Identifying and managing adverse environmental health effects: 2. Outdoor air pollution

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Abstract

AIR POLLUTION CONTRIBUTES TO PREVENTABLE ILLNESS AND DEATH. Subgroups of patients who appear to be more sensitive to the effects of air pollution include young children, the elderly and people with existing chronic cardiac and respiratory disease such as chronic obstructive pulmonary disease and asthma. It is unclear whether air pollution contributes to the development of asthma, but it does trigger asthma episodes. Physicians are in a position to identify patients at particular risk of health effects from air pollution exposure and to suggest timely and appropriate actions that these patients can take to protect themselves. A simple tool that uses the CH^2OPD^2 mnemonic (Community, Home, Hobbies, Occupation, Personal habits, Diet and Drugs) can help physicians take patients' environmental exposure histories to assess those who may be at risk. As public health advocates, physicians contribute to the primary prevention of illness and death related to air pollution in the population. In this article we review the origins of air pollutants, the pathophysiology of health effects, the burden of illness and the clinical implications of smog exposure using the illustrative case of an adolescent patient with asthma.

Review

Synthèse

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This article has been peer reviewed.

CMAJ 2002;166(9):1161-7

Case

A 16-year-old girl and her mother visit their family physician in July because the daughter woke up at 6 am that morning with shortness of breath, a cough and tightness in her chest. The girl has a history of asthma and used salbutamol soon after the onset of symptoms, with some but not total relief. She reports having had no symptoms during the previous month. She had a few episodes of wheezing the previous summer, which resolved with the use of salbutamol, and a cough that persisted for 2 weeks after an upper respiratory tract infection in the winter. She has no history of allergies, hayfever or other medical problems. She is a nonsmoker and has no family history of allergies. Audible wheezing is detected on physical examination, but the girl does not appear to be in distress. Her vital signs are normal, as are the results of the ear-nose-throat and cardiovascular examinations. Respiratory examination reveals wheezing throughout chest, no focal findings and a centrally placed trachea. The girl's calves are soft and nontender, and there is no evidence of ankle edema. Her peak expiratory flow is 240 L/min (expected for height 400 L/min). Spirometry testing is unavailable. Fifteen minutes after 2 puffs of salbutamol her peak expiratory flow increases to 320 L/min. To identify possible exposures that may have contributed to the asthma episode, the physician quickly takes an environmental exposure history using the CH^2OPD^2 mnemonic — Community, Home, Hobbies, Occupation, Personal habits, Drugs and Diet (Table 1).[1]

Questions surrounding this case: What was the patient's exposure to outdoor air pollutants? How should the patient and family be counselled about dealing with these trigger factors? What are the possible inducers and triggers from indoor air pollution? How can the patient and family find out about the status of outdoor air quality in their community?

Origins of air pollutants

Air pollutants are derived from both natural and human activities. Conventionally measured pollutants include ground-level ozone, suspended particulate matter, carbon monoxide, nitrogen oxides, sulfur dioxide and reduced sulfur compounds such as hy-

drogen sulfide. Predominant sources of these pollutants are summarized in Table 2. Other airborne pollutants of concern from a health perspective include metals (e.g., lead and mercury) and hydrocarbons (e.g., benzene, formaldehyde and trichloroethylene). These contaminants are not discussed in this article, which deals with the pollutants listed above.

Ground-level, or tropospheric, ozone is distinguished from stratospheric ozone. The latter forms a protective layer around the earth. The former is a colourless gas formed when its precursors, nitrogen oxides and volatile organic compounds or hydrocarbons, interact in the atmosphere in the presence of heat and sunlight.[2,3] Stagnant air masses allow ozone and its precursors to accumulate, which results in rising ozone levels. Nitrogen oxides are produced from fossil fuel combustion by motor vehicles and by power plants and other industries. Volatile organic compounds are produced by fossil fuel combustion, petroleum refining, surface coating (e.g., painting) and use of solvents. Another major source of these volatile compounds is natural forests and vegetation.[4]

Table 1: Environmental exposure history of sample case using the CH^2OPD^2 mnemonic[1]

Community	The Air Quality Index was 60 the day before the patient presented, and a smog advisory had been in effect for 3 days.
Home	There were no recent moves or renovations. The basement is damp. A gas range is used for cooking. The family has had a cat for 5 years.
Hobbies	No hobbies are reported that use glues or solvents or that make sawdust in the house.
Occupation	The patient recently started working as a waitress. Both parents have office jobs.
Personal habits	The mother smokes. The patient was involved in a baseball tournament for 2 days before her presentation.
Diet	The patient has no known food allergies or intolerances. She avoids "junk" food except on Saturday nights when she is out with her friends.
Drugs	The patient takes no medications other than salbutamol as needed and denies any use of recreational drugs.

