the observed health effects. Acidity of particles has been proposed as an important factor but similar results have been obtained for high acidity and low acidity

locations [89]. Seaton et al. (1995) [127] suggested instead that the number of ultrafine particles are a major factor contributing to the adverse health effects of particulate pollution. Peters et al. (1997) [89] thus elected to compare fine and ultrafine PM as well as PM<sub>10</sub> in terms of both number and mass concentrations. “Decreases in PEF and increased reporting of feeling ill during the day and of cough were associated with the number concentration and the mass concentration of the fine and ultrafine particles. As the number and the mass of the particles characterize different properties of the aerosol in the atmosphere, the goal of the analyses ... was to distinguish between their contribution to the health effects.” [89, pp. 1380-1381]

The largest decreases in PEF were observed for the 5 day means of ultrafine number concentrations. Peters et al. [89] suggest that all other observed associations might be spurious and caused only by the correlation with the number of ultrafine particles. In terms of the relation between personal and ambient outdoor concentrations, the authors note that Thatcher and Layton (1995) [134] “...were able to show that for submicron particles (0.3 to 1 $\mu$ m) in absence of indoor sources such as smoking or gas stoves the indoor/outdoor ratio was one for a California house without air conditioning.” [89, p. 1382]

It is important to note that particles and especially PM<sub>2.5</sub>, readily penetrate indoors (see, Braun-Fahrländer et al., 1997 [13]; Yu et al., 2000 [150]). The latter researchers cite a previous study that demonstrates that PM penetrates readily into a sample of homes in Seattle. “The actual air pollutant exposure level to ambient source pollutants for each study child is a function of the amount of time they spent outdoors, the pollutant-specific penetration rate, and building ventilation characteristics. ... For [particulates], ambient monitor measurements may reasonably represent personal exposure to their ambient source components.” [150, p. 1213]

## **V. PM<sub>10</sub> Levels Within Existing Regulatory Standards for Ambient Air Increase Lower Respiratory Symptoms and Medication Use in Children**

As noted above, there appears to be no safe threshold level for PM<sub>10</sub> under which no adverse health effects occur. Low levels of PM<sub>10</sub> in ambient air increase lower respiratory symptoms and medication use both in asymptomatic children as well as in symptomatic children. A recent diary study of 133 asthmatic children (aged 5 to 13 years) in Seattle, Washington by Yu et al. (2000) [150] examined both between-subject and within-subject effects of ambient air pollution. The latter refers to the effect of air pollutant excursions from typical levels in each child’s observation period on the odds of asthma symptoms. In single pollutant models, the authors found an 18% increase for a 10  $\mu$ g/m<sup>3</sup> increase in same day PM<sub>1,0</sub> and an 11% increase for a 10  $\mu$ g/m<sup>3</sup> increase in PM<sub>10</sub> lagged one day. Conditional on the previous day’s asthma symptoms, they estimated 14% and 10% increases in the odds of asthma symptoms associated with PM<sub>1,0</sub> and PM<sub>10</sub>, respectively. It is important to note that Seattle is in an air shed where SO<sub>2</sub> concentrations are very low and are not expected to aggravate asthma, as seen in some European studies.

In a study in Southern California, Delfino et al (1998) [27A] found that PM air pollution was associated with both symptoms and medication use in a panel of 25 children with asthma. Vedal et al. (1998) [140] reported that increased cough, phlegm production, and sore throat were associated with PM<sub>10</sub> in asthmatic children in Port Alberni, British Columbia, Canada during an 18-month period.

The PM results of Yu et al. [150] are consistent with findings from several previous studies. “For example, among 83 African-American children with asthma 7 to 12 years of age in Los Angeles, California, Ostro et al. [1995, cited in 150] reported a 9%

increase in the reporting of shortness of breath for a  $10 \mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{10}$ .” [150, p. 1213] In a follow-up study, Ostro et al. (2001) [82] made similar findings of a 7 to 18% increase in the onset of asthma symptoms (wheeze, cough, shortness of breath) with a  $10 \mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{10}$ . Similarly, Pope et al. (1991) [104] studied a health school-based sample of fourth and fifth graders in the Utah Valley and a sample of 34 asthmatic children who wheezed and/or were diagnosed with asthma by a doctor. The authors reported a 5.1% increase in lower respiratory disease, including trouble breathing, dry cough, and wheezing for a  $10 \mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{10}$ , even when  $\text{PM}_{10}$  levels were well below the 24 hour national ambient air quality standard. In this study, ambient concentration of other pollutants ( $\text{SO}_2$ ,  $\text{NO}_2$ , acid aerosols, and ozone) were very low. “ $\text{PM}_{10}$  pollution, therefore, was more explicitly implicated as the pollutant responsible for the observed associations.” [104, p. 673] In an earlier study of  $\text{PM}_{2.5}$  in Seattle, Koenig et al. (1993) [63] examined pulmonary function changes in 326 elementary schoolchildren (including 24 asthmatics). An approximate  $20 \mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$  was strongly associated with a decline in pulmonary function.

As part of the Children’s Health Study,<sup>24</sup> McConnell et al. (1999) [72] investigated the effect of ambient pollutants (including  $\text{PM}_{10}$ ) on the prevalence of bronchitis, chronic cough, and chronic phlegm among potentially sensitive children. The children were divided into those with a history of asthma, those with a history of wheezing but not asthma, and those with no history of either asthma or wheeze. As  $\text{PM}_{10}$  increased across communities, there was a corresponding increase in the risk of bronchitis (40% per interquartile range for both  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$ ) and all ambient pollutants except ozone were significantly associated with increased prevalence of phlegm. These results are consistent with previous studies that demonstrate acute exacerbation of childhood asthma by ambient  $\text{PM}_{10}$  pollution (unconfounded by exposure to other criteria pollutants) [97, 104, 140]. “The results of this study indicate that children with asthma are especially sensitive to the effects of air pollution in Southern California.” [72, p. 760]

Neas et al. (1994) [79] examined the effects of indoor levels of  $\text{PM}_{2.5}$  on respiratory symptoms and pulmonary function level in a cohort of white children aged 7 to 11 years who had been participants in the Harvard Six Cities study. Although specifically aimed at the risk of passive exposure to tobacco smoke, the results are enlightening with respect to  $\text{PM}_{2.5}$  in general. For 2,994 children with questionnaire-based exposure data, passive exposure to an additional pack of cigarettes smoked daily in the home was associated with a 25% increased incidence of lower respiratory symptoms. For 1,237 children with two consecutive 1-week measurements in both winter and summer, a  $30 \mu\text{g}/\text{m}^3$  increase in the annual average indoor concentration of respirable  $\text{PM}_{2.5}$  – “that is, approximately the effect of one pack per day of smoking” – was associated with a 13% increased cumulative incidence of lower respiratory symptoms. “Indoor  $\text{PM}_{2.5}$  was positively associated with restriction of activity due to chest illness and with four of the five lower respiratory symptoms [shortness of breath, persistent wheeze, chronic cough, bronchitis].” [79, p. 1094] Chest illness increased 26% for each  $30 \mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$ .

