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

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The adverse health impacts resulting from even small increases in ambient concentrations of inhalable particulate matter (PM_{10}) are quite significant and widely documented by public health professionals. These effects are particularly significant for the most susceptible populations, which include the elderly, those with pre-existing cardiovascular disease (CVD), those with pre-existing respiratory diseases, and those with other potentially fatal chronic illnesses such as diabetes, as well as children and infants. These effects on susceptible populations other than children are discussed in connection with the anticipated increases in PM_{10} concentrations that will result in Morro Bay, with the proposed new Morro Bay Power Plant (MBPP) compared to the existing plant. Morro Bay is a unique city in many ways, one of which is the relatively high number of elderly residents (24% of the total population).

All cause mortality resulting from increased PM_{10} concentrations is higher for this group and there are even greater increases in CVD mortality and respiratory disease mortality. This is found when examining the short term, e.g., daily mortality increases associated with daily increases in PM concentrations, as well as chronic mortality increases. These effects are supported by findings of increased risks of hospital admissions for these groups as well. These significant increases in daily mortality rates are not merely the result of mortality displacement (shortening one's life by a day or two). These significant increases in mortality risks are also linear, meaning a reduction of 1 $\mu g/m^3$ in $PM_{10}/PM_{2.5}$ has the same benefit whether the existing ambient concentration is at 15 or 90 $\mu g/m^3$.

In addition to the mortality risks, there are additive increased health effects in the form of increased hospital admissions and other measures of morbidity from increases in $PM_{10/2.5}$ of the magnitude expected with the new MBPP. These increased risks include increased hospital admissions for the elderly generally and for both CVD and various respiratory diseases, as well as for diabetes. Other documented significant adverse health effects associated with increases in $PM_{2.5}$ and PM_{10} include increased heart rate, decreased heart rate variability, increased incidences of dysrhythmia, and increased defibrillator responses, all of which are recognized risks for CVD mortality.

These increased risks will occur at a significant level in Morro Bay as a result of the increased $PM_{2.5}$ emissions and resulting concentrations from the new MBPP as compared to the existing plant. Moreover, these are all risks that can be avoided either with modifications to the MBPP as it is now proposed or with a "no project" alternative.

This report addresses the adverse health effects of particulate matter (PM) on susceptible populations other than children and infants. It then goes on to discuss related issues of mortality displacement and the existence of any thresholds below which there are no increased health risks from PM. This report should be read in conjunction with the authors' earlier report entitled "Effects of Particulate Air Pollution on Children: Potential Impacts of the Proposed New Morro Bay Power Plant" as revised December 2001. That report describes various data obtained from the pending Application for Certification (AFC) filed with the California Energy Commission by Duke Energy (and subsequent filings in that matter) regarding its proposed new Morro Bay Power Plant (MBPP). Included are comparisons of the MBPP to the existing plant in Morro Bay in terms of air pollutant emissions and resulting concentrations.

For purposes of this report, the most critical data regarding the new MBPP and the existing plant are the following:

- Overall $PM_{2.5}$ emissions from the MBPP will increase compared to the existing plant by 76 tons per year.

- The modeled maximum concentrations resulting from these increased $PM_{2.5}$ emissions in Morro Bay increase at least by 0.66 $\mu g/m^3$ (from 0.14 $\mu g/m^3$ including Morro Rock for the existing plant to 0.80 $\mu g/m^3$ with the new MBPP excluding the uninhabited Morro Rock) or by up to 0.76 $\mu g/m^3$ excluding Morro Rock in both cases, based on the annual average $PM_{2.5}$.[1]

- The modeled maximum 24-hour PM_{10} concentrations increase at least by 4.42 $\mu g/m^3$ (from 4.28 $\mu g/m^3$ including Morro Rock for the existing plant to 8.7 $\mu g/m^3$ with the new MBPP excluding Morro Rock) or by up to 8.31 $\mu g/m^3$ excluding Morro Rock in both cases.[2]

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1 This 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 $PM_{2.5}$ modeled concentration with and excluding Morro Rock (.8/2.7 $\mu g/m^3$ = 29.63%) to the existing plant maximum modeled annual $PM_{2.5}$ average concentration including Morro Rock (0.14 $\mu g/m^3$ x 29.63% = .04 $\mu g/m^3$) results in a modeled annual average increase excluding Morro Rock in both cases of 0.76 $\mu g/m^3$ (.14 - .04 = .10). 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 (450 ft. vs. 145 ft.) with both higher exhaust velocity and temperature than the new MBPP, suggesting that the portion of $PM_{2.5}$ 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.

2 This a very conservative calculation as well 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 $PM_{2.5}$ concentration including and excluding Morro Rock (8.7/.8 $\mu g/m^3$ = 9.2%) to the existing plant maximum modeled 24-hour PM_{10} average concentration (4.28 $\mu g/m^3$ x 9.2% = 0.39 $\mu g/m^3$) to derive the annual $PM_{2.5}$ average for the existing plant excluding Morro Rock results in a daily maximum increase excluding Morro Rock of 8.31 $\mu g/m^3$ (8.7 - 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.

- There is likewise a modeled annual maximum increase of 2.28 $\mu g/m^3$ of NO_2 and a 1-hour increase of 24.1 $\mu g/m^3$ in CO with the new MBPP relative to the existing plant.

- Duct firing at the new MBPP will result in disproportionately higher PM_{10} emissions than baseload operations.

The modeled annual maximum concentrations roughly approximate what the most typical average case will be in increased in concentrations from the new MBPP, but do not cover the extremes that may occur.

Susceptible Populations Other Than Children

Many studies by independent health researchers have found increased adverse health effects from inhalable particulate matter of an aerodynamic diameter of 10 microns are less (PM_{10}) and smaller or fine particles ($PM_{2.5}$) on what are generally described as susceptible subgroups of the general population.[3] These susceptible populations include the elderly, people with pre-existing cardiovascular disease ("CVD") and people with pre-existing respiratory disease, as well as children and infants.[4] For example, Schwartz and Dockery (1992) [74] demonstrated that the increased risks of death from PM was greater in the elderly, greater for chronic obstructive pulmonary disease ("COPD")[5] and pneumonia, and greater for cardiovascular disease (with about three times greater risk in the elderly) (see also, Schwartz, 1994 [66]). The elderly "are generally considered to be a sensitive subgroup because they have a higher prevalence of cardiorespiratory conditions, which together with age related declines in physiological reserves, places them at risk of dying when subject to additional factors – such as air pollution." (Bremner et al., 1999 [1], p. 244)[6]

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[3] It should be noted that these studies by and large are conducted by independent public health experts. The findings of these studies are generally reported in terms of statistical significance, meaning that the hypothesis of no effect was rejected at the 5% level. In other words, there is less than a 5% chance of estimating an effect of this size or greater just by chance. Equivalently, the effect is statistically significant if a 95% confidence interval for the effect excludes zero. Roughly speaking, a 95% confidence interval is a range of values within which it is 95% certain that the true effect lies.

[4] These are not the only populations or the only health impacts observed from PM. In addition, other health end points have been investigated with respect to fine particles ($PM_{2.5}$). For example, Korrick et al. (1998) [21] found significant deficits in pulmonary function of adult (age 18-64 years) hikers associated with $PM_{2.5}$ in the White Mountain National Forest of New Hampshire, where the median daily $PM_{2.5}$ concentration was 10 $\mu g/m^3$ with a maximum of 60 $\mu g/m^3$.

[5] Chronic obstructive pulmonary disease (COPD) is the nonspecific terminology commonly used to describe the spectrum of various diseases causing limitation of respiratory air flow, such as asthma, chronic bronchitis, and emphysema (Chen et al., 2000 [7]). Unless otherwise noted, definitions of medical terms included for the convenience of the reader are from Dorland's Illustrated Medical Dictionary (28th ed.)

[6] Interestingly, Bremner et al. (1999) [1] compared two groups of elderly persons in this regard, those aged 65 to 74 and those 75 and over, in terms of all causes of death, all respiratory deaths and all CVD deaths. They found the relative risks were generally higher in the younger elderly than in the older elderly group. However, the number of deaths was three to four times greater in the older elderly group, which means that the attributable deaths were considerably greater in the older group. Similarly, the numbers of cardiovascular deaths were two to three times higher than those of respiratory deaths. Therefore,

To place these studies in context, Samet et al. (2000) [51, 53] found a .51% increase in all cause daily mortality associated with a 10 $\mu g/m^3$ increase in PM_{10} (excluding accidental deaths and homicides) in a study examining 90 cities across the United States. Similarly, Katsouyanni et al. (2001) [19] investigated the relationship between all cause daily mortality in another multi-city study involving 29 European cities that measured PM_{10} (although in some of the cities PM_{10} was estimated from observations collected from a subset of days using collected TSP or black smoke monitors). Black smoke (BS) is considered to be an alternative measure of fine particles, particularly of elemental carbon.[7] These authors found an association between daily all-cause mortality and PM_{10} with an overall effect estimate of 0.6% per 10 $\mu g/m^3$. This study found higher levels of increased risk of 0.8% per 10 $\mu g/m^3$ for all cause mortality in the elderly.