Table 2: Common air pollutants and their main sources related to human activity

Pollutants	Main source
Carbon monoxide	Vehicle emissions, combustion of other fuels (e.g., wood)
Ground-level ozone	Interaction of nitrogen oxides and volatile organic compounds in the presence of sunlight
Nitrogen oxides	Vehicle emissions, combustion of other fuels (e.g., by power-generating industries)
Sulfur dioxide	Combustion of coal (e.g., power plants, metal smelting) and other fuels (diesel)
Suspended particulate matter	Combustion processes in vehicles and industrial facilities, road dust
Total reduced sulfur compounds	Paper production, coke ovens, refineries

Particulate matter comprises very small particles of solid or liquid matter that vary in size, chemical composition and source and that remain suspended in air for long periods. Fine particles (those less than 10 μ in diameter [PM10]) are considered inhalable, and smaller particles (less than 2.5 μ in diameter [PM2.5]) are considered respirable. When inhaled, fine particles are capable of being deposited in, and could damage the airways of, the lower respiratory tract and the gas-exchanging portions of the lung[5,6] and therefore are of most interest in terms of health effects.[2] These fine particles tend to be generated from human activities, particularly the combustion of fossil fuels, especially diesel fuel. In southern Ontario sulfate particles constitute 30%–40% of the total mass of smaller particles.[6]

Smog is the name given to the chemical soup produced from photochemical reactions in the atmosphere. Because of the role that heat and sunlight play in its production, highest levels of smog are recorded on hot, sunny days. Summer smog is composed mainly of ground-level ozone and particulate matter. Because ozone is not produced at high levels in cold weather, winter smog is composed mainly of particulate matter and sulfur dioxide.[7] Smog and its precursors can be transported long distances through the atmosphere by winds. In the summer up to 50% of ground-level ozone in Ontario[4,8] and significant amounts in the southern regions of Atlantic Canada[3] originate from the United States. The most frequent episodes of summer smog in Canada occur in the Windsor–Quebec City corridor. Other problem areas are the southern Atlantic region, especially southern New Brunswick,[9] and the lower Fraser Valley in British Columbia. Topography plays an important role in the Fraser Valley, where ozone precursors produced in Vancouver are blown inland by the westerly sea breezes and trapped by the mountains.[10] Although smog is commonly seen as strictly an urban problem, and sometimes even more narrowly as a "downtown" problem, levels of ozone can be higher in rural areas and suburbs.[11] This is related to the chemical equilibrium of ground-level ozone formation: high levels of the ozone precursor nitrogen oxides, formed secondary to heavy vehicle traffic, can "scavenge" ozone from the air and result in reduced local levels.

The Air Quality Index was developed to better inform the public about air quality.[12] It is a composite measure of 6 common air pollutants for which there is evidence of adverse effects on health and the environment: carbon monoxide, ground-level ozone, nitrogen dioxide, sulfur dioxide, suspended particulate matter (sometimes measured as a coefficient of haze) and total reduced sulfur compounds.[12] In summer, poor air quality is usually related to a measured increase in ground-level ozone, which is usually associated with an increase in fine particles in the air.[4] The Air Quality Index is reported on a scale of 0 to 100 or more, the number corresponding to the level of the pollutant that is highest relative to target levels. The scale ranges from "very good" air quality (0–15 on the index) to "very poor" quality (100 or more). For example, the report might state that the air quality is poor (50) due to ozone, which means that the ozone level is at 50 on the index.

A process has been initiated to revise the Air Quality Index to reflect changes in our understanding of the health effects of air pollution, including the occurrence of effects at levels previously thought to be safe and the effects of multiple pollutants acting concurrently.[13] Since 1993 Environment Canada, in cooperation with provincial and municipal officials, has issued smog advisories and alerts during the summer months when average regional levels of ground-level ozone are forecast to exceed a specified amount (usually about 82 parts per billion).[15]

Pathophysiologic effects

With respect to human exposure to air pollution, the inhaled dose of pollutant is increased during outdoor activities related to work, play and excercise.[15] During morning and evening rush hours, peak levels of pollutants from vehicle exhaust coincide with peak numbers of people making their way to and from work and school, resulting in maximum population exposure.[11]

Inhaled ozone causes an inflammatory response, manifested by increased airway permeability and bronchial hyperreactivity, and has been linked to reduced measures of pulmonary function, increased cough and chest tightness in controlled clinical studies. There is a marked individual variability in sensitivity to ozone, which cannot be predicted.[2] There is some evidence that ground-level ozone might potentiate the effects of allergens in people with asthma.[16]