The association between acute respiratory symptoms and  $\text{PM}_{10}$  in Utah Valley were assessed by Pope et al. (1992) [102] during the winter of 1990-1991 in symptomatic and asymptomatic samples of fifth and sixth grade students. Large associations between the incidence of respiratory symptoms, especially cough, and  $\text{PM}_{10}$  pollution were observed in both samples, with the stronger association for the symptomatic

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<sup>24</sup> The Children’s Health Study is a population-based investigation of respiratory health in schoolchildren (4<sup>th</sup>, 5<sup>th</sup> and 10<sup>th</sup> graders) from 12 primarily suburban communities in Southern California (including Atascadero and Santa Maria) with different mixes of air pollutants. [41, 44, 92, 93] Atascadero, interestingly, had the highest rates of asthma among all 12 communities (22% of the study sample).

sample. “This study indicates that the respiratory health of a wide range of children may suffer acute effects of respirable particulate pollution, with symptomatic children suffering the most.” [102, p. 1127]

Studies of children in Europe have made similar findings. Roemer et al. (1993) [108] investigated acute respiratory effects of ambient air pollution in a panel of 73 children (aged 6 to 12 years) with chronic respiratory symptoms from two small, nonindustrial towns in the Netherlands. The authors found a consistent positive association between  $PM_{10}$  with the prevalence of wheeze and bronchodilator use. Their results were nearly identical to those reported by Pope et al. (1991) [104].

Van der Zee et al. (1999) [138] studied both symptomatic and asymptomatic children (aged 7 to 11 years) in urban and non-urban areas of the Netherlands. The most consistent associations were with  $PM_{10}$  measures, including significant associations between  $PM_{10}$  and the prevalence of lower respiratory tract symptoms (LRS) and decrements in PEF. Particle concentrations were also associated with use of bronchodilators in the urban areas (29% increase with  $100 \mu\text{g}/\text{m}^3$  increase in  $PM_{10}$ ), but not in the non-urban areas. “After stratification by use of medication, stronger associations were found in children who used medication than in children who did not use medication. The magnitude of the estimated effects was in the order of a twofold increase in the use of bronchodilators, a 50% increase in LRS, and an 80% increase in decrements in PEF for a  $100 \mu\text{g}/\text{m}^3$  increase in the 5 day mean  $PM_{10}$  concentration.” [138, p. 802] Significant but smaller associations were found between  $PM_{10}$  levels and decrements in PEF in children without symptoms. These results suggest that children with symptoms are more susceptible to the effects of  $PM_{10}$  than children without symptoms, and that use of medication for asthma does not prevent the adverse effects of particulate air pollution in children with symptoms.

Peters et al. (1997) [90] likewise found an increase in short-term effects of particulate air pollution (particularly  $SO_2$ ) on respiratory morbidity in asthmatic children (aged 6 to 14 years) from the Czech Republic. In a study comparing schoolchildren (aged 7 to 13 years) in two communities in Israel (one with significant industrial  $PM_{10}$  pollution and the other without such pollution), Goren et al. (1999) [48] reported that odds ratio values calculated from logistic regression controlling for a variety of potentially confounding factors were 3.6 for cough without cold, 4.0 for asthma and 2.2 for asthma and/or bronchitis in the polluted area, compared with 1.0 in the low-pollution community, which are similar to the findings of Dockery et al. (1989) [33].

As was noted earlier, Romieu et al. (1996) [111] demonstrated that an increase of  $10 \mu\text{g}/\text{m}^3$  of  $PM_{10}$  was related to a 21% increase in lower respiratory illness among children with mild asthma in Mexico City (aged 5 to 7 years). For point of reference, the AFC contemplates a modeled maximum concentration **average** daily increase of  $17.09 \mu\text{g}/\text{m}^3$  with the new plant over the existing plant.

## **VI. Impacts on Immune Function and Respiratory Effects from Long-Term PM Exposure**

Recent studies have focused on the impacts of long-term PM exposure on immune functions in children. As part of the Central European Air Quality and Respiratory Health study, Leonardi et al. (2000) [65] collected blood and serum samples from school children aged 9 to 11 years in each of 17 communities in Central Europe. They found that the number of lymphocytes increased significantly as PM concentrations increased across the cities. Lymphocytes are the body’s immunologically competent cells and their precursors. The largest effects were for  $PM_{2.5}$  with small and non-significant effects for  $PM_{10-2.5}$  (i.e., coarse particles). A similar positive relationship was found between a specific

class of immunoglobulin (IgG) concentration in serum and  $PM_{2.5}$ , but not for  $PM_{10}$  or  $PM_{10-2.5}$ . These results suggest a PM effect on immune function more strongly due to ambient fine particles than coarse particle exposure.

Boezen et al. (1999) [11] investigated whether children with bronchial hyperresponsiveness (BHR) and relatively high serum concentration of total IgE, another specific immunoglobulin, are more highly susceptible to air pollution in a study involving 479 children (aged 7 to 11 years) living in rural areas of the Netherlands. Children with BHR and elevated serum concentrations of total IgE are not necessarily identified as especially vulnerable to air pollution effects because they do not yet present themselves with chronic respiratory symptoms. Of the panel, 26% had BHR and relatively high serum total IgE. In this group of children, the prevalence of lower respiratory symptoms increased significantly by between 32% and 139% for each  $100 \mu\text{g}/\text{m}^3$  increase in  $PM_{10}$  and decreases in PEF of more than 10% in that group were more common with increased  $PM_{10}$ . The authors emphasize that although their “odds ratios were rather low (range 1.16 - 2.39), the effect of air pollution on public health is likely to be substantial since these odds ratios refer to large numbers of people.” [11, p. 877]

A number of recent studies showing correlations between total air pollution, pulmonary function and allergic airway disease suggest that pollutants act by non-specific mucosal inflammatory effects [29]. For example, PAHs from diesel exhaust particles alone can enhance in vivo IgE production in the human upper respiratory mucosa, and when in the presence of allergens, specifically enhanced antigen-specific IgE. Diaz-Sanchez (1997) [29] concluded that these results suggest that diesel exhaust particles, by initiating and enhancing IgE production, may play an important role in the increased incidence of allergic airway disease.

Most recently, Mukae et al. (2001) [75] investigated the effect of repeated exposure to  $PM_{10}$  on bone marrow using rabbits. Their findings were in accord with those of Tan et al. (2000) [133], which had suggested that an episode of acute exposure to  $PM_{10}$  causes bone marrow stimulation in humans. Mukae et al. note the studies that have shown that exposure to ambient particulate matter is related to increased cardiopulmonary morbidity and mortality. Their study was designed to measure the effect of repeated exposure to urban air particles ( $PM_{10}$ ) on the rate of production and release of polymorphonuclear leukocytes (PMN), a type of white blood cell, from the bone marrow into the peripheral blood over a three-week period. The  $PM_{10}$  exposure increased the bone marrow pool of circulating PMN (band cells) and the  $PM_{10}$  was distributed diffusely in the lung and caused a mild mononuclear inflammation. Previous studies have shown that an increase in circulating leukocyte count is a predictor of total mortality, independent of smoking in large population-based studies [75, 133] and leukocyte movement disorders are classified as an immunodeficiency disorder.