Katsouyanni et al. (2001) [19] point out that Levy et al. (2000) [23] in a meta-analysis of 14 prior studies likewise found larger increases in mortality in populations with a proportion of persons over 65 years-of-age greater than 13% (0.77% all-cause mortality) than in those with a smaller proportion of elderly (0.64%). The latter authors estimated that overall mortality rates increased by an average 0.7% per 10 $\mu g/m^3$ increase in PM_{10}, with greater effects at sites with higher ratios of $PM_{2.5}$ to PM_{10}.

Mortality

Pre-existing cardiovascular disease is clearly a risk factor for PM-related mortality, as supported by numerous time-series studies (e.g., Samet et al., 2000 [53]; Ostro et al., 2000 [34]; Fairley, 1999 [13][8]; Schwartz, 1993 [67]) and the American Cancer Study Cancer Prevention Study ("ACS") (Pope et al., 1995 [47]) and the Harvard Six Cities Study (Dockery et al., 1993 [12]; Schwartz et al., 1996 [70]), which are chronic exposure studies. Summary estimates of increased risk of 1.4% per 10 ug/m^3 of PM_{10} have been reported for cardiovascular mortality (Schwartz & Dockery, 1992 [74, 75]; Schwartz, 1993 [67]).

Cardiovascular-specific mortality typically (with some exceptions) generates larger and more certain effect estimates for PM when compared with all-cause mortality (see, e.g., Lippman et al., 2000 [28] - total suspended particles [TSP], PM_{10}, $PM_{2.5}$; Schwartz, 1994 [66] - TSP; Schwartz & Dockery, 1992 [74] - TSP; Wichmann et al., 2000 [82] - PM_{10}, $PM_{2.5}$; Pope, 2000 [42] - $PM_{2.5}$; Daniels et al., 2000 [9] - PM_{10}; Samet et al., 2000 [51] - PM_{10}; Bremner et al., 1999 [1] - PM_{10}, black smoke; and the discussion of chronic morality studies below in "Mortality Displacement"). As pointed out by Pekkanen et al. (2000) [36] and other researchers, it is also important to bear in mind that "[a]lthough the relative risks of cardiovascular mortality are usually smaller than respiratory mortality, the largest number of excess deaths due to particulate air pollution are attributable to cardiovascular diseases, as the absolute risk of cardiovascular disease in western societies is

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although the relative risk estimates are greater for respiratory deaths, the attributable deaths are greater among cardiovascular deaths.

7 The authors opined that BS is a better marker of primary combustible particles and small particles than is PM_{10}.

8 In a study involving Santa Clara County, California, where sulfate concentrations are extremely low, Fairley (1999) [13] found a 1.1% increase for respiratory deaths for each 10 μgm^3 increase in PM_{10} and a 1.3% increase for each 10 μgm^3 increase in $PM_{2.5}$. He likewise found a 0.9% increase for cardiovascular deaths per 10 μgm^3 in PM_{10} and a 0.7% increase for each 10 μgm^3 increase in $PM_{2.5}$.

high." (p. 820; citing Dockery et al., 1993 [12]; Schwartz, 1994 [66] and Nevalainen & Pekkanen, 1998 [33]; see also, Bremner et al., 1999 [1] and fn 3 above).

Pre-existing respiratory disease likewise increases the risk for PM-related mortality and exacerbates the cardiovascular risks noted above. Various studies of acute air pollution exposure demonstrate the most likely sensitive adult subgroups. As one example, Schwartz (1994) [66] reported that respiratory conditions are more likely to be contributing causes of death on high PM days versus low PM days. Particulate matter was associated with increased deaths from respiratory conditions and increased deaths from other causes where respiratory conditions were a contributing factor.

In a recent daily mortality study in Montreal, Goldberg et al. (2000) [16] report that the association between $PM_{2.5}$ and mortality was elevated among those with acute lower respiratory disease, coronary artery disease, congestive heart failure, and any cardiovascular disease, but not those with other diseases such as upper respiratory disease, hypertension or stroke. The Goldberg et al. findings are very interesting as they relate to susceptible populations beyond those with CVD or respiratory diseases. For example, associations with both coefficient of haze (COH), which is an excellent indicator of ambient carbon, and predicted $PM_{2.5}$ (calculated from daily COH, sulfate, and airport visibility data, as well as actual $PM_{2.5}$ data where available) were reported for subjects with cancer. The interquartile range (IQR) for predicted $PM_{2.5}$ was 9.5 μgm^3. Over that IQR, predicted $PM_{2.5}$ was associated with a 1.4% increase in all cancer deaths and a 1.82% increase in lung cancer mortality.

The authors also found an association between both increased $PM_{2.5}$ and COH and increased mortality risks for a group of individuals who did not have any CVD, lower respiratory diseases or cancer. This latter group consisted of persons who had no interactions with the Canadian health care system one year before death (12%) and individuals with a wide variety of potentially fatal diseases (52%), including neurological conditions (12%), diabetes (8%), cardiac dysrhythmias (8%), dementia (6%), organic psychotic disorders (6%), and anemias (4%). Over the IQR, predicted $PM_{2.5}$ was associated with a 7.5% increase in diabetes mortality (see also, Linn et al., 2000 [25], who reported increased hospital admissions of diabetes with increased PM_{10} levels).

Bremner et al. (1999) [1] found the largest mortality effect of the air pollutants measured in their London study to be PM_{10} on respiratory mortality (4% increase in deaths of all ages for 10^{th} - 90^{th} percentile of approximately 30 $\mu g/m^3$), noting that the black smoke or fine particle fraction accounted for much of the effect of PM_{10}. Téllez-Rojo et al. (2000) [80] reported significant impacts from PM_{10} among the elderly in Mexico City, finding the total number of deaths from all respiratory causes and mortality for COPD were significantly related to $PM_{10,}$ specifically an increase of 2.9% and 4.1% respectively for a 10 $\mu g/m^3$ PM_{10} increase with a 3-day mean lag when death occurred outside of medical units. These relationships appeared linear over the range of ambient levels observed (23.4 - 175.3 $\mu g/m^3$ daily PM_{10}).

Schwartz et al. (1996) [70] specifically focused on the association between daily mortality and fine particles ($PM_{2.5}$) in six eastern cities (Boston. MA - mean $PM_{2.5}$ of 15.7 $\mu g/m^3$ and mean PM_{10} of 24.5 $\mu g/m^3$; Knoxville, TN - mean $PM_{2.5}$ of 20.8 $\mu g/m^3$ and mean PM_{10} of 32.0 $\mu g/m^3$; St. Louis, MO - mean $PM_{2.5}$ of 18.7 $\mu g/m^3$ and mean PM_{10} of 30.6 $\mu g/m^3$; Steubenville, OH - mean $PM_{2.5}$ of 29.6 $\mu g/m^3$ and mean PM_{10} of 45.6 $\mu g/m^3$; Portage, WI - mean $PM_{2.5}$ of 11.2 $\mu g/m^3$ and mean PM_{10} of 17.8 $\mu g/m^3$; and Topeka, KS - mean $PM_{2.5}$ of 12.2 $\mu g/m^3$ and mean PM_{10} of 26.7 $\mu g/m^3$). They found a 10 $\mu g/m^3$ increase in two-day mean $PM_{2.5}$ was associated with a 1.5% increase in total daily mortality with somewhat larger increases in deaths caused by COPD (3.3%), by ischemic heart disease (predominantly myocardial infarction) (2.1%) and pneumonia (4.0%) with slightly larger relative risks for those age 65 and older (overall mortality 1.7%). These findings were reanalyzed with stricter parameters on residence and place of death by Klemm et al. (2000) [20] producing similar results (1.3% vs. 1.5% increased all cause mortality for a 10 $\mu g/m^3$ increase in 2-day mean $PM_{2.5}$).

Mar et al. (2000) [29] examined the associations between air pollution and mortality outcome in elderly individuals in Phoenix using 1995-1997 data which included PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$ as well as other criteria pollutants and chemical components data. This is the first study to use detailed PM composition data in a time-series analysis of mortality. Phoenix has approximately 1 million residents (9.7% of whom are 65 or older). This compares to Morro Bay's population of approximately 24.2% elderly (based on 2000 U.S. census data). In this study, cardiovascular mortality was significantly associated with PM_{10}, $PM_{10-2.5}$ (coarse fraction), $PM_{2.5}$ and elemental carbon (EC), as well as CO, NO_2 and SO_2. All PM mass metrics were associated with an excess risk of cardiovascular death, with the strongest relation with $PM_{2.5}$ (6% increase, 1 day lag, for an interquartile increase from the 25th to the 75th percentile). $PM_{2.5}$ is the type of emission from the new and existing MBPP and the composition of the MBPP $PM_{2.5}$ emissions will be primarily EC (AFC § 2.2.3.10, p. 2-60). PM_{10} was associated with a 3% increase in all cause mortality. The overall annual range of $PM_{2.5}$ in Phoenix during 1995-1997 was 2 $\mu g/m^3$ to 39 $\mu g/m^3$.