Results from controlled laboratory studies of the effects of suspended particulate matter differ in some respects from observations in epidemiological studies. This is thought to be due to the difficulty in reproducing the complex chemistry of ambient particulate matter in the laboratory.[5] The pathophysiological mechanisms by which particulate matter affects the lungs and heart are not fully understood and are likely to be varied since, unlike ozone, particulate matter is chemically heterogeneous.[2,5,6] However, recent evidence has identified a variety of plausible mechanisms for the effects of particulate matter.[17,18] Some of these proposed mechanisms could explain the association of both particulate and gaseous pollutants with cardiovascular health outcomes; they involve changes in autonomic control as reflected in changes in heart rate,[19–23] blood coagulability and viscosity,[24–26] blood pressure[27] and bone marrow response.[28]

In controlled clinical studies nitrogen dioxide and sulfure dioxide have been linked to reduced lung function in people with asthma, and carbon monoxide has been associated with reduced exercise tolerance, particularly in people with coronary artery disease, because of the formation of carboxyhemoglobin.[2]

Burden of illness

Despite a decline in the levels of some air pollutants, recent epidemiological studies continue to demonstrate a link between ambient air pollution and illness. This has been observed even at the relatively low ambient concentrations currently measured in Canada[13,29–49] and elsewhere,[50–55] which suggests that air pollution still poses a significant public health risk to Canadians.

In 1987 Bates and Sizto[46] published an analysis linking "acid haze," consisting of ozone and sulfate aerosols, with hospital admissions because of respiratory conditions. Burnett and colleagues[47,48] subsequently replicated and expanded upon these findings. They examined the rates of hospital admission because of acute respiratory and cardiac illnesses in 168 Ontario hospitals and found a significant relation between increased admission rates and elevated levels of both ozone and sulfate aerosols in the vicinities of the hospitals over the 3 days before admission.

Numerous studies have also linked acute exposure to air pollution with increased mortality.[50,51,54,55] According to recent estimates, about 8% of all nontraumatic deaths in 11 Canadian cities are attributable to the combined effects of carbon monoxide, nitrogen dioxide, ground-level ozone and sulfur dioxide,[13] which translates into about 5000 preventable premature deaths annually.

Although there is a large body of literature linking air pollution to acute adverse health effects, the impact of long-term exposure is not as well understood. Increased mortality[56,57] and increased incidence of chronic bronchitis[58] and lung cancer[59] have been observed among adults and decreased lung function,[40,45,53,60] increased frequency of bronchitis[61] and increased prevalence of asthma[62] have been found among children in relation to air pollution. However, there is insufficient evidence to support a causal relation between ambient air pollution and the development of asthma in children and adolescents.[63] Air pollution is thus most appropriately viewed as a trigger of asthma.[64] It is complex and difficult to separate out the adverse health effects attributable to exposure to the different pollutants in the urban air mix, and recent studies in Canada have shown differing results.[13,65] Similarly, increasing attention is being paid to the relative contributions of air pollution and aeroallergens, including pollens and fungal spores, to the occurrence of cardiorespiratory morbidity.[35,42,66]

The Ontario Smog Plan Steering Committee[6] has estimated that, in Ontario, the total annual effects of particulate matter (over 90% due to respirable particulates [PM2.5]) include 1725 premature deaths, from both respiratory and cardiac causes, 1087 hospital admissions, 48 000 visits to emergency departments, 567 000 asthma-symptom days and 8.35 million restricted-activity days. These estimates are derived from numerous epidemiological studies, which illustrated a relation between daily variations in the level of particulate matter and acute health effects.

In a Toronto study nitrogen dioxide was found to be responsible for almost 40% of premature deaths related to air pollution and 60% of hospital admissions because of cardiorespiratory problems, with substantial mortality and morbidity being related to both carbon monoxide and sulfur dioxide. These 3 pollutants are present all year round, with higher levels in winter than in summer.[65] As shown in the results of the assessment of sulfur content in gasoline,[67] rates of prema-

ture deaths and hospital admissions reflect the tip of the iceberg relative to the larger spectrum of adverse health effects of air pollution. In fact, in a study from France, over-the-counter drug consumption represented the largest portion of the costs of respiratory illness related to air pollution.[68]

A number of assessments of the potential benefits of improved air quality in Canada have been conducted. An analysis of the benefits of the new regulations pertaining to sulfur in gasoline determined that reducing the sulfur content to 150 parts per million (ppm) by 2002 and to 30 ppm by 2005 would prevent, over a 20-year period, 2100 deaths, 2400 hospital admissions, 6500 visits to emergency departments, 7200 new cases of chronic bronchitis, 89 000 cases of bronchitis in children, 1.5 million restricted-activity days, 3.1 million asthma-symptom days and 11 million instances of acute respiratory symptoms.[67]

Besides affecting human health, air pollution also damages forests, crops and buildings.[8] It is important to appreciate that industries, homes and motor vehicles also emit carbon dioxide, thus also playing a part in global warming,[69] which itself has potentially huge effects on health at a global level.[70]

Clinical management

Asthma is a disorder of the airways characterized by paroxysmal or persistent symptoms such as dyspnea, chest tightness, wheezing, sputum production and cough and is associated with variable airflow limitation and a variable degree of hyperresponsiveness of airways to a variety of stimuli.[71] In the sample case described at the beginning of the article, the patient's signs and symptoms indicate reversible airflow limitation. (Although we have chosen to focus on asthma in the sample case, similar principles would apply to the management of other cardiorespiratory conditions exacerbated by air pollution exposure.)

