Comments on "Benefit Study of MRPO Candidate Control Options for Electricity Generation"

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1. Introduction and Overview

These comments pertain to the August 25, 2006 Report, "Benefit Study of MRPO Candidate Control Options for Electricity Generation," authored by Dr. Leland Deck, Stratus Consulting, for the Lake Michigan Air Directors Consortium. This report models and monetizes health benefits presumed (but not demonstrated) to result from reductions in ambient air concentrations of fine particulate matter ($PM_{2.5}$) and ozone (O_3). More than 90% of the estimated health benefits (expressed as dollars) are from "statistical lives" presumed to be saved because people will be breathing air with slightly lower concentrations of $PM_{2.5}$. Unfortunately, no one knows whether this presumption is correct — even qualitatively, let alone quantitatively. Indeed, as others have found, precisely the same sort of "damage function" analyses can "demonstrate" that statistical lives would be *lost* "due to" the same reductions in ambient air pollution (see the draft report, "Mortality Risk Reductions and Economic Benefits of Alternative SAMI Air Quality Strategies," prepared by Mathtech, Inc. (December 2001) for the Southern Appalachian Mountains Initiative (SAMI), utilizing damage function estimates from Lipfert *et al.*, 2000). The latter estimate was rejected by SAMI because it seemed biologically implausible, but the toxicologic and clinical data on ambient $PM_{2.5}$ (especially the specific forms of PM due to power plant emissions) indicate that the former result is also suspect.

The Stratus 2006 report asserts, "Changes in the ambient levels of ozone and $PM_{2.5}$ will affect the health of the population exposed to those changes." If the changes are to (or from) very high levels of pollution, this statement is true. At the very-low-level concentrations of pollutants of interest, however, this statement is highly uncertain: maybe the Emperor is wearing beautiful new clothes, or maybe he is naked. If the latter, should we quantify the costs and benefits of clothes that (almost certainly) don't exist?

The substantial size of the effect predicted by the assumptions in the Stratus 2006 report should give readers pause. If the predicted mortality effect included in the study were true, then ambient levels of $PM_{2.5}$ at about 15 $\mu g/m^3$ are responsible for approximately 10% of all deaths in the U.S. This is a quite remarkable claim, one that should require substantial evidence to demonstrate its truth. Moreover, since ambient air PM levels are much higher in Mexico, India, China, and elsewhere (see, for example, Han and Naeher, 2006), the approach taken in Stratus 2006 would predict that incredibly large percentages of deaths internationally are caused by fine PM in ambient air.

The toxicologic and related scientific literature contains numerous papers discussing the crucial uncertainties underlying the damage functions upon which Stratus 2006 depends. None of this literature is cited or otherwise acknowledged in the Report. This lack of citation is especially troubling because the Report does, for example, cite five papers by Moolgavkar (sometimes with others) that partially support the Report's analyses, but leaves out any mention or citation of Moolgavkar 2005, which paper systematically analyzes much of the literature on $PM_{2.5}$ and finds no adequate basis for the belief that current (or, in this case, the projected future) ambient concentrations are lethal.

Most of the analytical techniques employed in Stratus 2006 were present in Abt 2002, about which we have commented in detail (Crouch *et al.*, 2002, enclosed). Additional comments

on the relevant toxicologic issues are in Green *et al.* (2002), Green and Armstrong (2003; both enclosed), and presented here as follows.

2. General comments

The Stratus 2006 report gives little technical detail of the analysis performed; but this is largely because most of what was done was to apply the BenMAP program (from EPA) to process air quality modeling data provided by LADCO, apply cost estimates also obtained from LADCO, and incorporate EPA measures of the cost of various health effects. All the rest is simple arithmetic applied in the Tables. Replication and checking of the application of BenMAP would require access to the air quality modeling data provided by LADCO. Those data are summarized in the Stratus 2006 report, but only in the form of Figures from which quantitative information is difficult, if not impossible, to extract.

The health effects are estimated using standard dose-response curves included in BenMAP. The estimated health benefits of the various scenarios are dominated by estimated mortality (in EGU1, for example, 92% of the benefit estimate is due to the reduced mortality, essentially all of which is ascribed to changes in $PM_{2.5}$, not ozone). In EGU1, the mortality benefit is estimated as 3,010 deaths/year (Stratus 2006, Table 6). Using the dose-response curve (essentially a relative risk for total mortality above age 30 of 1.0058 per $\mu g/m^3$ change in long-term average $PM_{2.5}$), this implies that the population-weighted average change in $PM_{2.5}$ in EGU1 must be modeled as about 0.17 $\mu g/m^3$ (with a population of order 300 million, giving a death rate of around 3 million/year, the increment would be about $0.0058 \times 0.17 \times 3{,}000{,}000 \approx 3000$) . Unfortunately, we cannot positively check this because this essential statistic about the input data is not provided. The text (page 10) indicates that the maximum estimated change was 1.3 $\mu g/m^3$, but it is not specified to which control option this corresponds. Figure 3 suggests (from a very rough calculation) that the area-weighted average reduction in $PM_{2.5}$ is about 0.17 $\mu g/m^3$, indicating no substantial errors in calculation, at least for $PM_{2.5}$.

The BenMAP audit trails in Appendices A and B indicate that BenMAP was run using a Monte Carlo simulation with just 50 samples; and the tables suggest the same, since the cost estimates given in Table ES-6 are not obtained exactly as the product of the health effects in ES-4 and the cost estimates in ES-5 (although we did not checked the operation of BenMAP to see if this difference arises because of the use of a probabilistic analysis with so few samples). Since these appear to be mean estimates in both cases (it is not specified, and should be), Table ES-6 should be exactly obtained from the product of the values in Tables ES-4 and ES-5. While the overall differences are relatively small, around 2% of the total, and are completely negligible compared with the uncertainties involved in totals, they are in themselves larger than some of the entries in Table 6.

The failure to illustrate, discuss, or even properly acknowledge the uncertainties involved is perhaps the major criticism that can be made of the report. The coefficient of variation of the mortality estimate, for example, is about 37%, and that is based *solely* on statistical uncertainty from the original paper defining the dose-response curve. The actual uncertainty is vastly larger, as indicated by various expert evaluations (Industrial Economics Inc., 2004, 2006), and by abundant literature (about which more below). Importantly, the average estimate has not been

adjusted to account for the potential lack of causality in the dose-response curves; and some estimate of that should certainly be incorporated (NAS, 2002). Even the authors of the report on which the mortality dose-response curve is based do not give 100% probability for the result to be causal. Moreover, there is considerable uncertainty in the estimates for the unit cost associated with health effects. Table ES-5 does not indicate this. Indeed, the origin of the value given in Table ES-5 and Table 10 for premature mortality is not clear, at least to us. It is stated that the value given is "consistent" with the values used in the "CAIR RIA (U.S. EPA, 2004, 2005)". Consulting the second reference, we find (Table 4-11) that the central estimate given there varies from $5,500,000 to $6,400,000 (in 1999 dollars) depending on assumed income levels in various future years. More important than these small variations is the very large uncertainty — it is stated that the "[p]oint estimate is the mean of a normal distribution with a 95 percent confidence interval between $1 and $10 million" based on a meta-analysis[1] of the literature, giving a coefficient of variation of about 42%. Taking some account of the uncertainties for just the mortality component, the range of benefit estimates is extremely large[2] — from around $1 billion (due to other health effects; but these also need an uncertainty analysis) with a probability that depends on the probability of causation of mortality by $PM_{2.5}$, up to about $34 billion at around the 95th percentile.

3. The cental toxicologic uncertainties

With essentially no discussion of the vast uncertainties involved, Stratus 2006 "analyzes" $PM_{2.5}$ by assuming that:

(1) any and all forms of $PM_{2.5}$ in ambient air cause death;

(2) all such forms are qualitatively and quantitatively identical in their toxic actions and potencies;

(3) annual, average, mass-based concentrations are the best, relevant measure of $PM_{2.5}$ in air; and

(4) decreasing such concentrations of any or all forms of ambient $PM_{2.5}$ will decrease rates of death in some reliably quantifiable fashion.

To understand the problems with these assumptions, please consider the following.

The term $PM_{2.5}$ (in the context of ambient air regulations) is defined operationally as any atmospheric material, solid or liquid, with an effective diameter equal to or smaller than 2.5 microns (as collected by Federal Reference Method samplers and measured, by weight, under

[1] We would question any meta-analysis that assumed a normal distribution of values, since it seems likely that no-one would agree that the valuation should be negative.

