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Environmental
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Alan Abelsohn

Marg Sanborn

May , 2000

Foreword

As physicians practicing medicine in our communities and concerned with our patients' welfare, we cannot ignore the continuing degradation of our environment. Those of us who practice in large urban areas have witnessed our patients suffering from the adverse effects of air pollution. Walkerton, on the other hand, provides rural practitioners with a stark example of the lethal consequences of water pollution. The recent loss of life there poignantly underlines the urgent need for action. As our patients' advocates, we must work toward reversing the current deterioration of the environment in Canada and the United States, as well as in the rest of the world.

The hard work of the authors and the International Joint Commission should inspire us in this effort. Their Environmental Health in Family Medicine curriculum material is an invaluable resource, one which will help family physicians to cultivate a more detailed knowledge of environmental issues. We anticipate that family physicians, working in concert with other groups, will ultimately respond by raising the profile of health issues stemming from environmental degradation. Amid rising public awareness, voters will persuade politicians to overcome their intrinsic resistance to change.

On behalf of physicians and the public, I would like to thank the authors for their commitment to this vital cause. We hope that this curriculum material will inform all who make use of it, inspiring them to commitment and action.

Dr. Walter Rosser
Professor and Chair, Faculty of Community Medicine, University of Toronto
Past President, Ontario College of Family Physicians

Environmental HEALTH in Family Medicine

Introduction

Environmental
HEALTH
in Family Medicine

Introduction

"Environmental Health in Family Medicine" is a set of modules based on clinical cases which can be used for self-learning, or for teaching residents or practicing physicians.

The six modules are designed to cover content areas important to family medicine and environmental health skills relevant to primary care. These content areas and skills were derived from a needs assessment of family physicians, and from a consensus process which included family physicians and experts in environmental and public health.

Each module covers one content area and emphasizes different environmental health skill sets. The modules are weighted toward the three skill sets considered core to family medicine: taking an exposure history, knowledge of resources and risk communication.

The following grid indicates the relative emphasis of each module on a 1-5 scale: for example, the lead module emphasizes skills of taking an exposure history, utilization of lab tests, toxicology and risk communication.

Skill Sets

	Exposure Hx.	Exp./Risk Assessment	Env. Health Epidemiology	Lab Utilization	Toxicology	Knowledge of Resources	Risk Communication
Modules							
Lead	*****	**	**	****	****	***	****
Outdoor Air Quality	**	***	**	**	**	****	
Indoor Air Quality	****	*	**	**	***	****	
Pesticides	***	***	*	***	****	**	****
Clusters of Disease (Water Quality)	***	**	*****	**	*	****	**
Persistent Organic Pollutants (PCB'S)	****	***	**	****	**	*****	

Introduction(cont.)

Each module has objectives, clinical cases which illustrate environmental health skills and at-risk groups, a discussion of the cases, background information for teachers, references including websites, and proposed exam questions covering core content of each module.

The cases cover all age groups, and focus on the relevant at-risk groups for a particular exposure.The following is a guide for selecting cases by age group or at-risk group:

Pediatric Cases : Every module has at least one pediatric case. Lead, indoor and outdoor air quality, pesticides and PCB's have particular emphasis on effects in children.

Adult Cases: Lead, indoor air, pesticides, clusters-water quality and PCB's have adult cases. Cases in the lead, pesticide and PCB modules have content about reproductive health effects.

Geriatric Cases: The pesticide module includes one geriatric case, and a discussion on environmental health effects in the elderly.

This curriculum can also be downloaded free from the following websites:

International Joint Commission : www.ijc.org/boards/health.html
Ontario College of Family Physicians: www.cfpc.ca/ocfp/

For more information please contact the authors:

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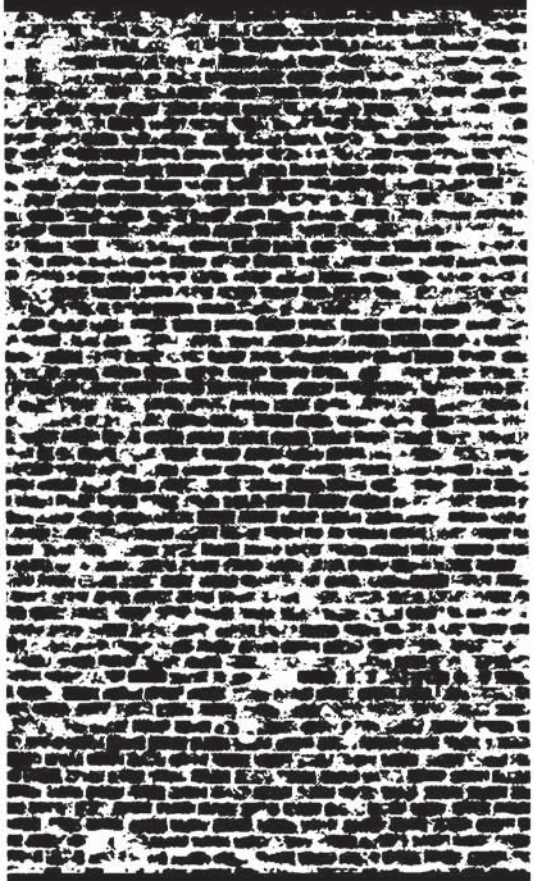
Environmental HEALTH in Family Medicine



Lead Module

Lead module

This module is one of six in the series Environmental Health in Family Medicine Curriculum Development Project

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The support of the Ontario College of Family Physicians and the Health Professionals Task Force of the International Joint Commission is gratefully acknowledged.

Objectives

1. To introduce and provide practice in a method for taking an environmental exposure history in the office.

2. To provide screening guidelines for identifying children in a practice who may be high risk and require blood lead levels.

3. To provide learners with cases which provide an opportunity to acquire skills in exposure assessment, lab utilization and interpretation of results, use of resources, and risk communication and management, for the area of lead poisoning.

4. To provide a selected resource list for reading and online information re: diagnosis and treatment of health effects of lead.

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Case # 1

A 5 year old boy with a nasal fracture

A 5 year old was brought to the office with a facial injury, which on x-ray revealed an undisplaced nasal fracture. His mother reported that he had stumbled while walking down the concrete steps outside the back door.

What are the relevant questions in the exposure history?

C - Community

H_2 - Home/ Hobbies

O - Occupation

P - Personal (Smoking, use of alternative medications)

Case # 1 (continued)
A 5 year old boy with a nasal fracture

CH2OP Exposure History

Community	- lives on farm - some pesticides used on fields surrounding house, no recent applications
Home / Hobbies	- 80 year-old farmhouse, presently stripping multiple layers of paint from kitchen area, using chemical and heat strippers - woodstove for heat - mother makes stained glass crafts as a hobby
Occupation	- mother babysits in own home - father drives truck and farms
Personal	- no risk factors for abuse identified - has 2 1/2 year old sister

The boy's physical exam is normal except for the nasal injury.

Questions

What are this 5 year old boy's exposure risks ?

Who else is at risk?

What lab test(s) would you order?

Case # 1 (continued)

A 5 year old boy with a nasal fracture

This family was exposed to lead through removal of multiple layers of leaded paint from the large kitchen, where they ate, and spent a great deal of time.

Lab Results : Blood Lead umol/L(ug/dL)

	First test	4-month follow-up
5 y.o. boy	.87 umol/L (18 ug/dL)*	.33 (6.8 ug/dL)
2 1/2 y.o. girl	.98 umol/L (20 ug/dL)	.52 (10.8 ug/dL)
Mother	.77 umol/L (16 ug/dL)	.35 (7.2 ug/dL)
Father	.31 umol/L (6.4 ug/dL)	no F/U

*Conversion factor ug/dL=$\frac{\text{umol/L}}{0.0483}$

Questions

How would you assess the health risk of each family member as a result of lead exposure?
5 y.o. boy
2 1/2 y.o. girl
Mother
Father

Are any other lab tests indicated?

If you received the first set of results, and felt unsure of their significance, where could you get advice?

At the 4-month follow-up, the mother asks you about planning another pregnancy. Are her present lead levels a risk for reproductive health?

Case # 1 (continued)

A 5 year old boy with a nasal fracture

Background

- Both children and their mother had lead levels which may be associated with adverse health effects. Testing the children for anemia would be appropriate. The two year old girl had an MCV of 80, and intermittent abdominal pain. The children in the home daycare were also at risk.

- Lead levels under 0.75 umol/L are recommended for women planning a pregnancy. There is also some evidence that high paternal lead levels increase the risk of spontaneous abortion.

- The first signs of lead poisoning in children are subtle neurobehavioral changes that affect social interaction. Impaired hearing, reduced learning capacity, hematologic effects, depressed Vitamin D metabolism, and possible hypertensive effects can all occur at the levels found in this family. Abdominal colic, encephalopathy, and renal damage occur at higher levels.

- A Canadian study in 1990 showed that in the general population, 5% of children had lead levels exceeding 0.5 umol / L, the current threshold level for known adverse health effects in children.

Other exposure sources:

There are other potential sources of lead exposure in this case:

- lead solder from old pipes contaminating drinking water
- mother's stained glass hobby, using lead solder
- leaded paint peeling from other areas of the house
- lead in the soil in outdoor play areas near the house or barn

Seasonal effects:

- Children's blood lead levels tend to increase during hot weather. A recent study showed that these increases were attributable to increased house dust from open windows, and increased exposure to lead in soil from outdoor summertime play (8).

- This suggests that summer may be a good time to test lead levels in children.

Case # 1 (continued)
A 5 year old boy with a nasal fracture

Treatment
Treatment consisted of immediate removal of the children from the house while clean-up was done. All surfaces were scrubbed with TSP twice and then weekly, and the house was vacuumed thoroughly. Follow-up levels on the children two years later were still above average (.33, .52), suggesting continuing exposure, perhaps in the drinking water.

Chelation, which removes lead from the body, is an important treatment for more severe lead poisoning. The preferred chelation agent for children is succimer, an oral preparation. Children requiring chelation should be referred to a pediatrician knowledgable in the procedure.

Succimer chelation: 10mg/kg every 8 hours X 5 days, then 10 mg/kg every 12 hours X 14 days

Preventing Lead Exposure in Drinking Water

* Lead pipes, or copper pipes soldered prior to 1989 should be replaced.

* Old brass pipes, faucets and fittings often contain lead inside.

* Water should be run vigorously for 30-60 seconds in older homes before use as drinking water. This flushes the pipes to remove standing water, and should be done if the tap has been unused more than 5 hours.

Case # 2

A man concerned about lead poisoning

A 45 year old man comes to your office because he is concerned about possible lead poisoning in his family. He has discovered during recent city repairs to the water line on his street that the water supply line to his home is a lead pipe.

He brings to you the following results of water samples in his home which he requested from the Works Department:

Test Results- Water(single samples)		**Lead** (ug/dL)
23 Williams Ave. - Washroom	Running:	**19**
	Standing:	**48**
Drinking Water Objectives- Running		**Max. 10**

Case # 2 (continued)
A man concerned about lead poisoning

Questions:

1. Why are the running and standing values so different? Do you have confidence in water test results from a single sample? If not, what do you recommend to this man?

2. Is patient testing for lead levels warranted by these results? If you decided patient testing should be done, what test(s) would you order? Who would you test?

3. What immediate advice would enable this family to reduce their lead exposure?

4. What other questions will allow you to assess this family's lead exposure risk?:
   Using the CH2OP mnemonic for exposure history:

   Community (C)

   Home/ Hobbies (H2)

   Occupation (O)

   Personal Habits (P)

Case # 2 (continued)
A man concerned about lead poisoning

You receive the following results from the lab:

	Blood Lead	Urine Lead	Random Urine Lead
Reference Range:	<0.5 umol/L (<10 ug/dL)	<0.58umol/L	<26umol/mol
Patients' Results:			
11 year old son	.16umol/L (3.3 ug/dL)	.12umol/L	10umol/mol
43 year old mother	.19umol/L (3.9 ug/dL)	.17umol/L	19umol/mol
45 year old father	.20umol/L (4.1 ug/dL)	.24umol/L	52.2umol/mol

The couple books an urgent appointment to discuss the results. By now the family is quite anxious about the health effects of their lead exposure.

Case # 2 (continued)
A man concerned about lead poisoning

Questions: A man concerned about lead poisoning

1. How would you explain these results and their health consequences to this family?

2. If you received these results and were unsure about their significance, who could help?

3. Is any repeat testing, or other follow-up, necessary for this family?

Case # 2 (continued)
A man concerned about lead poisoning

Discussion
Routes of Exposure

The family in this case had legitimate concerns about lead exposure based on their water testing. First, a **repeat water sample** should have been taken to replicate the elevated level in standing water. Secondly, one must consider **other sources of lead exposure.**

This can be done using the **CH2OP** exposure history. Pertinent questions about lead sources in the **community** (eg. smelter or battery recycling) are asked. They lived in a **home** built in 1922, so exposure from interior leaded paint, through contaminated soil from chalking of exterior paint, and from pre-1989 plumbing with lead solder would all be possible routes of exposure. **Hobbies** using lead (eg. stained glass or home furniture refinishing), parental **occupational** lead exposure, and the use of home remedies containing lead, would complete the exposure history.

In this case, the immediate advice is to reduce lead exposure by using only cold water for drinking, and flushing the standing water in the pipes by running it for 30-60 seconds, or until it feels cold, before use for cooking, drinking, or mixing beverages. Advice on inexpensive home devices for removing lead from drinking water can be obtained from the local health unit.

Lab Utilization

Only **blood lead** is a reliable indicator of current lead exposure. A zinc protoporphyrin (ZPP) test may be helpful if the exposure could be more than three months previously. Urinry lead is not recommended. If the blood lead is > 0.5 umol/L, a CBC is indicated to check for microcytic RBC changes and anemia. The medical officer of health can provide guidance about ordering and interpretation of lab tests for lead.

There are currently no known health effects with blood lead levels under 0.5umol/L

Case # 2 (continued)
A man concerned about lead poisoning

Communicating Risks

The family in this case was misinformed, or misunderstood, their family doctor's interpretation of their lab results. The father mistakenly concluded that his lead levels were too high, and could cause health problems. He replaced the lead pipe to his house at a cost of $4,000 , and unsuccessfully went to court to ask the city to pay this cost.

Environmental health concerns of patients often come accompanied by a high level of anxiety about possible adverse health effects.

Our role as family physicians is to: 1.) **conduct appropriate testing**, and 2.) **communicate clearly** to the patient whether a risk to health does exist, may exist, or does not exist-as in this case.

Simple advice on prevention of lead exposure, or advice on home remediation for patients with elevated levels, can reduce both health risks and anxiety for patients in the practice.

Environmental
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Lead Module

Background for Teachers

Screening for Lead Exposure in Children

Screening Children: When to Order a Blood Lead Test

Universal screening with blood levels in controversial. However, with the prevalence of elevated levels in studies of Canadian children ranging from 4-8%, it is desirable to identify those children most likely at high risk. Because chronic low-level exposure causes neurodevelopmental effects including lower IQ (3, 4, 5), it is important to reduce lead exposure for all children as much as possible, and to find the10-20 children in a typical family practice who have lead levels above the current guidelines (0.5 umol/L). The use of these quick screening questions will help identify the high-risk children under age 3 in a practice (3).

Quick Screening Questionnaire

A yes answer to any one of these 4 questions suggests a high-risk child, and should prompt the physician to consider ordering a blood lead level. (3)

1. Within the last 6 months, has your child lived in a house or apartment built before 1950?

2. Are there recent or ongoing renovations in the house, or painted surfaces which are peeling or chipping?

3. Have you ever been told that your child or a sibling has lead poisoning

4. Have you seen your child eating paint chips?

Diagnosing Lead Poisoning

HIGH-RISK GROUPS

There are two main risk groups:

1. **children aged 9 months -3 years**
   - absorb lead 5-10 times more efficiently than adults (respiratory and GI)
   - hand-to-mouth behavior
2. **those living near environmental sources**
   - leaded paint (pre-1980 housing)
   - lead-contaminated soil
   - lead-contaminated dust in homes - from paint or soil
   - industrial source in neighbourhood

Other risk factors

3. Renovation of old homes (often a middle-class activity)
4. Pre-war housing with leaded plumbing pipes, or lead-based solder used until 1989
5. Low socioeconomic status more likely to be living in lead-contaminated environments:
   - close to lead-emitting industry (battery recycling, smelting)
   - old house with peeling paint
   - close to high traffic areas with years of deposits from leaded gasoline
6. Urban and rural risk higher than suburban (due to older housing stock)
7. Poor nutrition - calcium and iron deficiency enhance lead absorption
8. Secondary exposure (child exposed through parent's work-clothes)
9. Parental hobby using lead - soldering, stained glass, home electronics, refinishing old painted furniture
10. Use of folk remedies containing lead eg. azarcon, paylooah, coral, ghasard (see list Ref 7)
11. Some household items made offshore, such as children's toys and backpacks, may be contaminated with lead.

Diagnosing Lead Poisoning (continued)

Children with excess lead levels usually show no unique features on physical examination.

But: Children with attention deficit disorder or other neurobehavioral symptoms, developmental delay, persistent hand-to-mouth activity (PICA), unexplained seizures, neurologic symptoms, anemia or abdominal pain may be showing clinical signs of lead poisoning. Questions directed at assessing lead exposure are indicated with these signs or symptoms, and blood lead testing should be considered.

Laboratory Tests

The appropriate test for diagnosis is a blood lead level. Zinc protoporphyrin (ZPP) or erythrocyte protoporphyrin may be useful in the cases where exposure occurred more than three months before testing. However, ZPP has no significant correlation with the blood lead, and false positives can be obtained in children with iron deficiency anemia. Levels in urine or hair are presently not useful. Hair levels reflect total body burden, and may be used clinically in the future. Dentine lead level in shed deciduous teeth provides an excellent measurement of lead exposure for research, but is not in use clinically (9). Children with blood lead levels > 0.5 umol/L (10 ug/dL) may require more tests to look for anemia, iron deficiency, and impaired renal function.

Steps in Office Management of Elevated Blood Lead Levels

The optimal follow-up and treatment of elevated lead levels requires cooperation between an informed family physician, a public health provider, and the child's family. The following steps are a guideline.

Step 1: A thorough exposure history to determine sources of lead exposure should be taken by the family doctor, if not done prior to testing. This includes questions on:

1. Age of the house
2. Age of the paint, and ongoing or recent renovations
3. Age of the plumbing: pre-1989 suggests lead pipes and/or solder
4. Outdoor play areas which could contain contaminated soil
5. Parental hobby or occupation causing lead contamination at home

At this point, the family physician should consider testing other family members who may be similarly exposed.

Step 2: The family physician contacts the local public health department with the exposure information, which can be used by public health to recommend an appropriate home remediation program, and give advice about whether referral is necessary. Home remediation may include removal or covering of lead-based paints, cleaning and vacuuming the home repeatedly using a special fine vacuum filter, and taking steps to reduce lead in the drinking water. The family can contact the health unit for detailed information. The children should be removed from the home during cleanup to prevent further exposure. In one study of children poisoned by leaded paint, home remediation with no other treatment produced a 23% reduction in blood lead levels (6).

Step 3: Since home remediation is time-consuming, inconvenient, and sometimes expensive, the family doctor has a further role in increasing compliance by supporting the recommendations, and communicating to parents the health risks and consequences of lead poisoning.

Step 4: The family doctor discusses repeat blood tests and other follow-up plans with the family. This may be done with public health and pediatric advice.