The authors further investigated the associations between the mortality outcomes and PM by evaluating the association between the mortality outcomes and the PM composition. "The $PM_{2.5}$ composition data analysis revealed that EC and TC [total carbon] were significantly associated with cardiovascular mortality (1 day lag)." (p. 350) Both EC and TC showed a 5% increased risk for cardiovascular mortality in this regard. The authors note that the association between PM_{10} and cardiovascular mortality is consistent with previous studies (Zmirou et al., 1998 [87] - 2% per 50 $\mu g/m^3$ increase for cardiovascular mortality and 4% increase for respiratory disease mortality; Pope et al., 1992 [48]; Schwartz, 1993 [67], 1994 [66]). In addition, the association between $PM_{2.5}$ and cardiovascular mortality reported by Mar et al. is similar to that of Schwartz et al. (1996) [70], which reported a 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 1.5% increase in total mortality and a 2.1% increase in mortality from ischemic heart disease.

Preliminary findings from another location with a similar air monitoring "superstation" in Atlanta, Georgia (Tolbert et al., 2000 [81]) have likewise demonstrated particular associations between EC (which is the primary component of $PM_{2.5}$ pollutant from the MBPP) and increased risks of dysrhythmia (9.9% per 2 $\mu g/m^3$ increase in EC) and all cardiovascular deaths (4.7% per 2 $\mu g/m^3$ increase). The PM_{10} characteristics in the Atlanta study showed a mean 24-hour PM_{10} of 30.1 $\mu g/m^3$, a median of 28 $\mu g/m^3$, and a range of 16 $\mu g/m^3$ at the 10th percentile to 47 $\mu g/m^3$ at the 90th percentile. This entire range is below the current California 24-hr PM_{10} standard of 50 $\mu g/m^3$.

Similarly, in a time series study, Hoek et al. (2001) [18] found air pollution was associated with specific cardiovascular causes of death in the Netherlands. They found that deaths due to heart failure, arrhythmia, cerebrovascular causes, and thrombocytic causes were more strongly associated with air pollution than cardiovascular deaths in general. Excess relative risks were 2.5 to 4 times larger for these categories than for total cardiovascular disease mortality. Heart failure deaths made up about 10% of all cardiovascular deaths, but were responsible for about 30% of the cardiovascular deaths related to PM, SO_2, CO and NO_2. The study analyzed air pollution and mortality in the Netherlands for 1986-1994. About 43% of the total daily deaths available for analysis were cardiovascular. This is very comparable to the figures in the Mar et al. (2000) [29] study of mortality and air pollution associations in Phoenix, where cardiovascular mortality accounted for 45% of the total nonaccidental deaths in the study region, and the Bremner et al. (1999) [1] London study, where 16% of total deaths were attributed to respiratory and 43% to cardiovascular causes. "The most consistent finding that emerges from [the Hoek et al.] study is that excess relative risks for heart failure were about three times higher than for total [cardiovascular disease] for all pollutants except ozone." (p. 356)

Increased Hospital Admissions

Studies examining hospital admissions have likewise demonstrated increased risks. For example, Zanobetti et al. (2000) [84] analyzed Medicare hospital admissions for heart disease, COPD and pneumonia in Chicago, Cook County, Illinois. The authors found that acute bronchitis and pneumonia increased (almost doubling) the risk for hospital admission with cardiovascular disease. Zanobetti et al. state: "One major finding of this study is that preexisting cardiovascular disease, particularly impaired autonomic control (conduction defects and dysrhythmias) and heart failure, substantially increased the risk of respiratory admissions with airborne particles." (p. 843)[9] The authors note that another consistent pattern in their data is of acute respiratory infections increasing susceptibility to airborne particles, which was consistent with previous animal studies showing enhanced influenza infections. They further suggest that asthmatics are more susceptible to PM_{10}-induced pneumonia exacerbation or to cardiovascular effects.

Schwartz (1997) [59] focused on hospital admissions for cardiovascular disease in Tucson for the elderly (aged \$ 65 years) to replicate findings of a number of earlier studies in a location where SO_2 concentrations are low and poorly correlated to PM_{10} and where PM_{10} concentrations peak in the winter when ozone is lowest to limit potential confounding. He found admissions increased by 2.75% for an interquartile range increase of 23 $\mu g/m^3$ in average daily PM_{10} and by 2.79% for an interquartile range increase of 1.66 parts per million (ppm) of CO. These associations were independent and additive. It should be noted that both PM_{10} and CO concentrations in Morro Bay increase with the proposed new MBPP, even though CO emissions will decrease relative to the existing plant. A direct comparison between this study's results and the modeled increased concentrations of CO from the new MBPP cannot be made in that the data provided by Duke for CO increases is measured only in $\mu g/m^3$.

Subsequently, Schwartz (1999) [58] examined the consistency of the association between PM_{10}, CO and hospital admissions for cardiovascular disease across eight urban counties (including 3 in the state of Washington with almost no measurable SO_2) with differing levels of correlation between pollutants and between the pollutants and weather. Daily variation in PM_{10} was associated with hospital admissions for heart disease in the elderly (2.48% increase for an interquartile range increase of 25 $\mu g/m^3$ in exposure). This association held in both humid and dry locations and was independent of the correlation between the pollutants and weather or other pollutants. "Overall, these results suggest that air pollution may be responsible for on the order of 5% of hospital admissions for heart disease." (p. 22).

Likewise numerous other studies focusing specifically on cardiovascular disease have shown increased hospital admissions based on short term (daily) fluctuations in PM_{10} pollution (see, e.g., Schwartz and Morris, 1995 [72]). Several of these studies specifically involved cities with very low SO_2 concentrations as is the case in Morro Bay

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9 The autonomic nervous system is that independently functioning portion of the nervous system concerned with regulation of the activity of cardiac muscle, smooth muscle and glands. See, the further discussion below in "Possible Biologic Mechanisms."

(Schwartz, 1996 [60] - Spokane, WA; Schwartz, 1995 [61] - Tacoma, WA; Schwartz et al., 1995 [73] - Seattle, WA).

Linn et al. (2000) [25] also used daily time-series analysis to evaluate the relationship between PM_{10} and hospital admissions for cardiopulmonary illnesses in metropolitan Los Angeles. These authors report increased hospital admissions for both cardiovascular and pulmonary illnesses with increases in daily PM_{10} levels, with the pulmonary effects generally smaller than cardiovascular effects. "Persons $ 65 years of age and diabetics showed somewhat increased cardiovascular disease effects as compared to others without those risk factors …" (p. 434) Linn et al. (1999) [26] earlier had found a significant relationship between increased ambient PM_{10} and increased blood pressure in 30 COPD patients in Los Angeles during relatively mild autumn and winter weather conditions (with ambient PM_{10} average 24-hr mean at 33 $\mu g/m^3$, ranging from 9 to 84 $\mu g/m^3$).

Prescott et al. (1998) [49] examined the relationship between black smoke and PM_{10} and mortality and cardiopulmonary hospital emergency room admissions in a 14.5 year time study in Edinburgh, Scotland. Their most significant findings were positive associations over the period between black smoke (another measure of fine particles and especially carbon) and daily all cause mortality (1.5% increase in mortality for a 10 $\mu g/m^3$ increase in black smoke) in the elderly and respiratory mortality in the elderly (3.9% increase in mortality for a 10 $\mu g/m^3$ increase). There was likewise a significant increase (4.8%) in cardiovascular admissions of the elderly with PM_{10} during 1992-1995 (with a mean 24-hr average of 20.7 $\mu g/m^3$).

Numerous studies focusing specifically on respiratory effects have shown increased hospital admissions or emergency room visits based on short term (daily) fluctuations in PM_{10} pollution (see, e.g., Schwartz, 1996 [60] - Spokane, WA; Schwartz, 1995 [61] - New Haven, CT and Tacoma, WA; Schwartz et al., 1993 [73] - Seattle, WA; Chen et al., 2000 [7] - Reno-Sparks, NV - COPD admissions). Likewise Schwartz (1994) [64] found that PM_{10} was a risk factor for pneumonia (RR = 1.17) and COPD (RR = 1.57) in Minneapolis-St. Paul for an increase of 100 $\mu g/m^3$ in daily PM_{10}. This translated to a 1.7% increase for pneumonia and 5.7% increase for COPD per 10 $\mu g/m^3$ increase. When days excluding the National Ambient Air Quality Standard were excluded (150 $\mu g/m^3$), the association remained for both pneumonia (RR = 1.18) and COPD (RR = 1.54).