[2] And this is accepting that the uncertainty on the mortality estimate is just 37% (assuming causality), and on the value of a statistical life 41%.

specific temperature and humidity conditions). As such, $PM_{2.5}$ refers to *thousands* of different things, both natural and man-made. Various forms of $PM_{2.5}$ differ with respect to (among other things): (1) size (with diameters ranging from a few nanometers to 2,500 nanometers), shape, and surface characteristics; (2) water solubility and pulmonary persistence; (3) chemical composition, including metal content, and related properties such as pH; and (4) biologic and immunologic properties and potencies. Clearly, it makes no more sense to think about estimating "*the* health effects of $PM_{2.5}$ in ambient air" than it does to consider estimating "*the* health effects of gases in ambient air." In the latter case, no one would assume that ambient mass concentrations of oxygen, nitrogen, ozone, carbon monoxide, mercury, phosgene, and sarin (a nerve gas) were all identically toxic, just because they are all gases and so capable of reaching deep into the lungs.

Moreover, not all ambient $PM_{2.5}$ is the direct or indirect result of pollution, since $PM_{2.5}$ includes thousands of species of viruses and bacteria, various molds and pollens, fragments of countless species of plants and insects, and bits of different types of sand and soil. Clearly, even restricting the discussion to "natural $PM_{2.5}$," small amounts of some forms, such as smallpox virus, can be deadly, while other forms are entirely benign.

Of particular interest to Stratus 2006 are the forms of PM that arise from emissions from fossil-fuel-fired electric generating units. As it turns out, these particular forms of PM — primarily ammonium sulfate and ammonium nitrate — are quite benign materials, and are expected to be harmless *at and considerably above* current concentrations in outdoor air. These sulfates and nitrates are simple, water-soluble salts, and vastly different — in terms of chemistry, physical properties, and impacts on the lung and health — than other forms of PM, such as silica or various chromate salts, that do, at sufficiently high concentrations, cause disease and death. The hypothesis that ambient or moderately elevated concentrations of relatively non-acidic, soluble, sulfates or nitrates *might* harm health has been repeatedly tested, using both human volunteers and laboratory animals, and found to lack merit (see, for example, Avol *et al.*, 1979; Utell *et al.*, 1983; Aris *et al.*, 1991; reviewed in U.S. EPA, 1996). The non-toxicity of even high-level concentrations of airborne sulfates is also suggested by the widespread use of sulfates in medicine. Many bronchodialators used to treat asthma, such as albuterol, metaproterenol, and terbutaline, are supplied as the sulfate salts (*Physicians Desk Reference*, 1998). One puff from a standard inhaler containing albuterol sulfate, for example, supplies an asthmatic with some 20 micrograms of sulfate, delivered at a concentration of some 10,000 micrograms of sulfate per cubic meter of inspired air (assuming 2 liters of air per deep inspiration). Medicinal chemists, pharmacologists, and others do not believe this to cause harm, let alone to hasten death.

Unfortunately, Stratus 2006 fails to recognize that sulfates and nitrates at relevant concentrations are harmless, and so promises health benefits it cannot deliver.

Virtually all aspects of U.S. EPA's Clean Air Act programs, such as its NESHAP program, recognize that PM is not PM is not PM. Thus, the federal list of 188 hazardous air pollutants (HAPs) contains scores of individual, specific, liquids and solids (that is, PM) present in air: there is nothing so crude as undifferentiated $PM_{2.5}$ or PM_{10} on such a list, nor should there be. In further refinements according to toxicity and exposure-levels, such as under EPA's Air Toxics Strategy, the Agency has identified 33 specific airborne chemicals or mixtures that pose "the greatest threats to public health in urban areas." This list again includes specific liquid and

solid forms of PM derived from both stationary and mobile sources; and again, there is, appropriately, no listing of fine PM as a whole.

Toxicologic, clinical, and epidemiologic studies of hundreds of forms of particulate matter demonstrate, as expected, vast differences in health-risks, according to specific chemical, physical, and biological properties. As a result, health-based recommendations and regulations concerning air in the workplace — such as those recommended by the National Institute of Occupational Safety and Health [NIOSH] or the American Conference of Governmental Industrial Hygienists [ACGIH], or promulgated by the Occupational Safety and Health Administration [OSHA] and the Mine Safety and Health Administration [MSHA] — are based on the specific chemical (and sometimes physical) forms of PM to which workers may be exposed. Thus, hundreds of disparate forms of PM — such as ammonium sulfamate, arsenic, barium oxide, beryllium, carbon black, carbaryl, cotton dust, crystalline silica, diesel engine exhaust particulate matter (DPM), mineral oil mist, nickel, phosphoric acid mist, pyrethrum, rhodium, sulfuric acid mist, and tin — are each regulated according to distinct, chemical-specific (or mixture-specific) standards intended to protect health. NIOSH-recommended exposure limits (RELs), for example, range from concentrations as stringent as 0.0005 mg/m^3, for beryllium, to levels as generous as 10 mg/m^3, for ammonium sulfamate.

Toxicologists, industrial hygienists, physicians, and others working in, or knowledgeable about, occupational health — and so aware of the very different kinds and extent of health effects and risks due to the hundreds of different kinds of industrially-derived PM — are mystified at U.S. EPA's current position (adopted by Stratus 2006) that all forms of $PM_{2.5}$ (or PM_{10}) in ambient air should be presumed to be alike. Such a notion seems to them and to me to be profoundly unscientific. More disturbingly, because compliance with such a standard could be achieved by reducing *any* harmless form of PM, such a standard is quite unlikely to improve public health, except perhaps by accident. It would be as if workplaces in which airborne levels of arsenic, for example, were too high, could "come into compliance" by substantially reducing airborne concentrations of sodium chloride (ordinary salt, such as from sea-spray). Total mass-based concentrations of respirable PM would indeed be reduced, but to no health benefit.

Clearly, health-based actions for particular matter in ambient air should be based on the specific chemical forms or mixtures of PM known or reasonably expected to harm health. Some forms of PM are or may be present in ambient air at significantly risky levels: the task for scientists and policymakers, then, is to determine which forms of PM these are, and then, for

example, to design one or more AAQS (if appropriate, and if not managed better under NESHAP or other programs) to control exposures.[3]

With regard to PM and morbidity (as opposed to mortality), numerous experimental, toxicological studies have found no causal relationships between moderate levels of power-plant-derived PM and respiratory disease, cardiovascular disease, cancer, or premature death. Regarding asthma — an important disease in America and elsewhere — time-trend data, prevalence data, and experimental data all fail to support the notion that moderate airborne concentrations of such PM either cause or exacerbate this disease. Additional details may be found at Green *et al.* (2002) and Green and Armstrong (2003), both enclosed.

The many, crucial differences still being investigated among various forms of ambient $PM_{2.5}$ mean that pollution control strategies intended to reduce risk to human health must be very specific, and must await clarification of the relationships among PM sources, compositions, and health effects.[4] Each industrial and mobile source of primary and secondary particulate matter generates different mixtures of PM types, and each type of control strategy (for example, bag houses versus electrostatic precipitators, not to mention variations within each of these classes of controls) also yields PM of varying composition.

4. Even concentrated forms of ambient PM do not kill or substantially harm laboratory animals

Fundamentally, if inhaled, low-level PM kills people, it must also kill laboratory animals. Stratus 2006 does not cite or mention any of the numerous toxicologic studies that have addressed this topic. Such toxicology studies on PM have been undertaken to probe whether and to what extent the observed epidemiologic associations are likely to be causal. Of course, epidemiologic study of ambient air pollution (the type of study upon which Stratus 2006 relies) is necessarily observational, not experimental or clinical. Scientists cannot perform

[3] As always, the extremes are easy to discern: certain forms of ambient PM clearly cause disease and death at current U.S. levels; other forms are apparently safe. In the first category are biological forms of PM, such as certain viruses and bacteria in air (both outdoors and indoors): indeed, influenza and pneumonia are currently the seventh leading cause of death in the United States. In the second category are airborne forms of PM such as sodium chloride and calcium chloride, which are enriched in ambient air near the Atlantic, Pacific, and Gulf coasts, and which are apparently harmless at ambient concentrations. Of course, each of these "extreme" forms of PM — microbial or marine — is due not primarily to pollution, and so not controllable by any AAQS program (or other regulatory interventions). Nonetheless, they are part of ambient PM as currently defined, measured, and studied.