Treatment and Prevention of Lead Poisoning: Practice Points

Removal of the child from further lead exposure is the main treatment for lead poisoning. Very few children require chelation therapy.

Leaded paint should be covered over with latex paint, or removed with chemical stripper using appropriate personal protection. Removal by heat stripping or sanding mobilizes the lead and causes greater exposure. The local health unit can offer advice on the safest way to deal with old lead paint during repairs or renovations.

To reduce lead in drinking water, use only the cold water for drinking, and run the water for 30-60 seconds, or until the water is as cold as it gets, for drinking and cooking water. This is especially important when preparing baby formula. The water should be flushed again if standing longer than 5 hours in the pipes. A Brita-type filter using reverse osmosis is also effective in removing lead from drinking water. The health unit will give advice about effective devices to reduce lead in drinking water.

Treatment and Prevention of Lead Poisoning: Practice Points (continued)

Lead poisoning is classified into 5 levels of severity (I-V) based on blood lead levels.

I. For any level > **0.5 umol/L**, an exposure history, and exposure reduction counselling and action is indicated. This may be done by a family physician with current knowledge on lead issues.

II. All children with levels >**.75 umol/L** should be referred to a pediatrician knowledgeable in treating lead poisoning, and the family physician should seek advice from the local public health department, or a community medicine specialist.

III, IV, V More information on treatment of the three levels > **1.0 umol/L** (severe lead poisoning), and on lead mobilization tests and chelation therapy, can be found in references 1, 2, and 6. These levels always require immediate action and referral.

Note: Any blood lead level over 0.5 umol/L requires a repeat level immediately, and in 3-6 months.

Levels > 2.1 umol/L require urgent management and possible hospitalization of the child.

Suggested Reading
Annotated References

1. Chao, J. and Kikano, G.E. Lead poisoning in children. American Family Physician 47:113-20, 1993.

An excellent and still current review from a family medicine perspective, which uses both the American (ug/dl) and the Canadian (umol/L) units of measuring blood lead. It includes guidelines for screening and management.

2. Health Canada-Great Lakes Health Effects Program. The Health and Environment Handbook for Health Professionals 1998: Contaminant Profiles: Lead, 7pp. The best single handbook on environmental health for office use. To request a copy, phone (613) 954-5995 or fax (613) 941-5366.

3. Rolnick, S.J. et al. A comparison of costs of universal vs targeted lead screening for young children. Env. Health Research 80: 84-91, 1999.

4. Needleman, H.L. et al The long-term effects of exposure to low doses of lead in childhood: An 11-year follow-up report. New England Journal of Medicine 322(2): 83-88, 1990.
This classic longitudinal study led to the 1991 lowering of acceptable blood lead levels in children, to <.5 umol/L. It demonstrated irreversible longterm neurodevelopmental effects of chronic low-level lead exposure, including a higher risk of having a reading disability (Odds Ratio=5.8), and of dropping out of high school (OR=7.4).

Suggested Reading
Annotated References (continued)

5. Silbergeld, E.K. Preventing Lead Poisoning in Children. Annu Rev Public Health 18:187-210, 1997.
This recent review has an excellent discussion of the sources of lead poisoning, and the relationship between lead levels and IQ. The cost-benefit analysis of various prevention strategies is relevant to both family and community medicine.

6. American Academy of Public Health Committee on Environmental Health. Screening for Elevated Blood Lead Levels Pediatrics 101(6): 1072-77, 1998.
This is the current American guide to screening as well as management of lead poisoning of children, with specific guidelines covering mild elevations through to cases requiring chelation therapy. All blood lead units are in ug/dL, necessitating conversion to the Canadian units umol/L by the following conversion factor:

Blood lead (ug/dL) X.0484=Blood lead (umol/L)

7. Why Barns Are Red: The Health Risks from Lead and their Prevention. 1995: 81 pp This comprehensive Canadian guide provides physicians with valuable clinical information including health effects of lead, a list of occupations which involve lead exposure, detailed advice about safe home renovations, and a list of folk medicines containing lead.
Resource manual available for $10.00 from:

Education and Research Division,
Toronto Public Health-North York Office
5100 Yonge St.
North York, Ont.
M2N 5V7
Phone: 416 395-7716 Fax: 416 395-7691

Suggested Reading
Annotated References (continued)

8. Yiin, L-H et al Summertime blues: Childhood lead exposure peaks in summer months. Env Health Perspectives 108(2): Feb. 2000.

9. Fergusson. David L et al Early dentine lead levels and educational outcomes at 18 years. Child Psychol Psychiat 38(4): 471-78, 1997
This longitudinal study of 1265 New Zealand children confirms many of Needleman's (Ref 4) findings on low-level lead exposure as a cause of longterm academic difficulties and underachievement extending at least to late adolescence.

Proposed Exam Questions

1. A mother brings her three-year-old boy to the office with concerns about lead poisoning, as a result of a TV program which stated that children living in old homes are at high risk. Which of the following statements about diagnosing and managing this case are true?

a) a brief screening history will provide a good risk assessment
b) findings on physical exam will be helpful in diagnosis in most cases
c) the single most helpful lab test is a blood lead level
d) office advice on home remediation for lead can have a significant effect in lowering children's' lead levels
e) lead poisoning in children is so rare that this mother can be reassured with no further assessment.

Answer: a, c, d

2. Which of the following statements about lead toxicity in adults are true?

a) there is no relationship between lead levels and pregnancy outcomes in women
b) heat stripping of leaded paint during home renovations should be avoided, as the vaporized lead is highly absorbed
c) adults absorb lead more efficiently than children, and are consequently at higher risk
d) ocupational exposure to lead should be considered in smelter and metal recycling workers
e) lead toxicity causes anemia by a combination of inhibiting heme synthesis, and increasing erythrocyte destruction

Answer: b, d, e

Environmental HEALTH in Family Medicine

Outdoor Air Module

Outdoor air module

This module is one of six in the series Environmental Health in Family Medicine Curriculum Development Project

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The support of the Ontario College of Family Physicians and the Health Professionals Task Force of the International Joint Commission is gratefully acknowledged.

Environmental
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Asthma and Air Pollution

Learning Objectives

1. To introduce and provide practice in a method for taking an environmental exposure history in the office.

2. To provide learners with cases which provide an opportunity to acquire skills in exposure assessment, risk communication and management, and use of resources related to the health effects of air pollution.

3. To provide a selected resource list for reading and online information re the health effects of air pollution.

Case # 3

Asthma and Air Pollution

S: A 16 year old girl is seen in the office in Toronto, in mid July. She is accompanied by her mother. c/o short of breath, cough, tightness in chest. This started when she woke up at 6am. She used Ventolin soon after the onset, with some relief, but is still symptomatic.

O: Some wheezing with respirations. No cyanosis, no anemia.
Temp. 37. RR 18. Pulse 88. BP 110/70
ENT: Normal
CVS: Normal. Resp: Wheeze throughout. Trachea central. No focal changes.
Adb: Normal
Calves. No tenderness. No ankle edema.
PEF: 240 (expected for height=400) Spirometry is not available. 15 minutes after 2 puffs of Ventolin, her PEF has increased to 320.

A: What is your diagnosis?
Is there any relevant differential diagnosis?

Case # 3 (continued)
Asthma and Air Pollution

Questions

Q1.What are the possible triggers for this episode of asthma? What else do you want to know about the medical or family history, or personal exposure history, to investigate this? Make sure you include environmental exposures that might be triggers, using the CH2OP mnemonic for exposure history,

Community,

Housing/Hobbies,

Occupation,

Personal.

Q2. What is the status of the outdoor air in your community?
How would you find this out?

Q3. What was her exposure to outdoor air pollutants?

Q4. How would you counsel the patient and family about how to deal with these trigger factors?

Q5. What where possible triggers from Indoor air pollution?

Case # 3 (continued)
Asthma and Air Pollution

Q1. What are the possible triggers for this episode of asthma? What else do you want to know in terms of history, or personal exposure history, to investigate this? Make sure you include environmental exposures that might be triggers, using the CH2OP mnemonic for exposure history,

Community,
Housing/Hobbies,
Occupation,
Personal.

1. History of present Illness.

Any preceding symptoms? Any URI?

Nil

Any associated symptoms? Specifically, any fever, cough, chest pain, swelling or pain in the legs/ calves?

Nil

2. Past Medical history.

including asthma, allergy, respiratory or cardiac problems?

She had a few episodes of wheezing last summer. They were treated with Ventolin puffer, and settled quickly, lasting a few days each. She was also successfully treated with Ventolin for a cough that persisted for 2 weeks after a URI in the winter. No allergies or hayfever. No other respiratory problems. No other medical history.

3. Medications ?

Nil, besides Ventolin described above.

4. Allergies?

Nil, not to any medications, and no history of hayfever/rhinorrhea.

5. Family history of asthma , allergy, hayfever?

Nil

6. Personal: smoker?

Nil

Case # 3 (continued)
Asthma and Air Pollution

7. What other triggers, especially environmental exposures?

Community. (C) The status of the outdoor air in the community
The AQI (Air Quality Index) yesterday had been 60, and a Smog Advisory had been in effect for the previous 3 days.

In Ontario from the Ministry of Environment (MOE) measures and reports on the status of air quality, which can be accessed by calling 1-800-387-7768. The Air Quality Index (AQI) is an index synthesizing the six most commonly measured air pollutants; Carbon Monoxide, Nitrogen Dioxide, Sulphur Dioxide, Suspended Particles, Total Reduced Sulphur, and Ozone, reported as Very Good, Good, Moderate, Poor, and Very Poor. In summer, a Poor AQI is usually related to an increase in Ozone, and this in turn is usually associated with an increase in fine particles in the air. An AQI of 60 is regarded as poor.

A Smog Advisory had been issued. This is issued when average regional levels of ozone are forecast to reach 80ppb with local peaks anticipated at 120ppb or greater.These levels of ozone are associated with health effects, and can exacerbate asthma, chronic respiratory disease, and cardiac conditions. On average about 5 such episodes, each a few days in duration, are likely to occur in the course of the summer. Elevated levels of fine particulates are usually found at the same time.
With the smog advisory, there had been warnings in the media as follows: During the episode individuals may experience eye irritation. Heavy outdoor exercise during the episode may cause respiratory symptoms. People with heart or lung disease (including asthma) may experience worsening of their condition. Outdoor activity should be reduced:
The Medical Officer of Health would also be a source of this information.

Housing (H1)
Have they moved to a new house? No
A new community? No
Any renovations? No
Any new furniture, or bedding? No
What is the state of the basement? Any moisture problems? Yes, it is a bit damp after heavy rains.

Case # 3 (continued)
Asthma and Air Pollution

How do they heat the house? Gas, with forced air.
How do they cook? Gas range.
Pets, or birds, in the home, or contact with other pets, and animals? Yes, they have had a cat for the past 5 years.

Hobbies.(H2)
Any hobbies that involve use of solvents? glues? No.

Occupation.(O)
Does she work? She has started working as a waitress in a restaurant.
What do her parents do? Both have office jobs.

Personal.(P)
Does she smoke? No.
Does anyone at home smoke? Yes, mom smokes 5 per day. Mostly smokes outdoors. Does not smoke in the car.
Activities? What outdoor activities has she done in the past few days?
She has been involved in a baseball tournament the previous two days.

Case # 3 (continued)
Asthma and Air Pollution

Q2. What is the status of the outdoor air in your community? How would you find this out?
see above under community

Q3.What was her exposure to outdoor air pollutants?
The AQI (Air Quality Index) yesterday had been 60, and a Smog Advisory had been in effect for the previous 3 days. A Smog Advisory indicates elevated Ozone levels, and this is almost always accompanied in Ontario by elevated levels of fine particulates (PM10 and PM2.5)
She has been involved in a baseball tournament the previous two days. Being outdoors increased her time of exposure, and physical activity , by increasing the respiratory rate and volume, increases exposure to pollutants in the air.

Q4. How would you counsel the patient and family about how to deal with these trigger factors?

The patient and family should be made aware of the **connection** between elevated levels of ozone and particulates ie smog, and exacerbations of asthma. It appears that smog is a trigger for her asthma. As such they should:

1. be aware of **smog advisories** in their area when they are issued via the media, and can also call the MOE Air Quality line.

2. attempt to reduce exposure to smog. They should understand that **outdoor activity** during smog advisories increases exposure, so that she should be careful with outdoor activities. She might need to stay indoors during smog advisories to reduce exposure if her asthma worsens significantly.

3. Increase monitoring of her Peak Flow during Smog Advisories, and follow her action plan by increasing her inhaled steroids and Ventolin if her Peak Flow drops.

4. It is also appropriate to give them information about the larger picture of how smog is formed, and what the individual can do **personally** and in an **advocacy role** to reduce the problem.

Case # 3 (continued)
Asthma and Air Pollution

Q5.What where possible triggers from Indoor air pollution?
These will not be discussed in detail here. See module on indoor air pollution for more detail. The factors relevant in this case are:

1.Environmental Tobacco Smoke, at home and at work (occupational exposure)

2. A damp basement, with possible exposure to mold or House Dust Mites.

3. A cat with allergens.

4. A gas range can emit Oxides of Nitrogen.

Asthma and Air Pollution

Discussion for residents

The initial issues to be addressed re the asthma are:
a) how severe is this clinically? and
b) how to treat medically? These two issues will not be addressed here. This module focuses instead on issues that are frequently not addressed after the initial medical treatment, namely the environmental factors that might affect asthma. The Canadian asthma consensus conference, (Canadian Respiratory Journal 1996;3(2):89-100) presents an asthma continuum to describe the recommended therapeutic approach. Environmental Control and education is stressed. "Improving the quality of the asthmatic's environment is of paramount importance for a safe and effective approach to management."

Environmental factors that affect asthma might be both indoor and outdoor

From the exposure history in this case, the issues relevant to asthma are:
Indoor: House dust mites (HDM), allergens from pets, mold in the damp basement, environmental tobacco smoke (ETS) at home, and at her workplace (occupational exposure), the oxides of nitrogen from the gas stove.
Outdoor: exposure to high levels of outdoor air pollution.

We will focus on the outdoor pollution. The indoor factors are dealt with in the module on indoor air.

First, we need to assess what was her exposure?

We know that she was involved in a baseball tournament. This means that she had spent the previous 2 days outdoors exposed to the air pollutants, and exercise, by increasing respiratory rate, further increases the exposure. So she was exposed to high levels of ground level ozone, and fine particulates. Both of these can trigger asthma episodes, such that the number of people presenting to family physicians and emergency rooms increases after smog episodes. Ozone causes inflammation of the airways. It is unclear how fine particulates, small particles less than 10 microns (PM_{10}), or 2.5 microns ($PM_{2.5}$) in size, exert their effect. These pollutants also increase reactivity to aeroallergens, and probably decrease resistance to respiratory infections.

Environmental
HEALTH
in Family Medicine

Outdoor Air

Background for Teachers

Asthma and Air Pollution

Discussion for Teachers

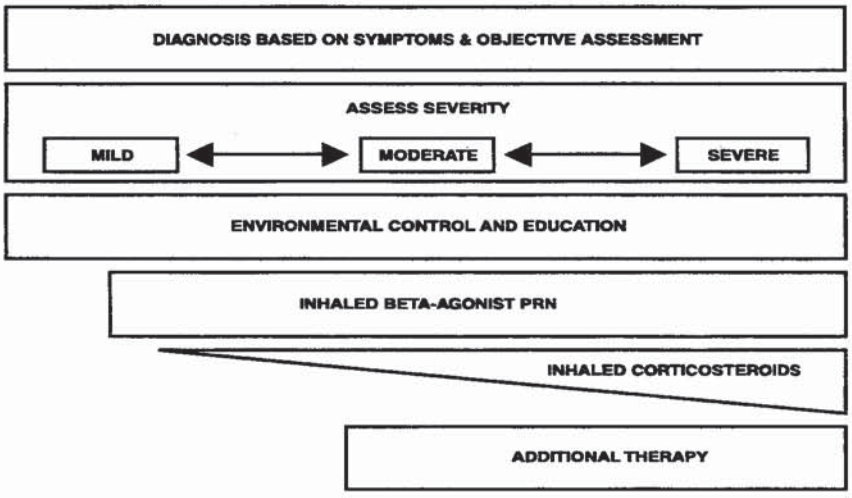
Asthma is a disorder of the airways characterized by paroxysmal or persistent symptoms (dyspnoea, chest tightness, wheeze or cough) with variable airflow limitation and airway hyperresponsiveness to a variety of stimuli.

This case presented demonstrates symptoms and objective evidence of reversible airflow limitation.

The first issues in dealing with this case, must include an assessment of a). How severe is this clinically? and b). How to treat medically?

However, in this discussion, we are less concerned with the management in terms of severity and drug treatment according to the concept of the asthma continuum/ good control, and more concerned with environmental factors affecting asthma and the appropriate control measures. The Canadian asthma consensus conference, (Canadian Respiratiory Journal 1996;3(2):89-100) presents an asthma continuum to describe the recommended therapeutic approach. Environmental Control and education is stressed. "Improving the quality of the asthmatic's environment is of paramount importance for the safe and effective approach to management."

In this module, the focus will be on outdoor air pollution. Please refer to the module on indoor air pollution, for more information and discussion on the indoor air issues of relevance to asthma.



Ernst P, et al. Canadian Asthma Consensus conference Summary of Recommendations. Can Respir J. 1996; 3(2): 89-100.

Asthma and Air Pollution

With respect to indoor air, we must consider allergies to house dust mite (HDM), pets, cockroaches, and molds and exposure to second hand smoke, household chemicals (cleaners, perfumes) and combustion appliances (gas stoves, portable heaters).
In the outdoors, asthma can be affected by pollen spores and other bio-aerosols, cold and air pollutants. These outdoor factors will be the focus of this case discussion.
However, it should be remembered that these factors can act synergistically. It is well known that ozone, a main component of smog, as discussed below, can sensitize the airways to the effects of aeroallergens and respiratory infections.

In the above case, besides outdoor air pollution, the patient is also potentially exposed to ETS, cat and dog dander, HDM, mold from the moist basement, oxides of nitrogen from the gas range, and other indoor exposures such as cleaners and perfumes.

This is an interesting case in terms of outdoor air pollution.

What had she been exposed to in the outdoor air that might trigger an asthmatic episode?
In terms of **exposure**, she had been playing in a baseball tournament the preceding 2 days. This means that she had been breathing outdoor air, compared to a normal day at school or work, where we spend most of our time indoors. Exercising increases the respiratory rate and respiratory volume, and thus increases exposure of the lungs to, and also the absorption through the lungs of, any pollutant in the air.
How do we find out **what pollutants are in the outdoor air**?
In Ontario, the Ministry of Environment and Energy (MOE) measures and reports on the six most common urban air pollutants: Carbon Monoxide, Nitrogen Dioxide, Sulphur Dioxide, Suspended Particles, Total Reduced Sulphur, and Ozone-through the Air Quality Index. This information can be obtained from the MOE, in Toronto 416-246-0411; Toll Free 1-800-387-7768 (English) or 1-800-221-8852 (French). Online www.ene.gov.on.ca. The AQI is reported as Very Good, Good, Moderate, Poor, and Very Poor. In summer, a Poor AQI is usually related to an increase in Ozone. Since 1993, Environment Canada and MOE have issued Smog Advisories when widespread elevated levels of ozone are forecast (average regional levels are forecast to reach 80ppb with local peaks anticipated at

120ppb or greater). On average, about 5 such episodes, each a few days in duration, are likely to occur in the course of the summer.