Mukae et al. (2001) [75] conclude that repeated exposure to  $PM_{10}$  stimulates the bone marrow to increase the production of PMN in the marrow and accelerate the release of more immature PMN into the circulation. The magnitude of these changes was related to the amount of particles ingested by alveolar macrophages. “These results show that chronic  $PM_{10}$  exposure induces a systemic inflammatory response that includes stimulation of the bone marrow and we postulate that this marrow stimulation is initiated by mediators released from the lung.” [75, p. 206]

As noted above, the biological mechanisms responsible for the association of  $PM_{10}$  exposure with adverse health effects (increased morbidity and mortality of respiratory and cardiovascular diseases) are not clear. “Seaton and colleagues proposed the hypothesis that the inhalation of fine particles provokes a low grade inflammatory response in the lung

that causes an exacerbation of lung disease such as asthma and COPD and change blood coagulability that results in increased pulmonary and cardiovascular deaths [127]. A previous study from our laboratory showed that a single instillation of small inert carbon particles directly into the lungs of rabbits stimulates the bone marrow and shortens the transit time of PMN through bone marrow.” [75, p. 207; other citation omitted] Tan et al. (2000) [133] demonstrated a transient increase in the number of leukocytes in the blood and an increase in circulating band cells in young military recruits exposed to an acute episode of air pollution during forest fires in Southeast Asia in the summer of 1997, suggesting that an episode of acute exposure to PM<sub>10</sub> causes bone marrow stimulation in humans. The Mukae et al. study “extends these findings by showing that the repeated deposition of low levels of particulate matter in the lung causes a systemic response that includes stimulation of the bone marrow.” [75, p. 207]

The pattern of bone marrow stimulation with repeated PM<sub>10</sub> exposure was distinctly different from that described with acute exposure to particles. Acute exposure caused an acute increase in leukocytes with a rapid release of PMN from the marrow and an accelerated transit time through all bone marrow pools [133]. In contrast, chronic PM<sub>10</sub> exposure increased the size of the bone marrow pools and released more immature PMN from the bone marrow. A similar bone marrow response occurred in rabbits repeatedly exposed to cigarette smoke. “This suggests that chronic low grade exposure to PM<sub>10</sub> stimulates the bone marrow through mechanisms that are similar to chronic cigarette smoking and that particle exposure is important in this response.” [75, p. 207]

The fact that PM<sub>10</sub> exposure causes an increase in the number of immature PMN cells in the circulation was thought to be significant by Mukae et al. (2001) [75] and Tan et al. (2000) [133]. “This increase in circulating immature PMN could be important in the pathogenesis of the heart and lung diseases associated with PM<sub>10</sub> exposure.” [75, p. 208]

## **VII. Pulmonary Function Decrements in Children Are Associated with PM<sub>10</sub> Increases**

Various studies examining effects of PM<sub>10</sub> in children have found significant decrements in peak expiratory flow (PEF) [41, 46, 52, 63, 76-78, 102, 120, 121, 138, 140], whereas others have not (e.g., Jalaludin et al., 1999 [56]<sup>25</sup>). In a four-year follow-up study to the original cross-sectional study by Peters et al. (1999) [93] as part of the Children’s Health Study, Gauderman et al. (2000) [41] modeled average growth of lung function in three groups of Southern California (including Atascadero and Santa Maria) schoolchildren (4<sup>th</sup>, 7<sup>th</sup> and 10<sup>th</sup> graders; total = 3,035) as a function of average exposure to ambient pollutants, including PM<sub>10</sub>, PM<sub>2.5</sub> and coarse particles. In the fourth grade group, significant deficits in growth of lung function (measured by FEV<sub>1</sub>, FVC, maximal midexpiratory flow [MMEF], and FEF<sub>7.5</sub>) were associated with exposures to particles. This indicates that pollutants may impair both large and small airway function, with greater deficits in small airways.

The estimated growth rate for children in the most polluted of the communities as compared with the least polluted was predicted to result in a cumulative reduction of 3.4% in FEV<sub>1</sub>, of 5.0% in MMEF and of 6.1% in FEF<sub>7.5</sub> over the 4-year study

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<sup>25</sup> This study examined the effects of the January 1994 Sydney, Australia bushfire on evening PEF rates in children with wheeze and found no association between PM<sub>10</sub> and evening PEF rate, although in a subgroup of children without bronchial hyper-reactivity, a significant negative association was present between PM<sub>10</sub> and evening PEF rate. This may have been in part as a result of medication use by symptomatic children.

period. “The estimated deficit in annual FEV<sub>1</sub> growth rate of 0.9% per year across the range of PM<sub>10</sub> exposure exceeds the 0.2% annual decrement that has been reported for passive smoke exposure in children. The results suggest that exposure to air pollution may lead to a reduction in maximal attained lung function, which occurs early in adult life, and ultimately to increased risk of chronic respiratory illness in adulthood.” [41, p. 1389; citation omitted]

The estimated deficits were generally larger for children spending more time outdoors. The estimated pollutant effects were also negative for most lung function measures in the seventh-grade and tenth-grade groups, but sample sizes were smaller and none achieved statistical significance. The authors note this indicates “that the deficits observed for children in the fourth-grade cohort are not likely to be reversed as they age through adolescence.” [41, p. 1388]

Romieu et al. (1996) [111], discussed above, also found that PEF rate was strongly associated with PM<sub>10</sub> levels. There an additional 20 µg/m<sup>3</sup> daily average PM<sub>10</sub> was associated with a 1.92 liter per minute decrease in the evening PEF rate which equates to 0.35% decrease in evening PEF with each increase of 10 µg/m<sup>3</sup> in the 24-hour PM<sub>10</sub> average. Gold et al. (1999) [46] likewise found significant associations between both PM<sub>10</sub> and PM<sub>2.5</sub> (as well as ozone) levels with reduction in PEF in schoolchildren (aged 8 to 11 years) in Mexico City.

Hoek et al. (1998) [52] reported the results of a reanalysis of data from two panel studies as to the association between PM<sub>10</sub> and decrements in pulmonary function measured in PEF rates in children. “The main findings of this study were that increases in the PM<sub>10</sub> concentration [of] 10 µg/m<sup>3</sup> were associated with an increase in the prevalence of 10% PEF decrements of 2.7%.” [52, p. 1310] The authors note that the findings of adverse effects on the more objective PEF measurements supports the report of the adverse symptom responses in panel studies (3% increase in the prevalence of lower respiratory symptoms with each 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>), and adds to the database of documented adverse effects of ambient particulate matter, including increased hospital admissions and mortality [30].