Initial management involves an assessment of severity and treatment response. In this discussion, however, we are less concerned with the acute management of asthma and instead focus on identifying the environmental factors affecting asthma and the implementation of appropriate control measures.[71] The most common factors affecting asthma include allergens, respiratory irritants and viral infections. Allergens and irritants are encountered both indoors and outdoors.[71]

The task of identifying a particular exposure that could be related to an exacerbation of asthma can be like looking for a needle in a haystack. An efficient approach involves searching for a change in the environment related to symptom onset or exacerbation rather than looking for a specific exposure. Since the timing of symptom onset or exacerbation is often linked to the source or setting of the exposure, it helps to organize the exposure history by the possible source or setting using the CH^2OPD^2 mnemonic — Community, Home, Hobbies, Occupation, Personal habits, Diet and Drugs (Table 1) — to direct general and more specific questions about possible exposures. [Information on where to find a detailed exposure history questionnaire appears at the end of the article.]

This approach can be elaborated by asking questions such as: Are the symptoms temporally related to being in the home, at work or outdoors? Was the onset of symptoms associated with any recent dietary or environmental changes such as a move, a new work location, smog advisories, renovations, or new hobbies, furniture, plants or pets? Are symptoms better on weekends? Are symptoms present when away from home or on vacation? Do they occur in other specific settings? Does anyone else at home or at work have these or other symptoms that started around the same time? Symptoms that worsen when a patient exercises outdoors, particularly during smog advisories, may signal outdoor air pollution as an exacerbating factor.

When outdoor air pollution is a factor, patients with asthma and their families should be educated about the association between smog and exacerbations of asthma. The Ontario Medical Association has published smog advisory health messages for physicians and patients, including special messages for children, people working and exercising outdoors and people sensitive to smog.[72] The messages emphasize the need to be aware of smog alerts and advisories issued in their area by the media and public health authorities and to reduce exposure. Information on how to obtain information on Air Quality Index readings and smog forecasts is also provided. Because outdoor physical activity increases the dose of the pollutant delivered to the respiratory tract, people should be advised to limit time spent outdoors during smog episodes, especially in the afternoon and evenings, when ozone levels tend to be highest.[2,3] The diurnal pattern of fine particulate matter is less pronounced, which makes it more difficult to know when to reschedule outdoor activities within a given day. Patients should monitor their peak expiratory flow during smog episodes and increase their medication according to an established action plan, if necessary; however, therapeutic regimens should not be adjusted without evidence of an adverse effect of air pollution on symptoms or lung function.[2] Finally, it might be appropriate to educate patients about how smog is formed and what they can do personally to reduce the problem. It must be emphasized, however, that actions taken during smog episodes will not produce immediate improvements in the environment. Also, patients should be advised not to walk or cycle, thereby increasing personal exposure, during smog episodes in an attempt to help reduce emissions of smog precursors.

Prevention of air pollution

In the long term, primary prevention includes a variety of individual actions to reduce air pollution,[68] such as reducing car use by using public transportation or car pooling; walking or bicycling when smog levels are not high; keeping cars well tuned, checking the emission control system, avoiding idling for long periods and turning off the

engine while waiting; buying a fuel-efficient vehicle, driving at moderate speeds and avoiding use of gasoline-powered vehicles and machines (e.g., motorbikes, motorboats and gas lawnmowers); considering energy efficiency at home; choosing alternatives to household cleaners and oil-based paints that emit volatile organic compounds; and disposing of toxic household waste appropriately.

Although it is important to provide information to motivate individual behaviour change, primary prevention also involves actions at a societal level.[73] These include fuel and motor vehicle emission standards, attention to urban planning and public transportation, and cooperative international action on transboundary air pollution. Recent examples include implementation of more stringent requirements to reduce the sulfur content in gasoline,[67] new Canada-wide standards for air quality[14,74] and the recently signed Ozone Annex to the Canada–United States Air Quality Agreement.[75]

The questions answered

What was the patient's exposure to outdoor air pollutants?

The patient in the sample case was involved in a baseball tournament for 2 days before symptom onset. She spent the time outdoors exposed to air pollutants and, by exercising and further increasing her respiratory rate, had increased her exposure. She was therefore exposed to high levels of ground-level ozone and fine particulate matter.

How should the patient and family be counselled about dealing with these trigger factors?

