Dear Sir;

I wish first of all to thank all the members and their staff for the thoroughness of their work and excellent presentations made for the public consultation at the Ste. Anne de Bellevue Library, Tuesday March 30, 7.00 pm.

Re: Maximiser la biodiversitéet augmenter la superficie des milieux naturals protegés a Montreal.
Favoriser une meilleure integration des ecosystemes et des paysages naturels dans les melieux de vie.
Assurer la pérennité des milieux naturals dans les parcs et favoriser la consalidation et la viabilité des écosystèms présents.

I asked a question at that meeting and I wish to pursue it further. RE: THE REGISTRATION OF PRIVATE PROPERTIES AS SANCTUARIES.

1. The registration could take place over aperiod of 2 years with the owner supplying proof(by photo & etc.,) of meeting the standards set up by government.
2. The registration fee could be $ 15- $25. dollars each year on succesive registrations. Paid by owner. And the reception of a written certificate of passing the standards set up.
3. A basic list of the criteria from either one to ten or 20 of standards set up by the Dept such as A) types of shrubs beneficial to birds or bees, or butterflies & etc, or a bat Sanctuary, or Blue bird houses with these birds, or a butterfly garden, or trees beneficial , protect from pollution, with years of life, and of a size for properties. Or a mixture of some of the above.

Benefits:

BENEFITS:

1. It would give much needed funding for the work on the protection o of the forests, and so forth.

2. Citizens would learn about the natural world around them. the earth and the fauna, their children would receive a beneficial learning experience for life.

3 It would encourage citizens not to use pesticides nor fertilizers nor insecticides on their properties.

4 It would benefit the Governments projects and at the same time benefit the owner , such as greater prestige for their properties, greater valu value when selling $ .Elevating the property value, benefiting and encouraging buyers, and at the same time eliminating their fears re contaminated land surrounding the house and on that property. Thus children encouraged to play on the property, safely, healthily. Thus the government benefits, and the owners benefit and so does our na natural world.

To me the whole project would be a teaching, learning, benefiting project and experience, at the same time protecting the land for future g enerations. Citizens would learn to care for the earth in knowledgeable ways whatever their expertise.

Yours Very Sincerely,

Maureen Murray R.N.

Maureen Murray R.N.

P.S. I have enclosed a photocopy of gardening tips that I cop copy and give out in our Town.

P.P.S. To stay on the register the owner must pay a fee of $10.00 dollars each successive year, if property sold that owner must pay the full fee e.g. 15 or 30 to register.

In the 1940s, my family found out that our yard was contaminated by lead from a gasoline station and car-painting factory next door. We had to dig up a foot of the soil and have it removed to a hazardous-waste dump. If we hadn't cleaned it up, we would have been creating problems for ourselves and for anyone who lived there after us.

FOR A FABULOUS LAWN THAT EVEN YOUR NEIGHBORS WILL LOVE, here's all you have to do: Plant a native variety of grass in soil enriched with compost, which adds all the nutrients you need. Cut the grass to about three inches high (longer than the norm) so that it can develop a strong, deep root system.

Water the lawn occasionally, but only if you must. Most people overwater their lawns, so the chemicals that remain on the grass wash into our rivers and streams, killing fish. The chemicals then go back into our water system, which we draw on to drink. Pesticides are also subject to vapor drift; that is, they are transported by air to other areas.

CLEANER

Better lawn and garden. BY MARIA RODALE

Fertilize your lawn—but only if it looks limp and pale and seems to need nutrients—by topping it with fresh compost and running over it with your lawn mower. Also, consider shrinking the size of your lawn by planting more shrubs and trees for wildlife.

Next, learn to live with bugs. This planet could survive without people, but it needs ants and bees. Ants aerate the soil and circulate nutrients even more than earthworms do. Honeybees tap 2 million flowers and fly 55,000 miles to make a pound of honey. In the process, they pollinate most of our fruits, vegetables, herbs, and flowers.

When you're faced with a mysterious invasion of bugs, it's tempting to think that a little bit of pesticide here or there won't matter. But pesticides don't kill only the bugs that you think are causing trouble; they also kill the ones that help you. I look at bugs as messengers. Beneficial insects have a job to do, and often their job is to eat and decompose diseased or malnourished plant material. If you have a bug problem, it's a symptom of a greater problem. Maybe your soil needs compost. Maybe you have been planting the same thing in the same place for too long. Maybe you planted things in the wrong place to begin with. Whatever your problem, get to the root of it; don't kill the messenger.

Finally, if you garden organically and observe and commit to learning from nature, you can't help ending up with a profound faith in nature and a respect for the role that each creature performs. So plant flowers for the bees, let the ants roam free, and listen to what the bugs are trying to tell you about your plants and your garden.

We all have a responsibility to do what we can to make the world a better place. For me there is no greater joy than going outside on a summer evening after work and lying in the warm grass with my kids. With a clean lawn and a clean yard, I can do so with a clean conscience. I have faith that I am doing the right thing for myself, for my family, and for nature. You can, too. ❧

Maria Rodale is the vice-chairwoman of Rodale Press and the author of Maria Rodale's Organic Gardening *(1998).*

The 20 All-Time-Greatest Organic-Gardening Tips

BY ELLEN PHILLIPS

In the past 50 years, the organic-gardening movement has contributed thousands of tips and techniques to gardeners everywhere. Here are 20 of the most enduring.

