ENVIRONMENTAL HEALTH CARE TOOLKIT


Created By:

ACAT
Alaska Community Action on Toxics

505 W. Northern Lights Blvd, Suite 205
Anchorage, AK 99503
www.akaction.org
Phone: (907) 222-7714
Fax: (907) 222-7715
E-mail: info@akaction.net

Project Funded By NIEHS Grant R25ES014308
Environmental Health and Justice for Norton Sound, AK Project
Contents: Toolkit for Community Health Aides

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Foreword

With our Environmental Health Care Toolkit, we would like to honor Annie Alowa and begin with her story. Annie Alowa was a former health aide from Savoonga, Alaska on St. Lawrence Island. After the military complex was built at Northeast Cape on St. Lawrence Island in 1952, she observed that her people began suffering from unusual numbers of miscarriages, cancers, diseases, and death. She noticed changes in the land and wildlife, and began to think these conditions were associated with contaminants from the military site at Northeast Cape.

Annie brought her concerns to state and federal agencies in Alaska and then worked with Alaska Community Action on Toxics to develop solutions. The enclosed DVD contains a short film, I Will Fight Until I Melt (Whanga Pillugaghlleqaqa Kenlangu Ughullemmun), which demonstrates Annie’s concerns and presents a call for others to respond to her concerns about the impact of contaminants from the abandoned military base on human health.

About Alaska Community Action on Toxics (ACAT)
ACAT is a statewide organization established in 1997 and dedicated to achieving environmental health and justice. Our mission is to assure justice by advocating for environmental and community health. We believe that everyone has the right to clean air, clean water, and toxic-free food. We work to stop the production and release of harmful chemicals by industry and military sources; ensure community right-to-know; achieve policies based on the precautionary principle; and support the rights and sovereignty of Indigenous peoples. ACAT’s programs include: Alaska Rural Environmental Justice, Environmental Health, Policy and Social Change, Alaska Youth and Community Outreach, and Wellness and Healing.

A main focus of ACAT’s work has been to address Annie Alowa’s concerns about the impact of military contaminants on the human health of St. Lawrence Island’s Yupik People. This work has led to a broadening of concern about other Alaska villages troubled by toxics. In 2005, ACAT was awarded a grant from the National Institute of Environmental Health Sciences (NIEHS) to find effective ways to limit the release and research the human health effects of contaminants in the Norton Sound region. The grant provides funding to develop an environmental health care training curriculum that addresses the prevention of harm, diagnosis and treatment of human health effects associated with environmental contaminants affecting Arctic Indigenous Peoples.

Increasing Awareness of Environmental Health and Contaminants in Alaska
Understanding the impact of environmental exposures on human health is a growing part of the field of public health. When considering preventable causes of disease, the primary focus is often on behavioral and lifestyle factors such as diet, smoking, alcohol use, exercise, and UV exposure. However, exposures
to chemical contaminants at work, home, outside, and even in utero, are now recognized as important, and reducing exposure can prevent human diseases.

Toxic chemicals from many sources are showing up in Alaska lands, waters, fish, and wildlife: hundreds of abandoned military sites, worldwide industrial pollutants carried north to the Arctic by wind and ocean currents, and waste sites full of fuels, plastics, and electronics contribute to exposures harmful to human health. More than 80,000 chemicals have been developed, distributed and released into the environment over the past 50 years. ACAT believes that every Alaskan has a right to know what contaminants are present in the air, water and soil in their environment, as well as in foods. If toxics are present and known to cause health problems, we believe ways to limit and avoid unnecessary exposure to those chemicals should be taken.

Knowledge about these contaminants is the first step to reduce and prevent exposure. ACAT has developed this toolkit to increase the understanding of environmental contaminants found in the North and Arctic regions that affect human health. Designed for Community Health Aides, this toolkit provides facts about contaminants that may be present in the environment, known or suspected symptoms and their human health effects. We offer this information as an additional guide to assessment of a health problem, but we recognize that making a direct link between exposure and disease is often difficult.

**Importance of Traditional Subsistence Foods**

Despite the presence of contaminants in the Arctic environment, maintaining a traditional diet and continuing to engage in the harvest of traditional foods is essential to the physical, cultural, social, and economic well being of Arctic residents. Subsistence foods are extremely health protective and nutrient rich (containing critical nutrients such as folic acid, vitamin C, vitamin D, iron and zinc), high in protein, fat (particularly omega-3 fatty acids) and antioxidants, and low in carbohydrates. In contrast, store-bought, processed foods may have limited nutritional value and possibly also contain harmful chemicals such as pesticides, preservatives, genetically-modified organisms (GMOs), and other substances that may be hazardous to health. Research shows that changes in diet from traditional foods to highly processed foods may have adverse health implications, including an increase in diabetes and heart disease among people of the Arctic, as well as possible declines in mental health and well being.1

**Working Together: Collaboration with Community Health Aides**

In the spirit of Annie Alowa, ACAT seeks to establish continuing communication with Community Health Aides about health concerns related to environmental exposures. Working together will allow us to cover more ground than we could on our own and, ultimately, make a difference. If you would like to join in this collaborative effort to restore health and justice to our people, please contact ACAT by calling (907) 222-7714 or emailing info@akaction.org.

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Introduction

What is Environmental Health?
The World Health Organization defines *environmental health* as follows: “Environmental health addresses all the physical, chemical, and biological factors external to a person, and all the related factors impacting behaviours. It encompasses the assessment and control of those environmental factors that can potentially affect health. It is targeted towards preventing disease and creating health-supportive environments.”

Put simply, where we live and how we live affect our daily and long-term health and add to our physical, mental and emotional well-being. The effects of the environment on human health have been researched and well established. In fact, the World Health Organization estimates that poor environmental quality is responsible for a quarter to a third of all preventable ill health in the world. In many cases, we are unaware that we are being exposed to chemicals that may affect our health. We assume that the air, water and food that we live off of are safe to us, yet a growing amount of evidence shows chemicals in the environment may add to the large increase in negative health effects such as infertility, learning disabilities, cancer, and other diseases. It is becoming more and more clear that toxic chemicals in the environment may have adverse effects on your patients’ health.

Both the physical and social environments affect environmental health. The physical environment is made up of air, water and soil. Exposures that come from the physical environment include chemical, biological and physical agents. The social environment means housing, transportation, urban development, land use, industry, and agriculture. Exposures that are caused by the social environment include work-related stress, injury and violence. Each of us has the right to know which contaminants in the environment may cause health problems. Chemicals used when making plastics, electronics, skin care products, cleaning agents, and building materials, and chemicals from landfills, burn boxes and waste sites, often have not been studied for their human health effects and how dangerous they are on health.

Precautionary Principle
Although we are learning more about the health effects resulting from exposure to toxic chemicals, it is difficult to prove that there is a direct cause between exposure to a contaminant and a disease. People may be exposed differently to contaminants and have varying sensitivities. In our daily lives, we are exposed to many different chemicals, so it is difficult to know how chemical mixtures react together in our bodies. Some health problems from exposure to chemicals do not become known until years after a person was exposed. Although scientists recognize uncertainties about the health effects of chemicals, do not assume that environmental exposures are harmless.

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The precautionary principle guides us to protect health by preventing exposures. A group of scientists developed a statement known as the Wingspread Statement on the Precautionary Principle: “When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically.” People in the health care profession know the phrase “first do no harm”—this is also another way of stating the precautionary principle. The precautionary principle protects people against danger or injury. The precautionary principle means that it is “better to be safe than sorry.”

Contaminants in the North/Arctic Region
Many villages in the North/Arctic are concerned about exposure to environmental contaminants from landfills, burn boxes, mining, formerly used defense sites, sewage, cigarette smoke, and dust. However, exposures can also happen from contact with certain household and work-related chemical use, as well as household products such as toys, plastics, cleaners, and electrical equipment used in most homes.

Why Do We Need to Assess Environmental Exposures?
Exposures to toxic chemicals in the household and environment are very common, and these exposures can cause diseases and disabilities. As a Community Health Aide, you are the first person patients come to for health care. Recognizing, diagnosing, treating, and preventing symptoms of contaminant exposure and making sure there are no other causes of illness are the most important steps in providing the best care to your patients and, most of all, improving the health care of Northern/Arctic residents.

What Is the Environmental Health Care Toolkit?
The materials in this toolkit will give you information on how to recognize and prevent human health problems linked to contaminants in the environment. This toolkit also includes materials you can hand out to your patients so they can learn about ways to protect their health.

ACAT has developed this Environmental Health Care Toolkit specifically for Community Health Aides in the North/Arctic region. The materials in this packet include:
1) An environmental health assessment form that will help you to diagnose your patients’ exposures and understand exposures more;
2) Resources for more information;
3) Fact sheets on environmental contaminants found in the North/Arctic, and symptoms and diseases that have been linked to these exposures;
4) Suggestions for less harmful alternatives and other ways to lower exposure;
5) Map: Villages and Federal Defense Facilities in the Norton Sound Region, Alaska; and
6) A poster with useful information for clinics and homes.

We hope that, together, we can make people more aware of contaminants and human health, benefit patient care, and help improve life in the North/Arctic Region.
Environmental Health History and Assessment

Screening your patient’s environmental health history is an important part of determining a correct diagnosis because the symptoms of environmental exposures are common and may seem like symptoms of a lot of other diseases and disabilities.\(^5,6\)

Below is a list of questions that will help you determine if your patient may be affected by an environmental exposure:\(^7,8,9,10\)

<table>
<thead>
<tr>
<th>Environmental Health History And Assessment Form</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>General Housing</strong></td>
</tr>
<tr>
<td>Where do you live and spend most of your time?</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Indoor Home Environment</strong></td>
</tr>
<tr>
<td>What is the major way that you heat the home or camp where you live now?</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Have you ever smoked tobacco?</strong></td>
</tr>
<tr>
<td>☐ Yes ☐ No ☐ Not sure</td>
</tr>
<tr>
<td>[See Environmental Tobacco Smoke fact sheet]</td>
</tr>
<tr>
<td>For how long?</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Does anyone in your family smoke tobacco?</strong></td>
</tr>
<tr>
<td>☐ Yes ☐ No ☐ Not sure</td>
</tr>
<tr>
<td>[See Environmental Tobacco Smoke fact sheet]</td>
</tr>
<tr>
<td><strong>Do visitors smoke tobacco in your home?</strong></td>
</tr>
<tr>
<td>☐ Yes ☐ No ☐ Not sure</td>
</tr>
<tr>
<td>[See Environmental Tobacco Smoke fact sheet]</td>
</tr>
<tr>
<td><strong>Do you have areas of water damage or mold in your home?</strong></td>
</tr>
<tr>
<td>☐ Yes ☐ No ☐ Not sure</td>
</tr>
<tr>
<td>[See Mold and Mildew fact sheet]</td>
</tr>
<tr>
<td><strong>Was the home you live in now built before 1978?</strong></td>
</tr>
<tr>
<td>☐ Yes ☐ No ☐ Not sure</td>
</tr>
<tr>
<td>[See Lead fact sheet]</td>
</tr>
<tr>
<td>If so, has it been tested for lead paint?</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>If lead paint has been identified in your home, is it flaking?</strong></td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>Environmental Health History And Assessment Form</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Toxic Chemical Exposures</strong></td>
</tr>
<tr>
<td>Do you use pesticides (including mosquito repellents, chemical bug traps, and lice and scabies treatments)?</td>
</tr>
<tr>
<td>Where do you keep household cleaners and other chemicals?</td>
</tr>
<tr>
<td>Please tell me all of the chemicals and toxic materials that you use in your home (Examples: cleaning products, solvents, glue, paint strippers, bleach, bathroom and tile cleaners for mold and mildew, gasoline and other petroleum products, mosquito repellents/bug spray and other pesticides, stain protectors for furniture, lead, etc.)</td>
</tr>
<tr>
<td>Please tell me all of the chemicals and toxic materials that you use at your camp (Examples: gasoline and other petroleum products, mosquito repellents and other pesticides, etc.)</td>
</tr>
<tr>
<td>Please tell me all of the chemicals and toxic materials that you use at work. (Examples: cleaning products, solvents, glue, paint strippers, bleach, gasoline and other petroleum products, varnish and marine paints, mosquito repellents/bug spray and other pesticides, lead, mercury, other metals, welding materials, etc.)</td>
</tr>
<tr>
<td>Is your home near a hazardous waste site (such as a formerly used defense site, current or past mine, etc.), landfill or burn box?</td>
</tr>
<tr>
<td><strong>Food and Water Contamination</strong></td>
</tr>
<tr>
<td>What is the major source of drinking water in the home where you live now?</td>
</tr>
<tr>
<td>What other sources of drinking water do you use, including camping sites?</td>
</tr>
<tr>
<td>Describe your traditional food diet. What kinds of foods do you eat (Examples: greens and berries, walrus, whale, seal, moose, caribou, seal oil, etc.)?</td>
</tr>
<tr>
<td>How much? How often? From where?</td>
</tr>
<tr>
<td>Do you wash fruits and vegetables before eating them or preparing meals?</td>
</tr>
</tbody>
</table>
### Occupations and Hobbies

**Where do you work?**

**Where do other adults in your household work?**

Do any adults in your household work around toxic chemicals? (Examples: glues, solvents, polish, fuels, chemicals associated with mining and oil development, etc.)