In Chicago, the EPA (Environmental Protection Agency) measures and reports the status of air quality. This can be accessed at 708-865-6320. The air Quality Index synthesizes the four most commonly measured air pollutants: Particulate Matter, Sulphur Dioxide, Carbon Monoxide, and Ozone. It is reported as Good, Moderate, Unhealthy, and Very Unhealthy. In summer, a poor AQI is usually related to elevated ozone, and this in turn is usually associated with an increase in fine particles in the air. An AQI of 100 or more is regarded as very unhealthy.

What are the pollutants of concern in the summer?

Although air quality in Canada has generally improved over the past 15 years, there is concern about the health effects of smog. Smog is the term given to the chemical "soup" that is the product of photochemical reactions. Because of this, highest levels are recorded on hot sunny days with clear blue skies and a light wind from the southwest; those beautiful summer days. It consists essentially of elevated concentrations of ground - level ozone and fine airborne particles (particulates). Together, these contaminants combine to give southern Ontario, or more precisely the Windsor-Quebec corridor, the worst chronic air quality problems in Canada, Other problem areas are the lower Fraser Valley and the South Atlantic region. This is a significant public health problem. It is estimated that 1800 premature deaths in Ontario occur annually because of air pollution.

Ozone

Ground level ozone, as opposed to stratospheric ozone (the ozone layer), is a colourless gas that is formed when its precursors, oxides of nitrogen and hydrocarbons, interact in the atmosphere in the presence of high temperatures and sunlight (photochemical reactions). Nitrogen oxide precursors (NOx) are emitted from the combustion of fossil fuels, mainly from motor vehicles exhausts, but also from power plants and industrial processes. The hydrocarbon precursors, also called volatile organic compounds or VOC's, are also produced by motor vehicle exhausts, industrial processes and by evaporation of gasoline from gas pumps, of surface coatings such as oil-based paints, and of solvents such as cleaners and barbecue starter fluid. Another major source of VOC's in Ontario is natural vegetation.

Asthma and Air Pollution

Particulates
These are fine particles, solid or liquid, which because of their small size, less than 10 microns (PM10) or less than 2.5 microns (PM 2.5), remain suspended in air, and when inhaled, penetrate deep into the airways, to the alveoli and bronchioles. Particulates vary in chemical composition, but a large fraction can be acidic, mostly sulphates from burning of fossil fuels. This acidic fraction, mostly made up of the finer PM 2.5 particles, called acid aerosols, is likely to be the most harmful to the bronchioles and alveoli. Particulates are produced by motor vehicle exhaust, especially diesel, coal burning power plants and wood smoke.

How do the components of smog, ie Ozone and Particulates, affect health?
When discussing the health effects of air pollution, it is important to keep in mind other environmental factors that impact negatively on respiratory health: smoking, second hand tobacco smoke, indoor air pollution at home, at school or at the office, and aero-allergens. In the case above, the following factors other than outdoor air pollution should be considered, and addressed with the patient and family:

1) tobacco smoke at home and in the workplace.
2) moist basement and likely presence of molds
3) possibility of pet, especially cat allergy
4) gas range, which give off oxides of nitrogen.
   These will be addressed in detail in the module on indoor air pollution.

Health Effects of Ozone
Levels of ozone and particulates measured in Ontario, especially during smog advisories, overlap with levels shown to have adverse effects on human health. Inhaled ozone causes an inflammatory response, manifested by increased airway permeability and bronchial hyperreactivity. Increased ozone exposure has been linked to reduced measures of pulmonary function, increased cough and chest tightness, exacerbations of asthma, and increased hospital admissions for respiratory diseases, including asthma. It is very likely that in this case, the asthmatic exacerbation was triggered by exposure while outdoors playing baseball to the elevated ozone levels. Recent studies have clearly linked ozone and particulates to increased mortality, and extrapolation of this data to Ontario has led the MOE to state that 1800 deaths are related to smog in Ontario each summer.
The evidence for chronic effects of ozone is unclear, although there is concern

Asthma and Air Pollution

that children living in more polluted areas have reduced pulmonary function indices compared to children in less polluted areas. There is a marked individual variability in sensitivity to ozone, which cannot be predicted.

Health Effects of Particulates

Elevations in particulate (PM10) levels of 100-150 ug/m3 have been associated with decreased PEF by up to 6%, a six-fold increase in medication use among asthmatics, 40% increase in school absenteeism, increased respiratory hospital admissions, and up to 16% increase in mortality, affecting both cardiac and respiratory deaths. The pathophysiological mechanisms of particulates are not understood.

It is likely that the effects of mixtures of pollutants are additive or synergistic, and probably also increase susceptibility to aeroallergens and infections.

Sensitive subgroups

There are sensitive subgroups; namely those with preexisting chronic cardiac and respiratory disease, including asthma, the very young, and the elderly. The inhaled dose of pollutant and thus harmful effects is increased during outdoor activity (school children, joggers, cyclists and outdoor workers, eg agriculture and construction). Children are more susceptible because they breathe more rapidly and inhale more pollutant per kg body weight than adults. They spend more time outdoors being physically active, often in the afternoon when ozone levels are highest, and they are less aware of the irritative symptoms that would inhibit adults.

How should the physician counsel this patient in terms of asthma and air pollution?

1. Awareness: The patient and family should be made aware of the connection between elevated levels of ozone and particulates ie smog, and exacerbations of asthma. They should be aware of smog advisories in their area when they are issued via the media, and that they can also call the MOE (Ontario Ministry of Environment) Air Quality line. In Toronto 416-246-0411; Toll Free 1-800-387-7768 (English) or 1-800-221-8852 (French). Online www.ene.gov.on.ca. In Chicago 708-865-6320. Patient information handouts are available (see Reference: OMA Smog Advisory health Messages for Phsicians and Patients).

Asthma and Air Pollution

2. Reduce Exposure: They should also understand that outdoor physical activity during smog advisories increases exposure, so that she should be careful with outdoor activities especially during the afternoon and early evening when ozone levels are at their highest, and stay indoors to reduce exposure if her asthma worsens significantly. Patients with asthma, chronic respiratory or cardiac disease, children and the elderly are more sensitive to the effects of smog, and all these groups should be counselled to reduce exposure in a similar way.
3. She should also be advised to be more careful in monitoring her peak flow during the smog advisory, and to increase her medication according to her action plan if necessary.
4. It might also be appropriate to give them information about the larger picture of how smog is formed, and what the individual can do personally and in an advocacy role to reduce the problem.
It must be emphasized that short term behaviour change (during smog episodes) will not produce immediate environmental improvement, and it is not wise to walk or cycle, thereby increasing personal exposure, in order to help reduce smog during smog episodes.

Primary Prevention
In the long term, primary prevention includes empowering patients to become part of the solution, by reducing emissions as follows:
*Reduce car use by using public transportation and car pooling whenever possible.
*Walk or ride your bicycle (wearing a helmet) when the smog levels are not high.
*Keep your car well tuned, and check the emission control system.
*Avoid idling for long periods, and turn off your engine while waiting.
*Driving at moderate speeds uses less fuel. Buy a fuel efficient vehicle.
*Use alternatives to other gasoline-powered vehicles and machines, such as motorbikes, motorboats and gas lawnmowers
*At home, think about Reducing, by considering energy efficiency.
*Solvents in household cleaners and in surface coatings like oil-based paints are a major source of VOC's. Choose alternatives such as water-based paints, and if it is necessary to use solvent-based products, handle and dispose with care.
Primary prevention also certainly involves advocacy and political action regarding the sulphur content of gasoline, motor vehicle emission standards, urban planning and public transportation issues, and federal action in terms of the regulation of transboundary transport of smog constituents from the USA.

Asthma and Air Pollution

Ecological Effects of Air Pollution - What can we do?

Air pollution, besides affecting human health, also damages forests, crops, and buildings. It is also important to appreciate that the emissions from industrial sources and cars that cause air pollution, especially CO2, also play a part in the global warming, which itself has potentially huge effects on health at a global level.

A recent Health Canada survey showed that Family Physicians were seen as the most trustworthy sources of environmental health information, but that our patients thought we were not doing a good job in this regard. We can inspire our students and future family physicians to be aware that they have a powerful voice in the primary prevention of these issues.

Case # 4
Asthma and Air Pollution

You live and work in a small rural community in South-Western Ontario. A mother comes into the office with her 2 children, a 10 year old daughter with no health problems, and a 7 year old son. He has asthma that has been difficult to control, but has recently settled down. He is on Flovent 125 ug one puff BID with a spacer, and Ventolin prn. They are well educated about asthma management, and compliant in following an action plan. They monitor his Peak Flow regularly, and she reports that he has done very well recently.
She is concerned however, because she heard on the radio that there has been a smog advisory issued for tomorrow. The kids have an athletics day at school tomorrow afternoon. They are both very good athletes, and very keen to compete.

Questions

1. What is Smog? What are it's components, and what are their health effects?

2.What is a smog advisory?

3.What is the health advice given with the smog advisory?

4.What should she do regarding her 2 children?

5.What should the school do regarding the sports event ?

Case # 4 (continued)
Asthma and Air Pollution

Discussion for Residents and Teachers
Q 1 and 2, re Smog, Smog advisories, and the related health advice were discussed in relation to case #3 above.

Q3. What should she do about her 2 children?
If they compete, the children will have a high exposure to ozone and the associated particulates, because a) they will be outdoors for a long period of time,and b) exercise, by increasing respiratory rate and respiratory volume, increases exposure. Both will be exposed with a high probability of being affected to some degree eg nose and throat irritation, and decrease in flow volume. Asthmatics are more sensitive to these effects, and he might experience a reduction in peak flow, and an exacerbation of his asthma, although individuals vary in their sensitivity to exposure. So it would certainly be prudent for her asthmatic son not to participate. Patient information handouts are available (see Reference: OMA Smog Advisory Health Messages for Physicians and Patients).

4. What should the school do regarding the sports event?
This is a difficult public health issue.
1) The prevalence of childhood asthma has increased. For example, a 1998 Health Canada survey reported the prevalence of current asthma among schoolchildren 5 to 9 years of age in the nine health units studied across the country to be 13%, ranging from 9.7% in Sherbrooke to 18% in PEI.
In the USA, annual health interview surveys indicate increases in asthma prevalence from 3.1% in 1980 to 5.1% in 1994. State specific prevalence rates range from 5.8% to 7.2%, but prevalence among impoverished inner city children has been much higher. Among inner city children 9-12 years of age in Detroit and San Diego, the combined prevalence of diagnosed and undiagnosed asthma has been 26% and 27% respectively (Sly R.M. Annals of Asthma and Immunology 82(3) Mar 1999). Persky et al (1998) reported a prevalence rate of 16% among children in low income neigbourhoods in Chicago. Thus a substantial number of children are at risk for potential exacerbation of their asthma.
2) Because ozone is made by a photochemical reaction, ozone levels increase during the day, with higher levels found in the afternoon and evening. Therefore these children exercising outdoors in the afternoon, will be exposed to the highest levels. Particulates tend to remain at a stable level throughout the day and night. A good argument could be made for postponing the event if possible, or for switching it to the morning, when the ozone levels as well as the temperatures and humidity, are likely to be lower.

Asthma and Air Pollution

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Bascom R et al. Health effects of outdoor air pollution. Am. J Respir Crit Care Med; 1996; 153:3-50 and 477-498. An extensive, in-depth review.

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Burnett R. et al. Effects of low ambient levels of ozone and sulphates on the frequency of respiratory admissions to Ontario hospitals. Environmental Research. 1994; 65: 172-194. An elegant epidemiological study.

Stieb DM, Pengelly LD et al. Health effects of air pollution in Canada: Expert panel findings for the Canadian Smog Advisory Program. Can Respir J 1995;2(3) 155-160. Notes on public advisories.

Thurston GD et al. Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario; consideration of the role of acid aerosols. Environ. Res 1994;65:271-290. Another epidemiological study in Ontario.

Health Canada September 1998. Childhood asthma in Sentinel Health Units. Report of the student Lung Health Survey Results 1995-96. Report on prevalence of asthma in Canada.

Ernst P, Fitzgerald JM, Spier S. Canadian Asthma Consensus Conference. Summary of Recommendations. Can Resp J. 1996; 3(2) 91-100.

Internet Sites

1. Cando Program. Ontario Lung Association.
www.web.net/cando/

2. ATSDR (Agency for Toxic Substances and Drug Registry)
www.atsdr.cdc.gov/atsdrhome.html

3. Great Lakes Health Effects Program, Health Canada.
www.hc-sc.gc.ca

Asthma and Air Pollution

4. Ontario College of Family Physicians, Environmental Health Committee
www.cfpc.ca/ocfp/commit.html

5. Ontario Medical Association
www.OMA.org/phealth/ground.html

6. www.oma.org
Interactive software "Illness Costs of Air Pollution in Ontario" (ICAP) which has data on the costs of air-pollution-related illness in different regions of Ontario.

Resources
Patient information handouts:
www.cfpc.ca/ocfp/commit.html or www.OMA.org/phealth
OMA Smog Advisory Health Messages for Physicians and Patients.

Proposed Exam Questions

1. With regard to smog advisories, which of the following statements are true?

a) Smog advisories in Ontario are issued when ozone levels are predicted to be above 200ppm.
b) Smog advisories are issued via TV and Radio.
c) Asthmatic patients should be advised to reduce exercise.
d) Elderly patients with COPD should be advised to stay indoors.
e) In Ontario, there are about 5 smog advisories issued per year, each a few days in duration.

answer: b,c,d,e.

2. In relation to the health effects of smog, which of the following are true?

a) Smog causes the onset of new cases of asthma.
b) Smog is composed mainly of ozone, particulates and carbon monoxide.
c) Particulates exacerbate asthma and lead to premature mortality.
d) Particulates lead to premature mortality by affecting both respiratory and cardiac patients.
e) Ozone causes inflammation of the airways.

answer: c,d,e.

Environmental HEALTH in Family Medicine

Indoor Air Module

Indoor air module

This module is one of six in the series Environmental Health in Family Medicine Curriculum Development Project

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Indoor Air Pollution

Learning Objectives

1. To introduce and provide practice in a method for taking an environmental exposure history in the office.

2. To provide learners with cases which provide an opportunity to acquire skills in exposure assessment, risk communication and management, and use of resources related to the health effects of indoor air pollution.

3. To provide a selected resource list for reading and online information about the health effects of indoor air pollution.

Indoor Air Pollution

This module deals with the health effects of indoor air pollution. It should be considered together with the module on outdoor air pollution.

Recent research indicates that Canadian children (< 12 years old) spend on average more than 70% of their time indoors at home, 10% indoors at school, and 8.5% outdoors. Adults spend even more time indoors, at home and in the workplace. It is therefore important that we consider the effects of indoor air pollution on health.

Many outdoor pollutants penetrate significantly into the indoor environment, eg fine particulates and carbon dioxide. Others, such as ozone, which is highly reactive, is found indoors at 30-60% of outdoor concentrations. Nitrogen dioxide penetrates indoors rapidly, and can be at higher concentrations indoors due to the added contribution from gas cooking ranges. These factors will not be discussed here. Please refer to the outdoor air pollution module.

Environmental tobacco smoke is also not addressed in this module, although the related health effects in the indoor environment are certainly relevant.

Environmental
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Case # 5

15 year old with frequent headaches

A 15 year old boy presents to your office with frequent headaches, which started 6 weeks ago in the late fall. Previously he "never" had headaches. He gets them 2 or 3 times a week, usually in the evenings, and often has them in the mornings as well. He describes it as a dull frontal headache. Associated symptoms include occasional dizziness, and sometimes nausea and difficulty concentrating. He often just feels tired. He had a headache last night and this morning.

Functional Inquiry
Unremarkable.

Past Medical History
Non contributory.
NKA

Medications
Acetaminophen and Advil to relieve the headaches.

Social
No smoking, no drugs, no alcohol use. Lives with his parents and 2 sisters.

Family History
Non contributory, beside mother has occasional migraine headaches.

Physical Examination
Gen well. ENT. Normal.
BP 120/80. CardioVascular: normal. Fundi normal.
Neurological Exam normal.
Musculoskeletal . Normal.

Case # 5 (continued)

15 year old with frequent headaches

Questions

1. What are the possible causes of these headaches, including indoor pollutants that can cause headaches?

2. What questions would you ask to sort out whether his symptoms might be related to indoor air pollution?

3. What other information would you want from investigations?

Case # 5 (continued)
15 year old with frequent headaches

Q1. What are the possible causes of these headaches, including indoor pollutants that can cause headaches?

This case study focuses on indoor air pollution. The major indoor air pollutant that can cause headaches is Carbon Monoxide. Environmental Tobacco Smoke (ETS) and Volatile Organic Compounds(VOCs) can also cause headaches, but irritant symptoms of the eyes and throat are likely to be more prominent.

Other conditions to be considered would include:

1. sleep apnoea, or Carbon Dioxide retention, although unusual in a teenager.
2. migraines
3. cluster headaches
4. paroxysmal nocturnal hemicrania. 1-4 are all more likely on waking.
5. tension headaches, chronic sinusitis, TMJ pain, or brain tumor causing increased intracarnial pressure would also be considered in the differential.

Q2. What questions would you ask to sort out whether his symptoms might be related to indoor air pollution?

It is important in environmental exposures to ask whether the timing of the symptoms relate to time spent in the home or work environment.

If the symptoms are more common at home, when did they start; were there any recent changes in the home such as renovations, new insulation, a move, new paint, furniture, plants or pets? Are symptoms related to hobbies, lighting a fire? Are they present when away from home, or on vacation? Do symptoms occur in other settings? Does anyone else at home have these symptoms?

No one else at home has headaches like these.

He moved down to the basement during the summer after he and his father insulated and drywalled the basement.

The house is heated by gas forced air furnace, and they have a fire in the fire-place a few times a week.

Q3. What other information would you want from investigations?

1. The family was advised that they should have the basement checked for Carbon Monoxide before spending another night there. They called the Gas Company, who sent someone to measure the level of Carbon Monoxide in the basement, and found it to be normal.

2. Venous blood was drawn for CarboxyHemoglobin level. The COHb level was 5 percent.

Case # 5 (continued)
15 year old with frequent headaches

Q4. How would you interpret this information, and how would you proceed?

There are two important findings.

1. COHB level of 5%.
   COHb in non-smokers ranges from 1-3%, while in smokers the level might reach 10-15%. A level of 5% in a non-smoker is definitely elevated, although headaches do not usually start until the level is over 16%. The half life of COHb is very short, about 3 hours, so it is very likely that his levels have fallen off substantially since he left the basement. A normal level would not definitively have ruled out CO poisoning as a cause for his headaches.

2. The CO level in the basement was normal.
   The important clue in this case is the timing of the headaches. They started in the fall, when the heating season starts, and the evening morning timing is also important. On further questioning, a relationship was established between the headaches and the evenings when the fireplace was lit. The mechanism is as follows: Lighting the fireplace, thus consuming oxygen, can cause a negative pressure in the house , which can draw exhaust gases from the furnace, including CO, into the house. The renovation that increased the insulation in the basement could also lead to decreased ventilation in the space, and increased concentration of the gas. A similar picture, perhaps more frequent and severe, could have resulted from a damaged chimney, vent or flue, improper installation or downdrafting of furnace gases because of excess exhaust, inadequate air supply or an airtight home.

Repeat testing with the fireplace lit showed a backdraft, and a CO level of 75ppm in the basement.