1. Make and use compost. Compost is a wonderful way to recycle kitchen and yard wastes and enrich your garden while keeping trash out of the landfill. Just heap grass clippings, shredded leaves, vegetable and fruit scraps, shredded newspaper, and other organic materials (no meat or fat, please) in a pile, and let it rot. In a year or less, you'll have rich, crumbly, odor-free organic fertilizer for your plants.

2. Mulch. Mulching retains soil moisture, so you don't have to water as often; it suppresses weeds and keeps the soil warm longer, extending your growing season; it enriches the soil as it breaks down; and it makes your garden look better. To mulch, cover the bare ground in your garden bed with a layer of shredded leaves, newspaper, grass clippings, pine needles, compost, or other organic material.

3. Don't treat your soil like dirt. Healthy soil is alive with microorganisms that break down organic matter to release plant nutrients; earthworms and other small creatures that aerate the soil as they burrow and enrich it with their droppings; and tiny feeder rootlets, invisible to the naked eye, which bring nutrients into the plant. Rather than

compacting the soil with heavy machinery and dousing it with toxic chemicals, treat it gently with hand tools, work in the paths rather than stepping on garden beds, and apply healthy doses of organic matter like compost.

4. Rotate your crops. Pests and diseases can build up when you grow the same crop in the same area year after year. By alternating crops, you don't give pest and disease populations a chance to grow.

5. Try companion planting. A big planting of a single crop like corn practically shouts at pests—from earworms to raccoons—"Eat me, eat me!" But when you plant squash and beans in the same plot with your corn, pests are confused and can't recognize the smell, appearance, and other allures of each crop as easily. As a result, you protect all three crops. Growing herbs and other aromatic plants like marigolds with your crops also throws pests off the scent. Adding nectar-producing flowers attracts beneficial insects to fight pests for you.

6. Use foliar feeding for a quick fix. When your plants need a fast-acting boost, spray nutrient-rich liquid seaweed, compost tea, or manure tea directly on the leaves. The nutrients will be absorbed into the plants' tissues immediately, resulting in perkier plants in a matter of hours. To make compost tea or manure tea, fill a five-gallon bucket half full of finished compost or well-rotted manure, top it off with water, and stir daily until the liquid looks like dark tea. Strain out the solids, and apply them to the garden as mulch.

7. Conserve water. Don't waste water and weaken your plants by watering them with a handheld hose or a sprinkler—you'll lose a lot of water to evaporation and encourage shallow rooting, which makes plants more susceptible to drought. Instead, use water-conserving soaker hoses or drip irrigation, which directs water to the plants' roots.

8. Invite garden allies to call your yard home. There's a whole world of creatures that are ready, willing, and able to help you in your fight against garden pests. You just have to invite them in. The easiest way is through water—set out a birdbath on a pedestal and one on the ground, and watch pest-eating birds, beneficial insects, and toads come around. Put up a bat house, and get some real pest-eating power—a bat consumes thousands of insects every night. Beneficial insects love plants with flat flower heads, like dill, fennel, Queen Anne's lace, and yarrow.

9. Discover heirloom flowers, fruits, and vegetables. These plants were originally developed or selected because they grew well in a particular place, had incredible flavor or fragrance, looked fantastic, or served a specific purpose (like cider apples). Today, when most plants are developed to withstand the bumps and bruises of shipping rather than for quality, heirlooms are a real find for the backyard gardener. Check specialty seed catalogues or join the Seed Savers Exchange (319-382-5990) for a wide selection.

10. Use tricks, traps, and homemade treatments to fight pests. When you do encounter pests, don't spray. Netting, sticky traps, shiny strips, beer bait, soap, oil, and garlic–hot pepper spray are just a few of the safe organic tools at your disposal.

11. Fight plant diseases with vinegar and baking soda. As with fending off pests, simple ingredients from your pantry are effective disease-fighters if you catch symptoms early. Among these are a tablespoon of vinegar or baking soda (or both) in a gallon of water. Compost and rhubarb-leaf tea are other safe ingredients.

12. Grow disease-resistant varieties. Check seed catalogues for resistant varieties of crops such as tomatoes and cucumbers. Nurseries often sell alternatives for flowers that are notoriously plagued by a disease—black spot on roses or powdery mildew on phlox or bee balm, for example. (For these three plants, resistant varieties include Stanwell Perpetual, Madame Plantier, and rugosa roses; David phlox; and Marshall's Delight bee balm.)

13. Raise your beds. Growing plants in raised beds rather than at ground level has many advantages. Drainage is improved. Paths are easy to find, so the soil in the beds is less likely to be compacted by feet. And perhaps most important, if your native soil is solid clay, hopelessly rocky, sandy, swampy, acidic, or otherwise impossible, you can create a custom soil for your raised bed that provides an instant fix. Gardeners in the desert Southwest, where raised beds would fry, may instead want to try a sunken bed to retain moisture and cool air.

14. Cover up. Nature abhors a vacuum—in this case, bare soil. Savvy organic gardeners grow cover crops, also called green manure, in their unused plots. These leafy crops—including alfalfa, clover, annual rye, and buckwheat—grow rapidly, outcompeting weeds. They prevent erosion. And when you turn them into the soil at the end of the season or the following spring, they add organic matter and nutrients, building better soil and rejuvenating your garden.