[4] Of course, it might also be the case that observed, positive correlations between ambient PM and populational mortality (which observations form the basis of U.S. EPA's $PM_{2.5}$ NAAQS) are *confounded entirely* by factors that vary with, but form no part of, ambient PM. If so, then differences among the hundreds of thousands of forms of ambient PM become differences without distinctions, and the confounding, non-PM causes of death are instead those to which attention should be turned.

double-blind, randomized, or placebo-controlled studies of the health effects of ambient air pollution. Nor can we rely on the sort of "natural experiments" that, for example, cigarette smokers perform on themselves, relative to nonsmokers, that allow observational epidemiologic study of the health effects of smoking to have substantial probative power. The only direct way to test whether ambient PM causes morbidity and mortality is to expose appropriate laboratory animal models (or, for short-term studies, human volunteers) to graded exposures of airborne PM and look for exposure-related symptoms and signs of disease. So far, such studies have failed to demonstrate that moderate levels of airborne PM cause (or substantially aggravate) respiratory disease, cardiovascular disease, cancer, or premature death. Our review of these studies is given as an appendix to these comments. Overall, these studies confirm that fine particles can indeed be inhaled and absorbed, but that such dosing leads to little or no clinical effect, even at levels much larger than ambient.

5. Cross-sectional studies also do not establish that current levels of ambient PM are lethal

Stratus 2006 depends heavily on estimates derived from one study — Pope *et al.*, 2002 . This is an epidemiologic, observational study of a cross-sectional design. The study — and the earlier Pope *et al.*, 1995 study — finds weak, positive associations between mortality in various metropolitan areas and ambient concentrations of PM in those areas. As noted above, not mentioned in Stratus 2006 is another, similar study which finds a weakly *negative* association between these two factors. In particular, a large cross-sectional study (Lipfert *et al.*, 2000) reported a relative risk for mortality of 0.94 for a 10-microgram of $PM_{2.5}$ per cubic meter of ambient air increase. If causal, this association would mean that increasing levels of airborne PM would save lives.

More generally, it is widely recognized that observational studies of this design, when yielding relative risk estimates close to 1.0, are far too likely to suffer from residual confounding (by true causes of mortality), and so should be discounted. In the case at hand, the cited observational studies (Pope et al., 2002, Pope *et al.*, 1995 and Dockery *et al.*, 1993, the last two reassessed in Krewski *et al.*, 2000), yield relative risk estimates on the order of 1.05 to 1.14 for each 10-microgram increase of $PM_{2.5}$ per cubic meter of ambient air. Such relative risk estimates are close to no association at all. The "statistical significance" obtained in the studies suggests that these relative risks are unlikely to be observed by chance, *if all the assumptions made by the analysts are correct*. That last caveat, however, is crucial.

As the epidemiologist Jamie Robins (2001) has noted:

> . . . in an observational study, every two variables have an unmeasured common cause, and thus there is always some uncontrolled confounding. . . . As epidemiologists, we should always seek highly skeptical subject-matter experts to elaborate the alternative causal theories needed to help keep us from being fooled by noncausal associations.

Indeed, it is the weakness of the associations from observational PM-mortality epidemiologic studies, combined with the lack of dose-relevant support from toxicologic or clinical studies, that make many experts wary of jumping to causal conclusions regarding low-level exposure to ambient PM and significant morbidity or mortality (Lipfert and Wyzga, 1995; Phalen and McClellan,1995; Moolgavkar and Luebeck, 1996; Vedal, 1997; Gamble, 1998; Phalen, 1998; Valberg and Watson, 1998; Chock *et al.*, 2000; Lippmann and Schlesinger, 2000; Lipfert and Morris, 2002; Green *et al.*, 2002; Moolgavkar, 2005). For example, Vedal (1997) has written:

> There is general agreement that the associations are weak, with estimates of effect being very small . . . Although a small effect may nevertheless be a real effect, the importance of observing only small effects is that a small estimate of effect is more likely to be due to confounding by factors not controlled by the investigators.

Evidence of such confounding is indeed present in the largest longitudinal cohort study on which Stratus 2006 relies for evidence of health effects due to PM. This study (Pope *et al.*, 2002) makes use of risk factor data, as assessed by only one questionnaire administered to each study participant in 1982, and mortality data (from 1982 through 1998) generated by the American Cancer Society (ACS) as part of its prospective Cancer Prevention Study II (CPS II). Notably, these studies were *not designed* to assess the effects of ambient air quality on disease and death; the data therein generated were put to such use only long after the study had been designed and completed. Moreover, examination of Pope *et al.*, 2002 reveals a peculiar finding. Although the investigators conclude that, "Each 10 $\mu g/m^3$ elevation in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively," examine Figure 4 of that paper (p.1139), and you will find that ambient air PM appears to be fatal (in any of these three ways) *only* for those people with no education beyond high school. Thus, for the 64% of the men and 55% of the women in the CPS II cohort who had any education beyond high school (Stellman and Garfinkel, 1986), ambient PM showed *no* association at all with rates of death.

If ambient PM is genuinely lethal, is it likely to kill only the less-educated? Or do these results suggest uncontrolled confounding? Recall that data on health risk factors for study subjects were gathered only once, in 1982, and that mortality status was ascertained for up to 16 years later. Could study subjects with education beyond high school differ from those without in some systematic ways with regard to temporal changes in these risk factors? Does level of educational attainment affect the likelihood that a smoker in 1982 quits smoking, or loses or gains significant weight, or develops high blood pressure, diabetes, or atherosclerosis over these years (up to16) of follow-up? The first of these uncertainties may be the most important, since cigarette smoking is, of course, a very strong risk factor for lung cancer, and an important risk factor for cardiopulmonary mortality and all-cause mortality as well; as is well known, quitting smoking reduces these risks.

Evidence of an "educational gradient" in the apparent lethal potency of ambient PM appeared in the earlier report of this study (Pope *et al.*, 1995) as well. The complete abolition of *any* PM effect on *any* measure of mortality in subjects with more than a high school education,

reported in the follow-up study (Pope *et al.*, 2002), should make careful readers wonder about the accuracy and generalizability of the findings.

Educational status, as measured in these studies, is arguably just a measure of socioeconomic status (SES), and is included as a catch-all potential confounding by SES effects. In the EPA's expert elicitation (Industrial Economics Inc., 2006) some of the experts (among whom were several of the authors of the Pope *et al.,* 2002 study) argued that the observed educational gradient in that study (and in the various re-analyses and extensions of the same basic data) should be used to infer a larger coefficient for the population as a whole, since the ACS population was more educated than the US population (Industrial Economics Inc., 2006). Others pointed out that such contextual SES variables could be confounders that might lead to a positive bias in the results (or a negative bias if they acted as surrogates for exposure to $PM_{2.5}$). Indeed, as recognized in that expert elicitation, Jerrett *et al.*'s (2005) analyses showed that including more contextual SES variables tended to diminish (and eliminated the statistical significance of) the observed $PM_{2.5}$ effect

The NRC (2002, p. 92) has pointed out that EPA (upon which Stratus 2006 relies) provides little information to judge the *plausibility* of the causal relationships assumed, and indicates that the uncertainty associated with the assumption of plausibility should be incorporated into the final benefits estimates when possible. EPA has recently (Industrial Economics, 2004, 2006) attempted to evaluate some experts' views of the plausibility and uncertainty of the mortality relationship. Not surprisingly, the opinions elicited in these attempts varied substantially.

6. Ambient PM and asthma

Although asthma morbidity is not a large component of the health benefits estimated in Stratus 2006, it bears mention that the presumed causal association between power-plant-derived PM and asthma is suspect. Many observational studies have indeed reported associations between airborne PM and asthma or other respiratory problems, but, for several reasons, these studies do not establish that reducing ordinary PM in ambient air will improve respiratory health.

Asthma is a health problem of significant proportions throughout America, and internationally. The vast weight of scientific evidence indicates that *non-biological* particulate matter (as opposed to particles from dust mites, pollens, rodent excreta, and other antigenic material) could play at most a minor role in either the incidence or the exacerbation of asthma, and may well play no role whatsoever.