The treatment in this case is not medical, but furnace-related. The downdrafting must be remedied by increasing ventilation. A carbon monoxide detector should be installed in the basement.

At a follow up appointment, the patient reports that he has no more headaches, and is feeling generally much better.

Environmental
HEALTH
in Family Medicine

Indoor Air

Background for Teachers

Background for Teachers
Carbon monoxide poisoning

Carbon Monoxide

Sources
CO is a product of incomplete combustion of hydrocarbons.The sources of CO that cause poisoning include motor vehicle exhaust fumes, poorly functioning heating systems, and inhaled smoke. Propane operated forklifts have been implicated as a cause of headaches in warehouse workers. Poisonings may also be intentional.

Pathophysiology
CO is a colourless, odourless and nonirritant gas that is easily absorbed through the lungs. The affinity of hemoglobin for CO, as Carboxyhemoglobin(COHb), is 200 to 250 times as great as its affinity for Oxygen. CO competes with O2 for binding to hemoglobin, and shifts the oxygen-hemoglobin dissociation curve to the left, resulting in decreased carrying capacity of O2 to the tissues, and cell hypoxia. The fetus is even more sensitive to the harmful effects of maternal exposure to CO.

Clinical signs and symptoms
The clinical symptoms and signs of non-lethal CO exposure are nonspecific, and may mimic those of a nonspecific viral illness, and include headache, dizziness, weakness, nausea, difficulty concentrating, visual changes and shortness of breath. Because of this, a high index of suspicion is essential for making the diagnosis. This can be confirmed by measuring the exposure levels in the home, and by the COHb level in venous blood.
COHb in non-smokers ranges from 1-3%, while in smokers the level might reach 10-15%. Headaches do not usually start until the level is over 16%. The half life of COHb is very short, about 3 hours, so a normal level does not definitively rule out CO poisoning, while an elevated level is diagnostic.

Carbon monoxide poisoning (cont.)

Treatment

The patient must be removed from the exposure, and the source must be dealt with. In the home the gas or furnace company will identify the problem, or the fire department if there is an emergency. If the patient is in distress, oxygen must be given. Hyperbaric oxygen may be indicated in severe cases.

Prevention

Primary prevention is aimed at decreasing production of, and exposure to, CO, via building codes and occupational health and safety regulations. Fuel burning furnaces require regular maintenance and appropriate ventilation. Motor vehicles should not remain in enclosed spaces with the engine running, including attached garages, and outdoor gas grills must not be operated indoors. Secondary prevention includes the recently required use of CO detectors.

Case # 6

32 year old office worker with fatigue and sore throat

A 32 year old female data entry clerk, whom you have known for 10 years in your practice, complains of progressive generalized tiredness, a feeling of weakness, frequent sore throats and sore eyes, and difficulty concentrating. The problems have been present for about 3 months.

Functional Inquiry
Occasional headaches only.
Menstruation normal.
She denies depression. No mood changes. Sleep and appetite normal. She does not nap during the day, and naps have not relieved her tiredness. She has no significant muscle or joint pains.
No history of sensitivity to smells, perfumes, or exposure to chemicals.

Past Medical History
Nil significant.
No medication. No Known Allergies.

Social
Non-smoker. Lives alone in an apartment. Has worked at this job for 18 months. Previously was an actor. Exercises regularly at a fitness club after work. One glass of wine daily.

Family History
Nil significant. No asthma.

Physical Examination
Unremarkable. No anemia or nodes. ENT clear. Chest Clear. Abd. Normal; no liver or spleen. CNS. normal.

Investigations
All normal, including: CBC, LFT, TSH, creatinine, EBV, ANF, ESR, Toxoplasmosis, and urine.

Case # 6 (continued)

32 year old office worker with fatigue and sore throat

Questions

1. What further points in history are important.? Remember, this case is focused on indoor air.

2. Any further investigations?

Case # 6 (continued)
Sick Building Syndrome

Further History

1. Temporality of symptoms.
She feels better at home in the evenings, on weekends, and had no symptoms during the Christmas vacation.

2. Others at work sick?
She knows of two other workers whose desks are near hers, who have complained of feeling unwell at work.

3. Changes in the workplace environment?
About 3 months ago, her desk was moved from a small area to an open area. New carpet was laid at the time. She works in a big downtown office building, built about 20 years ago.

4. Job satisfaction?
She finds the job boring, but is content to stay on to earn money and benefits. She enjoys her co workers, and gets on well with her manager.

5. Is she depressed?
She denies feeling depressed, and has no associated symptoms.

Investigations.

Nothing else was ordered.

Background for Teachers

Background for Teachers
Sick Building Syndrome

Discussion
The symptoms (irritative and neuropsychiatric), temporal association with workplace and symptoms in colleagues point to a likely diagnosis of Sick Building Syndrome.

The examination and investigations are normal.
This is typical of Building Related Symptoms (BRS). BRS, more commonly referred to as Sick Building Syndrome (SBS), has become an increasingly common problem seen in Family Physician's office. This increase is due to changes in indoor environments in the last 25 years. Buildings have become more energy efficient and more airtight, reducing heating costs, but also reducing fresh air exchange.
SBS, or workplace discomfort, is a collection of non-specific symptoms in 2 main categories:

1. Irritation- mainly of the eyes, nose, throat and skin

2. Neuropsychiatric- Headaches, difficulty concentrating, fatigue and dizziness.

Other symptoms might be respiratory, including shortness of breath, cough and wheeze, and chemosensory changes, such as enhanced or abnormal odour perception. The physical exam and laboratory tests are normal. There is no adequate pathophysiological theory for its occurrence. The most significant diagnostic findings are:

1. a temporal association between the symptoms and the workplace eg a pattern of symptoms being worse at work, and improving after work, on weekends and vacation, and

2. clustering among other inhabitants or co-workers.

Sick Building Syndrome (cont.)

It is important to distinguish SBS from Building related illness, which are diseases with a well defined link to specific indoor environmental factors, including

1. Type 1 allergic diseases, such as Allergic rhinitis, conjunctivitis, and asthma

2. Immune mediated diseases such as humidifier fever and hypersensitivity pneumonitis from contaminated humidifiers.

3. Building related infections such as Legionnaire's disease (from cooling towers), Tuberculosis and respiratory viral infections.

Question:

How would you manage the problem? Think in terms of

1. Patient management

2. Building management.

Sick Building Syndrome (cont.)

Management

1. Patient management.
Personal risk factors. Work related stress is one of the most important determinants of SBS, and this must be discussed with the patient. SBS is more common in clerical staff than managers, more common in women, and in smokers, or in the presence of ETS (Environmental Tobacco Smoke). This patient does not report significant work stress. Nor does she report significant personal stress, or depression.
Smoking and allergies are frequently found in patients with respiratory symptoms and diseases. This is not the case here.
She should be advised on how to explore building management issues, or perhaps to request a move of her office. If she does not improve after building management measures, she would have to consider the options of changing jobs, or managing in her situation as well as she can.

2. Building Management.
Building Risk Factors include warm, dry air, sealed windows, overcrowding, open work stations, high intensity light, glare, the use of VDTs, dust, areas of moisture damage and the presence of chemical substances.

In this case, her work station has recently been moved into an open area.

The possible role of these factors should be discussed, and she should be supported to discuss changes in her workstation with her manager. It is important to intervene in this way before making exhaustive special medical investigations. Delay in dealing with complaints may magnify the problem, by making the employee feel powerless, frustrated and angry. It is important to follow up with the patient.

Sick Building Syndrome (cont.)

A new carpet had been laid.

New carpets and furnishings, particle board, glues, solvents and paints can emit Volatile Organic Compounds (VOCs), which can irritate mucous membranes. This effect usually dissipates with time. However, rarely patients may develop multiple chemical sensitivities, where they develop symptoms in other environments.

Inadequate ventilation may be a factor affecting the microenvironment of the work site. A physician's recommendations may facilitate ventilation inspection and improvements. Building and ventilation inspection can be carried out by maintenance staff, health and safety officials or private consultants, and a walk through by a skilled person is usually helpful in identifying possible interventions.

Case # 7

A six year old with poor asthma control

A 6 year old girl, well known to you, with a history of asthma presents to your office as a management problem. She had been on Flovent 125ug one puff BID with aerochamber, and occasional Ventolin, and well controlled over the last year. The family moved into a new home (built in 1960) 2 months ago. Since then, she has been having much more problems, with frequent symptoms of cough(non-productive), wheeze and shortness of breath. She has been to emerg twice in the last 2 weeks, where she was treated with a mask, and settled down. Today she feels well.

Functional Inquiry. Nil else of note.
PMH.nil, besides asthma.
Family History. Mother has hayfever.

Questions:

1. What factors might have led to a worsening of her asthma?

2. What factors in the new house might have led to a worsening of her asthma?

3. What questions would you ask to further investigate these factors in indoor air in the new home?

Case # 7 (continued)
A six year old with poor asthma control

Factors that might have led to a worsening of her asthma:

1. Compliance, including proper use of her medication and devices. You check her technique, and find no problems. The medication is not expired.

2. Infection. There is no history of change in sputum color, feeling unwell, or fever. There was no URI at the beginning of the period of worsening 2 months ago. No fever or chest signs on exam.

3. Is she exposed to ETS (Environmental Tobacco Smoke)? Has someone started smoking indoors?

Factors in the new house:

A. Indoor allergens

1. House Dust Mites. Found in soft furnishings, eg. mattresses, sofas and carpets, particularly in warm and humid rooms
2. Pets, furry or feathered.
3. Fungi and Molds. Their concentrations are a function of humidity/moisture, and high levels are found in damp basements, and in damp areas around garbage containers, food storage areas, wallpaper, shower curtains and window mouldings.
4. Cockroaches. Worse in inner city houses and apartments.

B. Gases

Formaldehyde and VOCs (Volatile Organic Compounds) are respiratory irritants produced by many substances in modern homes, including insulation, fabrics, carpets, solvents, floor adhesives, particle board, wood stain, paint, cleaning products, polishers and room deodorants and fresheners.

C. Particulates

1. Environmental Tobacco Smoke (ETS)
2. Fireplace or woodstove.

D. In general, if the house is better insulated, the concentrations of all these indoor pollutants can increase.

Case # 7 (continued)
A six year old with poor asthma control

In fact, the issues discovered after questioning the parents were as follows:

1. The previous owners of the house had kept 3 cats. Cat allergen (Fel d 1) consists of small particles, which can persist in carpet and house dust for a long time.

2. The house had wall to wall carpeting throughout. This is a problem because of house dust mite.

3. The basement was damp, and on inspection, areas of mold could be seen.

4. They had recarpeted the living room. It is possible that the carpet or adhesive used was gasing off formaldehyde or VOCs. This is unlikely to persist for as long as 3 months.

5. The tenant in the basement smoked. ETS is a significant factor in asthma exacerbation.

Questions:

1. How would you counsel the parents to avoid the effects of these indoor factors? Do you have handouts re avoidance measures?

2. Does she need allergy testing?

3. How should they manage her while they institute environmental controls?

Case # 7 (continued)
Asthma and Indoor Air Pollution

Counselling re Environmental Controls

The discussion below will describe this in detail. Handouts are advised. See references below, especially the American Academy of Asthma, Allergy and Immunology, and the Canada Mortgage and Housing Corporation (CMHC) materials. The parents can also contact The Lung Association in their region for more information.

Does she need allergy testing?

Allergy testing, by skin prick, would likely be very useful in guiding the avoidance measures. For example, it is difficult for families to get rid of a cat, and this should only be advised with evidence of cat allergy.

How should they manage her while they institute environmental controls?

Her inhaled steroids can be increased in the short term, or a long-acting bronchodilator added, until hopefully environmental controls have helped reduce her reactivity.

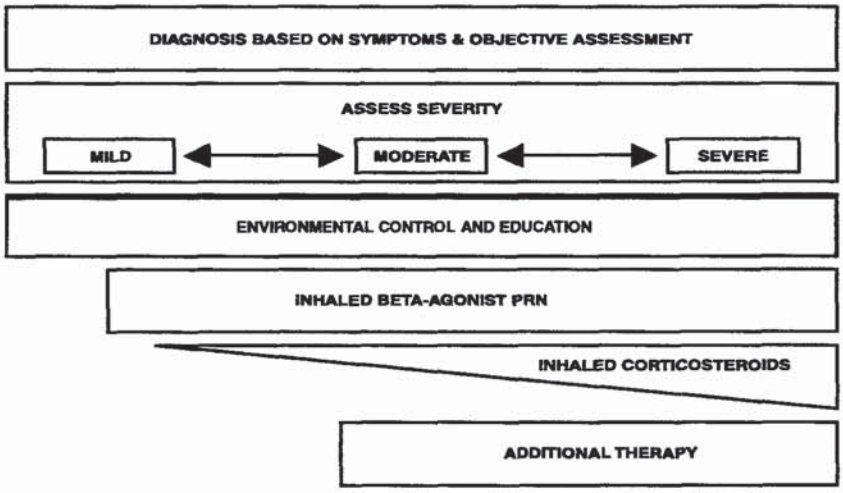
Background for Teachers

Background for Teachers
Asthma and Indoor Air Pollution

The prevalence of childhood asthma in Canada has risen sharply since the 1970s. A 1998 Stats Can report states that the percentage of children reported to have asthma in 1978/79 was 2.5%; the figure was 11.2%, or 672,000 children by 1994/95.

In the USA, annual health interview surveys indicate increases in asthma prevalence from 3.1% in 1980 to 5.4% in 1994. State specific prevalence rates range from 5.8% to 7.2%, but prevalence among impoverished inner city children has been much higher. Among inner city children 9-12 years of age in Detroit and San Diego, the combined prevalence of diagnosed and undiagnosed asthma has been 26% and 27% respectively (Sly R.M., 1999). Persky et al (1998) reported a prevalence rate of 16% among children in low income neigbourhoods in Chicago.

Although there is no clear evidence as to why this increase has occurred, there is some emerging evidence that indoor air quality is playing a major role in this trend. Below is a discussion of the indoor air pollutants of concern. The Canadian Asthma Consensus Conference of 1996 has recommended the asthma continuum for the ambulatory management of patients with asthma. This discussion will focus on the environmental control and education section.



Ernst P, et al. 1996. Canadian Asthma Consensus conference Summary of Recommendations. Can Respir J. 1996; 3(2): 89-100.

Asthma and Indoor Air Pollution (cont.)

Indoor air pollutants can be categorized as:
1. aero-allergens
2. indoor particulate matter
3. indoor gases.

It is estimated that Canadians spend more than 90% of their time indoors, both at home and at work or school. Over the last 25 years, there have been major changes in the indoor environment, both in homes, and office buildings, in part due to alterations in building design in response to the high costs of fuel during the 1970s oil crisis. Modern homes are better insulated, central heating and air conditioning units are common, wall to wall carpeting, and the use of synthetic building materials are more common. This has led to more comfortable living conditions, but has also resulted in warmer, more humid houses with poorer availability of fresh air, an environment where physical and chemical contaminants can build up, and biological agents flourish.

1. INDOOR ALLERGENS

The most important indoor allergens are dust-mite, cat and dog, cockroach and fungi.

Sensitization to one or more of these allergens contributes to asthma severity. The younger the patient, the greater the role of allergens in asthma.

House Dust mites are 8 legged organisms, relatives of spiders and ticks. They thrive in soft furnishings, eg. mattresses, sofas and carpets, particularly in warm and humid rooms. They are less common on the prairies. Their food source is human skin scales. The mites encase their droppings in a coating of intestinal enzymes, and it is a protein within these that is the principal allergen (Der p 1). The faecal particles are relatively large, and settle out of the air within 30 minutes. It is estimated that exposure to mite allergen may trigger attacks in up to 85% of asthmatics. There is also evidence that exposure to mite allergen particularly among infants may be an important factor in inducing the onset of asthma itself. A continuous dose-response relationship between the concentration of dust mites and the risk of developing childhood asthma has been reported.

Asthma and Indoor Air Pollution (cont.)

Domestic cats are an important source of allergen in the home. Cat allergen (Fel d 1)is found in saliva and skin. Because the particles that carry them are small (2.5 microns) they can be airborne for hours, and are widely distributed throughout the house, and can be carried on clothing into homes and schools. They can also be inhaled deep into the lungs, and can produce a rapid and severe asthmatic response. Exposure to allergens from dogs is less important generally, although can be significant for some asthmatics.

Allergy to cockroach is probably the most common cause of asthma in inner cities in North America. Roach allergens are found mostly in the whole body and faecal extracts.

Molds are the most common fungus in indoor environments, and their concentrations are a function of humidity/ moisture, and high levels are found in damp basements, and in damp areas around garbage containers, food storage areas, wallpaper, shower curtains and window mouldings. The most common health effects of molds are due to allergic mechanisms from inhalation of spores, causing asthma and allergy. Most common of this group are Alternaria, Cladosporium and Penicillium molds. A number of epidemiological studies have shown an increased risk for respiratory symptoms and airway reactivity associated with "home dampness", or signs of water damage and visible mold. Dampness is also a marker for House Dust Mites, and poor ventilation, as well as for molds. Besides allergic effects, some toxigenic molds produce mycotoxins and glucans, which can cause toxic effects. In a recent case in Halton region,a number of school portables which had been damaged by water, were found to be heavily populated with Stachybotris Chartarum (atra), a fungus that produces mycotoxins which have been associated with symptoms of malaise and mucosal irritation.

2. INDOOR PARTICLE MATTER

Small particles suspended in the air (respiratory particles less than 2.5 microns) can be inhaled deep in to the lungs and have been associated with an exacerbation of respiratory disease, including asthma. Contributors to indoor particulates are tobacco smoke, woodsmoke and outdoor particulates.

There is now overwhelming evidence that exposure to ETS(Environmental Tobacco Smoke) is associated with an increase in the severity of asthma in children, and that reducing ETS exposure is associated with improvement in asthma

symptoms and airway hyper-responsiveness. Recent studies have also shown that infants of smoking mothers have a significantly higher risk of developing asthma than those with nonsmoking mothers. It is likely that exposure to ETS acts as a co-factor in the induction of asthma in allergic people.

The use of open fires and woodstoves for cooking and heating can make a significant contribution to indoor particulate matter levels. This is a particularly important factor in terms of respiratory disease in developing countries.

Outdoor air pollution can also influence indoor air, especially respiratory particulates, less than 2.5 microns, which can penetrate indoors.

3. GASES

Gases of concern in indoor air are Nitrogen Dioxides, Formaldehyde and Volatile Organic Compounds (VOCs).

The major source of Nitrogen Dioxide is from gas cooking stoves and gas and kerosene space heaters. HIgher levels of NO2 are found during cooking in unvented kitchens. Exposure to NO2 may act as a trigger for asthma both directly and by increasing sensitivity to allergic responses.

Formaldehyde and VOCs (Volatile Organic Compounds) are produced by many substances in modern homes. Common sources include solvents, floor adhesives, particle board, wood stain, paint, cleaning products, polishers and room deodorants and fresheners. Both formaldehyde and VOCs are irritants. The irritating effects on the eyes and respiratory system may be one of the factors in sick building syndrome, and higher levels may cause asthma symptoms.

INDOOR ENVIRONMENTAL CONTROLS FOR ASTHMA PREVENTION

To date most of the asthma interventions have been designed to prevent asthma exacerbations (secondary prevention); few interventions to prevent the initial development of the condition (primary prevention) have been undertaken. The Canadian asthma consensus conference, (Canadian Respiratory Journal 1996;3(2):89-100) presents an asthma continuum to describe the recommended

Asthma and Indoor Air Pollution (cont.)

therapeutic approach. Environmental Control and education is stressed. "Improving the quality of the asthmatic's environment is of paramount importance for a safe and effective approach to management."