- Yes
- No
- Not sure

*See Benzene, Cyanide, Mercury and Gold Mining, Pesticides, Petroleum Products, PBDEs, PCBs, Solvents, and Uranium Mining fact sheets*

- If so, do they shower and change clothes before returning home from work?

- Yes
- No
- Not sure

- Do you or any family member have arts, crafts, ceramics, or similar hobbies?

- Yes
- No
- Not sure

*See Solvents fact sheet*

### Health-Related Questions

**What symptoms do you have that might be related to an environmental exposure?**

- Yes
- No
- Not sure

What do you notice your symptoms the most? What causes the symptoms to start? What causes them to stop?

Are family members, neighbors, and/or co-workers having the same symptoms?

- Yes
- No
- Not sure

Do you have symptoms that happen often like cough, headaches, fatigue, or unexplained pain?

- Cough
- Headaches
- Fatigue
- Unexplained pain

### Follow Up Questions

Are there environmental concerns in your village, job, child’s school, or child care setting?

Can you think of any way you might have been exposed to something harmful?
Resources for More Information

Non-Governmental Organizations (NGOs) (including Nonprofit Organizations, Grassroots Organizations, and Professional Associations)

**Alaska Community Action on Toxics (ACAT)**

**Mission/Background:** Alaska Community Action on Toxics is a statewide organization established in 1997 and dedicated to achieving environmental health and justice. The organization’s mission is: to assure justice by advocating for environmental and community health. We believe that everyone has the right to clean air, clean water, and toxic-free food. ACAT works to eliminate the production and release of harmful chemicals by industry and military sources; ensure community right-to-know; achieve policies based on the precautionary principle; and support the rights and sovereignty of Indigenous peoples. ACAT has four program areas: Military Toxics and Health; Northern Contaminants and Health; Pesticide Right-to-Know; and Water Quality Protection.

**Resources:** The organization provides information on environmental contaminants throughout the state of Alaska. Resources include fact sheets on contaminants, detailed maps of toxic sites, conference call recordings on topics regarding health and the environment, various reports, searchable website database, and other resources upon request.

**Contact Information:**
Alaska Community Action on Toxics
505 West Northern Lights Blvd, Suite 205
Anchorage, AK 99503
Phone: 907-222-7714
Fax: 907-222-7715
Email: info@akaction.net
[www.akaction.org](http://www.akaction.org)

**Alaska Native Science Commission**

**Mission/Background:** Alaska Native Science Commission (ANSC) was established in 1994 for the purpose of creating partnerships between the Native community with science and research sectors. It is a place where proposed research, ongoing and past research, and archives for research about the Native community culminate. The major goals of the commission are to integrate local and traditional knowledge into science and research, influence research priorities, incite Alaska Native involvement in the different levels of science, allow for the community to give and receive feedback on research activities, and ensure that Native people benefit economically from this professional field.

**Resources:** The commission provides information, referral and networking services for researchers who are in need of active partners in the Native community as well as communities who are seeking researchers. This includes an online database for funded research projects within the state of Alaska, as well as newsletters, presentations, and reports made by the commission. They also provide several links to sources about the key issues their organization focuses on.

**Contact Information:**
Alaska Native Science Commission
429 L Street
Association of Occupational and Environmental Clinics
Mission/Background: 55 clinics across the United States and Canada that specialize in occupational and environmental health issues.

Resources: Provides referrals to clinics for medical advice and care, conducts educational activities, and maintains a lending library.

Contact Information:
Association of Occupational and Environmental Clinics
1010 Vermont Ave, NW #513
Washington, DC 20005
Phone: 888-347-2632
Email: aoec@aoec.org
www.aoec.org

Breast Cancer Fund
Mission/Background: The Breast Cancer Fund was founded in 1992 to advocate for and bring attention to the need for eliminating the preventable environmental causes of cancer. This foundation provides public education, advocates for policy change, holds outdoor challenges, and creates other innovative campaigns in order to reach out to the public and ensure that change is made to prevent the proliferation of breast cancer.

Resources: The site provides information about their funds, events, linkages between the environment and cancer, information about breast cancer, and information about environmental health. They create reports, fact sheets, and other materials to educate the public about the linkage between breast cancer and the surrounding environment as well as how to reduce exposures to carcinogenic chemicals. They also follow closely with recent press coverage about the environmental causes of breast cancer.

Contact Information:
Breast Cancer Fund - San Francisco (Main Office):
1388 Sutter Street, Suite 400
San Francisco, CA 94109-5400
Phone: 415-346-8223 or toll-free 866-760-8223
Fax: 415-346-2975
Email: info@breastcancerfund.org
www.breastcancerfund.org
Breast Cancer Fund - Seattle (For Washington/Northwest Inquiries):
P.O. Box 15145
Seattle, WA 98115
Phone: 206-524-4405
Email: pamela@breastcancerfund.org

Canadian Association of Physicians for the Environment (CAPE)
Mission/Background: CAPE is a group of physicians, allied health care practitioners and citizens committed to a healthy and sustainable environment. CAPE aims to educate physicians on environmental issues and provide them with accurate information and a framework for thinking about environmental problems.

Resources: Online resources include links related to health and the environment, documents published by CAPE (articles, briefing papers, brochures, fact sheets, media releases, presentations, statements, and summaries), CAPE newsletters, an archive of letters and articles from CAPE, and information on how to join the mailing list.

Contact Information:
The Canadian Association of Physicians for the Environment
130 Spadina Avenue, Suite 301
Toronto, Ontario Canada M5V 2L4
Phone: 416-306-2273
Fax: 416-960-9392
Email: webmaster@cape.ca
www.cape.ca/

Collaborative on Health and the Environment (CHE)
Mission/Background: The Collaborative on Health and the Environment is a national non-partisan partnership of individuals and organizations concerned with the role of the environment in human and ecosystem health. CHE seeks to raise the level of scientific and public dialogue about the role of environmental contaminants and other environmental factors in many of the common diseases, disorders, and conditions of our time.

Resources: Monthly conference calls on environmental health issues, a searchable database (CHE Toxicant and Disease Database) that summarizes links between chemical contaminants and approximately 180 human diseases or conditions.

Contact Information:
The Collaborative on Health and the Environment
c/o Commonweal
PO Box 316
Bolinas, CA 94924
Email: info@healthandenvironment.org
www.healthandenvironment.org

Collaborative on Health and the Environment – Alaska (CHE-AK)
Mission/Background: The Collaborative on Health and the Environment – Alaska (CHE-AK) formed as a regional group in December 2005 following the Alaska Conference on Health and the Environment. CHE-AK invites
participation from health care professionals, researchers, health-affected and patient groups, students, educators, advocacy organizations, and any individual concerned about protecting the health of current and future generations from environmental harm.

**Resources:** Monthly conference calls on environmental health issues in Alaska

**Contact Information:**
Alaska Community Action on Toxics
505 West Northern Lights Blvd, Suite 205
Anchorage, Alaska 99503
Phone: 907-222-7714
Fax: 907-222-7715
Email: info@akaction.net
http://akaction.org/che.htm

**Environmental Working Group**
**Mission/Background:** Environmental Working Group (EWG) is a non-profit organization that was founded in 1993 by Ken Cook and Richard Wiles. The group seeks to protect public health and the environment by using public information to advocate for policies that shift subsidies and ensure that public health is protected. In 2002, the Environmental Working Group Action Fund was founded in order to advocate on Capitol Hill for these policy changes. Its major goals include seeking the protection for the most vulnerable members of the human population (children, infants, and unborn babies) from being exposed to toxic chemicals, as well as to enact policies that invest in conservation and sustainable development.

**Resources:** The organization conducts its own research to build the knowledge of consumers as well as to strengthen their push toward national policy changes. The major categories of study include health, information on toxics, issues dealing with farming, and natural resources. Their press releases and news articles are also available online.

**Contact Information:**
Environmental Working Group, Headquarters
1436 U St. N.W., Suite 100
Washington, DC 20009
Phone: 202-667-6982
www.ewg.org

Environmental Working Group, California Office
1904 Franklin St. Suite 703
Oakland, CA 94612
Phone: 510-444-0973

**Greater Boston Physicians for Social Responsibility (GBPSR)**
**Mission/Background:** GBPSR is an affiliate of Physicians for Social Responsibility (PSR), a national organization of over 26,000 physicians, health care professionals and supporters. PSR was established in 1961 to address health consequences of weapons of mass destruction. Since then, its mission has been expanded to include health consequences of environmental pollution and degradation, and also the reduction of violence and its causes.
GBPSR has over 1000 members in Eastern Massachusetts who individually and collectively work on various issues relating to health, including quality and access to health care, environmental pollution, militarism and war, community and personal violence, and social justice and human rights.

**Resources:** GBPSR offers online resources on the following projects: Generations at Risk, which addresses the association between reproductive health disorders and toxic chemicals; In Harm’s Way, which focuses on developmental disabilities and toxic chemicals; and the Pediatric Environmental Health Toolkit for health care providers on preventing exposures to toxic chemicals.

**Contact Information:**
Greater Boston Physicians for Social Responsibility
727 Massachusetts Avenue - 2nd Floor
Cambridge, MA 02139
Phone: 617-497-7440
Fax: 617-876-4277
E-Mail: psrmabo@igc.org
[http://www.psr.org/chapters/boston/](http://www.psr.org/chapters/boston/)

**Natural Resources Defense Council**
**Mission/Background:** The Natural Resources Defense Council (NRDC) was founded in 1970 by law students and attorneys who were inspired by the environmental movement. It is an environmental action organization that uses law, science, and member support to advocate for the protection of wildlife, wild places, and safety for the environment and those that live in it. They work to restore the integrity of air, land, and water as well as to ensure that every human has the right to voice their opinions on decisions being made about the environment.

**Resources:** This organization provides press releases, reports, papers, fact sheets, and information about legislation on some of the most pressing and recent environmental issues in the world. They also provide online newsletters and links to other environmental news outlets from across the nation and around the world. NRDC also has a collection of multimedia presentations produced by or for the organization.

**Contact Information:**
Natural Resources Defense Council, Headquarters
40 West 20th Street
New York, NY 10011
Telephone: 212-727-2700
Fax: 212-727-1773
[www.nrdc.org](http://www.nrdc.org)

**Washington Toxics Coalition**
**Mission/Background:** Washington Toxics Coalition (WTC) was founded in 1981 and seeks to protect public health and the environment by attending to a major root of the problem – toxic pollution. The organization promotes the use of alternatives, supports policy change, works to empower communities, and educates people about ways they can create a healthy environment for themselves.

**Resources:** WTC provides access to recent press releases and press clips about toxics and provides ways for the public to educate themselves about buying safer consumer products, creating healthy homes and garden
environments, and promoting sustainable agriculture. They also provide fact sheets about healthy alternatives, their campaigns, and other general information as well as pesticide action kits.

**Contact Information:**
Washington Toxics Coalition  
4649 Sunnyside Avenue N, Suite 540  
Seattle, WA 98103  
Phone: 206-632-1545  
Email: info@watoxics.org  
[www.watoxics.org](http://www.watoxics.org)

**Governmental Agencies**

**Agency for Toxic Substances and Disease Registry (ATSDR)**  
**Mission/Background:** ATSDR is a federal public health agency of the U.S. Department of Health and Human Services based in Atlanta, Georgia. ATSDR uses science, takes responsive public health actions, and provides health information to prevent harmful exposures and diseases related to toxic substances.

**Resources:** ATSDR provides information on toxic substances including ToxFAQs, a series of quick and easy to understand summaries on over 100 toxic substances. They also provide full toxicological profiles on over 250 substances.

**Contact Information:**
Agency for Toxic Substances and Disease Registry  
1825 Century Blvd  
Atlanta, GA 30345  
Phone: 800-232-4636  
E-mail: cdcinfo@cdc.gov  
[www.atsdr.cdc.gov](http://www.atsdr.cdc.gov)

**Alaska Department of Environmental Health**  
**Mission/Background:** The Division of Environmental Health (EH) faces issues concerning safe drinking water, food, and sanitation practices. They delineate standards for business to adhere to in order to promote environmental protection and ensure that food and drinking water is safe for all Alaskans.