Pope et al. (1992) [102], (1991) [104]<sup>26</sup> likewise found that elevated PM<sub>10</sub> levels were associated with small but statistically significant reductions in lung function as measured by PEF in both symptomatic and asymptomatic children living in the Utah Valley, as did Koenig et al. (1993) [63] in Seattle children and Neas et al. (1995) [78], (1996) [77] in children living in Uniontown and State College, Pennsylvania. Schwartz and Neas (2000) [121] in their reanalysis of the Uniontown and State College data found that PM<sub>2.5</sub> or fine particles (which were principally sulfates in that location) were associated with decreased evening PEF rates in children. Neas et al. (1999) [76] likewise found a significant relationship between fine sulfate particles and lower morning PEF in children in Philadelphia. Vedal et al. (1998) [140] also found significant decreases in PEF with each 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> in asthmatic children. These effects were observed even at PM<sub>10</sub> concentrations below 40 µg/m<sup>3</sup> (there were also only very low levels of other air pollutants in this study location, making confounding unlikely). SLO County is near or already exceeds the state 24-hour standard of 50 µg/m<sup>3</sup>.

In their study of acute effects of summer air pollution on the respiratory health of children in Amsterdam described above, Gielen et al. (1997) [43] likewise found a significant decrease in PEF (up to 4.2% in the morning and up to 2.9% in the evening) with

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<sup>26</sup> Pope et al. (1991) [104] note as well that their study provides additional information about the structure of the association between particulate pollution and lung function. Daily PEF measurements “suggest that the deficit in pulmonary function in response to particulate pollution episodes can be seen immediately but continue to accumulate for several days.” [104, p. 673]

increased levels of PM<sub>10</sub>. The authors note that these results are comparable to those they found in an earlier study (Roemer et al., 1993 [cited in 43]) and with the meta-analysis by Dockery and Pope (1994) [30] which calculated a mean decrease in PEF of 0.08% per 10 µg/m<sup>3</sup> in ten earlier studies of particulate matter and acute effects on lung function. Timonen and Pekkanen (1997) [135] studied children (aged 7 to 12 years) in Kuopio, Finland and found that PM<sub>10</sub> was significantly associated with decline in morning PEF among asthmatic children, even though the daily concentration of PM<sub>10</sub> was only 18 µg/m<sup>3</sup> in the urban area and 13 µg/m<sup>3</sup> in the suburban area, i.e., levels substantially below those in Morro Bay even before the proposed plant expansion.

Various studies focusing on air pollution effects from diesel emissions and lung function in children are also enlightening in that such emissions strongly impact PM<sub>10</sub> levels. For example, the authors of an epidemiologic study of children in the Netherlands (Brunekreef et al., 1997 [17]) explain:

Diesel exhaust particles contain large amounts of elemental carbon (EC), which contributes much to the “blackness” of particles as measured by OECD [Organization for Economic Cooperation and Development] method for BS [black smoke]. A side-by-side comparison of BS and EC near a busy road in Berlin, Germany, in 1991 found a close relation ( $R^2 = 0.96$ ). Apparently, BS measurements represent diesel exhaust particles (as opposed to tire abrasion, which results in organic carbon) well in such situations. [17, p. 302; citation omitted]

Although not addressed by Duke Energy in its AFC, a Duke Energy consultant indicated at a CEC public workshop on February 22, 2001, that the bulk of PM<sub>10</sub> (and presumably PM<sub>2.5</sub>) emissions from the new plant will likewise consist of elemental carbon, making these studies directly relevant to this analysis (see fn 6).

The Brunekreef et al. (1997) [17] study evaluated the extent to which lung function in 1,213 children living near freeways is related to air pollution generated on the motorways. The children (aged 7 to 12 years) were from 13 schools where their lung function was measured. In addition, in 12 of 13 participating schools, indoor measurements of PM<sub>10</sub> were conducted during school hours. Truck traffic (heavily diesel) density was also monitored by the researchers. For all children living within 1000 meters of the freeways, truck traffic density was related to a decrease of 8.0% for PEF per 10,000 trucks; these effects increased in the group of children living within 300 meters of freeways. The authors also note that diesel engines (like the natural gas turbines to be used by Duke Energy) emit large numbers of ultrafine particles.

A separate analysis of the respiratory symptom data collected in this study has shown that truck traffic density and black smoke concentrations were also associated with increased reporting of chronic respiratory symptoms (van Vliet et al., 1997 [139]). “Cough, wheeze, runny nose, and doctor-diagnosed asthma were significantly more often reported for children living within 100 m from the freeway. Truck traffic intensity and the concentration of black smoke measured in schools were found to be significantly associated with chronic respiratory symptoms. These relationships were more pronounced in girls than in boys.” [139, p. 122]

Other European studies (where nearly all truck traffic as well as a significant portion of automobile traffic results in diesel emissions) have also made similar findings. Duhme et al. (1996) [34] examined the association between self-reported symptoms of asthma and allergic rhinitis (inflammation of the nasal mucous membrane) and self-reported exposure to motor vehicle traffic in adolescents (aged 12-15 years) in Münster, Germany (an administrative city with little industry). They too found positive associations between both

wheezing and symptoms of allergic rhinitis during the past 12 months and self-reported frequency of truck traffic.

In a study examining the relationship between residence near major roads, traffic flow, and risk of hospital admission for asthma in children younger than 5 years of age living in Birmingham, United Kingdom, Edwards et al. (1994) [36] found that children admitted with an asthma diagnosis were significantly more likely to live in an area with high traffic flow located along the nearest adjacent segment of main road than were children admitted for nonrespiratory reasons or children from elsewhere in the community. “This study shows that children with asthma admitted to the hospital were between 13% and 74% more likely than children in the general community to live in areas with heavy traffic flow along the nearest adjacent segment of main road. ... It appears that traffic flow – and not distance from the road – is specifically associated with hospital admission for asthma.” [36, p. 225] In considering possible explanations for the observed relationships between hospital admissions for asthma and traffic flow, Edwards et al. note that children living near roads with heavy traffic are exposed to greater concentrations of exhaust emissions. “In particular, these emissions could involve fine particulate matter and nitrogen oxides, both of which have road traffic as a major source in urban areas ... and may cause more frequent or more severe asthma attacks that lead to hospital admission.” [Id.]

An earlier German study by Wjst et al. (1993) [146] of 6,537 fourth grade children in Munich likewise found that high rates of road traffic diminish forced expiratory flow and increase respiratory symptoms in children. After adjusting for various potential confounding factors (such as passive smoke exposure), PEF significantly decreased 0.71% per every 25,000 increase in cars passing through the school district on the main road. The cumulative prevalence of recurrent wheezing at the same exposure increased by 8% and the cumulative prevalence of recurrent breathing difficulties increased by 10%. Similar results were found by Weiland et al. (1994) [143] in an analysis of self-reported wheezing and allergic rhinitis in 2,050 seventh and eighth grade schoolchildren in Bochum, Germany and traffic density on street of residence. “There was a positive correlation between the prevalence of wheezing as well as allergic rhinitis and the indicators of traffic density, controlling for age, sex, nationality, passive smoking, active smoking, parental history for asthma, and so.” [143, p. 243]

### **VIII. Increases in PM<sub>10</sub> Result in Increased Absenteeism in Elementary School Children**

Given the significant detrimental health effects on children resulting from increased PM<sub>10</sub> air pollution described above, it is not at all surprising that increases in PM<sub>10</sub> have been demonstrated to result in increased absenteeism in elementary schoolchildren. Ransom and Pope (1992) [105] specifically assessed the association between school absenteeism and PM<sub>10</sub> in the Utah Valley for the six school years of 1985 to 1990. Weekly absenteeism data from the Provo School District and daily data from a single elementary school in the Alpine School District were analyzed for kindergarten through sixth grade. PM<sub>10</sub> concentrations during the study period averaged approximately 50  $\mu\text{g}/\text{m}^3$  (equivalent to the current California 24-hour standard), with a 24-hour maximum of 365  $\mu\text{g}/\text{m}^3$ . Absenteeism was regressed on PM<sub>10</sub> pollution levels, temperature, snowfall, and variables indicating day of week, month of school year, and days preceding and following holidays and extended weekends.