Indoor Allergen Avoidance

Given that exposure to allergens in early infancy may precipitate the onset of asthma,indoor allergen avoidance, together with ETS avoidance, is probably the most effective strategy for the primary prevention of asthma, as well as for secondary prevention.

Reducing the mite population is important but difficult. Controlled studies have shown a reduction in symptoms and improved airway reactivity following environmental cleanup.

a. Bedding. The single most effective strategy is to cover mattresses, duvets and pillows with plastic or vapour permeable fabric covers. Feather duvets and pillows, and cotton and wool blankets should be replaced with synthetic products. Bedding should be washed at least weekly in hot water, over 55C.

b. Flooring. Removing carpets and upholstered furnishings, especially in the bedroom, is advised. Chemical treatments are less effective. Vacuuming is recommended once a week. Vacuuming stirs up dust, and leads to a temporary increase in HDM allergen levels. It is more effective with high efficiency particulate air (HEPA) filters.

c. Humidity. Reducing humidity levels below 50%, and keeping temperature below 25C are important measures.

Pet allergen reduction is best achieved by removing the cat or dog. However, this is often not done. Keeping pets out of the bedroom, removing carpets, frequent vacuuming with HEPA filter, damp mopping, and regularly washing the pet reduces allergen levels.

Cockroaches are difficult to eradicate, although this is important. In apartments, the whole building needs to be treated. Be aware of, and minimize exposure,

Asthma and Indoor Air Pollution (cont.)

especially of children, to pesticides used for cockroach eradication.

Fungal growth is primarily dependant on humidity, and a source of organic matter. Dehumidifiers can be useful. Using chlorine bleach to clean mold-laden objects and surfaces, maintaining low humidity, sealing leaks and dealing with dampness in the basement, and regular washing of bathroom rugs are important interventions. Humidifiers, and HVAC (heating, ventilation and air-conditioning) systems must be regularly cleaned, as they can be a source of fungal growth.

INDOOR PARTICULATES AND GASES

The most important intervention is to stop smoking indoors. Parents must be clearly advised that children with asthma who have a parent who smokes have more frequent exacerbations and more severe symptoms.
Other important interventions include improving venting of furnaces, gas stoves and appliances, and by selecting building materials and indoor fittings such that emissions of pollutants such as formaldehyde and VOCs are as low as reasonably achievable.

Indoor Air Pollution

References

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6. CMHC.1993. The Clean Air Guide. How to identify and correct indoor air problems in your home. Copies for patients can be ordered from: CMHC,700 Montreal Road, Ottawa, ON K1A-0P7. Phone 1-800-668-2642 ($5.95).

7. Ernst P, Fitzgerald JM, Spier S. Canadian Asthma Consensus Conference . Summary of Recommendations. Can Resp J. 1996 ;3(2) 89-100.

8. Sly RM Changing prevalence of allergic rhinitis and asthma. Ann Allergy Athma Immunol 1999 Mar; 82(3); 233-48.

9. Persky VW et al. Relationships of race and socioeconomic status with prevalence, severity, and asthma in Chicago school children. Ann Allergy Asthma Immunol 1998; 81(3): 266-71.

Indoor Air Pollution

Internet Sites

1. Canada Mortgage and Housing Corporation (CMHC)
   www.cmhc-schl.gc.ca

2. Cando Program. Ontario Lung Association.
   www.web.net/cando/

3. American Academy of Asthma and Allergy
   http://www.aaaai.org/.
   For handouts, go to patient/public resource centre.

4. National Institute for Environmental Health Sciences (NIEHS)
   http://www.niehs.nih.gov/airborne/home.htm

5. Physicians for a Smoke Free Canada
   http://ventures.ca/smokefree/english/children.html

Video

CMHC. 1994. This Clean House. CMHC,700 Montreal Road, Ottawa, ON K1A-0P7 Useful instruction re practical steps to take to clean up the house.

Proposed Exam Questions

1. A teenager presents with headaches, and you suspect Carbon Monoxide poisoning. Which of the following statements about diagnosing and managing Carbon Monoxide poisoning are true?
   a) A level of Carboxyhemoglobin of 5% in a non-smoker is definitely elevated.
   b) In a smoker, the normal level of Carboxyhemoglobin can be as high as 15%.
   c) Carboxyhemoglobin persists in serum, and levels can be taken up to 24 hours after exposure.
   d) Carbon Monoxide shifts the Oxygen-hemoglobin curve to the right.
   e) Removal from the source of exposure, and Oxygen is the emergency treatment.

answer a,b,e.

2. Regarding asthma in children and indoor air pollution, which of the following are true.
   a) House dust mites, cats and cockroaches are all implicated as allergens in the exacerbation of childhood asthma.
   b) House dust mites feed on human skin scales, and live in soft furnishings eg bedding and carpets.
   c) Cat allergens are large particles, and settle out of the air to the ground quickly.
   d) Cockroach allergy is probably the most common cause of asthma in inner city children in North America.
   e) Reducing humidity levels below 50% is helpful in reducing house dust mites, as well as molds.

answer a,b,d, e.

Environmental HEALTH in Family Medicine

Pesticide Module

Pesticide module

This module is one of six in the series Environmental Health in Family Medicine Curriculum Development Project

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Objectives

1. To introduce and provide practice in the environmental exposure history through the use of clinical cases.

2. To provide diagnostic and treatment guidelines for the management of acute and chronic pesticide exposures.

3. To provide clinical cases and discussion material illustrating pesticide exposures in three groups with specific biologic vulnerability: children, pregnant women and the elderly.

4. To provide selected references relevant to the health effects of pesticide exposure in these three groups.

Contents

Environmental
HEALTH
in Family Medicine

Case # 8

A four year old with a rash, GI symptoms, and possible seizures

On a sunny, hot day in June, a four-year-old girl is brought to emergency with the following symptoms:

Rash on arms and face for 4 hours (started 1 hour after playing outside)
Vague abdominal discomfort and vomiting for 2-3 hours
Three brief (few seconds) episodes of shaking , staring and verbal unresponsiveness over past hour, with no LOC or incontinence.

Examination:
Afebrile, pulse 120, BP 80/60, resp rate 26
ENT normal.
Child is awake but not talking spontaneously. Neurologic exam otherwise normal.
Chest and abdominal exams normal.
Fine maculopapular rash on face, neck, arms, and very faint rash on lower legs.
After arriving in emergency, the child has a further episode of shaking and verbal unresponsiveness.

Questions
1. What is your working differential diagnosis for this presenting illness?

2. What are the important questions in the exposure history?
   Using the CH2OP mnemonic for exposure history:

Community (C)

Home / Hobbies (H2)

Occupational (O) (for children, work exposures brought into the home environment by parents)

Personal (P)

Case # 8 (continued)

A four year old with a rash, GI symptoms, and possible seizures

Community: Parents reported that the lawns on both sides had been sprayed with pesticides early that morning. The child played outside about three hours after the spraying. The rash started one hour later, and the other symptoms followed about 2-3 hours after playing outside.

Home / Hobbies: No access to drugs, alcohol, or other toxins in the house. No hobbies involving chemicals or solvents taking place in the house.

Occupation: Father is a computer programmer and mother works part-time as an accountant. No work-related exposures are evident.

Personal: She is receiving no medications or alternative medicines. Neither parent smokes.

Other history: The child has no previous history of seizures. There is no family history of seizures or metabolic disorders. The child is Caucasian.

Questions:

1. Does anything in the exposure history fit with this child's presenting illness?

2. What investigations would you order for this child?

3. Who would you consult for advice if needed?

Case # 8 (continued)

A four year old with a rash, GI symptoms, and possible seizures

Discussion

1. The child in this case had clinical findings in three systems which can be affected by pesticides: a rash, GI effects, and CNS effects. Symptom onset occurred after playing outside, which is suggestive of a pesticide exposure. Recent spraying of the two closest lawns was reported in the history.

2. Poison control incorrectly advised the treating emergency physician that there was no risk of the unknown pesticides producing such symptoms. No cholinesterase levels or other tests directed at finding a pesticide exposure were done.

The child had extensive other investigations including drug and alcohol levels, venous gases, CBC, electrolytes, urine, and a CT head scan, which were all normal.

3. Information on the specific pesticides sprayed is important in treating this child. In some municipalities, the lawn care company would be required to post a sign on the area sprayed naming the chemicals used. Otherwise, the parents should be asked to obtain this information from the lawn care company, specifically requesting Material Safety Data Sheets (MSDS Form) for all pesticides applied, while treatment of the child proceeds.

- Exposure to the herbicide 2,4-D may cause a dermatitis, as the acid causes skin irritation especially in combination with heat exposure. If the history suggests significant dermal exposure, a urine sample should be taken acutely and refrigerated for subsequent analysis of pesticide level.

- If further history revealed that insecticides had been sprayed adjacent to the child's play area, the appropriate diagnostic test for this child is a blood and serum cholinesterase. A depressed initial value is diagnostic; but the test is useful even if the initial value is normal. The RBC cholinesterase activity may take several days to reach its minimum, and 1-3 months to recover. Because serum cholinesterase has a wide normal range, follow-up testing in several weeks will often show a rebound increase of 25% or more, which is diagnostic of pesticide toxicity.

Case # 8 (continued)

A four year old with a rash, GI symptoms, and possible seizures

- If the child had symptoms suggestive of a cholinergic poisoning, eg. salivation, lacrimation , urination, diarrhea, or a productive cough, the probability of organophosphate poisoning is increased, and immediate atropine therapy is indicated. This poisoning could also show miosis on physical exam.

Case Outcome: With observation no fever and no further seizures occurred. The child was released from the ER without appropriate diagnostic tests or treatment for pesticide exposure.

There are several mnemonics for remembering clinical effects of organophosphate poisoning.

One is "MUDDLES"
- Miosis
- Urination
- Diarrhea
- Diaphoresis
- Lacrimation
- Excitation of central nervous system
- Salivation

Another is "SLUD"
- Salivation
- Lacrimation
- Urination
- Diarhea

Treatment of Acute Pesticide Poisoning

Health-care workers are at risk of significant exposure when treating pesticide poisonings. Rubber (not latex or vinyl) gloves should be worn when doing decontamination or handling body fluids.

1. Treatment must proceed based on exposure history and clinical findings, rather than awaiting confirmation by lab analysis.

2. Decontamination and resuscitation

- Airway protection, and decontamination measures including removal of clothing and vigorous repeated washing of skin, hair and nails with soap and water is indicated for exposure to all classes of pesticides. In general, gastric lavage, inducing emesis, and catharsis are not useful. Activated charcoal may be useful within 60 minutes of ingestion.

- If organophosphate or carbamate exposure is suspected, the cholinesterase inhibition should be treated. If cholinergic symptoms and signs are present, an organophosphate or N-methyl carbamate poisoning should be suspected and treated while confirmatory lab tests are in progress:

3. Atropine

This child may have benefited from treatment with atropine (0.05-0.1 mg/kg IV every 15 minutes) given after starting 100% oxygen, on admission. Atropine is especially helpful in reversing CNS effects such as coma, and the transient improvement in symptoms after atropine helps confirm that the clinical symptoms are related to poisoning with a cholinesterase-inhibiting insecticide. It can be given IM or via ET tube if necessary.

4. More severe insecticide poisonings, usually with organophosphates, may also require use of the specific antidote praladoxime chloride (PAM or 2-PAM), a selective antagonist which allows reactivation of cholinesterase by competing with the poison. Cholinesterase levels should be drawn prior to giving this drug. The dose for children under 12 years is 20-50 mg/kg IV, mixed in 100 cc of normal saline, infused slowly over at least 30 minutes. A repeat dose in 1-2 hours is usually required. Blood pressure monitoring is required as the drug may cause hypertension.

Treatment of Acute Pesticide Poisoning (continued)

5. Benzodiazepines should be used to control seizures:
Children: Diazepam 0.2-0.5 mg/kg IV every 5 minutes
Under age 5: max. dose 5 mg., over age 5 max. dose 10 mg.

OR Lorazepam 0.05-0.10 mg/kg IV over 2-5 minutes

Treatment Reference

Reigart, R and Roberts JR . Recognition and Management of Pesticide Poisonings, 5th Ed., Office of Pesticide Programs, U.S. Environmental Protection Agency, 1999.

As well as concise and current treatment information, this book has excellent information on health effects of the various classes of pesticides, which may be needed to match the exposure history with observed clinical signs.

Pesticides and Children

Vulnerability of Children

- Children are more vulnerable to the effects of pesticides. This is related to behavioral factors, such as hand-mouth behavior and play patterns , and to biologic factors such as the immature blood-brain barrier, large skin surface to body mass ratio, and increased sensitivity of CNS cholinergic receptors to pesticides.
- A recent study also suggests the fetus is vulnerable to pesticide effects as a cause of acute lymphoblastic leukemia (ALL). Children with specific genotypes in the detoxifying P-450 enzyme system have an increased incidence of ALL if the mother was exposed to indoor insecticides during the pregnancy (1).
- This biologic vulnerability of children is also reflected in increased rates for several other cancers in children aged 0-4 years exposed to pesticides (2,3,4).

- Farm children may be multiply exposed through playing on sprayed fields, spray drift, eating contaminated garden products, and through secondary exposure from contaminated spraying clothes brought into the home (5).

The clinical message is that in a whole family exposed to pesticides through lawn spraying or indoor fumigation of insect pests, young children may be the only family members with adverse health effects.

Pesticide Health Effects in Children- a Difficult Diagnosis

In one published series the diagnosis of pesticide poisoning in children was initially missed in 80% of cases at a major teaching hospital (6). In another series, a history of pesticide exposure was volunteered in only 3 of 25 confirmed cases of pesticide toxicity in children aged 3 months to seven years (7).

An analysis of all reported pesticide poisonings in the US showed that 56.9% of all cases involved children under 6, and only 7.6% were in the 6-17 age group. Exposures to insecticides and rodenticides accounted for most of the reports in children under 6 (66% and 28% of total). Unlike adult exposures, child exposures were almost always unintentional (8).

When clinical symptoms and signs are suggestive or the diagnosis is unclear, a specific history of pesticide exposure must be sought.

Prevention Messages

1. Cosmetic spraying of lawns and gardens may pose health risks, especially for young children , and should be avoided. While insecticides (eg. malathion) are most toxic, herbicides and fungicides also have subtle neurobehavioral and CNS effects(9), and non-specific effects such as fatigue, nausea, and diarrhea.

2. Young children receive some of their most severe pesticide exposures from indoor spraying of flea and tick pesticides (3,4). This can be avoided by the use of other flea control products, such as injections or pills, on house pets. Exposure to insecticides sprayed indoors should be avoided by pregnant women because of potential fetal effects(1).

   Removal of children from living quarters for an extended period may be necessary for safe fumigation of an infested building.

3. Insect repellants (active ingredient DEET) should not be used on children under age two. In older children, use on clothing or exposed skin- do not cover with clothing as this enhances systemic absorption.

4. Essential spraying in agricultural settings should be done with adequate protection for the applicator (respirator, overalls and gloves), and spray clothes should be laundered separately.
   Pesticides are readily absorbed through the skin, and should be washed off with soap or shampoo and warm water within 30 minutes to prevent absorption.

5. Agricultural spraying should be done with adequate warning to minimize incidental exposure of pregnant women and children.

6. Because of apparent paternal pesticide effects on pregnancy outcomes such as miscarriage and preterm delivery, it may be prudent to advise men with heavy seasonal pesticide exposures not to attempt to produce pregnancies during times of heavy exposure and for three months after (10).

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3. Daniels,JL Olshan AF Savitz DA Pesticides and childhood cancers. Env Health Perspectives 105 (10) 1069-77, 1997.

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8. Klein-Schwartz W and Smith GS Agricultural and horticultural chemical poisonings: Mortality and morbidity in the United States Ann Emerg Med 29(2) 232-38, 1997.

9. Guillette E et al An anthropological approach to the evaluation of preshool children exposed to pesticides in Mexico. Env. Health Perspect 106: 347-53, 1998.

10. Savitz, DA et al Male pesticide exposure and pregnancy outcome. Am J Epi 146(12):1025-36, 1997.

Environmental
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in Family Medicine

Case # 9

34 year old with second spontaneous abortion

A 34 y.o. woman had a spontaneous abortion at 17 wk. gestation. She required admission for a D&C.

Reproductive history was G5 P2 SA2 TA1.

She returned to her family doctor with her husband post D&C. The couple wanted to discuss what might have caused another miscarriage.

The fetal pathology report had been sent to the family doctor. The fetus had the following abnormalities: one stub for a leg, shortened umbilical cord , and no genitals.

What questions would be relevant in the exposure history? Using the CH2OP mnemonic for exposure history:

Community (C)

Home (H)

Hobbies (H)

Occupational (O)

Personal (P)

Case # 9 (continued)
34 year old with second spontaneous abortion

Exposure History
Community: -residential neighbourhood of 50 yr. old homes
-no factories or identified toxic waste problems

Home:
- the couple live in an apartment in a 40-year-old duplex
- both stripped some paint in kitchen during the winter

Hobbies:
- bingo, watching TV

Occupational:
34 year old woman:-seasonal worker in a seed retailing company which has a small seed packaging operation in the basement
-started work in March; got pregnant 1 month later; 40-hr. work week until early June, aborted in mid-July
- packaged treated seed, up to 500 kg. per season in unventilated basement area
-protection: latex gloves, paper surgical mask, street clothes
-ate lunch, smoked, and drank coffee near her work bench
-did not change or shower after work ; prepared dinner in her work clothes
husband: works as a postal worker delivering mail

Personal:
- smokes 1/2 PPD X 19 yr.
- couple has a 3 y.o. son and 7 y.o. daughter who are healthy

1. What are the potential exposures for this fetus?

Maternal
Paternal

2. How do you assess the risk of this woman's exposures in relation to the critical period of fetal development?
3. Who else might be at risk?
4. What other agencies may need to be involved in collecting information?

Case # 9 (continued)
34 year old with second spontaneous abortion

Further Information:

1.Potential Fetal Exposures

Maternal - Work exposure to five pesticides
-captan (fungicide)
-thiram (fungicide)
-methoxychlor (organochlorine insecticide)
-chloropyrifos (anticholinesterase insecticide)
-carboxin (Vitavax 34) (fungicide)
-protective equipment and hygeine practices were inadequate to prevent oral and dermal absorption
-no plasma cholinesterase monitoring was done although she worked with a cholinesterase-inhibiting insecticide.
- Lead from home renovations. No methyl chloride used to strip paint.
- Smoking

Paternal - Lead from renovations
- exposed to outdoor air pollution

2. The timing of this exposure to pesticides is high-risk, occurring during the embryonic period (3-8 weeks) and the early fetal period of organogenesis.

3. At-Risk Others - the couple's children, by secondary exposure
-other workers in her workplace
-pesticide applicators
-farmers and their children
-home gardeners and their families

4. Other Agencies - Occupational Medicine specialist 's consult letter:
"two [of the pesticides to which the worker was exposed] have teratogenic potential...captan which may cause a pattern of teratogenicity similar to the pattern in the affected fetus... [and] thiram, and it should be noted that a chemical compound closely related to thiram (disulfiram) is a suspected human teratogen"

34 year old with second spontaneous abortion

Further Information:

-WSIB : "has no policy on compensation of workers for adverse reproductive effects"
-Ministry of Labour should be notified when health effects are thought to be related to a workplace exposure
-Ministry of Environment (pesticide regulation)
-Farm Health and Safety Association

How would you answer this couple's question

" Can we try for another pregnancy right away?"