**Resources:** The EH gives the public access to current environmental conservation regulations and any changes that occur to those regulations. It also lists public notices, statutes, press releases, and links to other divisions of the Alaska Department of Environmental Conservation.

**Contact Information:**
Alaska Department of Environmental Health  
Phone: 907-269-7644  
Food Safety & Sanitation, Toll Free In-State Phone: 1-87-SAFE-FOOD  
Pesticide Control, Toll Free In-State Phone: 800-478-2577  
[www.dec.state.ak.us/eh](http://www.dec.state.ak.us/eh)
Alaska Department of Environmental Conservation

Mission/Background: The Alaska Department of Environmental Conservation (DEC) seeks to conserve, improve, and protect the natural resources and environment of the state of Alaska.

Resources: The Alaska DEC gives public access to databases that span the whole department as well as its individual divisions. Public notices are also posted on their website along with DEC press releases, public service announcements, environmental headlines about Alaska, department regulations and state statutes pertaining to the department’s responsibilities.

Contact Information:
Alaska Department of Environmental Conservation
www.dec.state.ak.us

Centers for Disease Control and Prevention (CDC)

Mission/Background: The CDC was founded in 1946 and has since been a leader in providing public health information. It works to prevent and control environmental health threats, infectious diseases, injuries, workplace hazards, and disabilities. It is recognized worldwide for conducting its own research and applying its findings to improve the daily lives of people around the world. It works through partnerships in order to maintain a health surveillance system which prevents disease outbreaks, enacts disease prevention strategies, and keep national health statistics up to date. Its four overarching health protection goals to become a better performance-based agency include focusing its efforts on healthy people, places, preparedness, and global health.

Resources: CDC conducts its own research, and the latest CDC news can be read on its website along with a list of major CDC event resources, press releases, and CDC publications. In addition to the research provided by the organization, the website has a list of tools and resources that are useful for the general public, educators, parents, and healthcare professionals.

Contact Information:
Centers for Disease Control and Prevention
1600 Clifton Rd
Atlanta, GA 30333
Phone: 800-311-3435
www.cdc.gov

National Institute of Environmental Health Sciences (NIEHS)

Mission/Background: “The mission of the NIEHS is to reduce the burden of human illness and disability by understanding how the environment influences the development and progression of human disease. To have the greatest impact on preventing disease and improving human health, the NIEHS focuses on basic science, disease-oriented research, global environmental health, and multidisciplinary training for researchers.”

Resources: The NIEHS website provides numerous public reports on results of its research on health conditions related to or affected by environmental exposures, successful public health interventions and policies, environmental health care materials for health care providers, and other resources.

Contact Information:
National Institute of Environmental Health Sciences
Physicians for Social Responsibility (PSR)

Mission/Background: PSR is a public policy organization with over 30,000 members representing the medical and public health professions and concerned citizens, working together for nuclear disarmament, a healthful environment, and an end to gun violence.

Resources: Website provides helpful fact sheets, articles and reports dealing with environment and health.

Contact Information:
1875 Connecticut Avenue, NW, Suite 1012
Washington, DC, 20009
Phone: 202-667-4260
Fax: 202-667-4201
Email: psrnat@psr.org
www.psr.org

Publications and Other Materials (books, websites, online databases, etc.)

Environmental Health Perspectives (EHP)

Resource: Published by the National Institute of Environmental Health Sciences (NIEHS), Environmental Health Perspectives is a monthly journal of peer-reviewed research and news on the impact of the environment on human health. Articles are available online free of charge.

Contact Information:
Environmental Health Perspectives
c/o Brogan & Partners
4011 Westchase Blvd, Suite 150
Raleigh, NC 27607
Phone: 866-541-3841
Fax: 919-828-5935
E-mail: ehponline@niehs.nih.gov
www.ehponline.org

In Harm’s Way: Toxic Threats to Child Development

Resource: The In Harm’s Way report investigates how toxic chemicals contribute to neurological, learning, and behavioral disabilities in children. Toxic exposures are a preventable cause of harm the work along with the complex interactions of genetic, environmental, and social factors that have an influence on child development.

Publishing Information:
Author: Greater Boston Physicians for Social Responsibility (GBPSR)
Is It In Us? Chemical Contamination in Our Bodies
Resource: The *Is It In Us?* project conducted biomonitoring on 35 Americans across the nation, including Alaska, to investigate the levels of contamination in our bodies. This project investigated whether toxic pollution found in consumer products is also found in people.

Contact Information:
Email: moreinfo@isitinusa.org
www.isitinusa.org

Nontoxic, Natural and Earthwise
Resource: A book written by an internationally recognized consumer advocate who identifies safe and environmentally responsible products.

Publishing Information:
Author: Debra Lynn Dadd
Date: September 1990
Publisher: G.P.Putnam’s Sons
ISBN-10: 0-87477-584-1
www.putnam.com/putnam

Our Stolen Future
Mission/Background: A groundbreaking book and now also a website, which report scientific discoveries about endocrine disruption, bringing awareness of how common contaminants may interfere with the natural signals controlling development of the fetus.

Resources: Website tracks recent developments relevant to contaminants and their effects on the endocrine system.

Publishing Information:
Title: *Our Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival?*
Authors: Theo Colborn, Dianne Dumanoski and John Peter Meyers
Date: March 1, 1997
Publisher: Plume
ISBN-10: 0452274141
www.ourstolenfuture.org/aboutOSF.htm

Contact Information:
Email: comments3@ourstolenfuture.org
www.ourstolenfuture.org
**Pediatric Environmental Health, 2nd Edition**

**Resource:** This handbook is an invaluable tool to help health care providers identify, treat and prevent pediatric environmental health hazards.

**Publishing Information:**
Editor Ruth A. Etzel, MD and Associate Editor Sophie J. Balk, MD
Date: 2003
Publisher: American Academy of Pediatrics
ISBN-10: 1581101112

**Skin Deep: Cosmetic Safety Database**

**Resource:** This database on cosmetic safety is provided by the Environmental Working Group. It matches ingredients in more than 25,000 commercially available cosmetic and personal care products with 50 toxicity and regulatory databases, since the FDA does not require companies to test these products for safety. The database is searchable by product, brand, or ingredient of concern, and lists information about toxicity and health effects.

**Contact Information:**
[www.cosmeticsdatabase.com](http://www.cosmeticsdatabase.com)

**Toxics A to Z: A Guide to Everyday Pollution Hazards**

**Resource:** A book written in two parts; part I has a vast amount of information on general issues concerning the hazards of toxics, and part II contains specific information about individual toxics.

**Publishing Information:**
Authors: John Harte, Cheryl Hodren, Richard Schneider, and Christine Shirley
Date: September 9, 1991
Publisher: University of California Press
POLYCHLORINATED BIPHENYLS IN SERUM
OF THE SIBERIAN YUPIK PEOPLE FROM
ST. LAWRENCE ISLAND, ALASKA

David O. Carpenter 1, Anthony P. DeCaprio 1, David O’Hehir 1, Farooq Akhtar 2,
Glenn Johnson 1,2, Ronald J. Scrudato 1, Lucy Apatiki 4, Jane Kava 5, Jesse Gologergen 5,
Pamela K. Miller 6, Lorraine Eckstein 6

1 Institute for Health and the Environment, University at Albany, Rensselaer, New York, USA
2 Department of Biometry and Statistics, School of Public Health, University at Albany, Rensselaer, New York, USA
3 University of Utah, Energy and Geoscience Institute, Dept. Civil and Environmental, Engineering,
University of Utah, Salt Lake City Utah, USA
4 Native Village of Gambell, St. Lawrence Island, Alaska, USA
5 Native Village of Savoonga, St. Lawrence Island, Alaska, USA
6 Alaska Community Action on Toxics, Anchorage, Alaska USA

Received 28 January 2005, Accepted 17 June 2005

ABSTRACT

Objectives. To determine serum levels of polychlorinated biphenyls (PCBs) in Siberian Yupik adults from St. Lawrence Island, Alaska, and to determine the relative contribution of atmospheric transport of PCBs and local contamination to body burdens.

Study Design. Siberian Yupiks of various ages were recruited from three populations: residents of the village of Gambell, residents of the village of Savoonga who did not have family hunting camps near the Northeast Cape (NEC), a Formerly Used Defense Site (FUDS) known to be contaminated with PCBs, and residents of Savoonga whose families had a hunting camp at the NEC.

Methods. Levels of PCBs were measured in serum samples from 130 people, ages 19-76. These Alaska Natives follow a traditional diet high in marine mammals and fish, which bioconcentrate organochlorine compounds that migrate to the Arctic via global air transport and ocean currents.

Results. The lipid-adjusted serum PCB levels of those members of families with hunting camps at the NEC had a mean lipid-adjusted PCB concentration of 1,143 ppb, whereas other residents of Savoonga had values of 847 ppb and residents of Gambell had values of 785 ppb.

Conclusions. Our observations suggest that atmospheric transport of PCBs contributes to levels in the Yupik people, but that the abandoned military site at the NEC may also contribute to the human body burden in those individuals who have either spent substantial time or consumed food from there.

(Int J Circumpolar Health 2005; 64(4):322-335.)

Keywords: Alaska, Eskimos, FUDS, atmospheric transport
INTRODUCTION

Levels of PCBs are declining in most of the developed world since the manufacture and use of these substances has been curtailed (1, 2). This is also true for at least some Northern dwelling communities (3). However, the polar regions are reservoirs for atmospheric transport of persistent toxic substances, which are carried by air currents and then condense out of the vapor phase in the cold of the polar regions and are deposited in snow (4). This fact, plus the high fat diet of many indigenous communities in the Arctic, raises concern that the intake of organochlorine compounds, which include PCBs, dioxins/furans and persistent organochlorine pesticides, may pose serious health risks to these populations (5, 6). The variety of diseases known to be associated with exposure to PCBs include cancer, immune suppression, neurobehavioral decrements, endocrine disruption of sex steroid and thyroid function, cardiovascular disease and diabetes (1, 7).

St. Lawrence Island, Alaska, is an island 104 miles long and averaging 20-30 miles across. It is located 38 miles off the Russian coast, about 150 miles below the Arctic Circle and about 130 miles west of Nome, Alaska. The population of St. Lawrence Island consists of about 1,400 Siberian Yupiks, living in two villages, Gambell and Savoonga. The Siberian Yupik people follow a traditional life-style, which includes significant consumption of marine mammals and fish, bird eggs, as well as local greens and berries. Storage of meats is primarily either by drying in the brief summer, freezing or fermenting in pits in the ground. The villages have electricity and most homes have indoor plumbing.

Two US military bases were constructed on St. Lawrence Island because of its strategic location. The military established a base at Gambell during World War II, and the Air Force and Army came in the early 1950s and operated there until the early 1980’s. Little is known about activities at Gambell, since data regarding activities there is still classified. Gambell is built on coarse gravel, and fuels, oils and other hydrocarbons that spilled have settled in a layer above the permafrost, some 8-10 feet below the surface. There is no documented history of the use of PCBs at Gambell, although some use is likely in generators, transformers and paint.

The other military base was located at the opposite end of the island, at the Northeast Cape (NEC) during the Cold War. While there is no permanent settlement at the NEC, the region has a number of hunting and fishing camps where members of some Savoonga families spend several months in the spring and early summer to hunt seal, walrus and whale, as well as to catch fish from the local rivers and streams and near shore areas and collect greens. When the military base at the
NEC was closed in 1972, most of the supplies and materials present at the site, including buildings, heavy equipment and generating facilities, as well as fuel containers, were abandoned, or buried on-site.

In addition to abandoned buildings, radar stations and air strips, the military left a range of contaminants, including fuels, PCBs and various trace metals, which had a severe impact on the local environment. The military complex established at the NEC is located along the Bering Sea. The soils are typical of tundra-covered areas, with numerous wetlands, ephemeral ponds and small streams draining from the granitic mountains. There was a documented diesel fuel spill of 160,000 gallons in the late 1960s that killed fish and other animals in the Suqitughneq (Suqi) River. The area around the power generating station and the soils and plants down-gradient of the main complex are highly contaminated with PCBs and volatile organics, as determined as a part of the ongoing remediation of the site under the direction of the United States Army Corps of Engineers (USACOE) and their contractors. While a multi-million dollar remediation of the NEC is currently being conducted by the USACOE, significant contamination remains (8).