Estimated associations between absenteeism and PM<sub>10</sub> pollution in both data sets were positive, statistically significant and robust.

PM<sub>10</sub> effects persisted for up to 3 or 4 weeks. Regression results from both data sets indicated that an increase in 28-day moving average PM<sub>10</sub> equal to 100 µg/m<sup>3</sup> was associated with an increase in the absence rate equal to approximately two percentage points, or an increase in overall absences equal to approximately 40%. Similar relationships were observed for all grade levels, although the response of absences to air pollution was generally greater for grades 1-3 compared with grades 4-6. [105, p. 204]

Associations between absenteeism and PM<sub>10</sub> pollution were observed even for levels below the national 24-hour standard of 150 µg/m<sup>3</sup>.

Several studies specific to the Utah Valley have evaluated the association between various indicators of health and respirable particulate pollution (PM<sub>10</sub>). These studies found substantial associations between PM<sub>10</sub> and mortality (Archer, 1990 [3]; Pope et al., 1991 [104]); respiratory hospital admissions (Pope, 1989 [98], 1991 [97]); and reported respiratory symptoms and lung function as measured by PEF (Pope et al., 1991 [104]; Pope & Dockery, 1992 [102]). In each of these studies, associations between compromised health and elevated PM<sub>10</sub> levels were below the federal ambient air quality standard (24-hour average -- 150 µg/m<sup>3</sup>).

Average absenteeism in all the schools studied was higher during time periods with relatively high PM<sub>10</sub> pollution versus periods with low PM<sub>10</sub> pollution levels. "Absenteeism at the Provo School District and Northridge Elementary averaged approximately 54 and 77% higher, respectively, on days with 28-day average PM<sub>10</sub> levels over 100 µg/m<sup>3</sup> relative to those with levels less than 50 µg/m<sup>3</sup>." [105, p. 209] During the school year when the local steel mill was closed, as compared with years when the mill was open, school absences were approximately 1% lower at the Provo School District and 0.68 percentage point lower at Northridge Elementary.

In their discussion of these results, Ransom and Pope (1992) [105] note that the highest PM<sub>10</sub> pollution levels occurred only during the winter months. "[A]bsenteeism tended to increase gradually through an individual school year except during and shortly following a high pollution episode when absenteeism would usually rise more sharply and then fall back to the previous trend." [105, p. 216] As noted above, multiple regression models consistently demonstrated strong statistically significant correlations between PM<sub>10</sub> air pollution and absenteeism, even when variables that accounted for weather, month of year, day of week, and holidays were included, and this association was consistent and robust across a wide range of model specifications and across two independent absenteeism data sets. Moreover, these associations were observed even for PM<sub>10</sub> levels below 150 µg/m<sup>3</sup>.

In the Utah Valley, pollution episodes may last for several days or even weeks and are often very close to each other, sometimes resulting in relatively high average PM<sub>10</sub> levels for several weeks at a time. "If health effects of respirable particulate pollution are determined by length of exposure as well as level of exposure, it is reasonable to expect to observe both concurrent and lagged PM<sub>10</sub> effects. The persistent lagged effects observed in this study may simply reflect that the most susceptible children will succumb to the pollution effects quickly, while others will succumb to the pollution effects only after more prolonged exposure." [105, pp. 216-217] The AFC indicates that Duke Energy intends to operate the new larger plant approximately 90% of the time (up to 7 days a week/24 hours a day). See, Sections 2.1.2 (p. 2-30) and 2.2.3.5 (p. 2-54) of the AFC.

Ransom and Pope (1992) [105] further note that, based on the estimated regression coefficient between PM<sub>10</sub> and absences and on average pollution levels in the study area, on average, about 1% of the students are absent each day as a result of exposure

to particulate air pollution. Based on the schools studied (with combined enrollments of 28,600 in 1990), PM<sub>10</sub> pollution is associated with nearly 300 school absences per day or just over 50,000 absences per school year on average in these two school districts. “On the average throughout an entire school year, this constitutes approximately 20% of all school absences.” [105, p. 217]

Studies conducted in other areas have found PM<sub>10</sub> exposure associations with reductions in pulmonary function, increased rates of bronchitis in children, increased incidence of respiratory symptoms, increased hospitalization for respiratory disease, increased absenteeism and limited activity due to illness in adults and increased mortality. Ransom and Pope (1992) [105] note that most of these studies have been confounded by the fact that during periods of high PM<sub>10</sub> pollution there were also high concentrations of SO<sub>2</sub>, NO<sub>2</sub> and/or ozone. The Utah Valley studies are somewhat unique because the association between school absences and other health endpoints and PM<sub>10</sub> has been observed in the absence of high SO<sub>2</sub>, NO<sub>2</sub> and ozone levels.

More recently, as part of the Children’s Health Study in Southern California, Gilliland et al. (2001) [44] investigated the relations between PM<sub>10</sub>, NO<sub>2</sub> and ozone and school absenteeism in a group of 1,935 fourth graders from 12 communities (including Atascadero and Santa Maria) during the first six months of 1996. Ozone levels had the highest correlation with increased school absences from both upper and lower respiratory illness with somewhat less effects of ozone in communities with high long-term average levels of PM<sub>10</sub>. Daily (24-hour) PM<sub>10</sub> was associated with all absences. “A change of 10 µg/m<sup>3</sup> in PM<sub>10</sub> was associated with a 22.8% increase in all types of school absences combined and with a 97.7% increase in non-illness-related absences, but a 5.7% increase in illness-related absences.” [44, pp. 48-49] These results are consistent with those of Ransom and Pope (1992) [105].

Other studies have likewise found associations between elevated levels of air pollution with increased prevalence of school absences (Peters et al., 1997 [90]) where particles associated with increased levels of SO<sub>2</sub> showed the largest effect estimates in the Czech Republic. Pönkä (1990) [95] studied the relationship between absenteeism and respiratory disease among children and adults in Helsinki relative to low-level air pollution. This study likewise demonstrated a significant association between levels of sulfur dioxide and absenteeism from day-care centers, schools and workplaces. As noted in the summary report, SO<sub>2</sub> pollution levels from the new plant will also increase relative to the existing plant, although such emissions are expected to be within both federal and state regulatory standards.