Discussion Points

Reproductive Hazards in the Environment

1. This case illustrates both a severe outcome of a multiple pesticide exposure in early pregnancy, and difficulties with the inadequacy of worker protection in this area.

Expert opinion suggested that this woman's fetus was affected by her occupational pesticide exposures. Her fetus had lethal anomalies leading to early fetal loss. However, she would be compensated only for the time off work with the miscarriage, and not for the fetal loss. A discussion with the patient should also include the information that fetal anomalies sometimes occur spontaneously.

2. Can we try for another pregnancy?

i) For prospective parents, it is recommended that blood lead levels are below 0.75 umol/ L at the time of conception. If you thought lead exposure was significant in this case due to the history of stripping leaded paint, maternal and paternal levels should be tested before another attempt to conceive.

ii) Results of testing for genetic abnormalities on the aborted fetus may also be helpful in answering this question, and should be requested.

3. Paraoccupational or secondary exposure is exposure of other family members to hazards in the workplace by means such as contaminated clothing or food. The children in this home may have had pesticide exposure from the mother's work clothes and skin. Farm children are exposed to currently and previously applied pesticides through home-grown food (8).

4. Adverse reproductive outcomes should prompt a search for environmental reproductive hazards, such as pesticides or lead. Other such outcomes are: impaired fertility, chromosomal abnormalities, prematurity, low birthweight, and neurobehavioural disorders or cancer in early childhood.

5. History-taking to find environmental causes of reproductive dysfunction must include maternal and paternal exposures. There are also some synergistic effects: eg. paternal lead exposure with maternal smoking increases the risk of spontaneous abortion in a synergistic way (1).

Discussion Points
(continued)

6. Establishing links between environmental risks and adverse reproductive outcomes may require specialized research and knowledge- as in the expert opinion provided in this case by the specialist in occupational health. However, recognizing the possibility that environmental factors may be a cause of illness is the essential first step.

The role of the family physician in this case is to:
i) suspect an environmental exposure causing a health problem
ii) acquire an adequate exposure history
- assess if there may be a significant exposure
- refer to an appropriate specialist in occupational or environmental health, and

iii) facilitate worker compensation for the patient

APPENDIX A: Summary of Environmental Risks for Spontaneous Abortion

1. Maternal Risks

- **Metals** (1)
- lead Odds Ratio = 0.8-1.9
  recommendation: blood lead < .75 uMol/ L for prospective parents
- **Solvents** (2)
- ethylene glycol ethers OR 1.1-3.4
- toluene OR 1.6-4.7
- **Other Chemicals** (3,4,5)
- **PCB'S** - increased risk with heavy contamination of food
- **pesticides**
  eg. methyl isocyanate in Bhopal (6)
  OR=4.3 for spontaneous abortion
  OR=4.0 for 21-28 wk. fetal death
- **Radiation** -ionizing (X-ray)-see Health Care Exposures below
  -non-ionizing – video display terminal data inconclusive (7)

- **Physical /Ergonomic Factors**
  - strenuous work eg. repetitive heavy lifting
  - noise, heat, cold, vibration exposure
  - prolonged standing
- **Health Care Exposures** (4)
  -antineoplastic drugs OR=1.7-2.8
  -anesthetic gases OR=2.0
  -ethylene oxide
  -formalin OR=3.5
- -X-ray - recommendations < 0.5 rads total in pregnancy
  < 0.05 rads in any one month

2. Paternal Risks

- **Metals** (1)
  -Lead OR 1.1-7.0
  - synergistic with maternal smoking
  -Mercury OR 1.1-2.3

APPENDIX A
(continued)

Solvents (2,4)

-data are inconsistent
-anesthetic gases OR=2.3

Odds Ratio (OR) estimates the relative risk of the outcome in exposed compared with non-exposed populations.

References- Appendix A

1. Anttila A and Sallmen, M . Effects of parental occupational exposure to lead and other metals on spontaneous abortion. J Occ Env Med 1995; 37(8): 915-21.

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APPENDIX A
(continued)

Other References

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9. Fedoruk JF. ACOEM Reproductive Hazard Management Guidelines. J Occ EnvMed 1996; 38(1) 83-90.

For clinical information re: exposures during pregnancy and breastfeeding:

1. Motherisk Program
   Hospital for Sick Children, Toronto
   9-5 weekdays
   Information specialist: (416)-813-6780
   OR: Clinical consultation by referral.

2. Safestart Program- McMaster University, Hamilton
   (905)-521-2100 Ext. 6788
   Dr. Elizabeth Chow-Tung
   Leave detailed message-receive call back.

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Case # 10

Ted, an 83 year old admitted with pneumonia on the weekend

You are doing rounds on your patients one Monday morning in winter, and find that Ted, an 83 y.o. from your practice, was admitted two days ago. Your chart review gives the following information:

Provisional Diagnosis: Pneumonia, complicating a possible stroke

History of Present Illness:
Found on the floor by landlord, incontinent of bowel and bladder, stated he had fallen and was too weak to get up. He was drowsy and unable to give a history in emergency. He is a non-smoker.

Past History: Type 2 diabetes mellitus on insulin; on no other meds
Remote left hip replacement
No history of cardiac, respiratory, or neurologic disease
No known history of alcohol abuse.

Admission Exam: Afebrile, BP 113/60, pulse 68, resp rate 30 and laboured, copious respiratory secretions. No signs of head injury or other trauma.

Chest: Diffuse rhonchi and crepitations; cardiac exam normal.
Abdominal exam normal except for incontinence.

Neurologic: Obtunded. Unable to answer simple questions or follow commands. Cranial nerves normal. Pupils 2 mm. Equal and reactive. Reflexes diminished; thought to be normal for age. Babinski reflexes equivocal bilaterally.

Investigations:
Hb 152; WBC 12,400 with "left shift"; glucose 9 mmol/L, BUN, creatinine, electrolytes normal, T4,TSH, B12 and folate all normal, urinalysis normal.
Blood gases(3 L/ min O2): pH 7.38, PaCO2 23.8, PaO2 83, Bicarb 24 mEq/L, O2Sat 96%
Drug screen negative for acetominophen, ASA, and alcohol

Sputum: gram stain negative. Blood and sputum cultures negative at 48 hours.
EKG normal. CXR: bilateral air-space opacification. CT scan head normal.

Case # 10 (continued)

Ted, an 83 year old admitted with pneumonia on the weekend

Initial Treatment: O2, IV cefuroxime and erythromycin, adjusted dose insulin, chest physio.

Re-examination: When you examine Ted on Monday, he is demented, poorly communicative, and has muscle fasciculations, pinpoint pupils, and a cough with clear copious secretions. The chest exam is unchanged from admission. The physio note states that he has "persistent, copious pulmonary and oral secretions", and "difficulty coughing and ambulating because of weakness".

Case # 10 (continued)

Ted, an 83 year old admitted with pneumonia on the weekend

Questions

1. Are you satisfied that the admitting diagnosis on your patient is correct?

What information to date confirms and conflicts with this diagnosis?

2. The combination of persistent CNS changes, miosis, muscle fasciculations, increased salivation, and generalized muscle weakness is highly suggestive of a cholinergic syndrome.

What are the possible causes of cholinergic syndrome in an elderly man?

3. What environmental exposure may cause this syndrome? What elements are needed to complete the exposure history?

What lab investigations would be helpful if you suspected an environmental exposure?

After an exposure-related diagnosis was made, Ted received a different treatment, slowly improved, returned to his prior health status,and was discharged home.

4. What treatment would you order for this patient? If you were unsure, where would you seek help?

Case # 10 (continued)

Ted, an 83 year old admitted with pneumonia on the weekend

Discussion

1. This case illustrates the difficulty of diagnosing exposure-related disease. Ted had many of the clinical features of cholinergic syndrome, in this case caused by chronic organophosphate poisoning (1). Because the clinical features may mimic presentations of other more common diseases, the diagnosis may be missed initially, or never made.

2. Diagnoses such as pneumonia, asthma, acute pulmonary edema, exacerbation of COPD, dementia, organic psychosis, hypoglycemia, stroke, or coronary artery disease with dysrhythmias may be suggested by the clinical signs.

3. Peripheral neuromuscular signs such as fasciculations, or generalized weakness may dominate the clinical picture, leading to differentials such as botulism, dermatomyositis or Guillain-Barre syndome (2).

4. Effective treatment of exposure-related disease requires an accurate clinical diagnosis. This patient was too confused to give a history, and the source of organophosphate exposure was not known. Lab diagnosis is rarely available quickly enough to direct timely treatment. However, correct treatment was started on the basis of clinical findings.

After consultation with the Poison Control Centre, the patient was given IV atropine and praladoxime, an organophosphate antidote. Ted improved gradually over 2-3 weeks, and left hospital "conversing well...and fully oriented, walking with the aid of one cane; he had normal-sized pupils, and no further fasciculations, or any evidence of focal weakness".

5. Laboratory diagnosis of organophosphate poisoning requires:
i) a lowered cholinesterase level in red blood cell or plasma on initial presentation, or
ii) a recovery (increase) on follow-up cholinesterase of 25% or greater.
Cholinesterase may take 1-3 months to recover.
Ted's first cholinesterase was 3.21 kU/L(normal 4.0-12.0). A repeat test one month later was in the normal range.

Case # 10 (continued)

Ted, an 83 year old admitted with pneumonia on the weekend

6. How was Ted exposed to a pesticide?
Ted lived alone in a small cabin on a mixed livestock and grain farm, but it was winter and his landlord thought it was unlikely he would have come in contact with pesticides. As sometimes happens in family medicine, the mystery was solved by a housecall. A visiting social worker found over two dozen empty cans of an organophosphate insecticide spray in Ted's bedroom. He had recently been applying the spray to his bed sheets in an effort to rid his room of insect pests.

References:

1. Lee, TK Is this patient poisoned? The Canadian Journal of Diagnosis. November 1997;123-30.
This is the original case report, including an excellent discussion of the pathophysiology, clinical diagnosis, and treatment of organophosphate poisoning.

2. LoVecchio, DO and Jacobsen,S Approach to generalized weakness and peripheral neuromuscular disease. Emergency Clinics of North America, 15(3):605-22, 1997.
This case-based article reviews the pertinent environmental exposures which cause neurologic symptoms, and includes a case of pesticide poisoning.

3. Reigart, R and Roberts JR . Recognition and Management of Pesticide Poisonings, 5th Ed., Office of Pesticide Programs, U.S. Environmental Protection Agency, 1999.
As well as concise and current treatment information, this book has excellent information on health effects of the various classes of pesticides- an important clinical tool to match the exposure history with observed clinical signs.

Environmental
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Pesticide Module

Background for Teachers

Pesticide Module: Background for Teachers

Classes of Pesticides

The main groups of commonly used pesticides include:

- Herbicides eg. glyphosate, 2, 4-D, atrazine,dinocap, paraquat
- Insecticides: 1) organophosphates eg. diazinon, chlorpyrifos
  2) carbamates eg. carbaryl, methomyl, propoxur
  3) biological eg. pyrethrins, rotenone
  4) repellants eg. DEET insect repellant
- Fungicides eg. captan, copper sulfate, folpet
- Fumigants eg. chloropicrin, methyl bromide
- Rodenticides eg. warfarin, diphacinone, crimidine

Basic Toxicology

Pesticide toxicity can result from ingestion, inhalation, or absorption through the skin. It is usual for several organ systems to be affected, and the pattern of effects varies widely according to the class of pesticide. A good history and physical, followed by treatment based on suspected exposure, is the most effective approach.

It is worth emphasizing that treatment must proceed based on suspected rather than lab-confirmed exposure to provide timely therapy.

Systemic Effects

The systemic effects of pesticide exposure range from acute poisonings with neurologic effects, diarrhea, and bronchial hypersecretion, to an 'intermediate syndrome' of delayed neurological symptoms, to chronic effects including dermatitis, neurobehavioral symptoms, and cancer. The effects vary greatly depending on the toxicology of the pesticide.

The range of clinical presentations for pesticide poisoning highlights the importance of taking a good exposure history, as the clinical presentation may mimic other common diseases.

At-Risk Patients in a Family Practice

1.Occupationally exposed workers such as pesticide applicators should have a baseline measure of plasma cholinesterase during a period of non-exposure. The WHO standards require removal from exposure if RBC cholinesterase levels are depressed to 70% of baseline levels.

2. Children are at increased risk because of exposure patterns and biologic vulnerability. This is reviewed in the Case #5 discussion. Other cases illustrating this general teaching point in environmental health can be found in the modules on lead, indoor air, and outdoor air.

3. The elderly may show health effects related to the long latent period between exposure and disease onset. For more information about these effects see the section on the next page:
"Chronic Health Effects of Pesticides That May Manifest in the Elderly".

4. Farmers and their families have multiple possible routes of exposure including occupational, secondary (work exposures brought into the home by the worker), and food, water and soil.

5. Home gardeners and their families have higher rates of pesticide-related tumors in a number of studies.

6. Pregnant women must be considered a high-risk group. Case #6 illustrates the vulnerability of the fetus to pesticide exposures.

The pesticide cases are designed to deal with three high-risk groups: children, pregnant women and the elderly.

Chronic Health Effects of Pesticides That May Manifest in the Elderly

Because of the long latency between exposure and diagnosis for many pesticide health effects, the elderly may be selectively affected. Agricultural chemical exposures are mixed, making etiology of specific diseases hard to determine. The current trends from many published studies are:

- pesticides appear to exert toxic effects on the bone marrow, and may cause hematopoetic cancers after a latency of 10-25 years
- pesticides may have cumulative neurotoxic effects which cause neurologic disease in combination with genetic susceptibility

Non-Hodgkins Lymphoma

Several epidemiologic studies have linked pesticide exposure to the development of this cancer, with a latency of 10-20 years. Herbicides and fungicides appear to present the greatest risk (1). High-risk patients in a family practice would include farmers, pesticide applicators and gardeners.

Prostate Cancer

A statistically significant increase in prostate cancer is seen in the majority of studies on men exposed to agricultural chemicals; a specific etiologic agent remains speculative. It is unclear if the risk may be related to weak estrogenic effects of pesticides, or to exposure to cadmium, a known prostatic carcinogen found in agricultural fertilizer, fungicides and insecticides (2).

Myelodysplasia (MDS)

Observations that leukemia in chemical-exposed workers had a distinct genetic profile led to a case-control study of 178 MDS patients. Exposure to pesticides or solvents for >2400 hours resulted in an increased relative risk of MDS:
(OR = 3.74;p<.00001). Those exposed also had a cytogenetically distinct form of MDS, with a shorter survival than non-exposed patients (3).

Parkinson's Disease with Dementia

Recent studies have focussed on an interaction between a genetic factor and pesticide exposure as a predictor for developing this form of Parkinson's disease (4).

Amyotrophic Lateral Sclerosis

In a Washington State study (5), lifetime exposure to agricultural chemicals increased the risk of developing ALS with the following Odds Ratios: Men ever exposed to agricultural chemicals: 2.4. Low exposure: 1.5. High exposure: 2.8

The latency period between peak exposure and diagnosis was 15-25 years.

References

Health Effects in the Elderly

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4.Hubble JP et al Gene-Toxin Interaction as a Putative Risk Factor for Parkinson's Disease with Dementia. Neuroepidemiology 1998; 17: 96-104.
5.McGuire V at al. Occupational Exposure and Amyotrophic Lateral Sclerosis. Am J Epi 1997; 145(12): 1076-88.

References for Teachers

References specific to the teaching cases are included at the end of each case.

Treatment

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General

Greenberg MI, Hamilton RJ, Phillips, SD. Occupational, Industrial and Environmental Toxicology. Mosby, 1997, 620 pp
This text provides a list of exposures by occupation, as well as readable toxicology information.

Pope AM and Rall DP Environmental Medicine: Integrating a Missing Element into Medical Education. National Academy Press, Washington, 1995.
This book has a large section of case material for teaching.

Schuman, SH Environmental Epidemiology for the Busy Clinician. Harwood Academic Publishers, Canada, 1997, 216 pp.
This book offers an organized clinical approach to assessing environmental health problems in primary care, and some case material for teaching.

Websites:

Canada

Environment Canada
www.ec.ca

Pest Management Regulatory Agency
www.hc-sc.gc.ca/pmra
National website and toll-free service for all Canadians on issues related to the use of pesticides. 1-800-267-6315

United States

Agency for Toxic Substances and Disease Registry (ATSDR)
http://atsdr1.atsdr.cdc.gov:8080

National Institute for Occupational Safety and Health (NIOSH)
www.cec.gov/niosh/homepage.html

Proposed Exam Questions

Proposed Exam Questions-Pesticide Module

1. Which of the following health effects have not been associated with pesticide exposure?

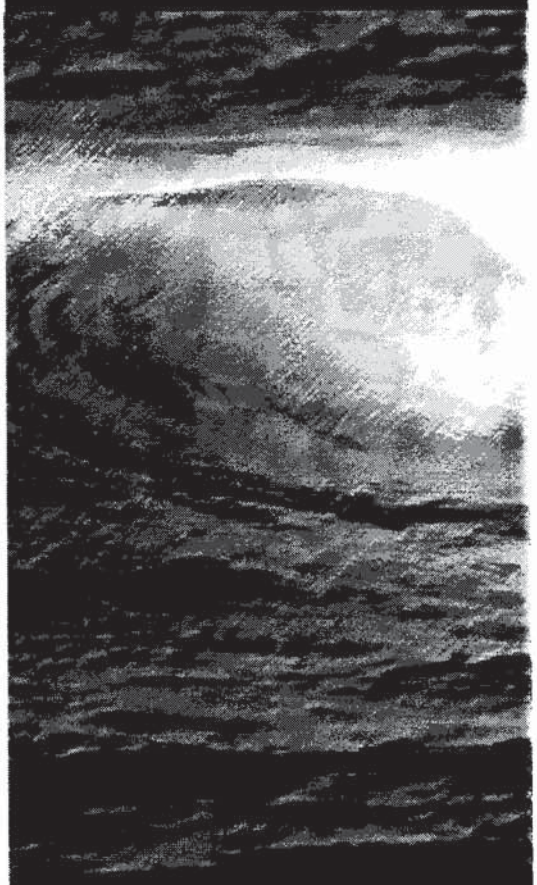
a) Acute elevation of platelets and leucocytes
b) Skin rashes
c) Acute coronary artery thrombosis
d) Seizures sometimes leading to coma
e) Prostate cancer

2. Which statements are true about the chronic human health effects of pesticides?

a) Individuals with "slow metabolizer" genotypes in the detoxifying P-450 system are more susceptible to some effects of pesticides.
b) Children are more susceptible than adults to pesticide effects through a combination of biologic and behavioral factors.
c) In spite of theoretical concerns, there are no known health effects of pesticides on the male or female reproductive systems.

Answers:
1. a,c
2. a and b are true

Environmental HEALTH in Family Medicine



Cluster Module

Clusters in Practice- Human Health and Water Quality

This module is one of six in the series Environmental Health in Family Medicine Curriculum Development Project

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The support of the Ontario College of Family Physicians and the Health Professionals Task Force of the International Joint Commission is gratefully acknowledged.