Asthma is a chronic respiratory disease characterized by episodic narrowing of the lumen of lung airways. *At sufficiently high levels of exposure*, many biological, chemical, or physical agents may initiate lung responses leading to airway constriction. Triggers capable of eliciting an asthmatic response include viral respiratory infection, allergens (such as pollen, animal dander, rodent excrement, mold, mites, cockroach components), exercise, weather (in particular, cold air), emotional stress, environmental tobacco smoke, and certain foods, drugs, or specific workplace chemicals.

A major problem with the hypothesis that ambient PM exacerbates asthma is that airborne PM has been *decreasing* over the same period of time (U.S. EPA, 2000) when asthma has been *on the rise* (Anderson, 1997). Illustrations of these opposite time trends are shown in Figures 6 and 7.

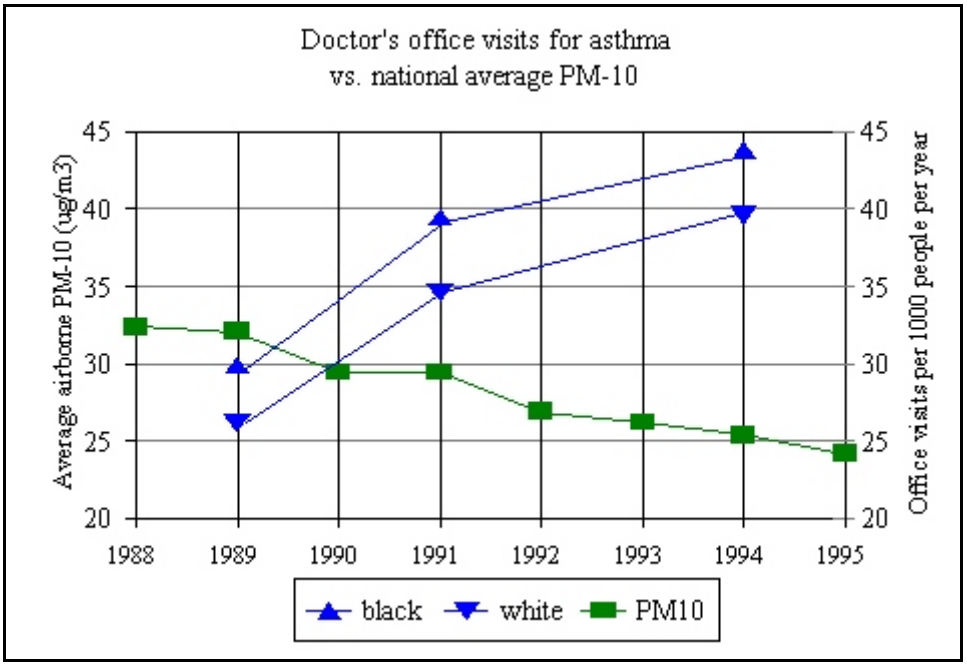

Figure 6. National rise in doctors' office visits for asthma symptoms per 1,000 people per year and the decline in national average PM levels from 1988 to 1995.[5]

[5] The asthma data are from Mannino, D.M., *et al.*, (1998) Surveillance for Asthma—United States, 1960-1995. *Morbidity and Mortality Weekly Report*, 47 (SS-1):1-27. The information was obtained from the National Center for Environmental Health's National Ambulatory Medical Care Survey. The values shown for 1991 and 1994 are the average values for the three years centered on the date shown. The national average PM_{10} values were taken from the U.S. EPA (Office of Air and Radiation) *1997 National Air Quality: Status and Trends* (December 1997).

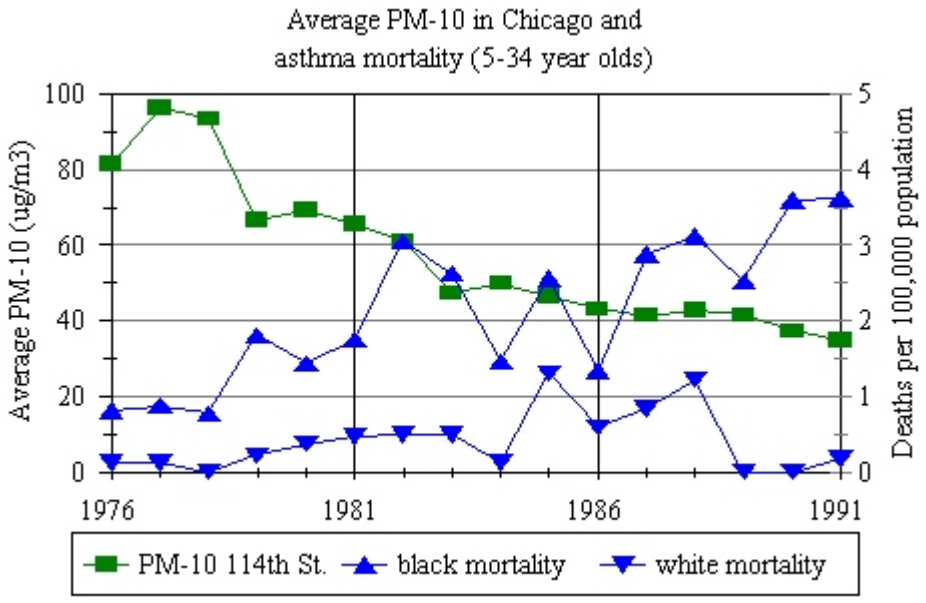

Figure 7. The rise in mortality of 5- to 34-year-olds in Chicago with asthma as the underlying cause and the decline in PM levels in Chicago from 1976 to 1991.[6]

The reasons for the rise in the prevalence and severity of asthma are not fully understood. Suspected factors include changing patterns of childhood illnesses, changing diet, increasing body mass index, changing exercise patterns, changing housing, increased vaccinations against childhood respiratory disease, and increased exposure to indoor-air allergens (see, for example, Burge, 1990; Sporik *et al.,* 1990; Platts-Mills *et al.,* 1997; and Crater and Platts-Mills, 1998). Vazquez-Garcia (2002) and others (Rodriguez *et al.*, 2002; Huovinen *et al.*, 2003) have noted that: (I) the rates of both obesity and asthma are increasing, in the U.S. and elsewhere; (ii) obesity appears to be a risk factor for asthma; and (iii) weight reduction can lessen the severity of asthma in some patients.

[6] The mortality data are from a figure in Targonski, P.V., *et al.*, (1994) Trends in Asthma Mortality among African Americans and Whites in Chicago, 1968 through 1991, *American Journal of Public Health*, Vol. 84, No. 11:1830-1833. PM_{10} data for Chicago from 1984 to 1991 were obtained from the U.S. EPA's on-line database AirsWeb and are for monitor #1700100022-1, located at 3535 E. 114th St. in south Chicago. Because there were no PM_{10} monitors in Chicago before 1984, the PM_{10} data shown are 0.55 times the annual average Total Suspended Particulate (TSP) levels for the same monitoring location. These were obtained by personal communication from Mr. Bob Swinford, Illinois Environmental Protection Agency, Springfield, IL.

A community-based study of asthma incidence in Rochester, Minnesota found that asthma episodes in children increased about 1.6-fold over the 20-year time period from 1964-1983 (Yunginger *et al.,* 1992). The authors concluded that this increase was "not likely to be caused by outdoor air pollution" but rather, may be linked to "energy conservation, which has resulted in increased exposure to indoor allergens."

Time trends for asthma in East *versus* West Germany show that the proportion of East Germans with atopy (a predisposition to allergic reactions) is increasing as the population becomes "Westernized" and industrial air pollution is reduced (Heinrich *et al.,* 1998).

Time trends aside, *geographical* variations in asthma hospitalization rates show sharp differences that cannot be attributed to differences in ambient air quality.

Studies around the world report low prevalence of asthma in countries with air pollution problems, such as Mexico, Eastern Europe, China, and Greece, whereas asthma rates are nearly 10 times higher in countries that have much better air quality, such as New Zealand, Australia, and Canada (Peat and Li, 1999; ISAAC, 1998). In the United Kingdom, regional differences in outdoor air pollution do not correlate with asthma prevalence (Anderson, 1997). In Europe, asthma rates are lower in more polluted regions than in regions with cleaner air (von Mutius *et al.*, 1994; Nowak *et al.,* 1996; Bjorkstein, 1997; and Nicolai, 1997).