## **IX. Personal Exposures of Children to PM<sub>10</sub> Have Been Found to be Higher than Ambient Air Concentrations and Classroom Concentrations May be Higher Than Ambient Concentrations**

Recent studies investigating the personal exposure to inhalable particulates (PM<sub>10</sub>) in children relative to ambient concentrations have found close correlations between personal exposure and ambient PM<sub>10</sub>/PM<sub>2.5</sub>. Janssen et al. (1997) [59] investigated the validity of outdoor concentration of PM<sub>10</sub> as a measure of exposure in time series studies and studied the extent to which differences between personal and outdoor PM<sub>10</sub> concentrations can be explained. Most studies of PM<sub>10</sub> effects utilize ambient air pollution concentrations from one or more permanent fixed site monitoring stations<sup>27</sup> as a surrogate for personal exposure measurements which are largely unavailable. “If the variation in

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<sup>27</sup> Duke Energy in its AFC likewise relies on PM<sub>10</sub> concentrations measured at a single monitoring station in Morro Bay.

outdoor concentrations of particulate matter is not tightly linked to variations in personal exposures, use of outdoor concentrations as a surrogate for personal exposures would tend to misclassify personal exposures and exposure-response relations could be attenuated.” [59, p. 888; citation omitted] The authors observe that because time series studies related day to day variations in outdoor concentrations to day to day variations of health end points, the correlation between personal and ambient concentrations within a person, over time, is more relevant than the variation between people.

Four to eight repeated measurements of personal and outdoor PM<sub>10</sub> concentrations were conducted for 45 children (aged 10 to 12 years) from four schools (two in nonindustrial areas and two in Amsterdam). Repeated PM<sub>10</sub> measurements in the classrooms were conducted in three of the schools. Averaging time was 24 hours for the personal and outdoor measurements and eight hours (daytime) and 24 hours in the classroom measurements. The authors found correlations between personal and outdoor concentrations of 63% for children with parents who did not smoke and 59% for children with parents who smoked. For children with parents who did not smoke, excluding days with exposure to environmental tobacco smoke (ETS) improved the correlation to a median 73%. The mean personal PM<sub>10</sub> correlation was 105 µg/m<sup>3</sup>, which on average was 67 µg/m<sup>3</sup> (i.e., three times) higher than the corresponding outdoor concentrations. “The main part of this difference could be attributed to exposure to ETS, to high PM<sub>10</sub> concentrations in the classrooms, and to (indoor) physical activity.” [59, p. 888]

The authors conclude that these results show a reasonably high correlation between repeated personal and outdoor PM<sub>10</sub> measurements within children, thus providing support for the use of fixed site measurements as a surrogate for personal exposure to PM<sub>10</sub> in epidemiological time series studies. They observe that the large differences between personal and outdoor PM<sub>10</sub> between classroom and outdoor concentrations probably result from a child’s proximity to particle generating sources and particles resuspended by the personal activities of children.

In all schools, PM<sub>10</sub> concentrations during school hours were much higher than during non-school hours and classroom concentrations for both school hours and 24 hours were much higher than outdoor concentrations. “The PM<sub>10</sub> concentrations in the classroom were another important cause of excess personal exposures.” [59, p. 893] PM<sub>10</sub> measurements conducted in 11 other primary schools in the Netherlands have confirmed this finding (Brunekreef et al., 1997 [17]). The latter study, discussed above, found PM<sub>10</sub> concentrations in the schools were highly variable and higher than those measured outdoors. In contrast, black smoke “concentrations derived from the PM<sub>10</sub> filters were comparable with outdoor concentrations and much less variable over time.” [17, p. 300]

In their subsequent study, Janssen et al. (1999) [58] discuss these differences between personal and outdoor PM<sub>10</sub> concentrations further with respect to the finding that personal exposures to PM<sub>10</sub> were typically about 67 µg/m<sup>3</sup> higher than the corresponding ambient concentrations. “Exposure to ETS contributed approximately 20 µg/m<sup>3</sup> to this difference. Another 24 µg/m<sup>3</sup> could be explained by high PM<sub>10</sub> concentrations in the classrooms; in the three schools in which PM<sub>10</sub> measurements were conducted, classroom concentrations during school-hours were, on average, 123 µg/m<sup>3</sup>, 46 µg/m<sup>3</sup>, and 102 µg/m<sup>3</sup> higher than ambient concentrations.” [58, p. 99]

The potential bias effects of using fixed site ambient air measurements in most studies as surrogates for personal exposure was addressed by Janssen et al. (1997) [59] as follows: “With the median [Pearson’s correlation] of 0.6 found in our study, this implies that the use of outdoor concentrations would result in a threefold underestimation of the relation between exposure and disease.” [59, p. 893; emphasis added] The authors

caution, however, that this reasoning depends strongly on the assumption that personal  $PM_{10}$  concentrations are the best measure of the relevant exposure (which has been the position of various industry experts, see, e.g., Moolgavkar & Luebeck, 1996 [74]; Gamble & Lewis, 1996 [40]; Kaiser, 1997 [60])<sup>28</sup>. For example, if fine particles or a specific component in  $PM_{10}$  and not  $PM_{10}$  mass is the causal agent responsible for the health effects found, personal  $PM_{10}$  mass may not necessarily be the best exposure estimate.

In a subsequent study using similar methodology, Janssen et al. (1999) [58] further investigated the validity of ambient fine particles ( $PM_{2.5}$ ) and  $PM_{10}$  concentrations as a measure of exposure in epidemiological time-series studies by establishing the association between personal and ambient concentrations within subjects over time in 13 children (aged 10 to 12 years) in a nonindustrial town in the Netherlands. Personal exposures to fine particle ( $PM_{2.5}$ ) concentrations had an even higher correlation to ambient concentrations (median Pearson correlation coefficient = 0.86) than did personal  $PM_{10}$  concentrations in the earlier study (median Pearson correlation coefficient = 0.63) [59]. This is important in light of recent studies suggesting  $PM_{2.5}$  are more responsible than coarse particles ( $PM_{10}$  minus  $PM_{2.5}$ ) for the observed associations between particulate matter air pollution and health effects [57]. Once again the  $PM_{2.5}$  concentrations in the classroom during school hours were approximately  $5 \mu\text{g}/\text{m}^3$  higher than the 24 hour averaged classroom concentrations and did not differ significantly from ambient concentrations, and classroom concentrations were highly correlated with ambient concentrations of both  $PM_{2.5}$  and  $PM_{10}$ .

Janssen et al. (1999) [58] likewise demonstrated that personal  $PM_{2.5}$  concentrations were highly correlated with ambient  $PM_{2.5}$  concentrations in children over time. “The median of the individual correlation coefficients was .86 for all children and .92 for non-ETS-exposed children. The estimated cross-sectional correlation coefficient for all children was considerable smaller ( $R = .41$ ).” [58, p. 98] Personal  $PM_{2.5}$  concentrations were typically about  $11 \mu\text{g}/\text{m}^3$  higher than ambient concentrations. Excluding the children who were exposed to ETS, the difference was  $5 \mu\text{g}/\text{m}^3$ .