Schwartz (1994) [63] investigated the association between PM_{10} and hospital admissions for respiratory disease in the elderly in Birmingham, Alabama. He found that inhalable particles were a risk factor for admission for pneumonia (RR = 1.19 for 100 $\mu g/m^3$ increase in daily PM_{10} concentration or 1.9% increase per 10 $\mu g/m^3$) and COPD (RR = 1.27 or 2.7% increase per 10 $\mu g/m^3$). Again, significantly, tests for nonlinearity in the relation between inhalable particles (PM_{10}) and admissions were not significant and nonparametric smoothing found no evidence of a threshold in the relation.

Schwartz (1994) [62] also examined the relationship between PM_{10} and increased respiratory hospital admissions in the elderly in Detroit, and found PM_{10} was associated with daily admissions for pneumonia (1.2% increase per 10 $\mu g/m^3$), COPD and emphysema (2% increase per 10 $\mu g/m^3$), but asthma admissions were not associated with either PM_{10} or ozone. "If these associations are causal, then the difference between the mean PM_{10} concentration in Detroit during the period and the mean concentration in the least polluted city in the Harvard Six City Study (18 $\mu g/m^3$) [Dockery et al., 1993 [12]] appears to have been responsible for 3.5% of the pneumonia admissions … in the elderly and 6% of the admissions for COPD. This would represent a nontrivial public health concern." (p. 652)

Wordley et al. (1997) [83] studied the relationship between short term variations in PM_{10} concentrations and hospital admissions and mortality in Birmingham, United Kingdom for a variety of respiratory, cardiovascular and circulatory conditions. They found significant associations between all respiratory admissions, cerebrovascular admissions, and bronchitis admissions and PM_{10} on the same day. In addition, pneumonia, all respiratory admissions and asthma admissions were significantly associated with the mean PM_{10} values for the past three days. Deaths from COPD, all circulatory deaths, and all cause mortality were significantly associated with PM_{10} 24 hours previously and COPD death also with PM_{10} on the same day. The effect of a 10 $\mu g/m^3$ increase in PM_{10} was estimated to represent a 2.4% increase in respiratory admissions, a 2.1% increase in cerebrovascular admissions, a 1.7% increase in circulatory mortality, and a 1.1% increase in all cause mortality. The mean 24 hour PM_{10} there was 25.6 $\mu g/m^3$. The authors found that associations between mortality and ambient PM_{10} were of lower significance than those for hospital admissions. Significantly, the increase in risk was almost linear with no evidence of threshold. In addition, Wordley et al. (1997) [83] found that the potential number of admissions or deaths saved increased at thresholds lower than 70 $\mu g/m^3$. They also note that the estimated size of health effect is similar to that found in studies from the United States.

The Delfino et al. (1997) [10] study found mean increases in daily emergency room visits of 16% (4 to 28%) with respect to the mean levels of PM_{10} and 12% (2 to 21%) with respect to the mean levels of the 1-hour maximum $PM_{2.5}$ levels, in a study involving 25 hospitals in Montreal, Canada. This study is particularly significant because PM_{10} levels never exceeded 51 $\mu g/m^3$ (which is very close to the present 24-hour standard in California), and the substantial majority of these levels were below 30 $\mu g/m^3$, with many days at 10 $\mu g/m^3$ or below. This compares to annual ambient concentrations of PM_{10} averages of 20.6, 14.6, 15.7 and 21.4 in Morro Bay for the years 1997-2000, respectively. At least two of these years exceed the newly proposed California annual standard for PM_{10} of 20 $\mu g/m^3$.

These increased daily emergency room visits from PM_{10} and $PM_{2.5}$ were highest with respect to the elderly. These findings are consistent with those of Schwartz (1994) [63] from a hospital study of Medicare patients, average age 64, in Birmingham, AL, in which an increase of PM_{10} by 100 $\mu g/m^3$ led to a significant 19% increase in pneumonia and a 27% increase in COPD admissions, i.e., a 1.9% and 2.7% increase, respectively, per 10 $\mu g/m^3$ PM_{10} increase. Delfino et al. (1997) [10] state: "A recent review of six papers [by Dockery and Pope (1994) [11]], which examined the acute respiratory effects of particles on respiratory ER visits of hospital admissions, reported an increase of 0.5 to 3.4% for each 10 $\mu g/m^3$ increase in PM_{10}, $PM_{2.5}$, or SO_4 effect levels that are smaller than those found in the present study (9.5% increase in respiratory ER visits for the elderly per 10 $\mu g/m^3$ increase in $PM_{2.5}$)." (p. 574)

Schwartz et al. (1993) [73] earlier had focused specifically on increased emergency room visits for asthma visits in persons under age 65 in Seattle and found that relative risk for a 30 $\mu g/m^3$ increase in PM_{10} was 1.12 (or 4% for a 10 $\mu g/m^3$ increase). The mean $\mu g/m^3$ there of 29.6 was also well below the federal standard as well as the California's current standard.

Chen et al. (2000) [7] found a statistically significant correlation (4.9% increase for an interquartile change of 26.6 $\mu g/m^3$ in 24-hr average level of PM_{10}) after adjusting for the effects of weather, day of week, seasons, and time trend, in another recent study examining the relationship between increased PM_{10} and hospital admissions for COPD in Reno-Sparks, Nevada, another area like Morro Bay with little SO_2 pollution. The daily average concentration of PM_{10} was 36.55 $\mu g/m^3$ over the four year period studied, which is well below both the federal and California daily PM_{10} standards. Given the area's population of approximately 307,000 people, the authors note that for every interquartile increase (26.6 $\mu g/m^3$) in the PM_{10} level, there would be 31 more hospital admissions, clearly a nontrivial concern. The authors concluded that the consistency of their study with other studies in different locations in the U.S., Europe and other countries, and with different populations "increases the likelihood that PM_{10} levels are a good predictor of daily hospital admissions for COPD and morbidity even below the current U.S. ambient PM_{10} standard." (p. 297)

Possible Biologic Mechanisms

In the last several years, there has been increased attention to what had previously been the often under appreciated, integral structural and physiological interplay of the heart and lungs within the cardiopulmonary system. Costa (2000) [8] emphasizes that from an air pollution health research perspective, the cardiac and pulmonary systems are inextricably integrated in both structure and function. He notes in this regard that: "Indeed, cardiopulmonary impairments may become apparent as a primary dysfunction in either the heart or lungs, but some dysfunction in the other organ frequently exists – if only secondarily. This is most apparent when the condition is chronic (e.g., emphysema or congestive heart failure) where poor gas exchange or perfusion lead to varying degrees of cardiac tissue damage or remodeling, including cardiomegaly [hyperatrophy of the heart], or extravasation [escape] of plasma fluids into the alveolar interstitium, or inflammation." (p. 37)

Humans have a substantial functional range (for example, allowing vigorous exercise even at high altitudes) through the interdependent function of the heart and lungs, which can compensate for much of the insidious deterioration of cardiac and pulmonary functions associated with aging and mild disease. Accordingly, in the healthy individual, low level PM exposures might well be accommodated by the functional reserve and pass largely undetected, which may also be the case even in individuals with mild disease. "On the other hand, a more advanced disease might have necessitated a reset of homeostasis [the tendency to return to stability] at the expense of reserve, with the result being the loss of that normal functional buffer due to either a lower response threshold or exacerbated response – because of overwhelmed or insufficient negative feedback control. An individual without reserve, under sufficient biologic stress or sufficiently imbalanced (e.g., autonomic cardiac control), may simply be incapable of withstanding random or repeated inhalant (in this case PM) challenge, and hence he or she would now be 'susceptible'." (p. 38)

Although the adverse cardiopulmonary impacts from PM are well established, research is continuing into the underlying biologic mechanisms linking PM and cardiopulmonary mortality. Several recent studies have focused on heart rate variability and the effects of increased PM_{10} and $PM_{2.5}$; more specifically, these studies have investigated the impacts of particulates in light of the growing recognition over the last two decades of the importance of the autonomic nervous system in cardiovascular death. Autonomic nervous system-activated changes in heart rate (HR), blood pressure, blood viscosity, and heart rate variability (HRV) may increase the likelihood of sudden cardiac death as noted by Pope et al. (1999) [46]. That study focused specifically on evaluating autonomic nervous system-related physiologic measures and particulate levels in elderly patients in the Utah Valley (an area of relatively low SO_2 concentrations). Pope et al. report that after controlling for differences across patients, elevated particulate levels were associated with several measures, including increased mean HR and decreased HRV. The associations between HRV and PM_{10} persisted even after controlling for mean HR. The authors believe that their "findings suggest that particulate pollution may exert a deleterious influence through changes in cardiac autonomic tone." (p. 898).

Seaton et al. (1995) [78] hypothesized that particulate air pollution (especially ultrafine particles) may provoke alveolar inflammation in susceptible individuals, resulting in the release of potentially harmful cytokines and causing exacerbation of lung disease and increased blood coagulability. Air pollution has been linked to elevated levels of blood viscosity in both men and women living in Augsburg, Germany during an air pollution episode where total suspended particles (TSP) was 20% above normal (Peters et al., 1997 [40], 2000 [39]). Peters et al. (1997) [40] suggested that altered blood rheology due to inflammatory processes in the lung that include an acute-phase reaction might therefore be part of the pathological mechanisms linking air pollution to mortality.