- Advise the patient to try to reduce exposure by limiting or avoiding outdoor activities during smog episodes and, if necessary, to stay indoors at these times.
- Remind the patient and her family that ground-level ozone is at its highest level during the afternoon and evenings and that outdoor activities should be limited during these times. Inform them that the diurnal pattern of fine particulate matter is less pronounced, and thus it will be more difficult to know when to reschedule outdoor activities in response.
- Instruct the patient to increase the monitoring of her peak expiratory flow during smog episodes and to adjust medications according to an established action plan.
- Advise the patient to consider changing her lifestyle habits to reduce the production of air pollutants.

What are the possible inducers and triggers from indoor air pollution?

The possible inducers in the case study are dust mites in the carpets, moulds in the damp basement and other moisture-damaged materials, and the cat.

The possible triggers are the tobacco smoke from the mother's cigarettes, and nitrogen oxides emitted from the gas range and other combustion appliances.

How can the patient and family find out about the status of outdoor air quality in their community?

They should be instructed to contact their provincial ministry of the environment or the local medical officer of health. Also, smog advisories and alerts are prominently announced in newspapers and on radio and television.

[A detailed exposure history questionnaire is available on the Ontario College of Family Physicians Web site (www.cfpc.ca/ocfp/index.html — click on "Exposure History Sheets in MS Word" in the scrolling menu located in the middle of the page). The different components (Community, Home and Hobbies, Occupation, Personal habits, Diet and Drugs) can be printed on coloured paper for easy identification in patient charts. The questionnaire may be given to a patient to complete at home and bring to the next appointment for review and interpretation.]

Competing interests: None declared.

Contributors: Dr. Abelsohn conceived of and drafted the article. Dr. Stieb was involved in the original design and contributed to the acquisition of data and the drafting of the article. Drs. Sanborn and Weir contributed to the original conception and design. All of the authors contributed to the revising of the manuscript and approved the final version.

References

1. Marshall L, Weir E, Abelsohn A, Sanborn MD. Identifying and managing adverse environmental health effects: 1. Taking an exposure history. *CMAJ* 2002;166(8):1049-55. Available: www.cmaj.ca/cgi/content/full/166/8/1049
2. Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. Health effects of outdoor air pollution [review]. *Am J Respir Crit Care Med* 1996;153:3-50.
3. Federal–Provincial Working Group on Air Quality Objectives and Guidelines. *National ambient air quality objectives for ground-level ozone. Summary science assessment document*. Ottawa: Health Canada and Environment Canada; 1999. Cat no En42-17/7-2-1999E. Available (pdf format): www.hc-sc.gc.ca/ehp/ehd/catalogue/bch_pubs/ozonesad/summary.pdf (accessed 2002 Mar 25).
4. *Towards a smog plan for Ontario*. Toronto: Ontario Ministry of Environment and Energy; 1996.
5. *National ambient air quality objectives for particulate matter: executive summary. Part 1: Science assessment document. A report by the CEPA/FPAC Working Group on Air Quality Objectives and Guidelines*. Ottawa: Health Canada and Environment Canada; 1998. Cat no H46-2/98-220. Available (pdf format): www.hc-sc.gc.ca/ehp/ehd/catalogue/bch_pubs/98ehd220.pdf (accessed 2002 Mar 25).
6. *A compendium of current knowledge on fine particulate matter in Ontario*. Toronto: Ontario Ministry of the Environment for the Ontario Smog Plan Steering Committee; 1999. Available: www.ene.gov.on.ca/envision/env_reg/er/documents/pa9e0008/list.htm (accessed 2002 Mar 25).
7. World Health Organization. *Regional office for Europe: acute effects of smog episodes*. Report on a WHO meeting; 1990. Copenhagen: The Organization; 1992. Report no 43.
8. *Air quality in Ontario: a concise report on the state of air quality in the province of Ontario, 1998*. Toronto: Ontario Ministry of the Environment; 2001.
9. *Smog*. Ottawa: Health Canada. Available: www.hc-sc.gc.ca/hecs-sesc/air_quality/smog.htm (accessed 2002 Apr 2).
10. *Air – air pollutants*. Vancouver: Greater Vancouver Regional District; 2001. Available: www.gvrd.bc.ca/services/air/pollution/pollution.html (last updated 2002 May 24; accessed 2002 Mar 25).
11. Campbell ME, Benson BA, Muir MA. Urban air quality and human health: a Toronto perspective. *Can J Public Health* 1995;86:351-7.
12. Outdoor air quality and human health. In: *Health and the environment. The health and environment handbook for health professionals*. Ottawa: Health Canada; 1998. Cat no H46-2/98-2111.
13. Burnett R, Cakmak S, Brook J. The effect of the urban ambient air pollution