Unlike  $PM_{10}$  concentrations [59],  $PM_{2.5}$  concentrations in classrooms did not differ significantly from ambient concentrations. An important part of the remaining  $33 \mu\text{g}/\text{m}^3$  difference in increased personal exposure to  $PM_{10}$  relative to  $PM_{10}$  ambient concentrations in the Janssen et al. (1997) [59] study, in the authors’ view, “... could be attributed to physical activity. The high classroom concentrations and the influence of physical activity found for  $PM_{10}$  (and not for [ $PM_{2.5}$ ]) are probably results of resuspension of coarse particles caused by the activity of the children.” [58, p. 99] Janssen et al. (1999) [57] likewise demonstrated that high  $PM_{10}$  mass concentrations in classrooms are probably due to resuspension of coarse particles or suspension of soil material caused by the activity of the children. These results thus suggest that proximity to  $PM_{2.5}$  generating sources (such as the power plant) is the strongest predictor of personal exposure.

Janssen et al. [58] further note that  $PM_{10}$  and  $PM_{2.5}$  ambient air concentrations are generally highly correlated in areas with few sources of coarse particles. “If personal [ $PM_{2.5}$ ] concentrations are highly correlated with ambient [ $PM_{2.5}$ ] concentrations, the high correlation between ambient  $PM_{10}$  and  $PM_{2.5}$  will also result in a high correlation between ambient  $PM_{10}$  and personal [ $PM_{2.5}$  concentrations].” [58, p. 99]

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<sup>28</sup> Zeger et al. (2000) [151] (Section 1 of HEI Report 94, Part I) recently did an extensive study on methodology issues relating to  $PM_{10}$  studies and their adverse impacts on health. One of the three issues addressed was exposure measurement error that can result from using stationary monitoring sites as opposed to personal exposure measurements. The authors offered a measurement error correction model and concluded that the generic criticism that such measurement errors render the results of such time-series models uninterpretable is incorrect.

## **X. Studies Have Shown an Increased Risk of Delivering Preterm, Growth-Retarded and Low Birth Weight Infants with Increased PM<sub>10</sub>/PM<sub>2.5</sub> Levels**

The effect of air pollution, including PM<sub>10</sub>, on preterm births among children in Southern California between 1989 and 1993 was investigated by Ritz et al. (2000) [107]. They calculated crude and adjusted risk ratios for premature birth by period-specific ambient pollution levels (measured at 17 air-quality-monitoring stations). The authors observed a 20% increase in preterm birth per 50 µg/m<sup>3</sup> increase in ambient PM<sub>10</sub> levels averaged over 6 weeks before birth and a 16% increase when averaging over the first month of pregnancy. Their results suggest that increased levels of ambient PM<sub>10</sub> and possibly CO during pregnancy may contribute to the occurrence of preterm births in Southern California. “Preterm birth is an indicator of prenatal disturbances of the placenta and of fetal development. Like low birth weight (LBW), prematurity is an important predictor of infant mortality, childhood morbidity, and possibly adult morbidity.” [107, p. 502; citations omitted] Preterm birth does not necessarily result in low weight or small-for-gestational-age babies.

The biological mechanisms by which air pollutants might cause preterm births remain to be determined, but the authors note they may include disturbances of the pituitary-adrenocortico-placental system, disturbances of the uterine blood flow, and/or increased maternal susceptibility to infections.

Air pollutants contributing to these pathogenic pathways could trigger premature contractions and/or premature rupture of membranes, resulting in preterm birth. For example, PM<sub>10</sub> exposure may increase maternal susceptibility to infections during the weeks before birth. Or, toxic components of PM<sub>10</sub> or unmeasured compounds that are correlated with particulate matter such as polycyclic aromatic hydrocarbons (PAHs) from vehicular exhaust could interfere with processes affecting the development and nutrition of the fetus and cause fetal distress. If such components are inhaled and absorbed into the maternal blood stream, they could affect the placental function or cross the placenta and affect the fetus directly. [107, pp. 507-508]

Other possible mechanisms are discussed further below.

When Ritz et al. (2000) [107] examined effect estimates for PM<sub>10</sub> by season of birth or conception, their data indicated a larger effect-per-unit increase for fall and winter births and conceptions. The authors suggested the observed fall/winter effects may reflect a larger contribution of primary and secondary fine combustion-source particles common during stagnant air mass conditions. The increased emissions from the MBPP will all be of the latter type, i.e., PM<sub>2.5</sub>.

One factor that has been found to potentially impact the ultimate health of children is intrauterine growth retardation (IUGR) or small-for-gestational-age babies. Dejmek et al. (2000) [26] recently reported the results of their study of the impact of the carcinogenic fraction of polycyclic aromatic hydrocarbons (c-PAHs)<sup>29</sup> and PM<sub>2.5</sub> and PM<sub>10</sub> on pregnancy outcome in a public health study in the Czech Republic, which was supported

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<sup>29</sup> Fresh combustion PAHs originate in ultrafine nuclei mode aerosol (about 0.07 µm) that secondarily coagulate, forming larger particles (0.1 to 2 µm), which are measured within the PM<sub>2.5</sub> fraction. “Ultrafine particles contribute only about 1% to 2% of the total particle mass, but they contain 40% to 60% of PAHs mass in urban aerosols.” [153, p. 121; citations omitted] In addition to possible carcinogenic effects, there is some experimental evidence that PAHs may induce reproductive and immunologic disorders. [1, 153]

in part by U.S. EPA grants. The authors note there is growing support for the idea that adverse pregnancy outcomes may result from parental exposures to airborne pollution. Dejmek et al. (1999) [27] had observed in a prior study a consistent relationship between maternal exposure to fine particles in early gestation and IUGR in Teplice, a highly polluted district of northern Bohemia. Increases of IUGR were associated with  $PM_{10}$  levels over  $40 \mu\text{g}/\text{m}^3$  and  $PM_{2.5}$  levels over  $37 \mu\text{g}/\text{m}^3$  during early pregnancy. “One possible explanation for this finding is that rather than particles, some associated copollutant such as polycyclic aromatic hydrocarbons (PAHs) may interfere with fetal development. Most of these compounds are usually adsorbed on the surface of fine particles.” [26, p. 1159; citations omitted]

This study involved a total of 4,883 European single live births occurring in a 4 year period in Teplice (high c-PAHs and fine particles) and Prachatice (high c-PAHs but low particle levels). Detailed personal data were obtained via questionnaires and medical records and the mean  $PM_{10}$ ,  $PM_{2.5}$  and c-PAHs levels during the 9 gestational months (GM) were estimated for each of the mothers. In the 4-year sample from Teplice, previously published results about increasing IUGR risk after exposure to particles in the first GM were fully confirmed but no such effects were found in Prachatice (where there were low particles levels). The results also demonstrated that exposure to c-PAHs in early gestation may influence fetal growth. An IUGR birth was defined as birth weight below the 10<sup>th</sup> percentile, by sex and gestational week, in the general Czech population. The results were obtained after factoring in numerous possible confounding factors.

Dejmek et al. (2000) [26] cite various medical studies suggesting that IUGR is triggered by the abnormal reaction of the trophoblast with uterine tissues during the implantation period. For each gestational month, the pollutant data were divided into three categories for analysis. For example, for  $PM_{10}$  these cutoffs were low (L)  $< 40 \mu\text{g}/\text{m}^3$ ; medium (M)  $40 \text{ to } < 50 \mu\text{g}/\text{m}^3$ ; and high (H)  $\geq 50 \mu\text{g}/\text{m}^3$ .