In a Massachusetts study, it was found that Merrimack Valley communities have widely disparate asthma hospitalization rates, even though they are in the same air-quality air shed (Declercq, 1998). For example, asthma hospitalizations in Lawrence were 7.5-fold greater than in Andover, even though these are bordering towns. Likewise, in Lowell, asthma hospitalizations were 68-fold greater than in nearby Dunstable.

Asthma hospitalizations across communities in the City of Boston were examined by researchers at Boston University School of Public Health (Gottlieb *et al.,* 1995). Among the neighborhoods of Boston, dramatic differences in asthma hospitalizations were evident. Some areas had hospitalization rates as high as 10 per 1,000. The more affluent "Downtown" and "Back Bay" areas were in the lowest group, with rates of about 1.3 per 1,000. Dorchester had a much greater asthma hospitalization rate than West Roxbury, despite the fact that outdoor air quality for these two nearby neighborhoods is very similar.

In New York City, several groups have analyzed the distribution and factors affecting asthma hospitalizations and mortality (Carr *et al.,* 1992; de Palo *et al.,* 1994; Claudio *et al.,* 1999). Asthma prevalence in New York City correlates strongly with socioeconomic status, and several factors link asthma with poverty. Factors that related to asthma risk in low-income areas were the number of occupants per apartment (related to bacterial and viral exposures), water leaks (related to fungal exposures), moist basements (related to fungal exposures), deteriorating building materials (related to fungal and mite exposures), and house dust exposure (containing insect parts, animal dander, and rodent excreta).

Experimental evidence from human volunteers indicates that asthmatics show little or no response during voluntary exposure to much higher levels of airborne PM than are characteristic of the outdoor environment. Studies in a number of laboratories (Avol, *et al.,* 1990; Hanley *et*

al., 1992; and Koenig *et al.,* 1989) have compared the pulmonary response of asthmatics to that of normal subjects in chamber studies, where volunteers are exposed to measured, controlled concentrations of various airborne substances. Although asthmatics were more sensitive to high concentrations of highly acidic aerosols than normal subjects, neither asthmatics nor normal subjects exhibited decrements in pulmonary function after exposure to mildly acidic airborne particulate. The concentrations used ranged from 100 to 1,000 $\mu g/m^3$ — much higher levels than found in outdoor environments in America today (which are on the order of 5 - 10 $\mu g/m^3$).

Genetic factors are also clearly important, at least with respect to the predominant, allergic type of asthma. Familial risk factors and racial and ethnic risk factors have been identified (Bleecker *et al.,* 1997; Joseph *et al.,* 2000). These findings suggest that some children may be predisposed to more severe asthma than others.

In summary, time-trend data, prevalence data, and experimental data fail to support the notion that moderate airborne concentrations of ordinary non-biological PM cause or exacerbate asthma.

7. Concluding remarks

Some might argue that it matters little whether we know what aspects of ambient $PM_{2.5}$, if any, seem to affect morbidity or mortality in many observational, population-based studies. Surely, they would argue, since ambient $PM_{2.5}$ generally correlates with rates of mortality, reducing any or all types of $PM_{2.5}$ must save lives.

This is incorrect, for at least three reasons. The first is that many methods of PM control serve to reduce mass concentrations of fine PM in ambient air but do not reduce, and in fact increase, *number* concentrations of fine and ultrafine PM in air (Pitz *et al.*, 2001). To the extent that insoluble ultrafine PM may be hazardous (Oberdörster, 2001), such actions could hardly be considered to be health-protective. Similarly, if sulfate in ambient air is benign, but vanadium-enriched ultrafine PM in air is not, what good would reducing the former do, especially if it comes at the expense of increasing the latter?

The second reason is that to the extent that the PM-mortality association is confounded (by factors that can vary with PM, but form no part of PM), reducing ambient concentrations of PM without reducing the confounding causes will do no good. Alcohol drinkers are well-known to be at increased risk for lung cancer, but this is because they also tend to smoke cigarettes at higher rates than non-drinkers. Would acting to reduce people's drinking (without acting to

reduce their smoking) lower people's risk of lung cancer[7]? Fundamentally, we cannot hope to reduce risks without identifying real causes.

The third reason is that actions based on insufficient evidence or knowledge are especially subject to the law of unintended consequences. Airbags designed to save adult lives kill infants. Gasoline reformulated with MtBE to clean up air fouls groundwater. DDT used to prevent millions of cases of malaria thins the shells of eagle eggs. Everyone can think of examples in which technologic "fixes" of one problem have created other, unintended problems. Moreover, if current concentrations of particulate matter and ozone don't kill, just whom are we saving by reducing them?

[7] Alcohol is itself a carcinogen, of course, and it may be that alcohol and smoking interact in increasing (some) people's risk of lung cancer. The majority of experts in this field reject this view, finding that smoking *per se* accounts entirely for the risk of lung cancer in drinkers who smoke (since when one controls for drinking in analyzing the results of epidemiologic studies, one finds that drinkers and non-drinkers with similar smoking histories incur similar risks of lung cancer), but the question is not entirely settled. Similarly, people who smoke cigarettes are at increased risk for cirrhosis of the liver, but this is because they also tend to drink alcohol at higher rates than non-smokers, and it is the alcohol *per se* that causes the cirrhosis. Acting to reduce people's smoking (without acting to reduce their drinking) would not lower their risk of cirrhosis (though it would, of course, greatly reduce their risks of many other, genuinely smoking-induced diseases).

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The views expressed here are our own. We take responsibility for errors.

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APPENDIX

Experimental evidence regarding the effects of exposure of compromised laboratory animals to concentrated ambient particles (CAP)[8]

Introduction and Overview

Controlled exposures of laboratory animals to concentrated ambient particles (CAP) have not yet significantly advanced our understanding of the toxicity of ambient particulate matter or validated findings of epidemiologic studies. Many of the experiments are difficult to interpret due to lack of control over CAP concentration and composition, and testing small groups of animals over months may introduce further complications in interpretation. Exposure time has been limited: few studies exposed animals for more than 18 hours over three days. Thus, the chronic toxicity of CAP (as opposed to diesel engine exhaust particulate matter, [DPM] which has indeed been extensively tested in chronic, lifetime bioassays in rats and other species) has yet to be examined).

Lethality is an epidemiologic association of great concern, yet CAP has been reported to cause death in only one of these studies (Godleski *et al.*, 1996), which was not published in the peer-reviewed literature nor apparently replicated by the investigators themselves, despite 6 attempts (Godleski, personal communication, July 12, 2002).

Several animal models of compromised respiratory or cardiopulmonary function (asthma, *cor pulmonale*, cardiomyopathy, etc.), as well as animals of different ages, have been exposed to CAP to test hypotheses regarding risk factors for particulate matter-induced morbidity. The studies are perhaps most notable for the *lack* of clinically relevant responses to CAP: relatively few parameters, of all the pulmonary, hematologic, systemic, and cardiac endpoints investigated, are statistically significantly affected by CAP exposure, and effects, when tested over time, are transient and fairly mild. Most of the experiments do not find that a CAP effect is larger in compromised animals than in healthy animals.

[8] Concentrated ambient particles (CAP) are generated experimentally by devices that concentrate ambient particles in the size range 0.1-2.5 microns by drawing outdoor air through a series of three virtual impactors. CAP exposures are typically on the order of 30 times more concentrated than outdoor air with respect to $PM_{2.5}$.

CAP studies of animal models of asthma or hypertension (high blood pressure)

Whether asthmatics are more sensitive than non-asthmatics to ambient air particulate matter has been studied in experimental animals by one group of researchers (Goldsmith *et al.*, 1999; Kobzik *et al.*, 2001). These researchers produced mice with some of the hallmarks of human asthma by sensitizing them when very young to ovalbumin through *i.p.* injections followed by three daily aerosol challenges with ovalbumin. Mice inhaling ovalbumin followed by filtered air developed airway hyperresponsiveness to methacholine, increased numbers of total cells and inflammatory cells in bronchoalveolar lavage (BAL) fluid, and pulmonary inflammation (as evidenced by histopathology), usually measured 24 or 48 hours after the last allergen challenge.