15. Try a chicken tractor. Small livestock, such as a few chickens or rabbits, can make a real contribution to your garden. Rabbits produce nitrogen-rich manure, which can be applied around plants, worked into beds at the end of the season, or added to the compost pile all year. If you let chickens range from garden bed to garden bed in a movable wire cage called a chicken tractor, they'll eat pests and weed seeds, loosen the soil, and deposit nitrogen-rich fertilizer at the same time.

16. Diversify. The more diverse your plantings, the richer your rewards. Don't just grow carrots—try three kinds of carrot, and have a family taste test. (Next year, pit the winner against two more varieties.) Grow a range of strawberry, blueberry, or raspberry varieties so you'll have berries from the earliest possible moment to the end of the season. Enrich your garden with herbs and flowers to attract beneficials and confuse pests. Spice up your landscape with a mix of trees, shrubs, ground covers, and vines for visual interest and to attract wildlife. Add a water garden and delight in the beauty of visiting birds, the call of resident frogs, and the flash of fish. Instead of planting the usual shade trees, choose fruit trees for beauty in flower and bountiful crops.

17. Stretch your seasons. Don't be daunted by a late spring or the threat of winter. You don't need to head South or invest in an energy-eating heated greenhouse to enjoy crops both earlier and later than your neighbors do. Instead, rely on solar power and a few simple devices like cold frames, row covers, cloches, growing pits, or a simple unheated greenhouse. Grow crops and varieties that can take the cold, such as salad greens, cole crops like kale and brussels sprouts, and root crops like carrots and parsnips. Use raised beds and dark mulch to warm up the soil.

18. Buy good tools. You don't need a shedful of tools to have a great garden. But when you purchase tools, choose them carefully and buy the best ones you can afford. Tops on many gardeners' lists are good gloves, a cherished weeder, and sharp pruners. My top three are my beloved rustproof trowel, garden fork, and poacher's spade (a narrow-bladed model that's great for working in between plants).

19. Go native. Native plants help preserve natural diversity and add beauty to a garden. (Consider the spring wildflower display.) They are already regionally adapted, so they'll thrive in your landscape with no fuss. Often pest- and disease-free, they provide food and shelter for native wildlife. Mix native plants in your ornamental beds and borders, or create a woodland or meadow garden especially for native plants. But be responsible—when you buy native plants, make sure they're nursery-propagated and not dug from the wild.

20. Get the big picture. As a homeowner and a gardener, you're responsible for caring for your piece of the earth. One way to start is to look at your yard as a whole, not each garden area as an isolated unit. How do the pieces of your landscape fit together? Have you provided sheltering corridors of trees, shrubs, and other plants so that birds and other wildlife can travel through your property unmolested? Are you avoiding the use of toxic chemicals? Once you begin seeing your whole yard as a garden, you've developed an organic perspective. Organic gardening isn't about "us and them"—us and pests, us and wildlife, us and weeds, our yard and other people's yards. As responsible gardeners, we must recognize that our actions and our choices link all of us together. ✱

Ellen Phillips is the executive editor of garden books at Rodale Press and the coauthor of Rodale's [illegible] Encyclopedia of Perenn[illegible]

Mrs. Murray

Increased Particulate Air Pollution and the Triggering of Myocardial Infarction

Annette Peters, PhD; Douglas W. Dockery, ScD;
James E. Muller, MD; Murray A. Mittleman, MD, DrPH

Background—Elevated concentrations of ambient particulate air pollution have been associated with increased hospital admissions for cardiovascular disease. Whether high concentrations of ambient particles can trigger the onset of acute myocardial infarction (MI), however, remains unknown.

Methods and Results—We interviewed 772 patients with MI in the greater Boston area between January 1995 and May 1996 as part of the Determinants of Myocardial Infarction Onset Study. Hourly concentrations of particle mass <2.5 μm ($PM_{2.5}$), carbon black, and gaseous air pollutants were measured. A case-crossover approach was used to analyze the data for evidence of triggering. The risk of MI onset increased in association with elevated concentrations of fine particles in the previous 2-hour period. In addition, a delayed response associated with 24-hour average exposure 1 day before the onset of symptoms was observed. Multivariate analyses considering both time windows jointly revealed an estimated odds ratio of 1.48 associated with an increase of 25 $\mu g/m^3$ $PM_{2.5}$ during a 2-hour period before the onset and an odds ratio of 1.69 for an increase of 20 $\mu g/m^3$ $PM_{2.5}$ in the 24-hour period 1 day before the onset (95% CIs 1.09, 2.02 and 1.13, 2.34, respectively).

Conclusions—The present study suggests that elevated concentrations of fine particles in the air may transiently elevate the risk of MIs within a few hours and 1 day after exposure. Further studies in other locations are needed to clarify the importance of this potentially preventable trigger of MI. (***Circulation.*** **2001;103:2810-2815.)**

Key Words: myocardial infarction ■ air pollution ■ heart disease ■ epidemiology

Epidemiological analyses throughout the world have shown that high 24-hour average levels of ambient particulate air pollution are associated with an increase in all-cause, respiratory, and cardiovascular disease mortality[1–4]; nevertheless, little information is available on the effect of shorter-term exposures. The harmful effects of elevation of ambient concentrations of particulate matter are well documented in multiple studies of hospital admissions and emergency department visits for respiratory diseases.[1,4] In addition, increased hospital admissions for cardiovascular diseases have been associated with particulate air pollution in studies of numerous American, Canadian, and European cities.[5–9] These results indicate that ambient particulate air pollution is a risk factor not only for respiratory diseases but also for acute cardiovascular events.