The perception in the Yupik community is that, in recent years, there has been a significant increase in the levels of various diseases, especially cancer and diabetes, and that this has occurred particularly among those individuals who have spent significant time at the NEC. The concern in the community is that these increases in disease have resulted from environmental contamination originating from the former military sites. One woman, Annie Alowa, whose family had a camp at the NEC and who was dying of cancer, established contact with the Alaska Community Action on Toxics, an advocacy organization based in Anchorage. This contact ultimately resulted in an NIEHS Environmental Justice grant for St. Lawrence Island, which has supported this project.

The residents of St. Lawrence Island have at least two possible sources of exposure to environmental contaminants. Certainly, the abandoned military sites comprise one of these. Although relatively localized, they are in areas of human activity and food collection. In addition, many organochlorines, including PCBs, dioxins/furans and persistent pesticides, travel to and concentrate in the polar regions by vapor phase transport followed by precipitation in the cold climate (global distillation), or by ocean currents (9). These lipophilic compounds then bioconcentrate in the food chain and are ultimately consumed by humans. Native Yupik people, whose diet includes the significant consumption of marine mammal fats, are susceptible to major exposure via this route. In addition, the consumption of polar bear is not uncommon among the Yupik people and, since seal blubber is a major food for the polar bear, PCB levels in polar bear meat, fat and organs are much higher than in the marine mammals.

At the request of the community, we initiated a study of serum levels of PCBs in 2001. We analyzed blood from residents of Gambell, residents of Savoonga who did not have family camps at the NEC, and residents of Savoonga who did have such camps. The hypothesis to be tested was that the results would allow us to distinguish contributions to the PCB body burden of global transport of these compounds from those arising from proximity to the military sites.
MATERIAL AND METHODS

Recruitment of the study population
The study population consisted of 40 residents of Gambell, 44 residents of Savoonga who had spent little or no time at the NEC, and 46 residents of Savoonga whose families had camps at the NEC and spent time there. Figure 1 shows a map of the island, and the relative distances between these sites. For those with family camps at the NEC, the duration of time spent there varied significantly, and not all family members went there every year. However, those who went frequently spent several months at the camp, and returned with food harvested from the vicinity of the NEC. Food in this culture is traditionally shared with the larger family members.

Subjects ages were between 19-75 years, with approximately equal numbers of men and women. Subjects were not randomly selected, but individuals were recruited by Yupik staff from willing participants balanced for age and gender. This should not have resulted in significant bias, given that diets and behaviors are very uniform in this population. There were no exclusion criteria. After signing an informed consent, subjects filled out a brief questionnaire (those who did not speak English were interviewed) documenting residential history, occupational history and time spent at the NEC. Subjects also provided an 8-hr fasting blood sample (two 10-ml specimens, collected in glass specimen tubes). Blood samples were obtained during August 2001, and between August and October in 2003. A few individuals who showed transient PCB congeners in their bloods in 2001 were asked to provide a second sample for analysis in 2003. All results were reported to the person who gave the blood by written communication, and most subjects also had private meetings with Dr. Carpenter during the summer of 2004 to discuss their personal blood results. The study was reviewed and approved by the Institution Review Board of the University at Albany. Detailed dietary information is currently being collected through other funding, but is not available at present.

Serum PCBs were determined as previously described (10). In brief, serum was
separated from clotted cells and extracted three times with diethylether/hexane. Polar lipids and other interferents were removed by adsorption onto Florisil, and 83 individual PCB congeners and 18 congeners co-eluting as pairs or triplets were identified and quantitated by simultaneous parallel dual-column gas chromatography with electron capture detection. Calibration was performed using a 1:1:1:1 mixture of Aroclors 1221, 1016, 1254 and 1260 (AccuStandard, Inc.), with internal standard-based quantitation. Values for individual congeners that were below the method detection limit (MDL) were given a value of zero. Total serum lipid concentration was determined by drying and weighing the residual of a hexane extract. A rigorous QA/QC program was applied as previously described (10).

Statistical procedures
Two-way ANOVA and Multiple Regression were used to analyze the data. For the regression model, the error terms were assumed to be independent, normal random variables with a mean of zero and with constant variance (Homogeneity of Variance). If the model is appropriate for the data at hand, the observed residuals should then reflect the properties assumed for error terms. The appropriateness of linear function for the data was analyzed from residual plots against the predictor variable (age). The residuals fell within a horizontal band around zero, displaying no systematic tendencies to be positive or negative. This indicates that there is no pattern of the residuals when plotted against the fitted values. Whether a linear function is appropriate for the data being analyzed can be studied from a residual plot against the fitted values to test the linear regression assumption that the variance of the error terms is constant. If the error variance increases with the predictor, this will give a megaphone-type plot. In our case, the error variance was constant, and we did not find any megaphone-type structure when we plotted the residuals against the fitted values.

RESULTS
The serum PCB levels of the 130 Yupik adults of St. Lawrence Island, aged from 19 to 75 years, ranged from 0.6 to 17.8 ng/g (ppb) wet weight, with a mean concentration of 4.6 ppb. After lipid adjustment, the mean value was 933 ppb. Table I shows the mean level of total PCBs (wet weight and lipid-adjusted) in serum from female and male residents of Gambell, residents of Savoonga that have family hunting camps near the NEC, and residents of Savoonga who rarely, if ever, go to the NEC. The lowest PCB levels were found in residents of Gambell, where the mean lipid-adjusted PCB level was 785 ppb. In contrast, residents of Savoonga had a mean lipid-adjusted serum PCB concentration of 847 ppb, while residents of Savoonga who had spent time at the NEC had the highest total serum PCB level of 1,143 ppb. In the two-way ANOVA, there is a marginally statistically significant difference between Gambell and NEC in serum PCB values (p < 0.06, SEM = 1.26), but the NEC levels were not significantly different from those in Savoonga (p < 0.16). In all groups, the mean levels were higher for males than females. This result was statistically significant (p < 0.0014, SEM = 2.25).

Figure 2 shows serum PCB level (wet weight) plotted against age for each of the
three Yupik subgroups. Serum levels of PCBs were generally positively correlated with age, while there is obvious variability. This is consistent with previous observations indicating that levels generally increase with age (1), but also may reflect a greater exposure to these very persistent compounds in past years. We have modeled this data by transforming it to a log-normal distribution. When modeled in a multiple regression analysis, we find that, for each one year increase in age, there is a predicted increase in serum PCB levels of 0.47 ppt wet weight after adjusting for all other variables (residence site and sex).

Table 1. Serum PCB levels (wet weight and lipid-adjusted in ppb) for female and male residents of Gambell, NE Cape and Savoonga.

<table>
<thead>
<tr>
<th></th>
<th>Gambell</th>
<th>NE Cape</th>
<th>Savoonga</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Female</td>
<td>Male</td>
<td>All</td>
</tr>
<tr>
<td>n</td>
<td>22</td>
<td>18</td>
<td>40</td>
</tr>
<tr>
<td>Age (mean)</td>
<td>39</td>
<td>47</td>
<td>43</td>
</tr>
<tr>
<td>Wet Weight Total PCB</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>2.642</td>
<td>5.726</td>
<td>4.030</td>
</tr>
<tr>
<td>Min</td>
<td>0.619</td>
<td>1.572</td>
<td>0.619</td>
</tr>
<tr>
<td>Median</td>
<td>2.119</td>
<td>5.841</td>
<td>2.844</td>
</tr>
</tbody>
</table>

| Lipid-Adjusted Total PCB |
| Mean     | 628     | 977     | 785      | 859     | 1343    | 1143     | 794     | 918     | 847      |
| Min      | 124     | 291     | 124      | 295     | 218     | 218      | 166     | 138     | 138      |
| Max      | 2816    | 2632    | 2816     | 2222    | 4963    | 4963     | 1882    | 1845    | 1882     |
| Median   | 370     | 972     | 706      | 814     | 1088    | 878      | 760     | 822     | 773      |

Figure 2. Serum PCB (wet weight) as a function of age for residents of Gambell, Savoonga and those with family camps at the NEC. The data from each individual in the study is plotted, as well as the linear regression best fit for each of the three populations. For Gambell the $R^2 = 0.4696$, for NEC $R^2 = 0.4713$ and for Savoonga $R^2 = 0.5134$. 
Analysis of covariance (ANCOVA) was carried out by adding the term "age" to the model. The Pearson correlation between age and serum PCB levels for females was 0.56 ($p < 0.0001$), while for males it was 0.63 ($p < 0.0001$). For running ANCOVA we tested the assumption that there should be no interaction between the continuous variable “age” at different locations, and we found no significant interaction, consistent with the selection of age ranges to be nearly the same at the three sites.

By use of the main effects model, using age as a continuous variable and location and sex as categorical variables, we found that the overall F statistic for the model is significant ($p < 0.0001$) and the adjusted $R^2$ is 59% for the full model. The means of serum PCB levels in Gambell is marginally significantly different from those at the NEC [$p < 0.06$ (CL = 0.25, 0.16)] with a Variance Inflation Factor (VIF) of 1.3, indicating no multicolinearity between predictors after adjusting for all other variables in the model. As in the two-way ANOVA, there was not a significant difference between the means from Savoonga and those from the NEC. Serum PCBs from males were significantly greater than those from females [$p < 0.0028$ (CL = -0.43, -0.01), VIF = 1.31]. As above, age, as a continuous variable, was significantly related to mean PCB levels [$p < 0.0001$ (CL = 1.21, 1.71), VIF = 1.03].

In the model with two-way interaction terms, the overall F is 20.10, with a p value of 0.0001 and an $R^2$ of 61%. The model explains about 61% of the variation in the data. The other measure of the fit of the model is residual analysis, where assumptions of homogeneity of variance of error terms was not violated. Age was found to be statistically significant [$\beta = 1.53, p < 0.0001$, degree of freedom = 1, SEM = 0.24, (CI = 1.06, 2.10), VIF = 4].

Table II lists each of the 101 PCB congeners that were measured, the MDL for each congener, as well as the average lipid-adjusted level and the number of individuals with levels above the MDL. The table also provides this information among persons in the age ranges 18-34, 35-50 and 51-75 years. Figure 3a shows the median pattern of the major PCB congeners detected in the Yupik samples. The higher chlorinated, persistent PCB congeners 153, 138(+163+164), 118, 170, and 180 dominate. Most of the congeners detected are relatively persistent, although the more labile PCBs 52, 95, 101(+90), and 110 are also present, consistent with recent and/or ongoing exposure. Figure 3(b) shows PCB patterns from three individual Yupik subjects, each of whom has a similar overall pattern, but with a greater contribution from lower chlorinated congeners. Since, in general, the lower chlorinated congeners are more transient, this is evidence of recent exposure. Another striking anomaly (not shown in this Figure, but seen in Figure 4) was the detection of PCB 22 (2,3,4′-triCB) from the NEC at concentrations ranging from 86 to 375 ppt in eight individuals. Four of these subjects had serum levels of this congener >300 ppt, while it was not detected in any of the subjects from the other two locations. This labile congener is not often noted in human samples, but has been previously reported in environmental media, such as soil and air at a PCB contaminated landfill (11, 12). The presence of this congener in serum is also consistent with recent PCB exposure.

Figure 4 shows results obtained from a single individual (a 35-year-old male from
Table II. Presence of individual congeners (levels greater than the MDL) in Yupik bloods [lipid-adjusted, ng/g (ppb)] in the full population and in three age groups. Values for hexachlorobenzene (HCB), DDE and mirex are also presented.

<table>
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<tr>
<th>Structure</th>
<th>IUPAC#</th>
<th>MDL</th>
<th>All age groups</th>
<th>Age 18-34</th>
<th>Age 35-50</th>
<th>Age 51-75</th>
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<tbody>
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<td>130</td>
<td>496</td>
<td>37</td>
<td>57</td>
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</tr>
<tr>
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<td>65</td>
<td>37</td>
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Figure 3. Mean PCB congener pattern in blood from 59 Yupiks aged 35 and over, analyzed in 2001 (A), with data normalized as percentages of total PCBs, and congener patterns from three individuals with elevated levels of transient PCB congeners (B). The mean pattern is very similar to that reported for the average US population (19), but the three individuals shown in B demonstrate the presence of a much higher proportion of lower chlorinated, transient congeners, indicative of recent exposure.