mix on daily mortality rates in 11 Canadian cities. *Can J Public Health* 1998;89(3):152-6.
14. Stieb DM, Pengelly LD, Arron N, Taylor SM, Raizenne ME. Health effects of air pollution in Canada: expert panel findings for the Canadian Smog Advisory Program. *Can Respir J* 1995;2(3):155-60.
15. Reiser K. General principles of susceptibility. In: Brooks SM, Gochfield M, Herzstein J, Schenker MB, Jackson RJ, editors. *Environmental medicine*. St Louis: Mosby; 1995. p. 351-60.
16. Molfino NA, Wright SC, Katz I, Tarlo S, Silverman F, McClean PA. Effects of low concentrations of ozone on inhaled allergen responses in asthmatic subjects. *Lancet* 1991;338:199-203.
17. Seaton A, MacNee W, Donaldson K, Godden D. Particulate air pollution and acute health effects. *Lancet* 1995;345:176-8.
18. Vincent R, Bjarnason SG, Adamson IY, Hedgecock C, Kumarathasan P, Guenette J, et al. Acute pulmonary toxicity of urban particulate matter and ozone. *Am J Pathol* 1997;151:1563-70.
19. Pope CA III, Verrier RL, Lovett EG, Larson AC, Raizenne ME, Kanner RE, et al. Heart rate variability associated with particulate air. *Am Heart J* 1999;138:890-9.
20. Peters A, Perz S, Döring A, Stieber J, Koenig W, Wichmann HE. Increases in heart rate during an air pollution episode. *Am J Epidemiol* 1999;150:1094-8.
21. Liao D, Creason J, Shy C, Williams R, Watts R, Zweidinger R. Daily variation of particulate air pollution and poor cardiac autonomic control in the elderly. *Environ Health Perspect* 1999;107:521-5.
22. Pope CA III, Dockery DW, Kanner RE, Villegas GM, Schwartz J. Oxygen saturation, pulse rate, and particulate air pollution: a daily time-series panel study. *Am J Respir Crit Care Med* 1999;159:365-72.
23. Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B, et al. Ambient pollution and heart rate variability. *Circulation* 2000;101:1267-73.
24. Peters A, Döring A, Wichmann HE, Koenig W. Increased plasma viscosity during an air pollution episode: A link to mortality? *Lancet* 1997;349:1582-7.
25. Pekkanen J, Brunner EJ, Anderson HR, Tiittanen P, Atkinson RW. Daily concentrations of air pollution and plasma fibrinogen in London. *Occup Environ Med* 2000;57:818-22.
26. Seaton A, Soutar A, Crawford V, Elton R, McNerlan S, Cherrie J, et al. Particulate air pollution and the blood. *Thorax* 1999;54:1027-32.
27. Peters A, Stieber J, Döring A, Wichmann HE. Is systolic blood pressure associated with air pollution? [abstract]. *Epidemiology* 1999;10:S177.
28. Tan WC, Qiu D, Liam BL, Ng TP, Lee SH, van Eeden SF, et al. The human bone marrow response to acute air pollution caused by forest fires. *Am J Respir Crit Care Med* 2000;161:1213-7.
29. Burnett RT, Dales RE, Brook JR, Raizenne ME, Krewski D. Association between ambient carbon monoxide and hospitalizations for congestive heart failure in the elderly in ten Canadian cities. *Epidemiology* 1997;8:162-7.
30. Burnett RT, Cakmak S, Brook JR, Krewski D. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. *Environ Health Perspect* 1997;105:614-20.
31. Burnett RT, Brook JR, Yung WT, Dales RE, Krewski D. Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. *Environ Res* 1997;72:24-31.
32. Burnett RT, Brook JR, Cakmak S, Philips O, Raizenne ME, Stieb DM, et al. The association between ambient carbon monoxide levels and daily mortality in Toronto, Canada. *J Air Waste Manag Assoc* 1998;48:689-700.
33. Burnett R, Smith-Doiron M, Stieb D, Cakmak S, Brook J. Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. *Arch Environ Health* 1999;54:130-9.
34. Burnett RT, Smith-Doiron M, Stieb D, Raizenne ME, Brook JR, Dales RE, et al. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. *Am J Epidemiol* 2001;153(5):444-52.
35. Dales RE, Cakmak S, Burnett RT, Judek S, Coates F, Brook JR. Influence of ambient fungal spores on emergency visits for asthma to a regional children's hospital. *Am J Respir Crit Care Med* 2000;162(6):2087-90.
36. Delfino RJ, Coate BD, Zeiger RS, Seltzer JM, Street DH, Koutrakis P. Daily asthma severity in relation to personal ozone exposure and outdoor fungal spores. *Am J Respir Crit Care Med* 1996;154:633-41.
37. Delfino RJ, Murphy-Moulton AM, Burnett RT, Brook JR, Becklake MR. Effects of ozone and particulate air pollution on emergency room visits for respiratory illness in Montreal. *Am J Respir Crit Care Med* 1997;155:568-76.
38. Delfino RJ, Murphy-Moulton AM, Becklake MR. Emergency room visits for respiratory illnesses among the elderly in Montreal: association with low level ozone exposure. *Environ Res* 1998;76:67-77.
39. Jorgensen B, Lundbye-Christensen S, Song XK, Sun L. A longitudinal study of emergency room visits and air pollution for Prince George, British Columbia. *Stat Med* 1996;823-36.
40. Raizenne M, Neas LM, Damokosh AI, Dockery DW, Spengler JD, Koutrakis P, et al. Health effects of acid aerosols on North American children: Pulmonary function. *Environ Health Perspect* 1996;104:506-14.
41. Stieb DM, Burnett RT, Beveridge RC, Brook JR. Ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. *Environ Health Perspect* 1996;104:1354-60.
42. Stieb DM, Beveridge RC, Brook JR, Smith-Doiron M, Burnett RT, Dales RE. Air pollution, aeroallergens and cardiorespiratory emergency department visits in Saint John, Canada. *J Expo Anal Environ Epidemiol* 2000;10:461-77.