In the more recent follow up study (Peters et al., 2000 [39]), the authors aimed at characterizing a subgroup of individuals who might be most susceptible to ambient air pollution. The resting heart rates in a cohort of 2681 men and women (aged 25-64 yr) were recorded during the winter 1984/1985 (when the air pollution episode occurred) and reexamined during the winter 1987/1988. Among persons whose plasma viscosity was above the 90th percentile, heart rates increased 5.1 beats per minute (bpm) during the air pollution episode. People with normal plasma viscosity only had an increase of 1.4 bpm during that episode. Elevated resting heart rate is a recognized risk factor for all-cause mortality as well as for cardiovascular mortality independent of other major risk factors. Elevated heart rates can serve as a marker for altered autonomic activity and thereby identify patients at higher risks of sudden deaths during ischemic events (Peters, 2000) [39].

Pekkanen et al. (2000) [36] likewise reported significant associations between increased air pollutant concentrations (including PM_{10}) and higher fibrinogen concentration in a cohort of office workers in London. Fibrinogen is an important risk factor for cardiovascular diseases, especially ischemic[10] heart disease, myocardial infarction[11] and thrombotic stroke and is a major determinant of plasma viscosity.

Gold et al. (2000) [15] likewise focused on heart rate variability and $PM_{2.5}$ in 21 active Boston residents (aged 53 to 87 yr). Mean 4-hr PM2.5 levels ranged from 3 to 49 $\mu g/m^3$. The protocol involved 25 minutes per week of continuous ECG monitoring, including 5 minutes each of rest, standing, exercise outdoors, recovery and 20 cycles of slow breathing. Significantly less heart rate variability was associated with elevated $PM_{2.5}$. As noted above, reduced HRV is a predictor of increased risk for cardiovascular mortality and morbidity, including higher risks of myocardial infarction and sudden cardiac death. This study suggests that fine particles may lead to short-term autonomic imbalance reflected by change in HR and HRV.

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[10] Ischemia is a deficiency of blood in a part, usually due to functional constriction or actual obstruction of a blood vessel.

[11] Myocardial infarction is gross necrosis of the myocardium (the middle and thickest layer of the heart wall, composed of cardiac muscle) as a result of interruption of the blood supply to the area; it is almost always caused by atherosclerosis of the coronary arteries (cholesteral plaques), upon which coronary thrombosis is usually superimposed. An infarct is an area of coagulation necrosis in a tissue due to local ischemia resulting from obstruction of circulation to the area, most commonly by a thrombus (an aggregation of blood factors, primarily platelets and fibrin with entrapineal or cellular elements, frequently causing vascular obstruction at the point of formation) or embolus (a mass of clotted blood or other formed elements, such as bubbles of air, calcium fragments, etc., brought by the blood from another vessel and forced into a smaller one).

Liao et al. (1999) [24] made similar findings regarding $PM_{2.5}$ on high pollution days (PM2.5 $ 15 $\mu g/m^3$) in a study involving 26 elderly individuals in Baltimore, MD. The authors conclude: "The data from this study suggest that elevated concentrations of $PM_{2.5}$ within an environmentally relevant range are associated with lower HRV in the elderly and that the association is more pronounced in elderly individuals with previous cardiovascular-related conditions." (p. 524) Pope et al. (1999) [45] also found increased pulse rates and increased odds of the pulse rate being elevated by 5 or 10 bpm associated with PM_{10} levels on the previous 1 to 5 days in elderly subjects in the Utah Valley. A 100 $\mu g/m^3$ increase in previous day PM_{10} was associated with an increase of 29% and 95% in the odds of experiencing an elevated pulse rates of 5 or 10 bpm, respectively. In a subset of the elderly subjects, HRV decreased with increasing PM_{10} concentrations.

Peters et al. (2000) [38] tested the hypothesis that patients with implanted cardioverter defibrillators (commonly referred to as pacemakers) experience potentially life-threatening arrhythmias (essentially any variation from the normal rhythm of the heart rate) after high air pollution episodes. The authors compared defibrillator discharge interventions among 100 patients with such devices in eastern Massachusetts according to variations in concentration of particulate matter and other pollutants. They also report that a 26 parts per billion (ppb) increase in NO_2 was associated with an 80% increased defibrillator intervention 2 days later. Patients with 10 or more interventions experienced increased arrhythmias in association with NO_2, CO, black carbon and $PM_{2.5}$. These results, in the authors' view, suggest that elevated levels of these air pollutants are associated with potentially life-threatening arrhythmia leading to therapeutic interventions by an implanted cardioverter defibrillator. "Increased heart rate and decreased [HRV] are indicators of altered autonomic control, specifically increased sympathetic stress. Raised sympathetic activity increases the risk of ventricular fibrillation,[12] a severe form of arrhythmia that, without intervention, leads to sudden death." (p. 11; citations omitted; footnote added)

Particles concentrations were modest in this study, with mean concentrations of 19.3 $\mu g/m^3$ for PM_{10} and 12.7 $\mu g/m^3$ for $PM_{2.5}$ at the South Boston site. Peters et al. (2000) [38] note in particular: "The odds of a therapeutic intervention to treat ventricular fibrillation or tachycardia [excessive rapidity in the action of the heart, e.g., 100+ bpm) in patients with at least 10 discharges nearly tripled in association with an increase of 26 ppb NO_2 and increased 60% in association with an increase in $PM_{2.5}$ concentrations of 22 $\mu g/m^3$." (p. 14) These associations were monotonical and nearly linear, and thus would equate to a 37% per each 10 $\mu g/m^3$ of $PM_{2.5}$. Defibrillator discharges did not follow exposures immediately but required an induction time of 1 or 2 days.

These findings help to explain one possible pathophysiologic mechanism for the time series analyses that have shown an association between mortality and hospital admissions for coronary disease with episodes of elevated levels of air pollution (see, e.g., Schwartz, 1999 [58]; Morris et al., 1995 [32] - examining only CO[13]; Schwartz & Morris, 1995 [72]). Stratification by diagnosis has shown specific associations between air pollution and ischemic heart diseases (Schwartz & Morris, 1995 [72] - elderly; Fairley, 1990 [14]) and congestive heart diseases (Schwartz & Morris, 1995 [72] - elderly; Morris et al., 1995 [32] - CO). Poloniecki et al. (1997) [41] tested for a significant association

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[12] Ventricular fibrillation is arrhythmia characterized by fibrillary contractions of the ventricular muscle due to rapid repetition excitation of myocardial fibers without coordinated contraction of the ventricle. Cardiac arrest is the sudden cessation of the pumping function of the heart with disappearance of arterial blood pressure, connoting either ventricular fibrillation or ventricular standstill.

[13] Morris et al. (1995) [32] found the increased risk of hospital admission for congestive heart failure associated with a 10 ppm increase in CO ranges from 10% in New York to 37% in Los Angeles.

between air pollution and emergency room hospital admissions for circulatory diseases in London, finding increased risks for acute myocardial infarction (2.5%) associated with black smoke, a surrogate for fine particles. They also found associations between black smoke and angina. The authors note in particular that cardiovascular morbidity, rather than mortality, has perhaps more consistent associations with particles. Peters et al. (2000) [38] notes that both ischemic and congestive heart disease are chronic diseases that are risk factors for acute tachycardia and ventricular fibrillation.

Applying the figures from Peters et al. (2000) [38] to Morro Bay with the modeled maximum projected 24-hour increases in $PM_{2.5}$ concentrations of 8.31 $\mu g/m^3$, excluding Morro Rock, could result in over a 30% increase in the odds of a therapeutic intervention to treat ventricular fibrillation or tachycardia. With respect to NO_2 and CO increased risks, it can only be noted at present that the modeled maximum concentrations of both pollutants resulting from the MBPP emissions increase in Morro Bay despite overall emission reductions with the new MBPP. The data provided by Duke to date is measured only in $\mu g/m^3$ increases rather than ppb or ppm.

More recently, Peters et al. (2001) [37] investigated whether high concentrations of ambient particles can trigger the onset of acute myocardial infarction (MI). The authors used a case-crossover approach to analyze the triggering of MI relative to $PM_{2.5}$ and other pollutants. This approach allows for control information for each patient based on his or her own past exposure experience. The risk of MI onset increased in association with elevated concentrations of $PM_{2.5}$ in the previous 2-hour period and an independent delayed response associated with 24-hour average exposure one day before the onset of symptoms was observed. An estimated odds ratio of 1.48 was associated with an increase of 25 $\mu g/m^3$ $PM_{2.5}$ during a 2-hour period before onset and an odds ratio of 1.69 was associated with an increase of 20 $\mu g/m^3$ $PM_{2.5}$ in the 24-hour period one day before the onset. Using the modeled maximum 24-hour increase in Morro Bay, excluding Morro Rock, of 8.31 $\mu g/m^3$ would equate to an increased risk of MI of .7%. These results are consistent with time-series analyses on hospital admissions for cardiac diseases (Burnett et al., 1995 [5]; Schwartz & Morris, 1995 [72]; Schwartz, 1999 [58], 1997 [59]; Poloniecki et al., 1997 [41]).