Objectives

1. To understand the family physician's sentinel role in reporting diseases which may be part of a cluster.

2. To learn skills to recognize possible disease clusters occurring in practice.

3. To learn about patterns of clinical disease related to exposure to contaminated drinking water and recreational water.

4. To know the three criteria which define a cluster of disease.

5. To learn skills in balancing patient concerns with scientific evidence when the community is concerned about an apparent cluster of disease.

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Case #11

You are working part-time at the health services centre on a university campus. During the afternoon, you see 15 patients including the following:

A. A 23 y.o. HIV positive man with a 6-day history of severe watery diarrhea, fever, and dehydration-referred to emergency for possible admission.

B. 21 y.o. usually healthy woman with 1 week of abdominal cramps and diarrhea, with weight loss of 3 kg.

C. 2 y.o. brought in from the university daycare centre with severe diarrhea, fever, and vomiting.

D. 19 y.o. man living in residence with a six-day history of severe nausea and diarrhea.

Case # 11 (continued)

Questions

1. What is your differential for exposures that may produce a gastrointestinal illness?

2. What other questions in the histories for these four patients would help to identify the source(s) of the illness? Using the CH2OP mnemonic:

Community (C)

Home/ Hobbies (H)

Occupation (O)

Personal Habits (P)

3. Do you think the four patients described above could represent a disease cluster? In thinking about disease clusters in primary care, how are they defined?

4. Are lab tests or other investigations helpful in this situation?
If so, what investigations would be important here?

5. What resources are available in any community to help the primary care physician with defining a cluster of illness?

Cluster Module- Human health and water quality: Further information(cont)

Cluster Module-Human health and water quality: Further Information

1. Exposures to consider when seeing multiple cases of gastrointestinal disease include food, water, and infectious agents acquired in the home, school, daycare, workplace, or through travel.

2. Further information was obtained from patients or families:
- All patients live within the city and drink municipally treated water
- The four patients have no common food source
  - no shared type of food in common, or shared market or grocery store
  - no shared eating place, such as a fast-food outlet or university cafeteria
- They live in different types of housing- old, new, on-campus and off-campus
- They have no shared hobbies or activities, such as swimming in the same pool
- Occupation: all are students or family of students. They share no common part-time workplace.
- One is a smoker, three are non-smokers
- None of the patients has a history of recent foreign travel or camping trips.

3. For a cluster of disease to be successfully documented, three criteria must be met:

a) The disease must be documented. This implies collecting good clinical information and documenting a cause for the GI symptoms by lab analysis of stool samples.

b) The exposure must be documented. There must be collection of information from a shared environment of these patients which defines a disease-causing agent, quantity, and time.

c) When the exposure or disease agent is related to the documented disease, there must be a plausible biologic mechanism for cause and effect, including documented plausible metabolic or toxicologic effects on target organs.

4. When a cluster of disease is suspected, prompt assessment by lab diagnosis is essential. Ideally, the physician in this case would order stools for culture, microscopy, and ova and parasites on all four patients. This may involve calling back patients for further lab tests after seeing more cases with similar symptoms.

The primary care physician plays an important role as a "sentinel" in the health-care system. Epidemics of strep pharyngitis, pertussis, influenza, RSV, and food poisoning are all examples where prompt recognition is important both for proper treatment and for prevention of more cases.

Cluster Module- Human health and water quality: Further information(cont)

5. Resources in the community include:
- Daycare providers who can supply information about illness and absenteeism
- School administrators who can provide information about illness and absenteeism
- Other physicians whose recent experiences may be pooled to find an illness trend in the community
- Health units can assist by calling back patients for further diagnostic tests. They may also identify a cluster by collating case reports from individual practices.

For primary care physicians, this type of detailed fact-finding is impractical and time-consuming. Our responsibility is to :
1. suspect a cluster of disease based on clinical observation; or suspect that individual cases may be part of a community cluster
2. make an accurate clinical and lab diagnosis
3. act as a sentinel in reporting the suspected cases or cluster to the local public health unit.

The E. coli outbreak in Walkerton provides a tragic example of the severe consequences of contamination of a municipal water supply, and has heightened physician and patient awareness of the possibility of water-borne gastrointestinal disease.

Cluster Module- Human health and water quality

Discussion

- This disease cluster occurred in an Ontario city, and over 200 confirmed cases were eventually reported.
- The causative agent was identified as the parasite cryptosporidium in the city's municipal water supply. The parasite originates in the feces of farm animals and is spread by fecal contamination of drinking and recreational water sources. Filter strips and other manure containment devices appear promising to prevent its entry into drinking water sources.
- Because it is ubiquitous in livestock feces, identifying a point source for an outbreak is usually impossible- as it was in this case.

- **Cryptosporidium** is a protozoa that exists in water as a thick-walled oocyst which is not eliminated by chlorination and is only partially sensitive to ozone treatment. It has caused large gastroenteritis outbreaks in Milwaukee (affecting over 400,000 people) and in Sydney, Australia. Special filtration is required to minimize its presence in treated water. Cryptosporidium causes a severe diarrheal illness with nausea,cramps and headache. The gastroenteritis may be life-threatening to people with HIV or other immunosuppressing medical problems. It has also caused disease outbreaks linked to swimming in community or motel swimming pools.

- **There is no drug treatment effective against cryptosporidium.** Since prevention is the only effective intervention to control outbreaks, early diagnosis and instituting effective public health measures are vital in controlling morbidity and mortality from the disease. The cryptosporidium oocysts are killed by boiling drinking water for 1 minute.

Public Health Units and the Investigation of Health Events Clusters

Public health units (PHU's) are well-situated to respond to, assess and prevent, reduce or eliminate factors leading to health events clusters, for a number of reasons:

- PHU's cover virtually all communities.
- They have the legal mandate to respond to environmental and infectious disease (ID) risks to protect public health.
- They have a range of resources necessary to undertake cluster investigations, e.g. Medical Officers of Health, epidemiologists, infectious disease staff, health inspectors.
- They have local, regional and provincial linkages where necessary, e.g. cross-PHU outbreaks, access to provincial health data registries, access to consultants.
- They have media and communications expertise where this is necessary to assess or respond to a cluster.
- They have the legal authority to institute control measures when this is necessary, e.g. boil water advisories, suspension of a water treatment plant functioning (as in the Waterloo Region cryptosporidiosis outbreak), treatment or exclusion orders in cases of certain IDs.

As indicated in the cluster module, the physician-public health interface is essential in recognizing and responding to clusters, through the primary roles physicians have in confirmation of diagnosis, recognition of abnormal patterns of events, their duty to report and the broader role of physicians in community education and interpretation of health issues.

Most physicians have a number of contacts within PHU's for discussing or reporting cluster concerns, but when in doubt, go to the top and speak to the Medical Officer of Health. It is reasonable, in the face of an ID cluster, for you to expect the following from your PHU:

- Information on your case(s) to permit local registries to be developed and contact tracing and assessment to begin.
- Active surveillance of hospital emergency departments, urgent care/walk-in clinics, schools, daycares, nursing homes, etc.

Public Health Units and the Investigation of Health Events Clusters

- Specific investigations, e.g. case-control studies, to examine possible etiologies (where this is necessary).
- Timely and effective actions to reduce or eliminate further health risks.
- Public and health professional communication and education on such issues as symptoms and signs, appropriate seeking of care, preventive measures, etc.

When confronted by environmental health clusters, e.g. birth defects, cancers, the PHU response and actions may be somewhat different. However, a number of principles are the same:

- Collection of accurate case information.
- Active surveillance through local surveys or use of provincial health data registries.
- Environmental and/or occupational exposure assessments when warranted.
- Public and health professional communication and education, especially related to whether or not a cluster does exist and, if not, what factor(s) could be leading to the perception of a cluster.
- Timely and effective actions to reduce or eliminate factors that may be or are associated with the cluster.

Environmental
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in Family Medicine

Cluster Module

Background for Teachers

Contaminants in Drinking and Recreational Water: Health Effects and At-risk Groups

Protozoa such as giardia and cryptosporidium are a very important source of illness clusters in municipally treated and private water sources. These organisms are highly infectious and resistant to chlorination. Bottled water is not routinely tested for these organisms. Many large outbreaks of gastroenteritis have been linked to these agents in Canada, the US and Australia.

Giardia is found in human and small animal feces, and produces intestinal illness with cramps and weight loss. It is treatable with anti-parasitic drugs.
Canadian water surveys have found the following levels of giardia contamination:
Raw water samples: 21%
Treated water samples: 18%
Pristine water samples in the Yukon: 32%. Campers beware!

Cryptosporidium is found in feces of cows and other domestic and wild animals, and causes an illness with watery diarrhea, cramps, nausea and headache. The incubation period is 2-25 days, and there is no approved drug treatment.

Both giardia and cryptosporidium may cause life-threatening illness in the immunocompromised and elderly.

Bacteria including E.coli, campylobacter, pseudomonas and enterococci may contaminate surface water, groundwater and bottled water. The gastrointestinal illnesses range from mild diarrhea to hemorrhagic colitis, depending on the specific bacteria and strain. Infants,children, the elderly and those with immunosuppression are most likely to develop severe disease.

Bacteria such as staph aureus and pseudomonas may cause clusters of dermatitis, conjunctivitis, respiratory disease and otitis externa in contaminated recreational waters. At-risk groups include swimmers, snorkellers, wind-surfers (more falls correlated with more infections), hot-tub users, and triathletes.
Other biologic contaminants include Schistosoma species, a flatworm which causes "swimmers itch" after swimming in warm freshwater lakes.

Nitrates and nitrites may contaminate surface or groundwater in rural or suburban areas, where it is caused by runoff from fertilizers and manure, or by contamination of water sources by leaking sewers or septic systems.

The important health effect of nitrites is methemoglobinemia. Infants under four months of age, and pregnant women around 30 weeks gestation are most sensitive to this effect.

Pesticides may contaminate groundwater or surface water as a result of agricultural or industrial runoff. Health effects may include increased incidence of some cancers. (See the pesticide module for further information).

Enquiry about the source of drinking water, and recreational water exposure are essential in the exposure history for gastrointestinal illness. The four main sources of water are: untreated surface water, untreated groundwater, municipally treated water (which may start as surface or groundwater), and bottled water.

Clusters of Disease

The role of the "alert clinician" or "sentinel" is very important in primary care. Here are some examples of successfully diagnosed disease clusters :

1. Two patients at a nursing home have upper respiratory symptoms. Stat nasal swabs are requested, and both are positive for influenza A. Other residents and staff are treated prophylactically, preventing an outbreak.

2. Two 12 year old girls are seen within two days in the office with severe spasmodic cough. They play on the same baseball team. Nasal swabs reveal B. pertussis, which allows timely treatment of the patients, family, and other team members with erythromycin.

3. Three cases of meningitis are reported to a health unit; lab studies confirm that the same strain of meningococcal bacteria is responsible. The health unit initiates a community education and immunization program.

4. The illegal spraying of the carbamate insecticide aldicarb on watermelons in California produced the largest outbreak of pesticide illness ever documented in North America, affecting over 1,000 people. An emergency physician recognized the symptoms of pesticide poisoning while treating three family members, and notified Poison Control of the probable connection to eating watermelon. The epidemic was then quickly controlled by a state-wide embargo on watermelon, and a field inspection program.

Goldman, LR et al Pesticide Food Poisoning from Contaminated Watermelons in California, 1985. Arch Env. Health 45(4): 229-36, 1990.

The effective management of many disease clusters is initiated only after case reporting by an alert clinician who has taken a thorough exposure history, and ordered the right diagnostic tests.

Disease Clusters and Public Perception

Clinicians are often approached by concerned community members who perceive a disease cluster.

Examples are:

- A local workplace has had three cases of breast cancer among female employees working in one division. The local breast cancer support group believes that chemicals used in the plant are responsible.

- A school has three teachers who developed chronic lung disease over a three-year period; one was a non-smoker. The teachers' union is concerned that asbestos in the walls and roof was a cause.

- In a local neighbourhood, two children have developed brain cancer and one has died of acute leukemia. A group of concerned parents asks you to support their concern that the cancers are being caused by some chemical in the neighbourhood environment.

Clinicians must strike a careful balance between respect for their patients' concerns, and respect for the process of science-based diagnosis and investigation. Going to the media with community concerns before the science is known is bad medicine.

The following role for community-based clinicians has been proposed:

"a) The better the diagnosis, the greater the chance of success. Confirm the case with expert help (health department, etc.).
b)Ask for investigation of a series of cases of the illness to identify the source and prevent further cases. Expert help is again required (health department, etc.).
c)Support the outcome of the objective investigation. Help the community accept the most likely science-based conclusion...
...Chance (is) the most likely 'cause' of clusters ".

Source: Stanley H. Schuman Environmental Epidemiology for the Busy Clinician, Harwood Academic Publishers, 1997.Chapt. 10: When the Community is the "Patient": Clusters of Illness.

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Proposed Exam Questions

1. Select the true statements:
   To diagnose a disease cluster,

a) an alert clinician must recognize that multiple cases of a similar illness could have a common cause;
b) the physician may need to call patients back for further lab tests after seeing other similar cases;
c) a thorough history must be taken on every patient, and a cause of the illness diagnosed before notifying public health;
d) a community physician will often need the expertise and resources of the public health unit.

2. Which of the following organisms is unlikely to cause a cluster of gastroenteritis ?

a) Cryptosporidium
b) Staph aureus
c) E. coli
d) Giardia

Answers:

1. a,b,d

2. b. Staph aureus is more likely to cause dermatitis or conjunctivitis.

Environmental HEALTH in Family Medicine

POP's Module

POP's module

This module is one of six in the series Environmental Health in Family Medicine Curriculum Development Project

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The support of the Ontario College of Family Physicians and the Health Professionals Task Force of the International Joint Commission is gratefully acknowledged.

Objectives

1. To introduce and provide practice in a method for taking an environmental exposure history in the office.

2. To provide learners with cases which provide an opportunity to acquire skills in exposure assessment, risk assessment, risk communication and risk management, specifically related to the health effects of persistent organic pollutants in the Great Lakes Area.

3. To understand the process of bioaccumulation of persistent toxics in the food chain and endocrine disrupting potential of POPs, and to become aware of PCBs and other contaminants in the Great Lakes basin fish and game.

4. To provide a selected resource list for reading and online information re: the health effects of persistent organic pollutants.

Environmental
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in Family Medicine

Case # 12

Case

You are a Family Physician in a small town on the shores of Lake Huron. You are seeing a 27 year old woman. She is wanting to go off the pill, to become pregnant. She is G1P1, with a 7 year old son who is presently being assessed by the school psychologist for a learning disorder. She saw on TV a clip about contaminants in fish affecting children's intelligence. She is concerned that she might have caused the learning disorder in her son, and about the health of her new baby, and asks advice about how to protect her fetus from environmental contaminants that might cause learning problems in children.

Q1. What in the history do you want to know? Besides a medical history, remember to include an exposure history (occupational and environmental).

Community.

Home/Hobbies.

Occupation.

Personal.

Case # 12 (continued)

1. Medical History

Past Medical History: Nil.

Allergies: Nil

Medications: Folate 0.4mg daily, started recently. Nil else.

Family History: Her Family: Diabetes in maternal grandmother, onset at 65. Father hypertensive and smokes. Mother and sibs well. Husbands Family: Husband is well. Nil of note in his family. No congenital abnormalities, early deafness or twins.
History of Pregnancy. G1P1. First pregnancy: normal pregnancy without complications, normal vaginal delivery at term, normal neonatal course, breast fed for 6 months. 7 year old son is well besides recent concern re school difficulties. He is currently being assessed with neuropsychological testing by the school psychologist for learning disorder.
Psychosocial Assessment. Married for 9 years. No significant marital problems. Supportive extended family.

2. CH2OP.

Community.
Live in small town of 5000 on shore of Lake Huron. Tourism, cattle and dairy farming, fishing.
Town water from Lake. Some smoggy days in summer. No polluting industry known.

Home/Hobbies.
Recently moved to a new home in new subdivision. Town water. Gas furnace, forced air heating. Previous home was an 80 year old house on main street, to which they did extensive renovations.
She gardens and knits. Her husband is an avid sports fisherman, including ice fishing in winter, and a hunter.

Case # 12 (continued)

Occupation.
She works as a Kindergarten teacher. Only other job was working in husband's store on weekends before her son was born. Husband co-owns a hardware store with his brother. He has always worked in the same store, which has been a family business for 2 generations.

Personal.
Neither she nor husband smoke. He quit 8 years ago, just before the first pregnancy.
Alcohol. She drinks beer occasionally. Plans not to drink when pregnant. He drinks 2 to 3 beers daily.
No drugs.

Questions:

Q1. What are the possible exposures to the planned pregnancy?

Q2. What lab tests would you order?

Q3. What contaminants can affect the neurobehavioural development of the fetus?

Q4. How would you address her concern about environmental exposures as the cause of her 7 year old's learning disability?

Q5. How would you counsel her to avoid exposure of her baby?

Case # 12 (continued)

Q1. What are the possible exposures to the planned pregnancy?

The clues are in the exposure history.

Housing: They had previously lived in and renovated an 80 year-old house. There must be suspicion about Lead toxicity, as exposure from interior leaded paint, especially mobilized during renovations; through contaminated soil from chalking exterior paint; and from pre-1989 plumbing with lead solder would be possible. This would be of concern especially for her son, as children are more exposed through contact with dust and hand to mouth activities.

Hobbies. Her husband is an avid sports fisherman and a hunter. It is important to know how much fish and game they eat.
She has been eating fish that her husband catches in Lake Huron at least weekly, for many years, and occasionally fish bought at the store. They also eat game meat that he has hunted. Last year he got a moose. She is unaware of any warnings or cautions about eating sports fish.

Fish and wildlife, being at the top of the food chain, can accumulate Persistent Organic Pollutants, such as dioxin, PCBs (polychlorinated biphenyls), and some Organochlorine Pesticides such as DDT. Other possible pollutants are organic metals such as methyl mercury. It is also possible that game can be polluted with lead, from the lead shot.

Q2. What lab tests would you order?
Blood lead levels for Mom and the 7 year old son were normal ie below 0.5 umol/L. Testing for PCBs is available, although difficult to arrange. It is unlikely to be of value in this case. It may be useful in high dose occupational exposures. Hair or blood mercury could be considered, although it would be advised to first estimate the probable mercury exposure from eating Lake Huron fish. This could be checked from the guide for sports fish (see below).

Case # 12 (continued)

Q3. What contaminants is she referring to in terms of affecting the neurobehavioural development of her fetus?

1. Lead

2. Other metals, such as methylmercury.

3. Persistent Organic Pollutants (POPs) such as PCBs and dioxin-like compounds.

1. The neurobehavioural effects of exposure to **lead** are described in the Lead module of this series.

2. What are the potential neurobehavioral effects of exposure to **mercury**? There is good evidence in animal as well as human epidemiologic studies that exposure to methylmercury has neurotoxic effects.
Mercury exists naturally in the environment, but levels have risen due to discharge from hydroelectric, mining and pulp and paper industries. Microbes in soils, and in river and lake sediments convert elemental mercury into Organic methylmercury by methylation. This methyl mercury is more bioavailable, and bioaccumulates in fish and other aquatic organisms. Thus methyl mercury is at highest concentrations in species at the top of the food chain, such as pike, walleye and bass in freshwater, and tuna, swordfish and shark in sea water.