In the first experiment (Goldsmith *et al.*, 1999), exposure to CAP (three daily exposures of five hours, beginning after ovalbumin challenge) did not worsen the pulmonary responses of ovalbumin-sensitized mice measured 24 or 48 hours after the last exposure. Three-day-average CAP exposures ranged from 395 to 1,479 $\mu g/m^3$, and averaged 787 $\mu g/m^3$ across all CAP-exposed groups.

In the second experiment (Kobzik *et al.*, 2001), in which three-day-average CAP exposures ranged from 78 to 939 $\mu g/m^3$, CAP was found to cause a small but statistically significant increase in airway responsiveness, measured immediately after exposure, of about 0.9% per 100 $\mu g/m^3$. This effect was dwarfed by the effect of ovalbumin sensitization, and was not seen 24 hours after exposure.

In summary, no marked effect of CAP on ovalbumin-sensitized mice has yet been seen, and thus the hypothesis that (human) asthmatics will be more affected by CAPs than non-asthmatics has not received strong experimental support.

Recently, Kooter *et al.* (2006) tested the effects of short-term (2-day) exposures to very fine, concentrated, ambient PM (fCAP) on sensitive, spontaneously hypersensitive rats. Exposure levels ranged from 400 to 3,600 $\mu g/m^3$. These exposures proved to be no different from room air with regard to pulmonary or systemic effects. Moreover, even some minor changes apparently due to CAP did not correlate with mass concentrations. These and other results strongly suggest that small and simple reductions in ambient PM concentrations will not affect health.

CAP studies of animal models of bronchitis

It is also hypothesized that chronic pulmonary diseases, such as bronchitis, predispose one to adverse effects of ambient particulate. This hypothesis has been investigated in rats rendered bronchitic by subchronic exposure to SO_2 (Godleski *et al.*,1996; Clarke *et al.*, 1999; Kodavanti *et al.*, 2000) and in bronchitic dogs (Godleski *et al.*, 2000). Bronchitic rats display mucus hypersecretion and goblet cell hypertrophy and hyperplasia but minimal airway thickening and inflammation.

In the first experiment, death was reported to have occurred in 37% of bronchitic rats (bronchitis produced by 250 ppm SO_2) exposed to CAP at levels ranging from 191 to 317 $\mu g/m^3$

CAP for six hours per day over three consecutive days (Godleski *et al.*, 1996).[9] Bronchitic animals not exposed to CAP, and healthy animals, exposed or not, did not die during the study. Animals did not show signs of irritation or distress during exposure. On necropsy, significant bronchoconstriction was seen in bronchitic rats exposed to CAP, compared with a lesser degree of inflammation in bronchitic animals exposed to air. In surviving bronchitic animals, CAP exposure caused a significant increase in BAL neutrophils.

In the second study of the same design by the same group (though with a few different investigators, including the lead author, Clarke *et al.*, 1999), pulmonary function and BAL fluid were evaluated immediately after or 24 hours after, respectively, the end of exposure to an average of 515 $\mu g/m^3$ CAP for six hours per day over three consecutive days. Lung tissues were also examined. Overall, the degree of bronchitis produced in this experiment (by exposure to 170 ppm SO_2) was mild. Bronchitic rats exposed to CAP developed significantly increased tidal volume compared to bronchitic rats exposed to clean air, while their BAL fluid contained increased protein, a decreased percentage of macrophages, and increased percentages and numbers of lymphocytes and neutrophils. The changes in protein content and numbers of lymphocytes and neutrophils induced by CAP in bronchitic rats were significantly larger than the changes induced by CAP in healthy rats. Histopathologic examination showed that inflammation in bronchitic rats was not exacerbated by CAP. No deaths were reported.

In the third investigation (Kodavanti *et al.*, 2000), BAL fluid was examined immediately following, or 18 hours following, the end of a two-day exposure of bronchitic rats (induced by 200 ppm SO_2) to CAP. Eighteen hours after exposure, no effect of CAP was detected in the single group examined at this time (exposed to an average of 590 $\mu g/m^3$). Immediately after exposure, no effect of CAP was detected in the bronchitic rats exposed to an average of 460 $\mu g/m^3$, but at the next highest exposure concentration of 640 $\mu g/m^3$, several BAL fluid parameters were statistically significantly altered (increased albumin and total protein and increased NAG activity). Bronchitic rats were more affected in this respect by CAP than were healthy rats; however, there was no consistent dose response, as BAL fluid parameters were not significantly affected by exposures of 870 or 910 $\mu g/m^3$. Some parameters were altered by bronchitis itself.

Dogs were rendered bronchitic by exposure to gradually increasing concentrations of SO_2 for five weeks (Godleski *et al.*, 2000). Pulmonary inflammation and mucus secretion were confirmed by BAL, and bronchitic and healthy dogs were then implanted with ECG leads and exposed to CAP after another three weeks of SO_2 exposure. SO_2 exposure caused a large shift in BAL cells from macrophages to neutrophils, but additional CAP exposure did not appear to cause further change. CAP exposure did not produce any consistent changes in cardiac or respiratory parameters, and this disease model was abandoned.

Godleski *et al.* (1996) is the only paper to have reported deaths after brief CAP exposure. The paper was prepared for a scientific colloquium, but has not appeared in a peer-reviewed journal, and apparently has not been corroborated, so perhaps some factor other than CAP was responsible for the deaths — or perhaps a virus or other acutely toxic agent was concentrated from the ambient air during that one set of experiments but during no others. If CAP exposure in

[9] It is not clear how many animals this percentage represents. The paper reports a "minimum of 10 rats per group." No number of deaths out of a group of 10 animals yields a mortality rate of 37%. Perhaps there were 11 rats and 4 deaths, which yields a mortality rate of 36%. If there were 12 rats and 4 deaths, the mortality rate would be 33%.

fact caused the deaths, the findings would seem to be inconsistent with the work of Clarke *et al.* (1999) and Kodavanti *et al.* (2000), who used larger CAP exposures, but in those studies, bronchitis was produced by lesser concentrations of SO_2. Conceivably, susceptibility to death depends on the severity of bronchitis, on the particular composition of CAP, or on some stresses associated with CAP exposure, but not control exposure, in certain experimental settings. Mortality aside, there are indications in these studies that some CAP effects are larger in bronchitic rats than healthy animals, but dose-response is lacking and effects may be transient.

CAP studies of animal models of cardiopulmonary disease

Epidemiologic studies suggest that exposure to ambient particulate increases deaths from cardiopulmonary diseases. It is hypothesized that people with preexisting cardiopulmonary conditions are particularly susceptible to exposure. Work by Gordon *et al.* (1998 and 2000) and Godleski *et al.* (2000) has investigated effects of CAP exposure on animals (rats, guinea pigs, and dogs) with natural or induced heart disease.

The findings of Gordon *et al.* (1998) are difficult to interpret, as the methods are insufficiently detailed and no data are presented. It appears that rats were injected with monocrotaline to induce pulmonary hypertension, a model for cor pulmonale.[10] Treated and untreated animals were then exposed to CAP for three hours, at concentrations ranging at least from 110 to 360 $\mu g/m^3$. CAP exposures (concentrations not specified) induced increases in circulating neutrophils and decreases in lymphocytes three but not 24 hours after exposure in both treated and untreated rats; whether changes were larger in monocrotaline-treated animals is not discussed. No signs of lung injury or inflammation were seen during necropsy. Cell counts, protein, and LDH were increased in BAL fluid of monocrotaline-treated rats at 360 $\mu g/m^3$ but not at lower concentrations, and never in untreated animals. Heart rate was increased for several hours, to a similar degree, after CAP exposure in animals both treated and not treated with monocrotaline. This increase in heart rate was more persistent after repeated CAP exposures.