43. Thurston GD, Ito K, Hayes CG, Bates DV, Lippmann M. Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. *Environ Res* 1994; 65:271-90.
44. Brauer M, Blair J, Vedal S. Effect of ambient ozone exposure on lung function in farm workers. *Am J Respir Crit Care Med* 1996;154:981-7.
45. Stern BR, Raizenne ME, Burnett RT, Jones L, Kearney J, Franklin CA. Air pollution and childhood respiratory health: Exposure to sulfate and ozone in ten Canadian rural communities. *Environ Res* 1994;66:125-42.
46. Bates DV, Sizto R. Air pollution and hospital admissions in southern Ontario: the acid summer haze effect. *Environ Res* 1987;43:317-31
47. Burnett R, Dales R, Raizenne M, Krewski D, Summers P, Roberts G, et al. Effects of low ambient levels of ozone and sulphates on the frequency of respiratory admissions to Ontario hospitals. *Environ Res* 1994;65:172-94.
48. Burnett RT, Dales R, Krewski D, Vincent R, Dann T, Brook JR. Associations between ambient particulate sulfate and admissions to Ontario hospitals for cardiac and respiratory diseases. *Am J Epidemiol* 1995;142:15-22.
49. Vedal S, Petkau J, White R, Blair J. Acute effects of ambient inhalable particles in asthmatic and non asthmatic children. *Am J Respir Crit Care Med* 1998;157:1034-43.
50. Katsouyanni K, Touloumi G, Spix C, Schwartz J, Balducci F, Medina S, 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. Air Pollution and Health: a European Approach. *BMJ* 1997;314:1658-63.
51. Morgan GS, Corbett J, Wlodarczyk J, Lewis P. Air pollution and daily mortality in Sydney, Australia 1989 through 1993. *Am J Public Health* 1998;88:759-64.
52. Morgan GS, Corbett J, Wlodarczyk J, Lewis P. Air pollution and hospital admissions in Sydney, Australia 1990 through 1994. *Am J Public Health* 1998;88:1761-6.
53. Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E, et al. Association between air pollution and lung function growth in Southern California children. *Am J Respir Crit Care Med* 2000;162;4:1383-90.
54. Pope CA III, Dockery DW. Epidemiology of particle effects. In: Holgate ST, Koren HS, Samet JM, Maynard RL, editors. *Air pollution and health*. San Diego: Academic Press; 1999. p. 673-705.
55. Thurston GD, Ito K. Epidemiological studies of ozone exposure effects. In: Holgate ST, Koren HS, Samet JM, Maynard RL, editors. *Air pollution and health*. San Diego: Academic Press; 1999. p. 485-510.
56. Dockery DW, Pope A, Xu X, Spengler JD, Ware JH, Fay ME, et al. An association between air pollution and mortality in six U.S. cities. *N Engl J Med* 1993; 329:1753-9.
57. Pope CA, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, et al. Particulate air pollution as a predictor of mortality in a prospective study of US adults. *Am J Respir Crit Care Med* 1995;151:669-74.
58. Abbey DE, Petersen FF, Mills PK, Beeson WL. Long-term ambient concentrations of total suspended particulates, ozone, and sulfur dioxide and respiratory symptoms in a non-smoking population. *Arch Environ Health* 1993;48:33-46.
59. Beeson WL, Abbey DE, Knutsen SF. Long-term concentrations of ambient air pollutants and incident lung cancer in California adults: results from the AHSMOG Study. *Environ Health Perspect* 1998;106:813-23.
60. Bates DV. Ozone: a review of recent experimental, clinical and epidemiological evidence, with notes on causation. *Can Respir J* 1995;2:25-31.
61. Dockery DW, Cunningham J, Damokosh AI, Neas LM, Spengler JD, Koutrakis P, et al. Health effects of acid aerosols on North American children: respiratory symptoms. *Enivron Health Perspect* 1996;104:500-5.
62. Guo YL, Lin YC, Sung FC, Huang SL, Ko YC, Lai JS, et al. Climate, traffic-related air pollutants, and asthma prevalence in middle-school children in Taiwan. *Environ Health Perspect* 1999;107:1001-6.
63. Raizenne M, Dales R, Burnett R. Air pollution exposures and children's health. *Can J Public Health* 1998;89(Suppl 1):S43-8.
64. Dolovich J, Hargreave F. The asthma syndrome: inciters, inducers, and host characteristics. *Thorax* 1981;36:641-4.
65. Toronto Public Health. *Air pollution burden of illness in Toronto*. Toronto: City of Toronto; May 2000. Available (pdf format): www.city.toronto.on.ca /health/hphe/pdf/burden_of_illness_technical_5.pdf (accessed 2002 Mar 27).
66. Platts-Mills TAE. The role of allergens in allergic airway disease. *J Allergy Clin Immunol* 1998;101:S364-S6.
67. Sulphur in gasoline regulations. *Canada Gazette Part II* 1999;133:1469-510.
68. Zmirou D, Deloraine A, Balducci F, Boudet C, Dechenaux J. Health effects costs of particulate air pollution. *J Occup Environ Med* 1999;41:847-56.
69. Last J, Trouton K, Pengelly D. *Taking our breath away: the health effects of air pollution and climate change*. Vancouver: David Suzuki Foundation; 1998.
70. Haines A, McMichael AJ, Epstein PR. Environment and health: 2. Global climate change and health. *CMAJ* 2000;163(6):729-34. Available: www.cmaj.ca /cgi/content/full/163/6/729
71. Boulet LP, Becker A, Berube D, Beveridge R, Ernst P, on behalf of the Canadian Asthma Consensus Group. Summary of recommendations from the Canadian Asthma Consensus Report, 1999. *CMAJ* 1999;161(Suppl 11):S1-S2. Available: www.cma.ca/cmaj/vol-161/issue-11/asthma/summary.pdf
72. Abelsohn A, Gray J, Boadway T. OMA Smog advisory messages for physicians and patients. *Ont Med Rev* 1999;May. Available: www.oma.org/phealth /99smog.htm (accessed 2002 Mar 27).