Figure 4. PCB congener pattern in blood taken from one individual (male, age 35, from the NEC group) in 2001 (upper) and from a repeated blood sample in 2003 (lower). Note the decrease in serum levels over time, and the shift in the PCB congener pattern.
the NEC group) who gave two blood samples, one obtained in 2001 and the other in 2003. His wet weight concentration was 5.7 ppb in 2001, but only 2.3 ppb in 2003, in spite of the general tendency for levels to increase with age. There was a decrease of between 34-36% in each of the three most persistent congeners (PCBs 138, 153 and 180) over this period of time. However, his congener profile also changed significantly over this period, with a significant reduction in the percentage of transient PCB congeners. This suggests significant recent exposure before the blood sample was drawn in 2001, followed by significant metabolism of the transient congeners. There was also some reduction in the more persistent congeners over time. This individual was selected for repeat analysis because of his high blood PCB 22 levels in 2001, but it was not present in any significant amount in 2003.

DISCUSSION

While there have been a number of studies of indigenous populations in many of the northern countries, there has been relatively little study in Alaska. The Arctic Monitoring and Assessment Programme (AMAP) was created in 1991 to begin monitoring human health in the Arctic. Hansen (13) reported lipid-adjusted levels of 14 PCB congeners and DDE in maternal plasma of Arctic residents of Canada, Greenland, Sweden, Norway, Iceland and Russia. The PCB values ranged from 167 μg/kg (ppb lipid) in Canada, to 571 ppb in Greenland. The AMAP (14) reported on PCB levels in several different districts of Greenland, finding an average total, lipid-adjusted PCB level of 2,160 ppb. They noted significant variation between different districts, with greater levels on the east than west coast. Thus, the level of PCBs in the Yupiks are within the ranges reported for other Arctic people, although there is considerable variation among the various groups studied.

One of the few reports from Alaska is that of Rubin et al. (15), who studied Alaska Native women in samples collected between 1980 and 1987. The mean total PCB level (sum of 28 congeners) was 7.56 ppb (1,153 ppb lipid-adjusted). They reported total PCB levels to be higher in the northern and southwestern parts of Alaska than in the interior or central south, perhaps reflecting a greater consumption of marine mammals in these regions. In the northwestern part, (which would include St. Lawrence Island, although residents here were not subjects in the study), the mean total PCB level was 9.48 ppb. Our PCB results are somewhat lower than those reported by Rubin et al. (15), perhaps reflecting a decrease in PCB levels over time. However, our results indicate that the Yupik people have significantly larger body burdens of PCBs than the background US and Canadian population.

PCB levels generally increase with age and, in the Yupiks over the age of 30, the mean PCB values were 7.5 ppb wet weight (1,506 ppb lipid-adjusted). The levels of PCBs in individuals from the general US population without unusual exposure was reported by the ATSDR (1) to be 0.9-1.5 ppb (wet weight). On average, fasting human serum contains about 0.6% fat, which means that a total PCB level of 1.5 ppb wet weight is equivalent to 250 ppb lipid. Thus, on average, the Yupik population of St. Lawrence Island shows elevated levels of total PCBs as compared to the general North American background.
Another comparison population, which we studied using identical analytical methods, is a sample of 753 adult Mohawk Native Americans from Akwesasne, located at the New York, Ontario and Quebec junction. While the Mohawks are traditionally fish eaters, the local fish are contaminated with PCBs from releases from aluminum foundries (16). Upon advice from tribal leaders some, but not all, of the Mohawks have reduced their consumption of local fish. We found that the Mohawks (30 years of age and older) had a mean PCB concentration somewhat lower than that of the Yupiks [3.2 ppb wet weight (706 ppb lipid-adjusted)], but also presented a much larger range of values (0.29-48.3 ppb) (17), probably reflecting the greater variability in diet.

Our study is not without limitations. Subjects were not randomly recruited, but were invited to participate by the project staff on the Island based on their age and whether, or not, their families had camps at the NEC. We did not obtain medical histories, nor information on medications, such as lipid-lowering drugs, as this was not a health effects study. We also do not, at present, have detailed dietary histories on the individuals studied. However, we do not believe that the procedures used are likely to have introduced significant bias in providing a profile of serum PCB levels as a function of age and residence.

The results provide some support for the community belief that the FUDS at the NEC contributes to the body burden of contaminants among those individuals who spend significant time at camps near the site. The mean serum PCB levels in those persons whose families have camps at the NEC tended to be higher than those of the other Yupiks, although the overall difference did not quite reach the level of statistical significance. Although ingestion of food is a likely route and source of exposure, it is not clear what is the dominant exposure pathway among these individuals. While detailed dietary information is currently being gathered, it is not available at present. A number of the men were employed at the military site during its operation and during the remediation of the site. Insofar as is known, PCBs were used primarily at the electrical generating facilities and are also known to be associated with the paint used at the sites. It is clear that contamination from metals and polyaromatic hydrocarbons has spread into the drainage basin of the Suqi River and into the Suqi estuary (8), although the extent of this spread is unclear. At present, there is little data on the spread of PCBs. Some fish collected from the Suqi River have shown measurable levels of PCBs (8), but most fish in the river are primarily anadromous, and would not be expected to be highly contaminated. The marine mammals would also not be expected to be contaminated from this site to any great extent, since none of them remain at the site for extended periods. Reindeer graze in the vicinity, but they are highly mobile and it is unlikely that they are the major source of contamination. Other produce from the NEC includes greens and berries. It is possible that contaminated particulates, including significant dust and suspended particles from the recent remedial activities being conducted, could contribute to the exposure if the produce were not carefully washed.

The presence of relatively high levels of transient, lower chlorinated congeners in a subset of Yupiks is an indication of recent exposure (18). From the data in Table II, one can get a good indication of how transient a
congener is by comparing levels in persons of different ages. For the more persistent congeners (PCBs 118, 138, 153, 180) the average levels clearly increase with age, whereas there is little, or no, age-dependence for the more transient congeners. The rather high amounts of PCB 22, a transient congener, in 8 of the 20 people with ties to the NEC is significant, as is the fact that the highest levels of PCB 22 were found in the youngest age group. However, in spite of personal interviews with most of the individuals with this congener, it was not possible to determine a common source of exposure. These individuals with PCB 22 were primarily members of three families. Some, while having spent significant periods of time at the NEC, reported that they had not been there for several years. However, all of them reported that relatives and friends who did go to the NEC brought back foodstuffs which were shared, most commonly salmon berries, greens, or reindeer. Thus, it is not certain that this exposure was tied to the NEC FUDS, even though no one in either of the other two groups showed this transient congener.

There is also a FUDS at the community of Gambell. While there is significant metal and volatile organic contamination at Gambell, there is little evidence for PCBs. The Gambell geology and geography are significantly different from those of the NEC. The FUDS and community of Gambell is situated on a highly permeable cobble spit and any spilled or released contaminants quickly migrate to the underlying permafrost, whereas at the NEC, the relatively impermeable soils retain the contaminants near the surface. It seems likely that the major source of PCBs for the residents of Gambell is global and regional atmospheric transport of these substances, which then bioconcentrate after depositing in the snow, soils and water. The mean PCB concentration of about 4 ppb (785 ng/g lipid) is somewhat lower than the wet weight values reported for Inuits of Greenland (13.3 ppb) (15), probably secondary to the fact that the levels of contaminants in marine mammals in the north Atlantic is greater than that in the north Pacific. The predominant air currents in far Western Alaska come primarily from Russia, Japan and China, whereas the northern Atlantic receives sources from the highly industrialized US, Canada and Western Europe.

The levels of PCBs were non-significantly greater at Savoonga than at Gambell, even though the mean age of the subjects was 2 years higher in Gambell. It is likely that the Yupik peoples tradition of sharing food is a factor here. Those residents with camps at the NEC live principally in Savoonga, and probably shared enough of the foods they brought back to Savoonga to raise the levels of other inhabitants.

In summary, our results suggest that the former military site located at the NEC on St. Lawrence Island may contribute to the PCB exposure of the native residents, but that the predominant source is global transport, deposition and bioconcentration in foodstuffs.

Acknowledgements
This study was supported by NIEHS Environmental Justice Partnerships for Communications, grant #R25 ES 10459 ZES (Ronald J. Scrudato, PI) and the Institute for Health and the Environment at the University at Albany.

All human studies were approved by the University at Albany’s Internal Review Board.
REFERENCES


David O. Carpenter, MD
Institute for Health and the Environment
University at Albany
One University Place, A217
Rensselaer, NY 12144, USA
Telephone: (518) 525-2660
Fax: (518) 525-2665
Email: Carpent@uamail.albany.edu
This retrospective cohort study evaluated adverse birth outcomes in infants whose birth records indicated maternal residence in villages containing dumpsites potentially hazardous to health and environment. Birth records from 1997 to 2001 identified 10,073 eligible infants born to mothers in 197 Alaska Native villages. Outcomes included low or very low birth weight, preterm birth, and intrauterine growth retardation. Infants from mothers in villages with intermediate (odds ratio (OR) = 1.73, 95% confidence interval (CI): 1.06, 2.84) and high (OR = 2.06, 95% CI: 1.28, 3.32) hazard dumpsites had a higher proportion of low birth weight infants than did infants from mothers in the referent category. More infants born to mothers from intermediate (OR = 4.38, 95% CI: 2.20, 8.77) and high (OR = 3.98, 95% CI: 1.93, 8.21) hazard villages suffered from intrauterine growth retardation. On average, infants weighed 36 g less (95% CI: 71.2, 0.8) and 55.4 g less (95% CI: 95.3, 15.6) when born to highly exposed mothers than did infants in the intermediate and low exposure groups, respectively, an effect even larger in births to Alaska Native mothers only. No differences in incidence were detected across exposure levels for other outcomes. This is the first study to evaluate adverse pregnancy outcomes associated with open dumpsites in Alaska Native villages.

Increased risks of adverse birth outcomes have been reported near individual landfill sites and in some multisite studies (1–7). Outcomes such as low birth weight, preterm birth, intrauterine growth retardation (IUGR), and other reproductive outcomes are considered to be sensitive indicators of potential health threats from environmental hazards (7–14). These studies have never been performed in rural Alaska. Historically, Alaska has had one of the lowest low birth weight rates in the United States, but low birth weight rates have increased from 1990 to 1998 (15). Low birth weight rates are still lower than the national average with 3.8 percent of singleton births to Alaska women classified as low and 0.9 percent classified as very low in 2000 (16). A recent study found no differences between Alaska Native and non-Native birth weights (15). Alaska Native women had a slightly higher proportion of preterm births (11.8 percent) than did the overall state proportion of 10.1 percent in 2000 (16).

In 2000, Alaska had 626,932 residents, 119,241 of whom were Alaska Native (17). In this work, Alaska Native includes any people indigenous to the Western Hemisphere: Alaska Native, Native mixed, Aleut, Eskimo, Canadian Eskimo and Indian, and American Indian (18). Many of these Alaska Natives are dispersed throughout federally recognized tribal villages.

Solid waste management is severely deficient in many of these remote villages, comparable to what is found in developing countries (19, 20). Over 95 percent of Alaska residents live in the 119 Native villages surveyed in this study.
Native villages use open dumpsites for solid waste disposal rather than landfills. An open dumpsite is a solid waste site that is not maintained, contains uncovered wastes, and has no boundaries (19). Open dumping can enable water and soil contamination, disease transmission, fire danger, and injury to site salvagers (21). In an attempt to reduce waste volume, dump fires are set, or nonseparated wastes are burned in metal containers in approximately 75 percent of villages, releasing potentially toxic fumes (22). Many Alaska Natives have subsistence diets, and there are concerns about contaminants getting into food and water supplies (23, 24). Many villages lack waste management services and are responsible for disposing of their own wastes, resulting in potential exposures to hazardous wastes and disposal methods. Approximately 45 percent of villages do not have running water to homes, and villagers must haul their own wastewater (25), often discarded at or near open dumps, increasing risks of exposure to pathogens when disposing of trash (25–27).

Negative birth outcomes were selected to evaluate potential environmental hazards posed by these dumpsites. The purpose of the study was to determine if women living in villages with open dumpsites ranked high hazard have a higher incidence of negative birth outcomes than women living in villages with sites that have lower hazard rankings.

MATERIALS AND METHODS

This was a population-based study that utilized a retrospective cohort design for the years 1997–2001. Birth records were obtained from the Alaska Bureau of Vital Statistics for all births to women living in federally recognized Alaska Native villages during 1997–2001. It is estimated that over 97 percent of births are electronically entered (18). Eligible pregnancies were those coded as live singleton births without congenital anomalies, whose mothers’ residences were listed as a Native village. Additionally, the women had to reside in villages for which there existed an evaluation of the hazard potential of the community dumpsite(s).

Negative birth outcomes included low birth weight (from 1,500 to <2,500 g), very low birth weight (<1,500 g), preterm birth (<37 weeks’ gestation), and IUGR (at least 37 weeks’ gestation and <2,500 g (28)). Outcomes were not mutually exclusive, although low birth weight births were excluded from the analyses involving very low birth weight births, and preterm births were excluded from analyses involving IUGR.