Inhaled particles could lead to acute exacerbation of cardiovascular disease through pulmonary inflammation triggering systemic hypercoagulability.[10] Increases in plasma viscosity[11] and C-reactive protein[12] were observed in randomly selected healthy adults after episodes of high particulate air pollution. Increased heart rate,[13,14] decreased heart rate variability,[15–17] and increased risk of implanted cardioverter-defibrillator discharges[18] associated with episodes of particulate air pollution indicate an autonomic nervous system response.

The US Environmental Protection Agency has promulgated a new ambient air quality standard for fine particles (particulate matter <2.5 μm aerodynamic diameter, $PM_{2.5}$).[19] This new standard regulates 24-hour and annual average concentrations and does not address transient elevations (minutes to hours) in fine-particle concentration. There are no published data on the risk of myocardial infarction (MI) in human populations after transient exposures to elevated concentrations of ambient fine particles.

We therefore evaluated the effect of short-term exposure to fine-particulate air pollution on the risk of acute MIs, comparing data from the Determinants of Myocardial Infarction Onset Study (Onset Study) with hourly measurements of fine particles in Boston. We used a case-crossover design[20,21] to specifically assess the risk of exposure to high levels of $PM_{2.5}$ and the timing of the impact of this exposure on the onset of MI.

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Circulation is available at http://www.circulationaha.org

Methods

Study Design

The design of the Onset Study has been described in detail elsewhere.[20–23] In brief, we used a case-crossover study design to assess the change in risk of acute MI during a brief "hazard period" after exposure to potential triggers of MI onset. An important feature of the case-crossover design is that control information for each patient is based on his or her own past exposure experience. Self-matching results in freedom from confounding by risk factors that are stable over time within an individual but often differ between study subjects.

Patient Population

The Onset Study is a multicenter case-crossover study conducted between 1989 and 1996 in 64 centers throughout the United States.[24] Participants were interviewed a median of 4 days after their MI. We analyzed data from 772 Onset Study participants living in the greater Boston area collected between January 20, 1995, and May 25, 1996. Data were collected in 6 centers with ≥50 cases (455 cases), 6 centers with 25 to 49 cases (209 cases), and 14 centers with <25 cases (108 cases).

Interviewers identified eligible cases by reviewing coronary care unit admission logs and patients' charts. For inclusion in the study, patients were required to meet all of the following criteria: symptom onset while in the greater Boston area, ≥1 creatine kinase level above the upper limit of normal for the clinical laboratory performing the test, positive MB isoenzymes, an identifiable onset of pain or other symptoms typical of infarction, and the ability to complete a structured interview. The protocol was approved by the Institutional Review Board at each participating center, and informed consent was obtained from each patient.

Detailed chart reviews and patient interviews were conducted by trained research personnel.[22,23] Data were collected on standard demographic variables as well as risk factors for coronary artery disease. The interview identified the time, place, and characteristics of MI pain and other symptoms.

Air Pollution Measurements

Daily air pollution measurements were collected at a Harvard School of Public Health–operated monitoring site in South Boston starting January 15, 1995.[18] $PM_{2.5}$ and PM_{10} concentrations were measured continuously with a Tapered Element Oscillating Microbalance (Rupprecht and Patashnick model 1400A TEOM). Elemental carbon concentration was determined continuously with an Aethalometer (Magee Scientific Inc), a light-absorption method to measure "black carbon." Ozone concentration was measured with a UV photometer analyzer (TECO model 49, Thermal Environmental). CO concentration was measured with a continuous nondispersive infrared analyzer (Bendix model 8501-5CA). Relative humidity and temperature were measured continuously (Vaisala model MP113Y). The Massachusetts Department of Environmental Protection measured concentrations of sulfur dioxide and nitrogen dioxide hourly in Chelsea, which is ~7.5 km north of the South Boston site. We calculated 24-hour mean values when ≥16 valid hourly measurements were available.

Statistical Analyses

The analysis of case-crossover data is an application of standard methods for stratified data analysis.[20,21] The stratifying variable is the individual patient, as in a crossover experiment. For each subject, 1 case period was matched to 3 control periods exactly 24 hours apart. Thus, by matching time of day for case and control periods, potential confounding by the circadian pattern of MI onset or diurnal patterns in the air pollution were controlled.

Conditional logistic regression analyses were used to analyze the data. Exposure to particles and gases were entered into the model as continuous variables. Odds ratios are expressed for a change in air pollution concentrations from the 5th to the 95th percentile for all measurements available. Separate models were constructed to evaluate the impact of hourly and 24-hour average air pollution concentrations on the onset of MI.