73. Stieb DM, Paola J, Neuman K. Do smog advisories work? Results of an evaluation of the Canadian Smog Advisory Program. *Can J Public Health* 1996;87:166-9.
74. Canadian Council of Ministers of the Environment. *Canada-wide standards for particulate matter (PM) and ozone*. Winnipeg: The Council; 2000. Available (pdf format): www.ccme.ca/pdfs/backgrounders_060600/PMOzone_Standard_E.pdf (accessed 2002 Mar 26).
75. *Canada–United States Ozone Annex*. Ottawa: Environment Canada. Available: www.ec.gc.ca/air/ozone-annex_e.shtml (updated 2001 Aug 1; accessed 2002 Mar 26).

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Weir E. Identifying and managing adverse environmental health effects: a new series. *CMAJ* 2002;166(8):1041-3.

Marshall L, Weir E, Abelsohn A, Sanborn MD. Identifying and managing adverse environmental health effects: 1. Taking an exposure history. *CMAJ* 2002;166(8):1049-55.

Additional resources

- Agency for Toxic Substances and Disease Registry: www.atsdr.cdc.gov/atsdrhome.html
- British Columbia Ministry of Water, Land and Air Protection air quality site: wlapwww.gov.bc.ca/air/airquality
- Environment Canada air quality information: www.msc-smc.ec.gc.ca/aq_smog/index_e.cfm and www.ec.gc.ca/air/introduction_e.cfm
- Greater Vancouver Regional District Air Quality Index report: www.gvrd.bc.ca/services/air/emissions/AQIndex/aqindex.html
- Health Canada's Health and Air Quality Web site: www.hc-sc.gc.ca/hecs-sesc/air_quality/index.htm
- Lung Association: www.lung.ca/asthma, tel (toll free) 800 668-7682
- New Brunswick air quality information: www.msc-smc.ec.gc.ca/aq_smog/NB/NB_e.cfm
- Ontario College of Family Physicians, Environmental Health Committee: www.cfpc.ca/ocfp/commit.html
- Ontario Medical Association (OMA) ground-level ozone position paper: www.oma.org/phealth/ground.htm
- OMA smog advisory health messages for physicians and patients: www.oma.org/phealth/99smog.htm
- Ontario Ministry of the Environment air quality information: www.ene.gov.on.ca/air.htm
- Toronto smog information: www.city.toronto.on.ca/health/smog/index.htm
- United States Environmental Protection Agency: www.epa.gov/ebtpages/air.html
- World Health Organization air quality guidelines: www.who.int/peh/air/Airqualitygd.htm