Exposure information was the hazard ranking of the dumpsite of the village that was indicated on the birth certificate as the mother’s residence. The Alaska Native Tribal Health Consortium ranks dumpsites on the basis of waste contents, average rainfall, distance to drinking water and domestic water source, site drainage, potential to create leachate at site, accessibility and exposure to the public and vectors, frequency of burning, and degree of public concern over the site (Appendix table 1) (20). Dump scores were categorized into high, intermediate, and low hazard potential to health and environment. During the time period of the study, 159 of the villages’ dumpsites had been scored, with 17 other sites being ranked as either high, intermediate, or low hazard. An additional 21 sites were ranked on the same hazard point factors as above, by use of data from the solid waste management database of the Central Council of Tlingit and Haida Indian Tribes of Alaska.

Covariate information was obtained from birth records and included gender, interpregnancy interval, parity, adequacy of prenatal care, smoking status, alcohol intake, race, mother’s age, and education. More information on the mothers’ villages was obtained from the state of Alaska Community Database (29). This included average family household size and income, percentage of population Alaska Native, percentage of population in poverty, and the land area of the village in square miles (1 square mile = 2.6 km²). Additionally, information was gathered on whether the whole village had piped water, part of the village had piped water, or none of the homes in the village were plumbed. Villages were also categorized into those that were isolated with restricted health-care options, villages with qualified emergency care centers, and regional centers with a qualified acute care facility.

For data analysis, chi-square tests were used to determine if the distribution of covariates was homogenous across exposure groups. Crude odds ratios and 95 percent confidence intervals were calculated for the effect of hazard ranking on each negative birth outcome. Crude odds ratios and 95 percent confidence intervals were calculated for each covariate on each negative birth outcome.

Logistic regression was used to determine adjusted odds ratios and 95 percent confidence intervals to quantify the relations between hazard potential and occurrences of low birth weight, very low birth weight, preterm birth, and IUGR. Because the outcomes of interest were rare, the odds ratios approximate the relative risks (30–32). Information on gender (female vs. male), interpregnancy interval, parity (no previous pregnancies, one or two previous pregnancies, and three or more previous pregnancies), adequacy of prenatal care, smoking status (did not smoke, smoked during pregnancy), alcohol intake (did not drink, drank during pregnancy), race (Caucasian, unknown and other, and Alaska Native), mother’s age (under 20 and over 39 vs. 20–39 years), mother’s education (less than 11 years, 12 years, and more than 12 years), year of birth, village health-care options (restricted, qualified emergency care center, and qualified acute care facility), and village water hookup (all households plumbed, some households plumbed, and no households plumbed) was used in models. Interpregnancy interval was categorized into two pregnancy endpoints in less than 2 years, two endpoints in 2 years or more, and no previous pregnancies. The Kessner Index (33) is a classification scheme of adequacy of prenatal care that accounts for the gestational month when prenatal care began and the number of prenatal visits with respect to length of gestation. The Kessner Index was used to define adequacy of prenatal care into categories of adequate, intermediate, inadequate, and unknown. The race of the baby was designated as the reported race of the mother in accordance with standards from the National Center for Health Statistics. For the
analysis on low birth weight, two different models were constructed. The first model included all records with birth weight information, while the second model adjusted for weeks of gestation.

Analyses of variance were used to compare mean birth weight (grams) and mean gestational length (days) in the three exposure areas by use of the least significant difference for multiple pairwise comparisons. Multivariate analysis of covariance was used to compare means for birth weight and gestation, while simultaneously adjusting for covariates. Birth weight and gestation were the outcome variables, with hazard ranking of the predictor variable and gender, interpregnancy interval, parity, adequacy of prenatal care, smoking status, alcohol intake, race, mother’s age (the mean of each 5-year age class), years of education (used continuously), village health-care options, and village water hookup used as covariates. The mean of years of mother’s education was used to replace missing values, and an additional categorical dummy variable was added to adjust for the missing data on education (34). The same analysis was performed again, restricted to Alaska Native births and substituting the race covariate with the percentage of the village population that was Native, used continuously.

RESULTS

Of the 199 Alaska Native villages that reported births during the study period, there were hazard rankings available for 197. There were 10,073 births to mothers from these villages determined to be eligible.

Chi-square tests revealed that the distributions of gender, alcohol use, and year of birth were roughly equal throughout exposure levels (table 1). The quality of prenatal care \( (p = 0.04) \) and cigarette use \( (p = 0.03) \) were less evenly distributed (table 1). There were disparities \( (p < 0.001) \) in the distributions of interpregnancy interval, parity, race, maternal age, maternal education level, type of health care, and water hookup to households (table 1). Mothers in villages with low hazard-ranked dumpsites tended to have had fewer short interpregnancy intervals and previous pregnancies, were more frequently Caucasian and between 20 and 39 years of age, completed more years of education, more often had access to acute care medical facilities, and were more likely to have households in their villages completely plumbed compared with mothers from villages with intermediate and higher hazard-ranked dumpsites. Other village-level covariates (villages’ size, percentage of population in poverty, and so on) are not displayed because these factors did not add any additional information to the models.

Low birth weight

Among the 10,073 infants born between 1997 and 2001 and included in the analyses, 10,056 had complete birth weight information in their records. A total of 353 (3.5 percent) of these infants were low birth weight (table 2). All factors except gender, year of birth, and type of health care available were associated with a change in risk for low birth weight.

Crude estimates revealed that mothers residing in villages with intermediate hazard rankings were at a mildly increased risk for low birth weight births (odds ratio (OR) = 1.73, 95 percent confidence interval (CI): 0.90, 1.84) compared with mothers residing in villages with low hazard rankings (table 3). Mothers residing in villages with high hazard rankings were 43 percent (95 percent CI: 1.12, 1.81) more likely to have low birth weight babies than were mothers who had low hazard-ranking villages listed on birth records (table 3). Adjusted estimates detected an increased risk for mothers residing in villages with both intermediate hazard rankings (\( OR = 1.73, 95 \) percent CI: 1.06, 2.84) and high hazard rankings (\( OR = 2.06, 95 \) percent CI: 1.28, 3.32) compared with the referent category. When weeks of gestation were added to the model, risks for intermediate hazard and low hazard villages rose to 2.69 (95 percent CI: 1.50, 4.84) and 2.20 (95 percent CI: 1.26, 3.85), respectively.

Very low birth weight

Of the 10,056 records that had complete birth weight information, 9,766 records were used for the very low birth weight analyses. Sixty-three (0.7 percent) of these infants were very low birth weight. Gender, parity, year of birth, and health-care options were not associated with a change in risk for very low birth weight infants (table 2). Interpregnancy interval, quality of prenatal care, cigarette use and alcohol intake, race, maternal education, and water hookup were associated with very low birth weight births.

Crude odds ratios revealed that mothers residing in villages with intermediate \( (OR = 1.28, 95 \) percent CI: 0.57, 2.89) and high \( (OR = 1.49, 95 \) percent CI: 0.87, 2.56) hazard rankings had slight increases in risk for very low birth weight births compared with mothers residing in villages with low hazard rankings (table 3). Adjusted estimates detected no risks for mothers residing in villages with intermediate hazard rankings \( (OR = 0.82, 95 \) percent CI: 0.25, 2.75) and high hazard rankings with a risk estimate of unity \( (OR = 1.02, 95 \) percent CI: 0.33, 3.12) compared with the referent category.

Preterm birth

Of the qualified births, 10,054 records had complete gestational information. A total of 734 (7.0 percent) of these infants were born preterm. All factors except gender and year of birth were associated with a change in risk for preterm birth (table 2).

Crude odds ratios revealed that mothers residing in villages with intermediate hazard rankings were at no appreciably different risk for preterm births \( (OR = 0.89, 95 \) percent CI: 0.68, 1.17) compared with mothers residing in villages with low hazard rankings. Mothers residing in villages with high hazard rankings were 45 percent (95 percent CI: 1.24, 1.70) more likely to give birth prematurely than were mothers from low hazard-ranking villages (table 3). Adjusted estimates indicated slightly reduced risks for preterm birth in mothers from intermediate hazard villages \( (OR = 0.70, 95 \) percent CI: 0.48, 1.01), while mothers from high hazard villages were at no increased risk \( (OR = 1.09, \)
### TABLE 1. The distribution and chi-square p values of individual-level and village-level characteristics across villages with low, intermediate, and high hazard potential dumpsites, Alaska, 1997–2001

<table>
<thead>
<tr>
<th>Covariates</th>
<th>Low hazard dumpsites (n = 4,369)</th>
<th>Intermediate hazard dumpsites (n = 1,247)</th>
<th>High hazard dumpsites (n = 4,457)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td><strong>Individual-level characteristics</strong></td>
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<td></td>
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<tr>
<td>Gender of infant</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>2,299</td>
<td>52.6</td>
<td>644</td>
<td>51.6</td>
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<td>Female</td>
<td>2,070</td>
<td>47.4</td>
<td>603</td>
<td>48.4</td>
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<tr>
<td>Interpregnancy interval</td>
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<td></td>
<td></td>
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<tr>
<td>&gt;2 years</td>
<td>2,714</td>
<td>63.9</td>
<td>781</td>
<td>63.9</td>
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<tr>
<td>≤2 years</td>
<td>413</td>
<td>9.7</td>
<td>170</td>
<td>13.9</td>
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<tr>
<td>No previous pregnancy</td>
<td>1,118</td>
<td>26.3</td>
<td>272</td>
<td>22.2</td>
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<tr>
<td>Parity</td>
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<td></td>
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<tr>
<td>1 or 2 previous pregnancies</td>
<td>1,793</td>
<td>41.2</td>
<td>478</td>
<td>38.4</td>
</tr>
<tr>
<td>0 previous pregnancies</td>
<td>1,117</td>
<td>25.6</td>
<td>272</td>
<td>21.8</td>
</tr>
<tr>
<td>≥3 previous pregnancies</td>
<td>1,445</td>
<td>33.2</td>
<td>496</td>
<td>39.8</td>
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<tr>
<td>Quality of prenatal care</td>
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<td></td>
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<tr>
<td>Adequate</td>
<td>2,166</td>
<td>49.6</td>
<td>612</td>
<td>49.1</td>
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<td>Intermediate</td>
<td>1,438</td>
<td>32.9</td>
<td>417</td>
<td>33.4</td>
</tr>
<tr>
<td>Inadequate</td>
<td>619</td>
<td>14.2</td>
<td>175</td>
<td>14</td>
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<tr>
<td>Unknown</td>
<td>146</td>
<td>3.3</td>
<td>43</td>
<td>3.4</td>
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<tr>
<td>Cigarette use during pregnancy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Did not report smoking</td>
<td>3,143</td>
<td>72.3</td>
<td>877</td>
<td>70.7</td>
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<tr>
<td>Reported smoking</td>
<td>1,203</td>
<td>27.7</td>
<td>363</td>
<td>29.3</td>
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<td>Alcohol use during pregnancy</td>
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<td></td>
</tr>
<tr>
<td>Did not report drinking</td>
<td>4,082</td>
<td>94.1</td>
<td>1,170</td>
<td>94.3</td>
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<tr>
<td>Reported drinking</td>
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<td>5.9</td>
<td>71</td>
<td>5.7</td>
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<tr>
<td>Race</td>
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<tr>
<td>Caucasian</td>
<td>2,002</td>
<td>45.8</td>
<td>265</td>
<td>21.3</td>
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<tr>
<td>Not reported or other</td>
<td>216</td>
<td>4.9</td>
<td>15</td>
<td>1.2</td>
</tr>
<tr>
<td>Alaska Native</td>
<td>2,151</td>
<td>49.2</td>
<td>967</td>
<td>77.5</td>
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<tr>
<td>Maternal age</td>
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<tr>
<td>20–39 years</td>
<td>3,687</td>
<td>84.4</td>
<td>1,016</td>
<td>81.5</td>
</tr>
<tr>
<td>&lt;20 years or ≥40 years</td>
<td>682</td>
<td>15.6</td>
<td>231</td>
<td>18.5</td>
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<tr>
<td>Maternal education</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;12 years</td>
<td>1,554</td>
<td>36.4</td>
<td>210</td>
<td>17.5</td>
</tr>
<tr>
<td>12 years</td>
<td>1,933</td>
<td>45.3</td>
<td>693</td>
<td>57.6</td>
</tr>
<tr>
<td>&lt;12 years</td>
<td>778</td>
<td>18.2</td>
<td>300</td>
<td>24.9</td>
</tr>
<tr>
<td>Year of birth</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1997</td>
<td>866</td>
<td>19.8</td>
<td>251</td>
<td>20.1</td>
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<td>1998</td>
<td>874</td>
<td>20</td>
<td>266</td>
<td>21.3</td>
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<td>1999</td>
<td>902</td>
<td>20.6</td>
<td>226</td>
<td>18.1</td>
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<tr>
<td>2000</td>
<td>876</td>
<td>20.1</td>
<td>240</td>
<td>19.2</td>
</tr>
<tr>
<td>2001</td>
<td>851</td>
<td>19.5</td>
<td>264</td>
<td>21.2</td>
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<td>Village-level characteristics</td>
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<tr>
<td>Available health care in village</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Qualified acute care facility</td>
<td>3,957</td>
<td>90.6</td>
<td>239</td>
<td>19.2</td>
</tr>
<tr>
<td>Qualified emergency care center</td>
<td>210</td>
<td>4.8</td>
<td>188</td>
<td>15.1</td>
</tr>
<tr>
<td>Restricted health care options</td>
<td>202</td>
<td>4.6</td>
<td>820</td>
<td>65.8</td>
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<tr>
<td>Piped water to households in village</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>All households plumbed</td>
<td>3,586</td>
<td>82.1</td>
<td>160</td>
<td>12.8</td>
</tr>
<tr>
<td>Some households plumbed</td>
<td>748</td>
<td>17.1</td>
<td>726</td>
<td>58.2</td>
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<tr>
<td>No households plumbed</td>
<td>35</td>
<td>0.8</td>
<td>361</td>
<td>28.9</td>
</tr>
</tbody>
</table>
TABLE 2. The distribution and risks associated with individual-level and village-level characteristics and outcomes of low and very
low birth weight, preterm birth, and intrauterine growth retardation, Alaska, 1997–2001†