We also evaluated the effect of hourly (2-hour average) and daily (24-hour average) exposures jointly in 1 model. Control periods were selected as multiples of 24 hours starting 3 days before the date and time of the onset of the symptoms. In addition, multivariate analyses adjusting for season, day of the week, and meteorological parameters on the same time scales were estimated. The final model included sine and cosine functions with periods of 1 year plus ½, ⅓, ¼, ⅕, and ⅙ of a year. It also included quadratic terms for minimum temperature and relative humidity during the 2-hour and 24-hour period of exposure and an indicator for the day of week. Results are presented as odds ratios (OR) and 95% CI.

The unidirectional case-crossover analyses might be sensitive to trends in the outcome and the exposure.[25,26] Therefore, control periods close to the event were chosen to minimize the impact of a potential trend. Particulate air pollution concentrations increased over time (0.4 $\mu g/m^3$ per 100 days, $P=0.0002$). Although there was weak evidence of a linear downward trend in the number of cases (−0.05 cases per 100 days, $P=0.23$), the sampling fraction of cases decreased substantially during 1996. Consequently, a downward bias of the estimates would have been expected. This could be demonstrated by choosing control periods >5 days before the event. The bidirectional design has been shown to give unbiased estimates when full case ascertainment was present.[26] Analyses of the present data, however, indicated a bias with the bidirectional design due to incomplete case ascertainment during 1996.

TABLE 1. Characteristics of the Study Population (n=772)

Age, y	
Mean±SD	61.6±13.4
<50	164 (21)
50–69	365 (47)
70+	243 (32)
Sex	
Male	489 (63)
Female	283 (37)
Medical history	
Prior myocardial infarction	237 (31)
Prior angina	174 (23)
Any coronary artery disease	302 (39)
Hypertension	319 (41)
Diabetes mellitus	143 (19)
Obese	261 (34)
Ever smoker	558 (72)
Current smoker	246 (32)

Values are n (%).

Results

The baseline characteristics of the study population are shown in Table 1. The distribution of 24-hour average and 1-hour average concentrations of the particulate and gaseous air pollutants is presented in Table 2. $PM_{2.5}$ and PM_{10} were highly correlated, whereas the coarse fraction of PM_{10}, ie, difference of PM_{10} and $PM_{2.5}$, and the gaseous pollutants were only moderately correlated with $PM_{2.5}$.

Figures 1 and 2 show results from the conditional logistic regression models, in which $PM_{2.5}$ was entered as a linear continuous variable. Odds ratios are expressed for an hourly change of 25 $\mu g/m^3$ in $PM_{2.5}$ (Figure 1) or a daily change of

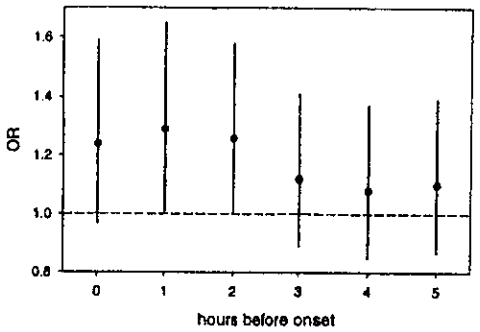

Figure 1. Univariate analyses for association between onset of MI and hourly concentrations of $PM_{2.5}$. Odds ratios and 95% CIs for an increase of 25 $\mu g/m^3$ $PM_{2.5}$.

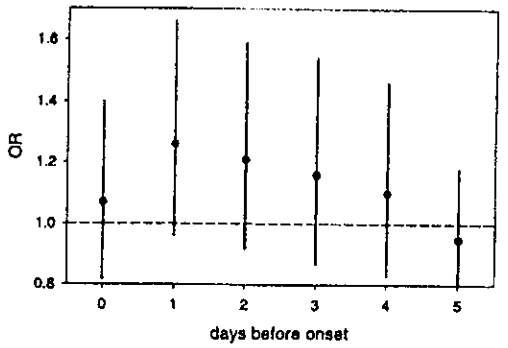

Figure 2. Univariate analyses for association between onset of MI and 24-hour average concentrations of $PM_{2.5}$. Odds ratios and 95% CIs for an increase of 20 $\mu g/m^3$ $PM_{2.5}$.

20 $\mu g/m^3$ $PM_{2.5}$ (Figure 2) corresponding the 5th to 95th percentile intervals (Table 2).

A positive association between the onset of MI and the concentrations of $PM_{2.5}$ was observed within the first 3 hours (Figure 1) that was statistically significant for the $PM_{2.5}$ concentrations 1 hour and 2 hours before the onset of symptoms of an MI. Exposures before this time period seemed to have little impact on the risk of acute MI. In addition, a more delayed response to air pollution was observed when 24-hour averages of the particles were considered (Figure 2). A positive association was observed with

TABLE 2. Distribution of the Air Pollutants for the Time Period January 15, 1995, to May 25, 1996, in Boston, Mass