3. What are the potential neurobehavioral effects of exposure to **Persistent Organic Pollutants (POPs)** such as PCBs and dioxin-like compounds?
Evidence for concern regarding POPs comes from animal laboratory studies, as well as epidemiological studies. Babies born to mothers who ate very large amounts of highly contaminated fish (PCBs were measured) from Lake Michigan had smaller birth weights, smaller head circumferences and shorter attention span than babies born to those not eating fish. As they have been followed over 11 years, these children have continued to do more poorly in a range of skills and development tests, including deficits in general intellectual functioning, short term and long term memory, and attention span. These effects are subtle, yet may be significant in vulnerable children, and like lead, may have significant public health importance. The effects are related to transplacental exposure, and not to exposure via breast feeding. It is postulated that the brain during fetal development is

Case # 12 (continued)

particularly sensitive to these contaminants.
Prenatal and postnatal exposures to PCBs and dioxin-like compounds have been shown to be associated with changes in thyroid hormone levels, which might be the mechanism by which these effects on neurobehaviour are mediated.

Other health effects of POPs are discussed below.

Q4. How would you address her concern about environmental exposures at the cause of her 7 year old's learning disability?

There is no way to know for sure if PCBs from her consumption of fish had a role in the problems of her 7 year old. At worst PCBs would have contributed to the boy's problem. The PCB effect is not large enough to produce clinically significant neurobehavioural effects on its own. Even at much higher exposures than Great Lakes exposures, in a group of children exposed to much higher doses of PCBs, the Yu-Cheng cohort, there was a maximum loss of 6 IQ points. Learning disorders are multifactoral, and a clear understanding of the causes in this case is impossible. A discussion about management of her son, via special educational and behavioral initiatives, as well as an assessment re possible medication use, is appropriate.

Q5.How would you counsel her to avoid exposure of her baby?

1. Lead. Her lead levels are normal. The risk was related to the old house, and especially in renovations, where old leaded interior paint can be a risk as it is removed, with the potential for ingestion and inhalation of lead dust.

2. However, the main issue to discuss is her diet.

She eats **sportsfish** regularly. This is a risk for exposure to mercury and Persistent Organic Pollutants such as PCBs and dioxin like compounds. She should be advised to get a copy of the Fish Advisory in her province or state. In Ontario, the Ministry of Environment and Ministry of Natural Resources publish the "Guide to Eating Ontario Sports Fish", which contains advice on species type, recommended consumption levels, and location of sportsfish species by body of water. These recommendations are distributed at beer stores and with fish licens-

Case # 12 (continued)

es. However, they are often not brought home by fishermen, or not read.

Consumption advice is stricter for all children, and girls and women until after childbearing age, because these contaminants persist in the body, and can be transferred to the fetus across the placenta and to the child through breast milk.

Fish caught and sold commercially is routinely monitored by government agencies, and is therefore considered safe for consumption. For sportsfish, contamination varies by species and body of water. However, there are many nutritional benefits to eating fish, and the patient must be reassured that besides the above restrictions, her family can eat fish, and is encouraged to do so regularly.

She also eats game meat.

Wildlife, such as ducks, geese, crayfish, turtles, moose, caribou, bear, seals and whale can be contaminated with organochlorines, methyl mercury, and lead from lead shot.

3. The question of whether she should delay getting pregnant should be discussed. A waiting period would not reduce potential elevated PCB or dioxin levels. These compounds are very persistent. However, waiting 3 to 6 months would make a significant difference in her mercury level, and therefore significantly reduce exposure of mercury to her fetus.

Environmental
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in Family Medicine

POP's Module

Background for Teachers

Background for Teachers
Persistent Organic Pollutants (POP's)

1. What environmental contaminants affect the neurobehavioural development of the fetus?

1. Lead

The main environmental contaminant that has neurotoxic effects on the developing fetus and child is exposure to lead. Exposure of the fetus ie transplacental transfer, does occur. In fact lead is mobilized from the bones of the mother during pregnancy, and is therefore available in the serum for transfer to the fetus. Exposure prior to age 3 may be critical, because this is the period of rapid neuronal growth and network formation. Most lead in the environment has come from leaded gasoline, and exposure is to the infant or child from contaminated paint chips and dust, and contaminated soil. Lead levels in children have fallen along with the phaseout of lead in gasoline. Lead levels in children are still a concern in many communities in the USA, particularly related to deteriorating paint in older housing. Levels are lower in Canadian children. For a more complete discussion on lead exposure and health effects, please see the module on Lead.

2. Mercury

There is good evidence in animal as well as human epidemiologic studies that exposure to methylmercury has neurotoxic effects.

Mercury exists naturally in the environment, but levels have risen due to discharge from hydroelectric, mining and pulp and paper industries. Microbes in soils, and in river and lake sediments convert elemental mercury into Organic methylmercury by methylation. This methylmercury is more bioavailable, and bioaccumulates in fish and other aquatic organisms. Thus methylmercury is at highest concentrations in species at the top of the food chain, such as pike, walleye and bass in freshwater, and tuna, swordfish and shark in sea water.

3. PCBs and Dioxin-like compounds.

These compounds are often called POPs, Persistent Organic Compounds. They also include a group of organochlorine pesticides. Besides being toxic to humans, they are of special concern because they are persistent, lipophillic, bioaccumulative, and at low dosages they act as endocrine disrupters.

Persistent refers to the fact that these contaminants are not readily broken down in the environment, persisting for long periods of time, and traveling long distances in the environment, moving toward colder climates, especially the Arctic.

POP's (cont.)

For example, DDT sprayed on crops in central America, can persist and travel north, until it can be measured in the fat of arctic seals and whales, and the breast milk of Arctic mothers.

In animal tissue, they are **lipophillic**, so they persist and accumulate in the fat of living organisms, with a half life of 5-10 years. In the food chain they **biomagnify**, ie their concentrations increase up the food chain, and highest levels are found in predators at the top of the food chain, such as predator fish, birds and sea mammals. People eating more of these species, especially in aboriginal/Native American communities, and sports fisherman, including some immigrant communities, are more exposed. Breast milk serves as an indicator of past human exposure, and extensive breast milk monitoring programs are in place, especially in the Arctic. They show, for example, that DDT levels are declining in southern Canada, but not in Inuit women of northern Quebec.

2.What are Persistent Organic Pollutants (POPs)?
Persistent organic pollutants have been grouped together as a class for special attention by the UNEP, United Nations Environment Program, as well as responsible agencies in the USA and Canada. The most toxic 12 POPs, the "dirty dozen", include PCBs, dioxin-like compounds and chlorinated pesticides, such as DDT, dieldrin and heptachlor. International negotiations to phase out their use are in progress.

These three groups will be discussed further.

a. Dioxins and furans There are 75 dioxins , a group of 210 similar chemicals (dioxins and furans). They are the unwanted byproducts of industrial activity, such as the manufacture of some pesticides, the bleaching of wood pulp with chlorine for white paper, and copper smelting. They are also produced by the incineration of waste, including medical waste, from medical equipment made from PVC, eg IV bags, tubing and instruments, and municipal waste. Dioxin is a human carcinogen, Class 1 as classified by IARC.
b. PCBs (poly-chlorinated-biphenyls) are a mixture of 209 similar chemicals that were widely used in electrical equipment because they are so stable and heat resistant. The most common PCB trade name is Aroclor, manufactured by Monsanto. Their production, import and use have been phased out in both the

POP's (cont.)

USA and Canada, and in many other countries. However, PCBs are still present in old transformers and electrical equipment, and as such there is still leakage into the environment. PCBs are classified as probable human carcinogens (Class 2A).

c. Organochlorine pesticides,such as DDT, dieldrin and heptachlor. DDT has been used extensively for vector control, eg against malarial mosquitoes, and in agriculture, especially cotton production. Although its use has been banned in 34 countries, it is still in use in some developing countries to control malaria. It can be measured ubiquitously, in fatty food throughout the world, and in breast milk. Levels of many POPs in Great Lake ecosystems decreased due to industrial phase-out during the 70s and 80s. However they seem to have reached a plateau since then.

3. How do these contaminants get from the environment into the mother, fetus or newborn?

As discussed above, PCBs and dioxin-like compounds persist in the environment, and they therefore can be transported long distances, and build up over time in the environment. Being lipophillic, they are stored in fat in the body, and biomagnify once they have entered the food chain. The highest levels are thus found at higher trophic levels ie predator fish, sea birds or marine mammals. Drinking water from the lakes is not a risk for significant exposure, as concentrations in water are so low.

The risk is greatest in the great lakes, as opposed to smaller lakes.
Methylmercury is different, affecting fish in inland smaller lakes as well as the great lakes and the oceans. It attaches to protein, accumulating in livers and kidneys, as opposed to fat.
In the Great Lakes region, the most important exposure source for humans is by eating fish, especially:

1. Top predator species such as lake trout, walleye and salmon.
2. Older larger fish. Longer lived species such as salmon are generally more contaminated than shorter lived species like perch .
3. Bottom feeders, such as brown bullhead, carp, and white sucker, are also likely to accumulate more toxins.
4. In preparation, trimming and discarding fat and the skin will reduce organochlo-

POP's (cont.)

rine contaminants (although not methylmercury).
5. These contaminants are not detectable by smell or taste or appearance of the fish.
6. In terms of commercial fish, the Canadian guidelines recommend that swordfish and shark not be eaten more than once a week, due to mercury contamination. A number of states have issued fish consumption advisories, suggesting that pregnant women limit their consumption of canned tuna to 7ozs per week, and not to eat any swordfish or shark, to avoid exposure of the fetus to mercury. Commercial fish are tested by regulatory agencies for POPs, and are not considered a risk.
7. The greatest consumers of fish, and therefore at risk for higher body burdens, are in aboriginal/Native American communities, and sportsfisherman, including some immigrant communities, especially southeast Asians.
8. Provincial and state environmental and health agencies bordering the Great Lakes have issued guidelines and advisories regarding fish consumption, with respect to both POPs and Methylmercury levels.
9. Other wild foods, such as waterfowl eggs, turtles and turtle eggs, muskrat, otter, moose and deer might be a problem if consumed regularly.
10. In the Arctic, consumption of fish and marine mammals has been studied. There are high concentrations of POPs in some fish species, predator birds and their eggs, and sea mammals, and whale and ringed seal whose blubber is eaten. However, in assessing risk benefit, noting the many nutritional benefits to eating traditional diets, the public in the Arctic has been advised to continue eating traditional diets and to continue breast feeding their babies.

POPs cross the placenta, and are transferred in mothers milk.

4. What are the health effects of concern caused by POPs?

Evidence for concern regarding the health effects of POPs comes from wildlife studies, animal laboratory studies, as well as human epidemiological studies. There are also studies from high dose exposures from poisonings and occupational exposures. There are difficulties in conducting research, and therefore gaps in knowledge, and areas of uncertainty. A common approach is to combine all 3 areas of research, the weight of evidence approach.

Wildlife studies clearly show reproductive problems, altered sexual development and reproductive behavior, effects on endocrine and neurological development of

POP's (cont.)

embryos, an increase of tumors, and immunosuppression.

The POPs such as PCBs, Dioxins and Organochlorine pesticides have been implicated in other significant health effects. They act as **endocrine disrupters**, or hormone mimics. Since their molecular structure resembles that of hormones, they can attach to hormone receptors, and are biologically active by switching them on, or blocking the receptor eg estrogenic or anti-estrogenic effects and also anti-androgenic effects. There is considerable evidence from wildlife studies, that POPs cause birth defects, various cancers, immune system dysfunction and reproductive problems. Although the evidence regarding these effects of POPs on human health is uncertain, there is concern re potential for adverse effects (cryptorchidism, hypospadias), carcinogenicity, and fertility problems.

The following is a summary of the human health effects of POPs from low dose exposures that we are discussing.

1. Developmental and Reproductive effects.
In the low dose levels from fish eating in the Great Lakes, there have been no significant findings in reproductive or developmental effects, besides some suggestion of smaller birth weight and head circumference, and a modest association in a study in Michigan anglers with conception failure, after trying for at least 12 months, in men only.

2. Neurobehavioral or Neurodevelopmental Effects.
Babies born to mothers who ate very large amounts of highly contaminated fish (PCBs were measured) from Lake Michigan, had smaller birth weights, smaller head circumferences and shorter attention span than babies born to those not eating fish. As they have been followed over 11 years, these children have continued to do more poorly in a range of skills and development tests,including deficits in general intellectual functioning, short term and long term memory, and attention span. These effects are subtle, yet may be significant in vulnerable children, and like lead, may have significant public health importance because of the numbers of children exposed. The effects are related to transplacental exposure, and not to exposure via breast feeding. It is postulated that the brain during fetal development is particularly sensitive to these contaminants.

There is good evidence in animal as well as human epidemiologic studies that

POP's (cont.)

exposure to methylmercury has neurotoxic effects.

3. Immunological Effects.
Breast milk POP levels are associated with subtle alterations in immunological status in the newborn, and in a recent study of Inuit in the Canadian Arctic, with increased incidence of otitis media.

4. Other systemic health effects.
A single study has found a correlation between PCB levels and diabetes and liver disease. A number of studies have shown a relationship between POP levels in plasma and breast milk, and thyroid hormone levels in adults and infants. This is an area of great concern, and intense research.

5.Advice/counseling for patients about POPs
In the discussion with the patient, the risks of eating contaminated fish must be balanced against the benefits. Fish provide a diet high in protein, unsaturated fatty oils, and low in saturated fat. Some studies suggest that eating fish once a week is helpful in preventing heart disease.

The patient should therefore not be warned off fish altogether, but she should be advised to avoid certain fish in order to best protect her fetus. It should also be pointed out that although she ate fish prior to and during her first pregnancy, many factors likely contribute to learning disabilities, and the role of contaminants in diet, and other possible environmental factors, is unclear, and likely of limited significance in each individual case, ie maximally a 6 IQ point loss, and probably less in most cases.

The patient should be advised to get a copy of the Fish Advisory in her province or state. In general, sports fish advisories a) encourage sportsfish consumers to eat fewer fish species and sizes known to contain elevated levels of chemical contaminants, and b) recommend the use of cleaning and cooking methods that can substantially reduce fat-soluble contamination in a fish meal.
In the US and Canada, state and provincial governments respectively are primarily responsible for managing potential risks through issuing fish and wildlife advisories. In the US, go to http://www.epa.gov/ost/fish, for a national listing of fish and wildlife advisories, and links to specific state advisories. In Ontario, the Ministry of Environment and Ministry of Natural Resources publish the "Guide to

POP's (cont.)

Eating Ontario Sports Fish", which contains advice on species type, recommended consumption levels, and location of sportsfish species by body of water. They are distributed at beer stores and when buying fishing licenses.
In the US, 40% of the nation's rivers, lakes, and streams are not suitable for fishing and swimming. 47 states have issued public health warnings or advisories to avoid or limit fish consumption. 60% of health warnings against fish or shellfish consumption are related to mercury contamination. PCBs are the most common reason for the issuance of advisories for Great Lakes fish.
In Canada, the provinces and territories are responsible for issuing fish and wildlife advisories. In 1997, there were 2625 advisories in effect, related to mercury, PCBs, dioxins/furans, and the chlorinated pesticides, Mirex and Toxaphene. 96% of the advisories resulted from mercury centamination of fish.

Consumption advice is stricter for all children, and girls and women until after childbearing age, including pregnant and nursing mothers, because these contaminants persist in the body, and can be transferred to the fetus across the placenta and to the child through breast milk. It should be emphasized that although breast milk contains POPs, the benefits of breast feeding to the infant clearly outweigh the risks from pollutants.

The advisory information is further laid out in question 2 above.

However, US data indicate that people who are most at risk, especially women and minority groups, are the least informed about existing fish advisories. Physicians can play an important role in improving this risk communication.
While it is important to counsel and advise patients, and especially those at high risk because of their diets, to follow the guidelines re fish and game consumption, the long term solution lies in pollution prevention. In many cases, this requires changing industrial processes, waste incineration and power generation to virtually eliminate the release of these pollutants, and the clean-up of already contamnated sites and sediments. International efforts, under the auspices of UNEP (United Nations Environment Program) to severely limit or eliminate altogether the emissions of POPs into the atmosphere are in progress.

POP Module

Resources

1. Health Canada. Great Lakes Health Effects Program. The Health and Environment Handbook for Health professionals. 1998: A comprehensive Canadian look at Great Lakes Environmental Health Issues. Free copies available. Accessible style.

2. Muckle G, Dewailly E, Ayotte P. 1998. Prenatal exposure of Canadian Children to Polyclorinated Biphenyls and Mercury. Candian Journal of Public Health 89 Suppl 1:S20-25. An interesting and informative article.

3. Johnson, B.L., Hicks H.E. et al. 1998. Public Health Implications of Persistent Toxic Substances in the Great Lakes and St. Lawrence basins. J Great Lakes Res. 24(2):698-722. An excellent state of the art review of the evidence about exposure to and health effects from Persistent Toxic substances in the Great Lakes Basin. Also covers Advisories and Public Health Policies.

4. Jacobson, JL and Jacobson, S.W. 1996. Intellectual Impairment in children exposed to polychlorinated biphenyls in utero. NEJM 335(11):783-789. The latest update at 11 years of age of the cohort of children born to fish eating mothers in Michigan.

5. A comprehensive website re Endocrine Disrupters
http://www.som.tulane.edu/ecme/eehome/

6. An excellent, comprehensive report published by PSR in Boston about Toxic threats to child development. http://www.igc.org/psr/

7. EPA website of US National Listing of Fish and Wildlife Advisories, with contact numbers for state agencies to obtain specific advisories within a state.
http://www.epa.gov/ost/fish

Proposed Exam Questions

1. People eating sports fish caught in the Great Lakes region in excess of the fish advisories are at risk from the following pollutants
a) Mercury
b) PCBs
c) Lead
d) Dioxin
e) chlorinated pesticides

Answer: a,b,d,e.

2. Health effects found in adults or their children, from eating fish and game containing PCBs and Dioxin-like compounds, include the following:
a) anemia
b) cognitive deficit
c) immunologic deficit
d) asthma
e) altered thyroid status

Answer: b,c,e.

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Evaluation

Reader Survey- Please return to Env. Health Committee, OCFP, 357 Bay St. Toronto, Ont. M5H 2T7
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1. Please indicate your uses for the curriculum (check all that apply):

Teaching in a university setting - clinical ___
- non-clinical ___
Teaching in a community-based setting ___
Self-learning tool ___
Tool for remedial work with learners ___

Other _____
_____

2. Please rate your overall satisfaction with Environmental Health in Family Medicine in the following categories:

	Very Satisfied	Satisfied	Neutral	Unsatisfied	Very Unsatisfied
Information evidence-based	☐	☐	☐	☐	☐
Format easy to use	☐	☐	☐	☐	☐
Covers main topics in environmental health	☐	☐	☐	☐	☐
Practicality of information to clinical practice	☐	☐	☐	☐	☐
Format useful for Teaching	☐	☐	☐	☐	☐

3. How useful are the following modules:

Modules	Very Useful	Useful	Somewhat Useful	Of Little Use	I Have Not Used the
Lead	☐	☐	☐	☐	☐
Outdoor Air	☐	☐	☐	☐	☐
Indoor Air	☐	☐	☐	☐	☐
Pesticides	☐	☐	☐	☐	☐
Clusters of Disease (Water Quality and Human Health)	☐	☐	☐	☐	☐
Persistent Organic Pollutants	☐	☐	☐	☐	☐

4. Please write your other comments, and ideas for new module topics, on the reverse of this page and fax both sides. Thank you.