As described above, existing evidence suggests that most of the more severe adverse effects of PM_{10} and $PM_{2.5}$ are likely to be experienced by elderly people with preexisting heart or lung disease. In general, PM has a disproportionate effect on the elderly. For example, a study in Santiago, Chile (Ostro et al., 1996 [35]) found that 65% of all mortality occurred in those 65 and older, but 79% of the PM-related mortality occurred in this group. Similarly, in a study in Brisbane, Australia (Simpson et al., 1997 [79]), the rates were 81% and 90%, respectively.

At this point it is useful to set forth a summary of the above-described increased risks that will occur in Morro Bay as a result of the increased modeled concentrations of $PM_{2.5}$ from the increased emissions of the new MBPP as compared to the existing plant. All of these risk figures below are obtained from the studies cited above and are set forth in a range based on the lowest figure reported applied to the $PM_{2.5}$ increase (assuming conservatively that the average annual increase is the same as a typical daily $PM_{2.5}$ increase) and the highest figure reported applied to the modeled maximum daily increase of $PM_{2.5}$ in Morro Bay, in both cases excluding Morro Rock.[14]

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[14] The increased risk figures taken from PM_{10} 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 $PM_{2.5}$. In many cases, these numbers are quite comparable to the figures obtained in $PM_{2.5}$ studies directly. E.g., compare Samet et al. (2000) [51, 53] with Klemm et al. (2000) [20].

Table 1. Increased Health Risks from Increased $PM_{2.5}$ in Morro Bay

Adverse Health Impact	.76 $\mu g/m^3$ Increase	8.31 $\mu g/m^3$ Increase	Citations
Daily Mortality -			
All Cause - All Ages	.08%	1.08%	[20, 51, 53, 79]
All Cause - Elderly	.12%	4.82%	[19, 70, 80]
Respiratory - All Ages	.10%	6.65%	[1, 13, 70]
Respiratory - Elderly	.29%	6.81%	[49, 80]
CVD - All Ages	.05%	3.49%	[13, 29, 70, 83]
Cancer - All Cancers	.11%	1.25%	[16]
- Lung Cancer	.14%	1.58%	[16]
Diabetes - All Ages	.60%	6.65%	[16]
Chronic Mortality -			
All Cause - All Ages	.60 - 1.06%	N/A	[12]
Daily hospital admissions -			
Elderly - CVD	.18%	3.99%	[49, 59, 83]
Elderly - All Respiratory	.72%	7.89%	[11]
- COPD	.30%	8.97%	[62, 64]
- Pneumonia	.18%	3.16%	[62, 63]
Incidents of Dysrhythmia	3.76%	41.13%	[81]

Although some of these percentage increases may appear to be 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.

Mortality Displacement

The above described increased mortality risks for the elderly, and those with preexisting cardiovascular and/or respiratory disease, were established primarily in studies evaluating the impacts of daily or very short term time series studies. The question has been raised as to whether these observed PM_{10} mortality associations represent premature mortality by only a few days among those already near death (see, e.g., McMichael et al., 1997 [30]; Lipfert & Wyzga, 1995 [27]). This advancing of death by only a few days has been referred to by the unfortunate term "harvesting" or mortality displacement. If associations between increased mortality and PM levels reflect only short-term mortality displacement, the daily time-series studies may be showing an effect of limited public health impact (Schwartz, 2000 [56]; Schwartz, 2001 [54]; Schwartz, 2000 [55, 57]; Brunekreef & Hoek, 2000 [3]; Zeger et al., 2000 [85], 1999 [86]).

The studies noted above have undertaken investigations as to whether the mortality impacts are only mortality displacement (i.e., harvesting) or of longer term significance and have provided direct evidence for a nontrivial reduction in life expectancy by statistically controlling for the phenomenon of mortality displacement. They have shown that most air pollution-associated mortality is not due to mortality displacement.

Through the National Morbidity, Mortality and Air Pollution Study funded by the Health Effects Institute, an approach using the daily time-series data was proposed to assess short and long-term scales. "Two closely related methods for analysis of daily time-series death were developed, both testing for air pollution-mortality associations on varying time scales. The approaches are unified by the underlying concept that mortality displacement should introduce association on shorter time scales while longer term scales should be mortality-displacement resistant." (Samet et al., 2000 [52], pp. 8-9) Schwartz (2001) [54] notes the consistency of findings between the two methods is "reassuring because although both approaches (filtering in the time and frequency domain to remove short-term rebounds) are similar, they are quantitatively different." (p. 60)

Zeger and colleagues (Zeger et al., 2000 [85], 1999 [86]) conducted a simulation study using three time series of mortality generated with differing underlying assumptions about the length of residence time spent in the frail state (3, 30 and 300 days), but assuming the same relative risk from pollution. Mortality displacement introduced association between mortality and pollution only at shorter time scales. This approach applied frequency domain regression to data for Philadelphia (1974-1988).

Schwartz (2000) [55, 56] used Cleveland's STL (seasonal and trend decomposition using LOESS [locally weighted smoother]) filtering algorithm to separate the time series of daily deaths, air pollution and weather into long wave-length components, mid-scale components, and residual very short time scale components. The long term component was assumed to represent effects of time trends and seasonal fluctuations (and was thus set aside by statistical means) and the short-term components were assumed to reflect short-term mortality displacement. The remaining midscale information was used to assess the association between air pollution and mortality without confounding from longer-term effects or liability to detecting mortality displacement. The midscale components were then examined with smoothing windows of 15, 30, 45 and 60 days. Schwartz (2000) [55, 56] used data for Boston (1979-1986), and Schwartz (2001) [54] used the same methodology to analyze both daily deaths and hospital admissions in Chicago (1988-1993). Schwartz (2000) [55, 56] utilized four estimates of mortality risk (all-cause, pneumonia, COPD, ischemic heart disease).

Zeger and colleagues found little evidence for mortality displacement in assessing the effect of PM_{10} in the Philadelphia data. To the contrary, the association is present on longer time scales and tends to diminish at the shorter time scales on which mortality displacement would operate. Schwartz (2000) [55, 56], using the Boston data, found the pattern of midscale association varied by cause of death. An effect that diminished from the shorter-term to the longer-term scales was interpreted as evidencing mortality displacement, whereas persistent or increasing effects at the longer-term scales were interpreted as an effect beyond mortality displacement.

The pattern of COPD (consisting primarily of emphysema and chronic bronchitis) was consistent with mortality displacement; the pattern for pneumonia was consistent with a mixture of mortality displacement and a longer-term effect; and the patterns for ischemic heart disease and total mortality were consistent with longer-term effects without evidence for mortality displacement. More specifically, Schwartz (2000) [55, 56] found that the percent increase in all deaths associated with a 10 $\mu g/m^3$ increase in $PM_{2.5}$ rose from 2.1% to 3.75% as the focus moved from daily to monthly patterns. The equivalent pattern for total mortality was observed with frequency domain regression in Zeger et al.'s (2000) [85], (1999) [86] analyses of the Philadelphia data.

The findings of the long-term prospective cohort studies[15] of air pollution and mortality – the Harvard Six Cities Study (Dockery et al., 1993 [12]) and the ACS Study

15 Künzli et al. (2001) [22] analyzed the use of risk estimates of death attributable to air pollution based on time studies compared to cohort studies. The authors distinguished four categories of death

(Pope et al., 1995 [47]) – were considered to offer critical evidence countering the mortality displacement hypothesis (Samet et al., 2000 [52]). These studies used individual-level data of approximately 558,000 individuals in 151 cities in the United States so that other factors that affect mortality could be characterized and adjusted for in the analyses. More specifically, these studies were able to control for mortality risks associated with differences in body mass, occupational exposures, current and past smoking, alcohol use, age, and gender. After determining the effects of the individual-level factors, the models then investigated whether longer-term city-wide averages in PM (measured as PM_{10}, $PM_{2.5}$ or sulfates) were associated with different risks of life expectancies and mortality, including various cause-specific categories such as lung cancer, cardiopulmonary, and all other causes. Both of these studies report robust and statistically significant associations between several years of exposure to PM and various measures of mortality, as discussed further below.