Monocrotaline-treated rats were also exposed to CAP in the investigation of Gordon *et al.* (2000). Unfortunately, healthy rats were not always exposed to the same CAP atmospheres as monocrotaline-treated rats, so whether CAP responses differ according to pulmonary disease status is sometimes unclear. In treated rats, a single exposure to 400 $\mu g/m^3$ CAP, but not 217 $\mu g/m^3$, caused a statistically significant increase in cell count, protein content, and LDH activity in BAL fluid, compared to values in air-exposed treated rats; but the highest CAP exposure used for healthy animals in this part of the investigation was 184 $\mu g/m^3$. In monocrotaline-treated rats, exposure to at least 217 $\mu g/m^3$ CAP, but not 157 $\mu g/m^3$, caused a statistically significant increase in the percentage of neutrophils and decrease in the percentage of lymphocytes in blood. Similar changes occurred in healthy animals at lower CAP exposures, but not at concentrations of 212 to 350 $\mu g/m^3$. These hematologic changes were present three hours after, but not 24 hours after, the single six-hour CAP exposures. In monocrotaline-treated rats exposed to 400 $\mu g/m^3$ CAP, but not 127 $\mu g/m^3$, heart rate was statistically significantly increased during the first post-exposure hour. In normal rats, exposure to CAP of 132 $\mu g/m^3$ or more caused an increase in heart rate over various post-exposure intervals. CAP exposure of 219 $\mu g/m^3$ produced no observable histologic alterations in lungs of healthy or monocrotaline-treated rats, and exposure to 181 $\mu g/m^3$ produced no spirometric changes. Overall, there is little evidence in this study that CAP exposure had a more detrimental effect on monocrotaline-exposed rats than healthy rats.

[10] Cor pulmonale is an enlargement of the right ventricle that occurs because of pulmonary hypertension from lung disorders (commonly chronic bronchitis or emphysema).

These investigators also exposed healthy guinea pigs, and guinea pigs with a genetic cardiomyopathy, to CAP. Few statistically significant alterations in lung or hematologic parameters were observed, and ill hamsters did not appear more sensitive to CAP effects (Gordon *et al.*, 2000).

Healthy dogs, and dogs with balloons implanted in cardiac arteries to permit occlusion of the arteries, underwent three-day CAP exposures in the study of Godleski *et al.* (2000). Pulmonary findings were virtually nil, but certain measures of heart function and electrical activity were modified by CAP exposure, particularly the low- and high-frequency powers of heart rate variability. Although healthy and occluded dogs were not co-exposed to CAP (and, thus, exposure concentrations and composition differed), the investigators believed the CAP responses in occluded dogs were larger and more significant than responses in healthy dogs, compared to responses in dogs exposed to filtered air.

CAP studies of aged animals

Some epidemiologic studies of particulate exposure and mortality suggest that the elderly are particularly vulnerable. One experimental investigation (Clarke *et al.*, 2000a) has specifically compared the responses of old and young rats to CAP exposure; another (Gordon *et al.*, 2000) used young and somewhat older rats (treated with monocrotaline) but did not compare responses. In the Clarke *et al.* study, young (two-month-old) and aged (17-month-old) rats were exposed to CAP five hours per day for three days at an average of 100 $\mu g/m^3$, after which BAL fluid and blood were collected. CAP, independent of age, caused an increase in BAL neutrophils and an increase in peripheral blood eosinophils in both groups, and increased BAL lymphocytes and macrophages in young rats only. CAP did not affect any parameter in aged rats only. The increases in BAL neutrophils and blood eosinophils following CAP exposure were less in old rats than in young rats. The study hypothesis, that aged rats would show more severe pulmonary inflammation and hematologic changes than young rats in response to CAP, was not supported by the data.

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10. *Curricula vitae* of authors

LAURA C. GREEN, Ph.D., D.A.B.T.

EDUCATION:

1981. Massachusetts Institute of Technology, Cambridge, Massachusetts. Ph.D. from the Department of Nutrition and Food Science (currently the Biological Engineering Division). Ph.D. thesis, "Nitrite and Nitrate: Toxicity, Metabolism, and Biosynthesis." Discovered that nitrate is biosynthesized *in vivo* in humans and in rats by a mammalian process. Investigated the toxicology and pharmacokinetics of nitrate and nitrite. Designed and built a novel automated system for the analysis of nitrate and nitrite in biological and environmental samples.

1975. B.A. with honors in Chemistry, Wellesley College, Wellesley, Massachusetts. Also studied Biology, Physics, Philosophy, and Literature. Honors included: Phi Beta Kappa, Sigma Xi, American Institute of Chemists Student Award, and Wellesley College Scholar.

BOARD CERTIFICATION:

Certification in general toxicology — Diplomate of the American Board of Toxicology (D.A.B.T.), 1988; recertified 1993, 1998, and 2003.

PROFESSIONAL EXPERIENCE:

1986-Present. Lecturer, Biological Engineering Division, Massachusetts Institute of Technology.

1989-Present. Senior Scientist and President, Cambridge Environmental Inc., Cambridge, MA.

1985-1989. Vice President for Environmental Health and Toxicology, Meta Systems Inc., Cambridge, MA.

1983-1986. Research Affiliate and Project Coordinator for a five-year grant from the American Cancer Society for work in biochemical epidemiology, Department of Applied Biological Sciences, Massachusetts Institute of Technology.

1983-1985. Research Director of the Scientific Conflict Mapping Project, Harvard University School of Public Health. Developed a new method for making scientific and regulatory decisions about toxic and carcinogenic chemicals in the workplace and environment. Co-authored a book entitled, *In Search of Safety: Chemicals and Cancer Risk* (Harvard University Press, 1988).

1978-1985. Consultant in toxicology and risk assessment, self-employed.

1981-1983. Postdoctoral Fellow in Environmental Toxicology, Massachusetts Institute of Technology. Research with Professor Gerald Wogan directed toward developing dosimeters for carcinogenic chemicals. Studied the covalent modification of hemoglobin and albumin by carcinogens. Determined that blood protein adduction was quantitative and sensitive, and therefore of use in assessing actual human exposures.

1975-1981. Research Assistant, Teaching Assistant, and Predoctoral Trainee, Department of Nutrition and Food Science (currently the Division of Toxicology), Massachusetts Institute of Technology.

Summer, 1974. Research Chemist, Dow Chemical Company, Wayland, Massachusetts. Developed a direct oxidative synthesis method for propylene oxide.

OTHER PROFESSIONAL ACTIVITIES:

Toxicologist on the Massachusetts Department of Public Health's Medical Review Panel on Formaldehyde-related Claims
Invited peer reviewer, Agency for Toxic Substances and Disease Registry
Invited peer reviewer, United States Environmental Protection Agency
Invited peer reviewer, *Cancer Epidemiology, Biomarkers & Prevention*
Invited peer reviewer, *Epidemiology*
Invited peer reviewer, *Inhalation Toxicology*
Invited peer reviewer, *Risk Analysis*
Invited peer reviewer, *Solid Waste and Power*
Invited member, Visiting Committee, Whitaker College of Health Sciences and Technology, M.I.T.
Invited member, Visiting Committee, Division of Bioengineering and Environmental Health, M.I.T.
Invited member, Board of Scientific and Policy Advisors, American Council on Science and Health
Invited lecturer in Toxicology, Harvard School of Public Health
Invited member, Massachusetts Department of Environmental Protection, Science Advisory Panel.

PROFESSIONAL ORGANIZATIONS:

American Association for the Advancement of Science
American Chemical Society
Society of Toxicology

SELECTED CONSULTING PROJECT EXPERIENCE:

Quantitative methods in risk assessment

- Evaluated, developed, and applied various methods intended to estimate low-dose risks to human health from various exposures. Combined evidence from rodent and human studies; developed and applied probabilistic, Monte Carlo techniques; and developed holistic risk profiles.

Trichloroethylene and related chlorinated hydrocarbons

- Analyzed and critiqued literature on toxicity of trichloroethylene and related compounds. Studied trichloroethylene-induced carcinogenesis and neurotoxicity associated with trichloroethylene and its products of degradation. Provided detailed quantitative commentary to the Agency for Toxic Substances and Disease Registry (ATSDR) and the National Toxicology Program (NTP) on the toxicology and epidemiology of trichloroethylene.

Polychlorinated dibenzodioxins, dibenzofurans, and polychlorinated biphenyls

- Studied the fate, transport, and impacts of emissions of polychlorinated dibenzo(*p*)dioxins (PCDDs) and related compounds. Analyzed and developed various extrapolation models intended to predict low-dose risks to human health. Performed specific quantitative risk assessments for various exposures to PCDDs; focussed on excess risks of cancer and on risks of reproductive toxicity. Assessed risks associated with emissions of PCDDs and associated compounds from incinerators, paper mills, and other sources. Evaluated exposures to and risks from PCBs in a number of settings.

Benzene and related compounds

- Developed and applied expertise in benzene-induced leukemia. Performed qualitative and quantitative assessments of risk associated with various routes and levels of exposure. Evaluated risks of acute myelogenous leukemia and other hematopoietic disorders from known and suspected causes, including various chemicals, drugs, and radiation.