<table>
<thead>
<tr>
<th>Covariates</th>
<th>Low birth weight (cases = 353)</th>
<th>Very low birth weight (cases = 63)</th>
<th>Preterm birth (cases = 734)</th>
<th>Intrauterine growth retardation (cases = 98)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Odds ratio [95% confidence interval]</td>
<td>Odds ratio [95% confidence interval]</td>
<td>Odds ratio [95% confidence interval]</td>
<td>Odds ratio [95% confidence interval]</td>
</tr>
<tr>
<td>Gender of infant</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Female</td>
<td>1.19 [0.96, 1.47]</td>
<td>1.13 [0.69, 1.85]</td>
<td>0.89 [0.76, 1.03]</td>
<td>0.67* [0.44, 0.99]</td>
</tr>
<tr>
<td>Interpregnancy interval</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;2 years</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>≤2 years</td>
<td>1.39* [1.03, 1.86]</td>
<td>2.10** [1.59, 2.58]</td>
<td>1.30** [1.07, 1.65]</td>
<td>0.8 [0.40, 1.62]</td>
</tr>
<tr>
<td>No previous pregnancy</td>
<td>1.24 [0.98, 1.57]</td>
<td>1.03 [0.59, 1.79]</td>
<td>0.99 [0.83, 1.19]</td>
<td>1.2 [0.76, 1.90]</td>
</tr>
<tr>
<td>Parity</td>
<td></td>
<td></td>
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<td>1 or 2 previous pregnancies</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>0 previous pregnancies</td>
<td>1.32* [1.01, 1.72]</td>
<td>1.03 [0.57, 1.86]</td>
<td>1.08 [0.89, 1.32]</td>
<td>1.13 [0.69, 1.86]</td>
</tr>
<tr>
<td>≥3 previous pregnancies</td>
<td>1.31* [1.03, 1.66]</td>
<td>1.4 [0.80, 2.45]</td>
<td>1.29** [1.08, 1.53]</td>
<td>1.26 [0.76, 1.90]</td>
</tr>
<tr>
<td>Quality of prenatal care</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adequate</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Intermediate</td>
<td>1.42** [1.11, 1.82]</td>
<td>0.91 [0.48, 1.73]</td>
<td>1.25** [1.05, 1.49]</td>
<td>1.55 [0.99, 2.44]</td>
</tr>
<tr>
<td>Inadequate</td>
<td>2.12** [1.60, 2.82]</td>
<td>2.66** [1.45, 4.87]</td>
<td>1.79*** [1.46, 2.19]</td>
<td>1.65 [0.93, 2.95]</td>
</tr>
<tr>
<td>Unknown</td>
<td>2.44*** [1.48, 4.00]</td>
<td>3.42** [1.29, 9.02]</td>
<td>1.83** [1.25, 2.67]</td>
<td>2.37 [0.92, 6.08]</td>
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<tr>
<td>Cigarette use during pregnancy</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Did not report smoking</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Reported smoking</td>
<td>1.79*** [1.45, 2.20]</td>
<td>2.27*** [1.46, 3.09]</td>
<td>1.60*** [1.38, 1.86]</td>
<td>1.22* [1.04, 1.43]</td>
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<tr>
<td>Alcohol use during pregnancy</td>
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<tr>
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<td>1</td>
<td>1</td>
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<tr>
<td>Reported drinking</td>
<td>2.66*** [1.98, 3.67]</td>
<td>4.01*** [2.30, 6.99]</td>
<td>2.00*** [1.58, 2.53]</td>
<td>1.12 [0.93, 1.34]</td>
</tr>
<tr>
<td>Race</td>
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<td></td>
</tr>
<tr>
<td>Caucasian</td>
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</tr>
<tr>
<td>Not reported or other</td>
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<td>1.19 [0.15, 9.54]</td>
<td>1.5 [0.90, 2.51]</td>
<td>1.43 [0.50, 4.11]</td>
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<tr>
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<td>1.66*** [1.26, 2.19]</td>
<td>2.54** [1.21, 5.34]</td>
<td>2.02*** [1.64, 2.48]</td>
<td>0.96 [0.61, 1.50]</td>
</tr>
<tr>
<td>Maternal age</td>
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<td>20–39 years</td>
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<tr>
<td>&lt;20 years or ≥40 years</td>
<td>1.68*** [1.32, 2.15]</td>
<td>1.49 [0.83, 2.67]</td>
<td>1.24* [1.03, 1.50]</td>
<td>1.47 [0.92, 2.35]</td>
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<tr>
<td>Maternal education</td>
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<td>&gt;12 years</td>
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<tr>
<td>12 years</td>
<td>1.77*** [1.28, 2.38]</td>
<td>2.50* [1.12, 5.60]</td>
<td>1.43*** [1.16, 1.76]</td>
<td>1.36 [0.78, 2.36]</td>
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<tr>
<td>&lt;12 years</td>
<td>2.20*** [1.58, 3.07]</td>
<td>3.15*** [1.39, 7.13]</td>
<td>1.93*** [1.54, 2.41]</td>
<td>1.83 [0.99, 3.41]</td>
</tr>
<tr>
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<td>1</td>
<td>1</td>
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</tr>
<tr>
<td>1998</td>
<td>0.94 [0.67, 1.32]</td>
<td>0.69 [0.32, 1.50]</td>
<td>0.96 [0.76, 1.22]</td>
<td>1.14 [0.58, 2.23]</td>
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<tr>
<td>1999</td>
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<td>0.51 [0.22, 1.19]</td>
<td>0.88 [0.69, 1.12]</td>
<td>1.28 [0.65, 2.44]</td>
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<tr>
<td>2000</td>
<td>1.03 [0.74, 1.43]</td>
<td>0.65 [0.30, 1.44]</td>
<td>1.02 [0.81, 1.30]</td>
<td>1.84 [0.99, 3.41]</td>
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<tr>
<td>2001</td>
<td>1.09 [0.78, 1.51]</td>
<td>1.19 [0.60, 2.34]</td>
<td>1.12 [0.89, 1.41]</td>
<td>1.07 [0.53, 2.14]</td>
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<td>Available health care in village</td>
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<td>1.07 [0.45, 2.52]</td>
<td>1.14 [0.90, 1.46]</td>
<td>0.76 [0.40, 1.44]</td>
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<td>1.47*** [1.25, 1.73]</td>
<td>0.86 [0.56, 1.31]</td>
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<td>Piped water to households in village</td>
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<td></td>
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<tr>
<td>All households plumbed</td>
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<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Some households plumbed</td>
<td>1.35* [1.06, 1.72]</td>
<td>2.13** [1.21, 3.75]</td>
<td>1.27** [1.07, 1.51]</td>
<td>0.92 [0.58, 1.50]</td>
</tr>
<tr>
<td>No households plumbed</td>
<td>1.32* [1.00, 1.74]</td>
<td>1.47 [0.73, 2.93]</td>
<td>1.41*** [1.17, 1.71]</td>
<td>1.18 [0.71, 2.00]</td>
</tr>
</tbody>
</table>

* p < 0.05; **p < 0.01; ***p < 0.001.
† The number of records for each analysis varied from 9,766 to 10,056.
95 percent CI: 0.78, 1.51) compared with the referent category.

### Intrauterine growth retardation

Of the qualified full-term births, 9,221 records had complete birth weight information. Ninety-eight (1.1 percent) of these infants were born with IUGR (table 2). Only male gender and smoking were significantly associated with IUGR, although quality of prenatal care, alcohol use, maternal age, maternal education of less than 12 years, and birth year in 2000 also indicated IUGR.

Crude odds ratios revealed that mothers residing in villages with intermediate hazard rankings were at an 80 percent increased risk for IUGR (OR = 1.78, 95 percent CI: 1.01, 2.84) compared with mothers residing in villages with low hazard rankings. Mothers residing in villages with high hazard rankings had no appreciable difference in risk for IUGR (OR = 1.15, 95 percent CI: 0.73, 1.80) compared with mothers who had low hazard-ranking villages listed on birth records (table 3). Adjusted estimates detected a fourfold increase in risk for mothers residing in villages with intermediate hazard rankings (OR = 3.99, 95 percent CI: 1.95, 8.15) and a slightly lower estimate in the villages with high hazard rankings (OR = 3.68, 95 percent CI: 1.72, 7.87) compared with the referent category.

### Average birth weight and gestational length

The average birth weight was 3,569.9 g for infants born to mothers from low hazard villages, 3,565.5 g for those from intermediate hazard villages, and 3,542.1 g for those from high hazard areas (table 4). Infants in the high hazard villages weighed, on average, 27.8 g less than did infants in the low hazard villages (95 percent CI: 1.7, −0.01 g). The average gestational length (days) is displayed in table 4 for each hazard category. The gestational length was 273.4 days for pregnancies in mothers from low hazard villages, 272.5 days for those from intermediate hazard villages, and 270.9 days for those from high hazard villages. Mothers from the intermediate hazard villages had pregnancies that, on average, lasted 0.9 days less than did mothers from the low hazard villages (95 percent CI: −3.0, −1.9 days) and 1.6 days less than did
The multivariate analysis revealed adjusted mean birth weights of 3,584.9 g, 3,565.5 g, and 3,529.5 g for births to mothers from low, intermediate, and high hazard villages, respectively (table 4). Infants born to mothers from high hazard villages weighed, on average, 36.0 g less than did infants whose mothers were from intermediate hazard villages (95 percent CI: 71.2, 0.8 g) and 55.4 g less than did those from low hazard villages (95 percent CI: 95.3, 15.6 g). The adjusted mean gestational length was 272.7 days in low hazard villages, 272.8 days in intermediate hazard villages, and 271.6 days in high hazard villages. Mothers in the high hazard villages had pregnancies that, on average, lasted 1.2 days less than did mothers in intermediate hazard villages (95 percent CI: 2.0, 0.3 days). Mothers in the

<p>| TABLE 4. Crude and adjusted average birth weights and gestational lengths, as well as mean differences, across low, intermediate, and high hazard exposure categories, and 95% confidence intervals, Alaska, 1997–2001 |
|---------------------------------------------|----------------|-----------------|-----------------|-----------------|-----------------|</p>
<table>
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<tr>
<th>Model</th>
<th>No.</th>
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<th>Contrast</th>
<th>Mean difference</th>
<th>95% confidence interval</th>
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<td>Weight (g)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td>−23.4</td>
<td>−59.0, 12.1</td>
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<td>3,542.10</td>
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<td>−1.6***</td>
<td>−2.4, −0.8</td>
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<td>270.9</td>
<td>High-low</td>
<td>−2.5***</td>
<td>−3.0, −1.9</td>
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<td>Weight (adjusted†) (g)</td>
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<td>3,584.90</td>
<td>Intermediate-low</td>
<td>−19.4</td>
<td>−64.4, 25.6</td>
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<td>1,245</td>
<td>3,565.50</td>
<td>High-intermediate</td>
<td>−36.0*</td>
<td>−71.2, −0.8</td>
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<td>−55.4**</td>
<td>−95.3, −15.6</td>
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<tr>
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<td>Intermediate-low</td>
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<td>High-low</td>
<td>−1.0*</td>
<td>−2.0, −0.1</td>
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<td>−97.9, −13.3</td>
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<tr>
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<td>Weight (adjusted‡) (g)</td>
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<tr>
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<td>Intermediate-low</td>
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<td>269.8</td>
<td>High-low</td>
<td>−1.9*</td>
<td>−3.7, −0.1</td>
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* p < 0.05; ** p < 0.01; *** p < 0.001.  
† Adjusted for gender, interpregnancy interval, parity, adequacy of prenatal care, smoking status, alcohol intake, race, mother’s age and education, health care options, piped water, and missing values.  
‡ Adjusted for gender, interpregnancy interval, parity, adequacy of prenatal care, smoking status, alcohol intake, percentage of village population that is Alaska Native, mother’s age and education, health care options, piped water, and missing values.
high hazard villages also had pregnancies lasting, on average, 1.0 day less than did mothers in the low hazard villages (95 percent CI: −2.0, −0.1).