							Correlation With $PM_{2.5}$	
	n	Mean	SD	5%	50%	95%	1-Hour	24-Hour
Particles								
$PM_{2.5}$, $\mu g/m^3$								
1-hour	11 457	12.1	8.9	2.6	9.7	29.6	1.00	
24-hour	490	12.1	6.8	4.6	10.4	24.3		1.00
PM^{10}, $\mu g/m^3$								
1-hour	11 698	19.4	12.8	4.5	16.7	43.7	0.87	
24-hour	497	19.4	9.4	7.8	17.6	37.0		0.90
Coarse mass, $\mu g/m^3$								
1-hour	11 357	7.3	6.6	−0.7	6.2	19.4	0.33	
24-hour	490	7.4	4.4	1.6	6.8	15.2		0.38
Black carbon, $\mu g/m^3$								
1-hour	11 466	1.34	1.21	0.27	0.97	3.71	0.68	
24-hour	488	1.35	0.72	0.49	1.18	2.83		0.74
Gases								
Ozone, ppb								
1-hour	10 884	19.8	14.8	1	18	46	−0.03	
24-hour	460	19.9	10.0	6	19	36		0.10
Carbon monoxide, ppm								
1-hour	11 843	1.09	0.58	0.30	1.00	2.10	0.54	
24-hour	497	1.09	0.40	0.49	1.07	1.78		0.57
Nitrogen dioxide, ppm								
1-hour	11 671	0.023	0.013	0.007	0.021	0.047	0.51	
24-hour	494	0.024	0.009	0.011	0.022	0.039		0.60
Sulfur dioxide, ppm								
1-hour	11 796	0.007	0.010	0	0.005	0.023	0.38	
24-hour	497	0.007	0.007	0.001	0.006	0.020		0.43

TABLE 3. Odds Ratios for 2-Hour (1 Hour Before Onset) and 24-Hour Average (on the Previous Day) Concentrations of $PM_{2.5}$ Considered Jointly

	Quintile					Trend Test *P*
	I	II	III	IV	V	
2-hour average $PM_{2.5}$						
Range, $\mu g/m^3$	0–5.2	5.3–7.9	7.9–11.5	11.6–17.0	17.1–74.8	
Odds ratios (CI)	1.00	1.15 (0.90, 1.48)	1.09 (0.84, 1.41)	1.27 (0.99, 1.64)	1.44 (1.12, 1.87)	0.025
24-hour average $PM_{2.5}$						
Range, $\mu g/m^3$	1.6–6.4	6.5–8.6	8.7–11.5	11.6–16.2	16.3–52.2	
Odds ratios (CI)	1.00	1.12 (0.87, 1.45)	1.15 (0.89, 1.48)	1.31 (1.01, 1.69)	1.32 (1.01, 1.72)	0.008

elevated concentrations between 24 and 48 hours before the onset of the symptoms.

A combined analysis considered 2-hour averages (between 60 and 180 minutes before the onset of symptoms) and 24-hour averages (between 24 and 48 hours before the onset of the symptoms) jointly, with pollution levels divided into quintiles (Table 3). When concentrations of $PM_{2.5}$ were elevated immediately before the onset of symptoms as well as 1 day before the onset of symptoms, the risk of an MI was increased.

Table 4 summarizes the association between ambient air pollution as a continuous measure and the risk of onset of MI. The estimates of the combined analyses of 2-hour averages and 24-hour averages were larger than the analyses considering the time periods individually. Statistically significantly elevated risks of MI were observed for $PM_{2.5}$. The coarse

TABLE 4. Odds Ratios for 2-Hour and 24-Hour Average Concentrations of Single Pollutants Estimated Jointly.

	Increase (5th to 95th Percentile)	Unadjusted OR (95% CI) (n=772)	Adjusted* OR (95% CI) (n=764)
Particles			
$PM_{2.5}$, $\mu g/m^3$			
2-hour	25	1.43 (1.13, 1.81)	1.48 (1.09, 2.02)
24-hour	20	1.44 (1.11, 1.86)	1.62 (1.13, 2.34)
PM_{10}, $\mu g/m^3$			
2-hour	40	1.45 (1.11, 1.88)	1.51 (1.06, 2.15)
24-hour	30	1.31 (0.99, 1.73)	1.66 (1.11, 2.49)
Coarse mass, $\mu g/m^3$			
2-hour	15	1.13 (0.92, 1.40)	1.16 (0.89, 1.51)
24-hour	15	1.18 (0.85, 1.64)	1.39 (0.89, 2.15)
Black carbon, $\mu g/m^3$			
2-hour	3	1.32 (1.06, 1.65)	1.27 (0.97, 1.68)
24-hour	2	1.08 (0.84, 1.39)	1.21 (0.87, 1.70)
Gases			
Ozone, ppb			
2-hour	45	1.05 (0.76, 1.46)	1.31 (0.85, 2.03)
24-hour	30	1.21 (0.88, 1.67)	0.94 (0.60, 1.49)
Carbon monoxide, ppm			
2-hour	1.0	1.27 (0.98, 1.63)	1.22 (0.89, 1.67)
24-hour	0.6	0.99 (0.77, 1.27)	0.98 (0.70, 1.36)
Nitrogen dioxide, ppm			
2-hour	0.040	1.20 (0.91, 1.59)	1.08 (0.76, 1.53)
24-hour	0.030	1.03 (0.77, 1.39)	1.19 (0.81, 1.77)
Sulfur dioxide, ppm			
2-hour	0.020	1.00 (0.87, 1.14)	0.96 (0.83, 1.12)
24-hour	0.020	0.92 (0.71, 1.20)	0.91 (0.67, 1.23)

Estimates are calculated for a change from 5th to 95th percentile of the pollutants.
*Adjusted for season, meteorological parameters, and day of the week.

fraction of PM_{10}, black carbon, and the gaseous air pollutants including carbon monoxide, NO_2, SO_2, and ozone showed positive associations, but none were statistically significant.