- Assessed hazards associated with exposures to benzene, toluene, and xylenes emanating from leaking underground storage tanks and above-ground spills of gasoline.

Mercury

- Performed an in-depth study on sources of mercury in municipal solid waste. Assessed toxicity to mercury given current environmental exposures and under extreme scenarios. Studied mechanisms of bioaccumulation of mercury in fish.

Municipal solid waste: environmental aspects of waste-to-energy and of landfills

- Performed or participated in many in-depth assessments of risk associated with management of municipal solid waste. Developed and applied expertise on risks associated with airborne emissions from waste-to-energy plants and from solid waste landfills. Applied technical knowledge to criticism of various proposed regulations concerning landfills, waste-to-energy plants, and various Superfund sites.

Hazardous waste: environmental aspects of incineration and of land disposal

- Performed and peer-reviewed in-depth assessments of risks associated with incineration of hazardous waste. Helped to develop regulatory approaches to modeling the fate and transport of contaminants released during incineration. Evaluated impacts to public health and the environment from land disposal of various types of hazardous wastes.

Food microbiology and food toxicology

- Evaluated matters involving salmonellosis from ingestion of poultry, alleged salmonellosis from ingestion of eggs, illnesses arising from *E. coli* in contaminated apple cider, and related incidents.

- Served as consultant to National Academy of Sciences, Committee for a Study of Saccharin and Food Safety Policy. Developed relative risk assessment for the food additive uses of nitrite in cured meats; quantified and balanced risks of botulism (were nitrite absent from the food product) against risks of cancer (from N-nitroso compounds formed during cooking in the presence of nitrite).

Miscellaneous risk assessment and risk communication

- Participated in scores of public hearings on various matters of environmental health and safety. Presented testimony on general toxicologic matters, as well as on specific aspects of waste water discharges, pesticide applications, and workplace exposures to various chemicals.

ORIGINAL REPORTS:

Ames, M., Zemba, S.G. and Green, L.G. (2005). Results of an emission and air dispersion modeling study and public health evaluation of the Virginia Paving Company Facility, 5601 Courtney Avenue, Alexandria, Virginia. Cambridge Environmental Inc.

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Green, L.C., Crouch, E.A.C., Armstrong, S.R., and Lester, R.R. (1995). Comments on *Health assessment document for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds: review draft*. Cambridge Environmental Inc.

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Zemba, S.G., Lash, T.L., and Green, L.C. (1993). A health risk assessment of the Recycle Energy System (RES) Facility in Akron, OH. Cambridge Environmental Inc.

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EDUCATION:

1975. Ph.D., University of Cambridge, England, High Energy Physics, (Thesis: "The Algebraic Structure of Some Dual Resonance Models").

1972. B.A., University of Cambridge, England, Natural Sciences (Theoretical Physics).

PROFESSIONAL EXPERIENCE:

1989–Present. Senior Scientist, Cambridge Environmental Inc., Cambridge, Massachusetts.

1987–2005. Associate of the Department of Physics, Harvard University, Cambridge, Massachusetts.

1992-1994. Lecturer in the Department of Epidemiology, Harvard University School of Public Health.

1990–1992. Assistant Professor of Community Health, Tufts University School of Medicine, Boston, Massachusetts.

1987–1989. Senior Scientist, Environmental Health and Toxicology Group, Meta Systems Inc., Cambridge, Massachusetts.

1979-1986. Research Associate In Physics, Jefferson Physical Laboratory, Harvard University, Cambridge, Massachusetts.

1977-1979. Research Fellow in Physics, Jefferson Physical Laboratory, Harvard University, Cambridge, Massachusetts.

1984-1986. Consulting Associate in Risk Assessment, Meta Systems Inc., Cambridge, Massachusetts.

1974-1977. Research Fellow in the Energy Research Group, Cavendish Laboratory, University of Cambridge, Cambridge, England.

NATIONAL ACADEMY OF SCIENCE COMMITTEES

Committee on Health Effects of Waste Incineration, Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council. 1995–1999. (*Waste Incineration and Public Health*, National Academy Press, 2000).

Committee on Risk-Based Criteria for Non-RCRA Hazardous Waste, Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council. 1998–1999. (*Risk-Based Waste Classification in California*. National Academy Press, 1999).

Committee on Toxicology, Subcommittee on the Atsugi Incinerator, Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council. 2000. (*Review of the US Navy's Human Health Risk Assessment of the Naval Air Facility at Atsugi, Japan*, National Academy Press, 2001).

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Committee on An Assessment of Centers for Disease Control and Prevention Radiation Studies: Review of a Research Protocol Prepared by the University of Utah, Board on Radiation Effects Research, Division on Earth and Life Studies, National Research Council. 2001–2002. (*Review of a Research Protocol Prepared by the University of Utah: Letter Report*, National Academy Press, 2002).

Committee to Review Methods for Estimating Radiation Doses to Workers at Hanford, Board on Radiation Effects Research, Division on Earth and Life Studies, National Research Council. 2001. (*Letter Report: Review of Methods for Estimating Radiation Doses to Workers at Hanford*, National Academy Press, 2002).

Committee to Review the Identification of Radionuclides released from the Hanford Nuclear Reservation's Facilities to the Columbia River, Board on Radiation Effects Research, Division on Earth and Life Studies, National Research Council. 2002 (*Letter Report: Review of the Identification of Radionuclides Released from the Hanford Nuclear Reservation's Facilities to the Columbia River*, National Academy Press, 2002)

Committee to Review the CDC-NCI Feasibility Study of the Health Consequences to the American Population from Nuclear Weapons Tests, Board on Radiation Effects Research, Division on Earth and Life Studies, National Research Council. 2002. (*Exposure of the American Population to Radioactive Fallout from Nuclear Weapons Tests*, National Academy Press, 2003)

Committee on Superfund Site Assessment and Remediation in the Coeur D'Alene River Basin, Board on Environmental Studies and Toxicology, Division on Earth and Life Studies, National Research Council, 2004–2005. (*Superfund and Mining Megasites — Lessons from the Coeur d'Alene River Basin*, National Academy Press, Prepublication copy, July 2005).

SELECTED PROJECT EXPERIENCE:

- Managed and wrote several site risk assessments for Massachusetts and Federal Superfund sites.

- Managed and wrote several risk assessments for Waste-to-Energy plants.

- Designed and wrote the probabilistic risk assessment for FSIS, USDA, on the risk of *C. Perfringens* in Ready-to-Eat and Partially Cooked Foods.

- Designed the theory and implemented in computer code a site assessment tool (called RISK-ON-SITE™) that uses Voronoi diagrams to assist in estimating risks from soil and groundwater on a site. Provided suitable color displays to allow rapid evaluation of the available data on a site. Extended the methodology to give risk-based estimates of required clean-up levels.

- Developed simple algorithms (theory and computer implementation) for air-dispersion modeling to account rapidly and adequately for area sources. Applied these and full EPA air dispersion algorithms in RISK-ON-SITE™ for a site in California.

- Performed statistically correct comparisons of metals concentrations in groundwater upgradient and downgradient of a Superfund landfill site, and showed that the entire apparent difference in mean concentrations was accounted for by the skewness of the distribution and the larger number of samples taken downgradient.

- Derived and used in risk assessments and in support of various litigations many quantitative exposure assessments involving transport and fate of chemicals in the natural (for example: air, soil, groundwater, surface water) and man-made (for example: indoor and outdoor air) environment.

- Wrote and maintain a software package (MSTAGE) to evaluate the results of carcinogenesis bioassays in laboratory animals. This package is flexible enough to incorporate the standard EPA methodologies, but may also be used with more advanced methodologies incorporating uncertainty correctly.

- Performed full uncertainty analyses of cancer risk assessments for several chemicals and in several situations, incorporating all the known uncertainties in a consistent fashion.

- Provided expert technical comments on proposed EPA rules in such areas as exposure assessment (for example: for landfill leachate and landfill gas), and risk assessment methodology (for example, as applied to the Hazard Ranking System).

SELECTED ORIGINAL REPORTS:

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Green, L.C., Crouch, E.A.C., Ames, M.R., and Lash, T.L. (2002). What's wrong with the National Ambient Air Quality Standard (NAAQS) for fine particulate matter ($PM_{2.5}$)? *Regulatory Toxicology and Pharmacology 35*:327-337.

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