Schwartz (2000) [55] discusses the support provided for his findings by the long-term prospective cohort studies by Dockery et al. (1993) [12] and Pope et al. (1995) [47]. In that regard, he noted that Pope et al. (1995) examined the relation between fine particle exposure on a scale of years and deaths, reporting a 10 ug/m^3 increase in $PM_{2.5}$ concentration was associated with a 6.6% increase in all-cause mortality. Pope et al.[16] "attributed the difference between that effect estimate and results such as the 2.1% estimate seen in the original time series from Boston (Schwartz et al., 1996 [413]) as suggesting a greater effect of long term exposure, possibly due to the development of chronic disease." (Schwartz, 2000 [56], p. 56)

In a commentary on the Schwartz (2000) [55] study, Brunekreef (who had been one of the authors who earlier had expressed doubt about using daily mortality studies to judge longer term impacts in McMichael et al. (1998) [30]) and Hoek (2000) [3] describes Schwartz's approach and results as remarkable. "One important conclusion is that for all-cause mortality and ischemic heart disease death, mortality displacement in this data set seems to be at least 2 months." (p. 450) The two months limitation results from the specific methodology used to avoid confounding factors such as seasonal changes. Even more intriguing to Brunekreef and Hoek (2000) [3] than the absence of mortality displacement is the finding that most effect estimates even increased with increasing window sizes. "The implication seems to be that cumulative exposures over periods of 1-2 months

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associated with air pollution: A) air pollution increases both the risk of underlying disease leading to frailty and the short term risk of death among the frail; B) air pollution increases the risk of chronic diseases leading to frailty, but is unrelated to timing of death; C) air pollution is unrelated to risk of chronic diseases, but short term exposure increases mortality among persons who are frail; and D) neither underlying chronic disease nor the event of death is related to air pollution exposure. Time-series approaches capture deaths from categories A and C, whereas cohort studies access cases from categories A, B and C. The authors further note that years of life lost can only be derived from cohort studies, where time to death is the outcome, while in time-series studies, death is a once-only event (with no dimension of time assessed). Overall, Künzli et al. (2001) [22] conclude that time-series analyses underestimate the cases of death attributable to air pollution.

16 This study (Pope et al., 1995 [47]) using the ACS cohort and the Harvard Six Cities Study (Dockery et al., 1993 [12]) estimated mortality effects of approximately 4 to 7% per 10 μg/m^3 increase of long-term exposure to PM_{10} are much larger than those effects associated with daily exposure (approximately 1% per 10 ug/m^3 overall). These studies likewise provided a basis for calculating reductions in life expectancy associated with PM exposure. The results suggest that the 24 ug/m^3 difference in $PM_{2.5}$ between the cleanest and dirtiest cities in the studies is associated with about 1.5 years difference in life expectancy in the two cities (Pope, 2000 [42]).

are actually more harmful (in terms of the daily number of deaths associated with them) than short exposures of similar magnitude." [Id.] These authors applaud this approach as moving beyond the "body count" approach that is so prevalent in the popular press' interpretation of time series studies on air pollution and death to what is really important in their view, i.e., the impact of air pollution on life expectancy.

Brunekreef (1997) [2] used a life table for men in the Netherlands and estimated a difference of 1.11 years in life expectancy between the two extreme cities in the ACS study, whose median $PM_{2.5}$ concentrations range from a 9.0 ug/m^3 to 33.5 ug/m^3 (Pope et al., 1995 [47]). When the life tables were extended to include two more five-year age groups, overall life expectancy decreased further from 1.11 to 1.51 years. A similar analysis using the life table of United States white men resulted in an estimated difference in life expectancy of 1.31 years. Moreover, the difference in life expectancy of a person who actually died from diseases associated with $PM_{2.5}$ was estimated to be about 10 years (Künzli et al., 2001 [22]). This is because air pollution-related deaths only make up a small fraction of total deaths in any given city.

The likelihood of significant loss in life expectancy is reinforced by studies indicating that deaths occurring outside of a hospital had larger (two- to four-fold) and stronger associations with PM than did deaths occurring inside hospitals (Schwartz, 2001 [54]; Schwartz, 2000 [57]). These results are important because they suggest that some of the PM_{10} impacts occur among a subgroup that is not under intensive medical care, and thus may not necessarily be at the end-stage of their disease.

Schwartz (2001) [54] examined harvesting or morbidity displacement using the Chicago data for both mortality and hospital admissions using his methodology described above. In baseline analyses, a 10 ug/m^3 increase in PM_{10} was associated with increased daily deaths (0.89%) and hospital admissions for heart disease (1.27%), pneumonia (1.45%) and COPD (2.00%). Using smoothing to look at the effects net of short-term rebounds (i.e., mortality displacement), the effect-size estimates for daily deaths outside of the hospital increased (four-fold) and for COPD hospital admissions more than doubled and remained unchanged for pneumonia and heart disease admissions. "These results are consistent with air pollution increasing the size of the risk pool and for most of the deaths being advanced by months to years." (p. 55)

PM_{10} was associated with increased risk of all of the death outcomes (all deaths, deaths in hospital, deaths out of hospital) and admissions outcomes (heart disease, COPD, pneumonia). These mortality results confirmed those previously reported in Boston and Philadelphia for mortality that when short-term mortality displacement effects are averaged out, the effect size of PM increases rather than decreases. "Moreover, this finding is supported by the alternative approach of examining deaths inside and outside of the hospital separately. Deaths in the hospital, where short-term harvesting is most likely, are less sensitive to air pollution than are deaths outside of the hospital." (p. 59)

In his 10 city analysis, Schwartz (2000) [57] likewise found that daily deaths associated with a 10 ug/m^3 increase in PM_{10} occurring outside of hospitals was almost double (0.89%) that of increased deaths in hospitals (0.49%). "This suggests that most of the PM_{10}-associated deaths are not in people who are desperately ill and hence that, in most cases, increased mortality is not a result of time of death simply being reduced by a few days." (p. 566) These findings demonstrate that sudden death may thus be an important factor in PM_{10}-related mortality, i.e., it effects more than those who are already chronically ill and hospitalized.

Schwartz (2001) [54] notes regarding his mortality displacement model that his results would follow if air pollution were to increase the net recruitment rate in the risk pool by more than the death rate our of the risk pool. If the airborne particles can only deplete the pool of susceptibles as in pure mortality displacement, his results would be inexplicable. He explains: "Air pollution could increase the size of the risk pool or, viewed continuously, shift the susceptibility of the population [closer to death] if it, for example, increased the intensity of infectious illness. Air pollution has been associated with increases in physician visits [Schwartz et al., 1991 [76]] and hospital admissions [Pope, 1989 [44]] for infectious respiratory illness and with increased school absences in children [Ransom & Pope, 1992 [50]]." (p. 59) He cites various animal studies involving pneumonia and influenza infections and ambient PM that support this explanation as well. Schwartz (2001) [54] likewise cites the recent epidemiologic studies described above in "Suscepticle Populations Other than Children – Possible Biologic Mechanisms" reporting that airborne particles are associated with changes in heart rate variability and increased defibrillation discharges. "Hence, there is a biological basis for believing that airborne particle exposure increases the risk of sudden death, which may then be triggered by other factors. That is, it may increase the risk pool." (p. 60). In addition, air pollution may reduce the transition out of the risk pool to the healthier population (see, Schwartz, 1992 [68] reporting increased duration of respiratory illnesses with increased pollution).

In Schwartz's (2001) [54] view, the analysis of deaths inside and outside of the hospital is informative in two ways.

> First, the baseline results themselves speak to the harvesting issue. Persons who are lingering on the edge of death, who will die in a few days if they do not die today, are disproportionately in the hospital today. If most air pollution-related deaths were short-term harvesting, we would expect a greater impact on this population. In fact, airborne particles had a greater impact on deaths outside of the hospital in Chicago. … Moreover, the increase in effect size seen as we move to longer time scales was seen only for the out-of-hospital deaths. This result is consistent with particle exposure increasing the risk of sudden death (due to other triggers) in ambulatory subjects with apparently good health but underlying heart disease. (p. 60)

The latter should be of direct interest to those in Morro Bay (with its 24% elderly population) who do not already exhibit severe disease symptoms. Finally, evidence of a significant loss of life-years from air pollution is provided by the studies of infants and children addressed in detail in Churney and Soderbeck's (2001) report entitled "Effects of Particulate Air Pollution on Children: Potential Impacts of the Proposed New Morro Bay Power Plant."

Absence of Thresholds

No threshold of response has been observed in the PM-mortality studies. Several direct and indirect approaches described further below have consistently found that non-threshold, linear models (i.e., the relationship between mortality and PM_{10} level is a monotonic or a straight line relationship) provide the best fit to the data. This is critical to an understanding of the cumulative impacts of the increased $PM_{2.5}$ concentrations resulting from the proposed MBPP above the levels existing previously in Morro Bay. This means that the effects of particulates are linear, i.e., the dose-response function is a straight line relation. Thus, not only is there no threshold, but any reduction, even starting from relatively "low" ambient concentrations is beneficial. For example, a linear dose-response relation implies that the benefit from a reduction of an initial ambient PM_{10} concentration of 20 $\mu g/m^3$ to 10 $\mu g/m^3$ is as great as it is when going from 80 $\mu g/m^3$ to 70 $\mu g/m^3$. The data set forth in the AFC Table 6.2-37 (as revised by the Applicant's response to CAPE's Data Request No. 26) indicates the PM_{10} annual concentration in Morro Bay was 20.6 ug/m^3 in 1997 and 21.4 ug/m^3 in 2000, which generally reflects the mean average daily PM_{10} levels, and would violate the currently proposed California annual standard of 20 $\mu g/m^3$.