The first gestational month was found to be the critical period for the effects of pollution on fetal growth whether related to  $PM_{10}$  or c-PAHs. “The altered growth may arise from defective trophoblast invasion, resulting in suboptimal placentation [i.e., attachment of the placenta to the uterus] and maternal hemodynamic [i.e., pertaining to the movements involved in blood circulation] maladaptation.” [26, p. 1162; citations omitted] This can alter growth and development of the fetus. See also, Dejmek et al. (1999) [27]; Ritz et al. (2000) [107]. The trophoblast is a layer of nutritive ectoderm, which is the outer layer of cells of an embryo from which the nervous system, skin, etc. are developed, by which the fertilized ovum is attached to the uterine wall and the developing embryo receives its nourishment. The authors go on to explain that “PAHs may directly affect early trophoblast proliferation due to their reaction with growth factor receptors. In this way, fetoplacental exchange and, consequently, fetal nourishment and growth may be impaired.” [26, p. 1163]

The question about the respective role of particles and c-PAHs should be studied further in the authors’ view.

[T]he results based on the Teplice data alone are consistent with the idea that the effects of c-PAHs and particles on IUGR could be combined, probably in an additive rather than synergistic manner. However, another hypothesis, that only one of the two factors – either particulate matter or c-PAHs – may be the major etiological factor, is also compatible with the results. [Id.]

Dejmek et al. [26] go on to detail various observations that support the idea of a primary role of c-PAHs and their reactive derivatives in the observed slow-down of fetal growth. Nonetheless, these

...arguments in favor of c-PAH influence on fetal growth do not rule out a simultaneous influence of particles. Airborne particles always originate in combustion processes together with polynuclear organic compounds. Thus, c-PAHs and airborne particulate matter always operate simultaneously. The critical point seems to be the proportion of organic compounds adsorbed on fine particles. The observed association between particles and IUGR could be at least partly explained by the presence of c-PAHs and their highly biologically active derivatives. Fine particles might influence the transport, penetration, and deposition of organic compounds. [26, p. 1163]

Because this issue has not been definitively resolved and because the new power plant will be emitting greater levels of fine particles than the old plant and emitting c-PAHs for the first time (see Tables 6.2-17 and 6.2-18 of Attachment 6.2-1.1 of the AFC), much more information needs to be obtained from Duke Energy addressing this public health consideration.

In a study conducted in Poland, Perera et al. (1999) [87] used biomarkers to measure the internal and bioeffective dose of toxicants and individual susceptibility factors in exploring the effects of in utero exposure to common environmental contaminants, including PAHs and particulate matter. Citing several studies suggesting that the fetus may be more prone to genetic damage and clears toxicants less efficiently than an adult, the authors explain:

In particular, the developing nervous system is an extremely sensitive target. The central nervous system (CNS) and the peripheral nervous system both comprise highly specialized organs and tissues that are vulnerable to deficits in oxygen and nutrients and to damage by toxic chemicals. During the prenatal period, the developing fetal brain undergoes tremendous growth and differentiation as cells divide and migrate to form structures in many areas simultaneously. Neurotoxicants present in fetal circulation or tissues will have ready access to these activated cells, as the blood brain barrier does not develop until after birth. [87, p. 451]

Because many brain areas are developing simultaneously, prenatal exposures to toxicants might be expected to produce rather general effects on growth and development. "Because brain development continues after birth, postnatal exposures to toxicants may be no less damaging than prenatal exposure but are likely to be more specific." [87, p. 452]

Ambient air pollution was significantly associated with the amount of PAH bound to DNA (PAH-DNA adducts) in both maternal and infant cord white blood cells (WBC). Newborns with elevated PAH-DNA adducts (i.e., greater than the median) had significantly decreased birth weight, birth length and head circumference compared to the newborns with lower adducts. Levels of PAH-DNA were higher in newborns than in mothers.

These results document that there is a significant transplacental transfer of PAH ... constituents from mother to fetus; that PAH-DNA adduct levels in maternal and newborn WBC were increased with environmental exposure to PAH from ambient pollution; and that the fetus is more sensitive to genetic damage than the mother. The study also provided the first molecular evidence that transplacental PAH exposure to the fetus is compromising fetal development. If confirmed, these findings could have

significant public health implications since a number of studies have found that reduction of head circumference at birth correlates with lower intelligent quotient as well as poorer cognitive functioning and school performance in childhood. [87, p. 451]

With respect to head circumference, the authors note that most head growth occurs during the prenatal period, with more than 60% of adult head circumference attained at birth. PAH-DNA adducts were inversely correlated with head circumference both before and after controlling for birth weight, suggesting asymmetrical growth retardation. “The finding of higher [PAH-DNA] adduct levels in the infant compared to the mother suggests increased susceptibility of the developing fetus to DNA damage.” [87, p. 457]

As Dejmek et al. (1999) [27] note, using low birth weight (LBW) or very low birth weight (VLBW) as the health outcome to be investigated tends to focus on births with shorter gestations, whereas IUGR tends to focus on small births at all gestations. Given their findings of increased infant mortality with increased PM, Bobak and Leon (1999) [9] investigated the question of whether air pollution was also related to pregnancy outcomes such as birth weight and stillbirths in the Czech Republic. Stillbirth rate was not significantly associated with any indicator of air pollution (TSP, SO<sub>2</sub>, or NO<sub>2</sub>). Crude prevalence of LBW showed highly significant associations with several socioeconomic factors. After controlling for such factors, the odds ratio per 50 µg/m<sup>3</sup> increase in TSP was 1.04. They concluded that further investigations were necessary in this area.

More recently, Rogers et al. (2000) [109] examined the association of very low birth weight with exposures to environmental SO<sub>2</sub> and TSP in 29 counties, including Atlanta, Georgia, in a population-based case-control study. VLBW babies, most of which are preterm, weigh less than 1,500 grams (approximately 3.3 pounds) at birth. Exposures less than or equal to 9.94 µg/m<sup>3</sup>, the median of TSP and sulfur dioxides combined for the controls, were used as referent exposures. Exposures to atmospheric TSP and sulfur dioxide above 56.75 µg/m<sup>3</sup> were almost 3 times as likely to result in VLBW (odds ratio 2.88) and such exposures between 25.18 and 56.75 µg/m<sup>3</sup> led to a 27% increase in VLBW. One drawback of this study is that it focused almost entirely on combined TSP/SO<sub>2</sub> levels. The authors did note, however, that when they modeled TSP alone (as to which the referent level was 5.93 µg/m<sup>3</sup>, the 75<sup>th</sup> percentile was 16.98 µg/m<sup>3</sup> and the 95<sup>th</sup> percentile was 43.6 µg/m<sup>3</sup>), the estimated odds ratio for women with exposures of more than 43.6 µg/m<sup>3</sup> was 2.36. The authors state: “From the exposure estimates, we were able to demonstrate an association between environmental TSPSO<sub>2</sub> and women’s risk of having a VLBW newborn, while controlling for many risk factors with previous links to low birth weight.” [109, p. 611]

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