A strong seasonal pattern was observed, with increased risks of MI between May and December. Temperature and humidity immediately before the onset of symptoms were not associated with the onset of symptoms, but the 24-hour averages of higher temperatures and lower humidity 1 day before the onset of symptoms showed an increased risk. After adjustment for seasonal and meteorological conditions, the association of $PM_{2.5}$ with the onset of MI was sustained (Table 4).

Discussion

Elevated concentrations of fine particles ($PM_{2.5}$) were associated with a transient risk of acute MI onset. High 24-hour average concentrations of fine particles were also associated with an elevated risk of MI with a 24-hour delay. The elevated risks during 2 separate time periods appear to be independent of each other. In addition, even changes from low to moderate ambient concentrations were associated with an increased risk of MI, although $PM_{2.5}$ concentrations were below the new standards.[23] Particles >2.5 μm, which consist primarily of resuspended crustal material, showed a substantially smaller association than particles <2.5 μm. Other pollutants, such as black carbon, carbon monoxide, nitrogen dioxide, and sulfur dioxide, showed positive associations, but none of them achieved statistical significance in the single-pollutant multivariate analyses.

These results are consistent with time-series analyses on hospital admissions for cardiac diseases.[5–9] Hospital admission data collected for administrative purposes were positively associated with 24-hour average particle mass concentrations collected for regulatory compliance monitoring. The effect of ambient particles on hospital admissions was reported to vary between an immediate response on the same day[5–7,9] and a 1-day lagged response.[8]

There are several biological effects of ambient particles that may lead to cardiac events. First, particles deposited in the alveoli lead to activation of cytokine production by alveolar macrophages[27] and epithelial cells[28] and to recruitment of inflammatory cells.[29] Second, increases in plasma viscosity[11] and C-reactive protein[12] have been observed in randomly selected healthy adults in association with episodes of high particulate air pollution. Third, acceleration of heart rates and diminished heart rate variability in association with air pollution have been documented in elderly persons[13,15–17] and in a random population sample.[14] One study reported that heart rate variability started to decrease within hours of exposure.[17] Controlled-exposure experiments in dogs exposed to concentrated ambient particles indicated changes in the ECG within an hour of the onset of exposure.[30] Fourth, ambient concentrations of $PM_{2.5}$ have been associated with ventricular fibrillation and an increased number of therapeutic interventions in patients with implanted cardioverter-defibrillators.[18]

A proposed mechanism for triggering of MI is that onset occurs when a vulnerable but not necessarily stenotic atherosclerotic plaque disrupts in response to hemodynamic stress; thereafter, hemostatic and vasoconstrictive forces determine whether the resultant thrombus becomes occlusive.[31] As reviewed above, particulate air pollution is associated with hemodynamic and hemostatic alterations, which may contribute to MI onset.

Previous studies have shown that physical[23,32] and psychological[24] stress as well as substances such as cocaine[22] can trigger the onset of MI. In this report, we demonstrate that transient exposures to an environmental factor, ie, ambient air pollution, appear to increase the risk of an acute MI.

The available evidence suggests that the mechanisms responsible for the impact of ambient particles on MI may be similar to the mechanisms responsible for triggering by other stressors. If these findings are substantiated, susceptible subgroups could be identified and possible pharmacological interventions could be developed to protect the public from transient exposures to ambient particles, such as that experienced during rush-hour traffic.

Limitations

The case-crossover design controls for chronic risk factors for MI such as sex, age, and hypertension. Confounding may occur because of time-varying risk factors,[26] such as time of day, season, or weather. These potential confounders, however, were considered in the multivariate analyses.

Another potential limitation of the study is that only 1 air pollution monitoring site was available. Air pollution measurements throughout the east coast indicate that the elevated concentrations of particulate matter during the summer months are due to regional transport.[33] For 11 months, starting in October 1995, concurrent $PM_{2.5}$ measurements were collected every other day in South Boston and 3 other sites in eastern Massachusetts. There was high concordance between these 24-hour samples, with Pearson correlation between South Boston and downtown Boston (Beacon Hill, 3 km northwest) of 0.86, Lynn (16 km north) of 0.86, and Brockton (27 km south) of 0.81. On a larger scale, a high correlation (0.76) was found between daily concentrations of fine particles measured at sites 200 km apart in Washington and Philadelphia.[33] Data on the correlation between hourly concentrations of fine particles at different locations within a metropolitan area are not available.

Conclusions

Knowledge of the induction time between the exposure to particulate air pollution and adverse health effects is crucial to understanding the biological mechanisms responsible for these associations and to setting of standards that reduce the risk for the population. The present study suggests that elevated concentrations of fine particles may transiently increase the risk of MI for several hours as well as for several days after exposure. As a consequence, 24-hour averages might underestimate the association between air pollution and acute cardiovascular events.