For short-term exposure to PM, there are two methods available to address the issue of the existence of a threshold, i.e., an ambient level of PM below which there would be no risk of a significant adverse health outcome. First, the existence of a threshold can be examined indirectly by considering data sets with very low mean ambient concentrations. The second method of determining the existence of a threshold for PM effects is direct by developing statistical tests that carefully model the shape of the concentration-response function. Studies using both of these approaches consistently indicate the lack of an observable threshold.

As to the first method, several studies have been conducted in cities with low ambient PM_{10} concentration, including Morgan et al. (1998) [31] for Sydney, Australia (mean = 18 ug/m^3), Gwynn et al. (2000) [17] for Buffalo and Rochester (mean = 24 ug/m^3), Schwartz et al. (1996) [70] for the Harvard Six Cities (mean = 25 ug/m^3), Burnett et al. (2000) [4] for the eight largest Canadian cities (PM_{10} mean = 26 ug/m^3; $PM_{2.5}$ mean = 13.3 ug/m^3), and Wordley et al. (1997) [83] for Birmingham, UK (mean = 25 ug/m^3). Several cities used in the 90 cities data set used by Samet et al. (2000) [53] likewise had mean concentrations in the mid-teens and low twenties (e.g., Honolulu - mean = 15.3 ug/m^3; Albuquerque - mean = 16.9 ug/m^3; Madison - mean = 19.9 ug/m^3). The results consistently indicate that the concentration-response functions are not driven by the high concentrations and that the slopes of these functions do not appear to increase significantly at higher concentrations.

Among the statistical approaches, Schwartz et al. (2000) [57] examined the concentration-response relationship in 10 U.S. cities by restricting the data to only days where PM_{10} concentrations were less than 50 ug/m^3. The effect was not only significant, but even greater on those days than for the entire data set. "These results are inconsistent with a threshold for PM_{10} at any concentrations except those substantially < 50 ug/m^3. Indeed, they suggest that the PM_{10} slope increases at lower concentrations rather than approaching zero." (p. 567)

This same tendency for a lower slope at higher concentrations has been noted in other air pollution studies as well (see, e.g., Schwartz & Marcus, 1990 [77] - London; Samet et al. (2000) [53] - 90 U.S. cities; Schwartz et al., 1996 [70] - $PM_{2.5}$ in six U.S. cities;[17] Ostro et al., 2000 [34] - $PM_{2.5}$, $PM_{10-2.5}$, PM_{10} in the Coachella Valley[18]). Schwartz et al. (2000) [57] conclude that the "public health benefit of each incremental reduction of 1 ug/m^3 appears to be higher at the lower air pollution levels that prevail on most days." [Id.] Similarly, Samet et al. (2000) [53] found that the effect size of increased

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[17] Schwartz et al. (1996) [70] state: "Our finding of a highly significant association on days when $PM_{2.5}$ concentrations were below 25 ug/m^3 suggests that any threshold in this association must occur well below that concentration." (p. 935) Specifically, on days when $PM_{2.5}$ was below 25 ug/m^3, $PM_{2.5}$ as a risk factor increased daily deaths, with a somewhat larger effect size than when the higher concentration days were included (2.2% compared to 1.5% overall) per 10 ug/m^3 increase in 2-day mean $PM_{2.5}$.

[18] Ostro et al. (2000) [34] found evidence of a slight diminution of effect at the higher PM concentrations in the Palm Springs/Indio area, where there are relatively high levels of the coarse fraction of PM_{10}.

PM_{10} on hospital admissions in 14 U.S. cities using only the 2-day mean of PM_{10} for days with PM_{10} concentrations less than 50 ug/m^3, the effect size increased by 20% or more for all three outcomes (cardiovascular disease, COPD and pneumonia).

Two other papers have addressed the issue of whether existing statistical techniques could identify a threshold if one existed. Based on actual data from Toronto, Cakmak et al. (1999) [6] simulated data with varying amounts of exposure measurement error. They investigated whether statistical models commonly used in most air pollution epidemiologic studies (including locally weighted smoothing techniques in Poisson regression models) would be able to detect thresholds in the PM-mortality association. The authors concluded that if a threshold existed, it is highly likely that the existing statistical modeling would detect it (which has not occurred). Many mortality studies have investigated the shape of the concentration-response function and found that overall a linear (non-threshold) model fit the data well (Pope, 2000 [42]; see also, Schwartz, 1994 [63] with respect to similar findings regarding hospital admissions). Fairley (1999) [13] likewise found no evidence for a threshold, where the daily PM_{10} concentrations ranged from 6 to 165 ug/m^3 and daily $PM_{2.5}$ ranged from 2 to 105 ug/m^3.

An alternative statistical approach was used by Schwartz and Zanobetti (2000) [69] in their analysis of data for the association between PM_{10} and daily deaths in 10 U.S. cities with daily PM_{10} levels. The authors combined concentration-response curves across the cities, after first demonstrating this methodology produced unbiased estimates. Predicted values of the response function were estimated at 2 ug/m^3 intervals. The results showed no evidence for any threshold effect. Attached as Exhibit A is an enlargement of Figure 2 included in this study. This figure shows the estimated dose-response curve between PM_{10} and daily deaths in the 10 cities and illustrates the concept of linearity. "There is no evidence of a threshold in the relation, down to the lowest levels of airborne particles observed in our study. The curve indicates that an increase from 5 ug/m^3 (its baseline) to 30 ug/m^3 is associated with about a 2% increase in daily deaths." (p. 669) It should be noted that these increases are for all deaths, as opposed to the typically higher death rates per dose increase found in the elderly and those with cardiopulmonary disease.

A different statistical approach was used by Daniels et al. (2000) [9] to test for the evidence of a threshold using the PM_{10} data from the 20 largest cities in the United States. They analyzed three alternative log-linear regression models. Model 1 used a simple linear term for PM_{10}, which could then be used as a basis of comparison with the other models. Model 2 used a spline-dose-response model which would allow for non-linearity in PM_{10} that might represent a threshold function. Model 3 presumed a threshold that assumed no relation between PM_{10} mortality up to a threshold and a linear retention after the threshold, in which a grid search would be used to test for a construction that would support a threshold.

The results of Daniels et al. (2000) [9] demonstrated that for the second model, which can allow for a threshold if the underlying data suggest one, the best fit to the data was a linear specification. In addition, analysis using the third model, suggested that there was no threshold for either total mortality or cardiopulmonary mortality. Finally, using a well established goodness-of-fit test to compare the simple linear no-threshold Model 1 with the other models, the authors reported that there was no evidence to prefer the threshold models to the linear model. The estimated threshold suggested by their data for respiratory mortality was 15 ug/m^3, which is lower than the mean annual average of PM_{10} in Morro Bay. This in turn indicates the increased impacts of $PM_{2.5}$ concentrations attributable to the new MBPP would not be below any threshold for respiratory mortality effects.

In his commentary on the Daniels et al. (2000) [9] study, Pope (2000) [43] states: "From at least one perspective, these results are good news – not because exposure to PM contributes to cardiopulmonary disease mortality even at relatively low concentrations, but because it may represent a preventable cause of death." (p. 411) This means a true reduction in $PM_{2.5}$ concentrations in Morro Bay, as opposed to the increase that will occur with the currently configured new MBPP, would indeed save lives as well as result in corresponding health improvements.

Conclusions

The increased daily and chronic mortality risks associated with even small increases in $PM_{10/2.5}$ are well established. These increases are even greater for susceptible populations such as the elderly (24% of the Morro Bay population), and those with pre-existing chronic diseases such as CVD, respiratory disease and diabetes. There likewise is substantial evidence supporting a direct relationship between $PM_{10/2.5}$ concentration levels and increased hospital admissions of these same populations. In addition, there are increased risks of elevated heart rate, decreased heart rate variability, increased instances of dysrhythmias, and increased defibrillator activity associated with increased $PM_{2.5}$ and PM_{10}. The advance in mortality associated with PM is significantly beyond the hypothesized mortality displacement effects and there is no threshold, with a 1 $\mu g/m^3$ increase in $PM_{10/2.5}$ having the same or even higher adverse health impacts at the lowest levels of ambient concentrations (well below current California statutory standards) than occur at the higher end of the range.

There is no justification for any increase in PM_{10} emissions that may result in increased ambient concentrations of $PM_{2.5}$ and PM_{10} in Morro Bay. The MBPP could reduce its PM_{10} emissions in a number of ways, such as eliminating duct burning and agreeing to further daily operational limitations. The $PM_{2.5/10}$ concentrations in Morro Bay resulting from the increased $PM_{2.5}$ emissions could likewise be reduced by design modifications to the proposed MBPP, such as taller stacks that would result in a more broadly distributed PM_{10} emission stream. These taller stacks for the new MBPP could be substantially shorter than the existing 450 ft. stacks and would still achieve improvements in local air quality.

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