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References

1. Bascom R, Bromberg PA, Costa DA, et al. Health effects of outdoor air pollution. *Am J Respir Crit Care Med.* 1996;153:3–50.
2. Katsouyanni K, Touloumi G, Spix C, et al. Short term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project. *BMJ.* 1997;314: 1658–1663.
3. Schwartz J. Particulate air pollution and daily mortality: a synthesis. *Public Health Rev.* 1991;19:39–60.
4. Pope CA III, Dockery DW. Epidemiology of particle effects. In: Holgate ST, Samet JM, Koren HS, et al, eds. *Air Pollution and Health.* San Diego, Calif: Academic Press; 1999:673–705.
5. Burnett RT, Dales R, Krewski D, et al. Associations between ambient particulate sulfate and admissions to Ontario hospitals for cardiac and respiratory diseases. *Am J Epidemiol.* 1995;142:15–22.
6. Schwartz J, Morris R. Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. *Am J Epidemiol.* 1995;142:23–35.
7. Schwartz J. Air pollution and hospital admissions for cardiovascular disease in Tucson. *Epidemiology.* 1997;8:371–377.
8. Poloniecki JD, Atkinson RW, Anderson HR. Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK. *Occup Environ Med.* 1997;54:535–540.
9. Schwartz J. Air pollution and hospital admissions for heart disease in eight US counties. *Epidemiology.* 1999;10:17–22.
10. Seaton A, MacNee W, Donaldson K, et al. Particulate air pollution and acute health effects. *Lancet.* 1995;345:176–178.
11. Peters A, Döring A, Wichmann HE, et al. Increased plasma viscosity during the 1985 air pollution episode: a link to mortality? *Lancet* 1997; 349:1582–1587.
12. Peters A, Fröhlich M, Döring A, et al. Particulate air pollution is associated with an acute phase response in men. *Eur Heart J.* In press.
13. Pope CA III, Dockery DW, Kanner RE, et al. Oxygen saturation, pulse rate, and particulate air pollution. *Am J Respir Crit Care Med.* 1999;159: 365–372.
14. Peters A, Perz S, Döring A, et al. Increases in heart rate during an air pollution episode. *Am J Epidemiol.* 1999;150:1094–1098.
15. Liao D, Creason J, Shy CM, et al. Daily variation of particulate air pollution and poor cardiac autonomic control in the elderly. *Environ Health Perspect.* 1999;107:521–525.
16. Pope CA III, Verrier RL, Lovett EG, et al. Heart rate variability associated with particulate air pollution. *Am Heart J.* 1999;138:890–899.
17. Gold DR, Litonjua A, Schwartz J, et al. The relationship between particulate pollution and heart rate variability. *Circulation.* 2000;101: 1267–1273.
18. Peters A, Verrier RL, Schwartz J, et al. Air pollution and incidences of cardiac arrhythmia. *Epidemiology.* 2000;11:11–17.
19. EPA. *National Ambient Air Quality Standards for Particulate Matter.* Washington DC: Environmental Protection Agency; 1997.
20. Maclure M. The case-crossover design: a method for studying transient effects on the risk of acute events. *Am J Epidemiol.* 1991;133:144–153.
21. Mittleman MA, Maclure M, Robins JM. Control sampling strategies for case-crossover studies: an assessment of relative efficiency. *Am J Epidemiol.* 1995;142:91–98.
22. Mittleman MA, Maclure M, Tofler GH, et al. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion. Determinants of Myocardial Infarction Onset Study Investigators. *N Engl J Med.* 1993;329:1677–1683.
23. Mittleman MA, Maclure M, Sherwood JB, et al. Triggering of acute myocardial infarction onset by episodes of anger. Determinants of Myocardial Infarction Onset Study Investigators. *Circulation.* 1995;92: 1720–1725.
24. Mittleman MA, Mintzer D, Maclure M, et al. Triggering of myocardial infarction by cocaine. *Circulation.* 1999;99:2737–2741.
25. Navidi W. Bidirectional case-crossover designs for exposures with time trends. *Biometrics.* 1998;54:596–605.
26. Bateson TF, Schwartz J. Control for seasonal variation and time trend in case crossover studies of acute effects of environmental exposures. *Epidemiology.* 1999;10:539–544.
27. Crystal RG. Alveolar macrophages. In: Crystal RG, West JB, eds. *The Lung.* New York, NY: Raven Press, Ltd; 1991:527–535.
28. Finkelstein JN, Johnston CJ, Barrett T, et al. Particulate-cell interactions and pulmonary cytokine expression. *Environ Health Perspect.* 1997; 105(suppl 5):1179–1182.
29. Driscoll KE, Carter JM, Hassenbein DG, et al. Cytokines and particle-induced inflammatory cell recruitment. *Environ Health Perspect.* 1997; 105(suppl 5):1159–1164.
30. Godleski JJ, Verrier RL, Koutrakis P, et al. Mechanisms of morbidity und mortality from exposure to ambient air particles. *Res Rep Health Eff Inst.* 2000;91:5–88.
31. Muller JE, Abela GS, Nesto RW, et al. Triggers, acute risk factors and vulnerable plaques: the lexicon of a new frontier. *J Am Coll Cardiol.* 1994;23:809–813.
32. Willich SN, Lewis M, Lowel H, et al. Physical exertion as a trigger of acute myocardial infarction: Triggers and Mechanisms of Myocardial Infarction Study Group. *N Engl J Med.* 1993;329:1684–1690.
33. Spengler JD, Koutrakis P, Dockery DW, et al. Health effects of acid aerosols on North American children: air pollution exposures. *Environ Health Perspect.* 1996;104:492–499.