**Average birth weight and gestational length among Alaska Native women only**

There were 7,147 Alaska Native women in this study. The average birth weights of infants born to Alaska Native mothers were 3,587.6 g, 3,597.5 g, and 3,545.6 g for mothers from low, intermediate, and high hazard villages, respectively (table 4). Infants in the high hazard villages weighed, on average, 82.1 g (95 percent CI: −142.4, −21.8 g) less than did infants in the low hazard villages and 55.6 g (95 percent CI: −142.4, −21.8 g) less than did infants born to mothers from intermediate hazard villages. The gestational length was 271.8 days for pregnancies in mothers from low hazard villages, 271.5 days for those from intermediate hazard villages, and 269.8 days for those from high hazard villages. Mothers from high hazard villages had pregnancies lasting, on average, 1.9 days less than those from low hazard villages (95 percent CI: −3.8, −0.1 days) and 1.8 days less than did mothers from intermediate hazard villages (95 percent CI: −2.2, −0.9 days).

The multivariate analysis revealed the adjusted mean birth weights as 3,616.6 g, 3,587.6 g, and 3,539.4 g for births to mothers from low, intermediate, and high hazard villages, respectively (table 4). Infants born to mothers from high hazard villages weighed, on average, 48.4 g less than did infants whose mothers were from intermediate hazard villages (95 percent CI: −90.8, −5.9 g) and 77.3 g less than did those from low hazard villages (95 percent CI: −138.1, −16.6 g). The adjusted mean gestational lengths were 271.8 days in low hazard villages, 271.5 days in intermediate hazard villages, and 269.8 days in high hazard villages. Mothers in the high hazard villages had pregnancies that, on average, lasted 1.9 days less than did mothers in intermediate hazard villages (95 percent CI: −3.7, −0.1). Mothers in the high hazard villages also had pregnancies lasting, on average, 1.7 days less than did mothers in the low hazard villages (95 percent CI: −3.0, −0.4).

**DISCUSSION**

This work detected a meaningful increase in risk of low birth weight births to mothers who resided in villages with intermediate and high hazard dumpsites compared with villages with low hazard sites. There was also an increase in risk estimates with a higher level of exposure. Although this apparent dose response disappeared when adjustment was made for gestation, the estimates of risk increased. The risk estimates were higher in the exposed group than in another similarly sized study (4).

There was also a meaningful birth weight reduction detected when comparison was made of average birth weights in infants born to mothers from high, intermediate, and low hazard villages. There was evidence of dose-response grouping of birth weights with respect to exposure level. These reductions are similar to those found in a comparable study in California (35). When restricting the analyses of births to Alaska Native women, we found that the reductions in mean birth weight were greater, although the infants weighed slightly more. However, even the difference in weight between low and high hazard villages would be clinically significant only for the smallest infants, and it was approximately one third of the 200-g reduction in weight predicted by smoking during pregnancy (36).

No dumpsite hazard effects were detected for very low birth weight babies. Table 3 shows that the covariates associated with low birth weight were the same as those associated with very low birth weight, but the effect estimates were higher in the very low birth weight group. Very low birth weight infants comprised less than 1 percent of the study population, so any differences across exposure levels may have been difficult to detect. Additionally, no information was available on other potentially confounding variables, such as drug use and maternal health status. Some risk factors, such as structural abnormalities of reproductive organs, may affect the incidence of very low birth weight infants more than that of moderately low birth weight infants (37).

This study also detected no excess risk of preterm births. In fact, births to mothers in intermediate hazard villages bordered on being protected from preterm birth. Interestingly, the 7 percent preterm birth rate was lower than that reported in Alaska. Other studies detecting decreases in low birth weight across environmental exposure levels have not found differences with respect to very low birth weight or preterm births (4, 35). The multivariate analysis of the covariance predicted an approximate 1-day difference in mean gestational length when comparison was made of births to mothers residing in high hazard villages with those to mothers residing in low hazard villages and 2 days when examining only births to Alaska Native women. This would be clinically significant only in the most premature of infants and apparently was not enough of a reduction to have an effect on the incidence of preterm births.

Information about gestation was obtained from the calculated gestation entered on the birth record. This variable is based on the mothers’ estimate of the date of last menses and could be subject to reporting errors. Gestation was estimated in weeks but converted to days for analyses, so a reduction of 1 day should be interpreted cautiously. Risk factors for preterm birth, such as urogenital infections and placental abnormalities, were not adjusted for in the analyses and could differ across strata as the study population was not homogenous.

Women from intermediate and high hazard villages were more likely to have babies afflicted with IUGR than were women from low hazard villages. Effect estimates were slightly higher in babies from intermediate hazard villages than in those from high hazard villages. This is similar to the decreases in length of gestation in the intermediate and high hazard villages compared with low hazard villages and the effects observed when adjustment was made for gestation in the low birth weight models. These results contrast with the dose response observed when we examined low birth weight and birth weight reduction. Possible explanations include the different risk factors for low birth weight and preterm birth.
Perhaps these inconsistencies could be reconciled with a more specific definition of exposure. The assumption behind this study was that women living in villages with intermediate or high hazard dumpsites were all exposed to these sites via proximity and waste disposal throughout their pregnancies, although this is impossible to ascertain with the current study design. Dumpsites were ranked in generally the same time period as the study period, but the quality of the dumpsites could have been labile. It is not known what proportion of their pregnancies women spent in their villages. Women living in isolated villages in Alaska typically spend the last 3–6 weeks in prematernal homes in regional centers that have access to hospitals. Approximately 70 percent of the mothers who lived in high hazard villages would have left those villages for prematernal homes during the last portion of their pregnancies. These prematernal homes are located in villages that generally have lower hazard scores. Birth weight reductions are often associated with factors that occur later in pregnancy (7, 38), although other research demonstrates that first trimester exposures can also affect birth weight (39).

Covariates were distributed differently throughout exposure levels. Hazard categories could have been surrogates for other factors affecting birth weight. For example, important risk factors such as underlying health conditions and occupational exposures were not adjusted for in the analyses. Another concern is that studies performed in other states have found that birth record information often does not correlate with information on patients’ medical records (40, 41).

Future studies examining the potential health effects associated with open dumpsites in Alaska Native villages should include measurements that are more precise in nature. Misclassification errors are inherent in studies with crude exposure measurements. Several contaminants identified in and proximal to individual dumpsites (arsenic, lead, methyl mercury, and several petroleum hydrocarbons) are associated with negative birth outcomes (7, 42, 43). During examination of the health effects associated with environmental exposures from hazardous waste sites, it is always preferable, although rarely possible, to identify direct pathways of exposure. Other birth outcome studies with well-defined exposure routes have found more convincing associations (3, 5). Reproductive outcomes can be sensitive indicators of environmental insults, as the reproductive system often fails before other systems (7, 11, 44). The most important aspect of this study is that it is the first to attempt to characterize the relation of these adverse pregnancy outcomes among residents of Alaska Native villages to open dumpsites.

ACKNOWLEDGMENTS

The authors would like to acknowledge Phillip Mitchell of the Alaska Bureau of Vital Statistics for providing birth record data, the Central Council of Tlingit and Haida Indian Tribes of Alaska and the Alaska Native Tribal Health Consortium for exposure information, and Zender Environmental Science and Planning Services.

Conflict of interest: none declared.

REFERENCES

TABLE 1. Guidelines from the Indian Health Service for classifying the possible threat to health and the environment posed by the solid waste site*

<table>
<thead>
<tr>
<th>Hazard point factors</th>
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<td></td>
<td>201–600 feet (4 points)</td>
<td></td>
</tr>
<tr>
<td><strong>Site drainage</strong></td>
<td>Site drainage increases the likelihood of ground or surface water contamination (8 points)</td>
<td>Effects of moderate drainage, limited ponding, and drainage are largely neutral (2 points)</td>
<td>Site drainage contributes to protection of ground or surface water</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Potential to create leachate at site</td>
<td>High probability (4 points)</td>
<td>Moderate probability (2 points)</td>
<td>Low probability</td>
</tr>
</tbody>
</table>

*Table continues*
APPENDIX TABLE 1. Continued

<table>
<thead>
<tr>
<th>Hazard point factors</th>
<th>High factors</th>
<th>Moderate factors</th>
<th>Low factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distance to domestic water source</td>
<td>&lt;1,000 feet (4 points)</td>
<td>1,000–5,000 feet (2 points)</td>
<td>&gt;5,000 feet</td>
</tr>
<tr>
<td>Site accessibility</td>
<td>Unrestricted access with residences nearby (&lt;1 mile†) (4 points)</td>
<td>Unrestricted access but remote from population (2 points)</td>
<td>Restricted, controlled access</td>
</tr>
<tr>
<td>Frequency of burning</td>
<td>Frequent burning (weekly) (4 points)</td>
<td>Infrequent burning (monthly) (2 points)</td>
<td>Burning never occurs</td>
</tr>
<tr>
<td>Site materials’ exposure to public and vectors</td>
<td>Surface materials, no cover, scavenging by public (4 points)</td>
<td>Materials in open trenches, limited scavenging (2 points)</td>
<td>Materials covered, no scavenging</td>
</tr>
<tr>
<td>Degree of public concern over site aesthetics</td>
<td>Frequent expressions of public concern over site (4 points)</td>
<td>Little public concern, government awareness only (2 points)</td>
<td>No concern expressed by any entity</td>
</tr>
<tr>
<td>Hazard score</td>
<td>Points total 30 or more (high)</td>
<td>Points total 14–29 (intermediate)</td>
<td>Points total 13 or less (low)</td>
</tr>
</tbody>
</table>

† Metric equivalents: 1 inch = 2.54 cm; 1 foot = 30.48 cm; 1 mile = 1.61 km.
FETAL AND NEONATAL DEATHS AND CONGENITAL ANOMALIES ASSOCIATED WITH OPEN DUMPSITES IN ALASKA NATIVE VILLAGES

Susan Gilbreath, Philip H. Kass

Department of Population Health and Reproduction, University of California, Davis, USA

Received 15 November 2005; Accepted 8 February 2006

ABSTRACT

Objective. To determine if women living in Alaska Native villages with open dumpsites ranked as higher hazard have higher rates of adverse pregnancy outcomes than women living in villages with sites that have lower hazard rankings. Adverse pregnancy outcomes examined included fetal and neonatal death and congenital anomalies.

Study Design. A population-based retrospective cohort study.

Methods. Birth records from 1997-2001 were used to identify the 10 360 eligible infants born to mothers who resided in 197 Alaska Native villages with dumpsite rankings. Exposure variables were derived from hazard rankings of dumpsites. Covariates were obtained from both birth certificate information and village-specific characteristics.

Results. Neither crude, nor adjusted estimates detected a statistically significant difference in rates between exposure levels, although adjusted estimates were positive in all congenital anomaly categories, except gastrointestinal defects. Infants born to mothers residing in villages with high hazard dumpsite contents were more likely (RR=4.27; 95% CI: 1.76, 10.36) to have anomalies classified as other defects. Other hazard factors were not significant predictors for any of the adverse outcomes examined.

Conclusions. This is the first study to evaluate fetal and neonatal deaths and congenital anomalies associated with open dumpsites in Alaska Native villages. Problems with the study include a population-based exposure measurement, small sample size, and biases related to birth record information. Future studies should include more comprehensive registries of congenital anomalies.

(Inf J Circumpolar Health 2006;65(2):133-147)

Keywords: Alaska Native, dumpsites, hazardous waste, neonatal death, stillbirth, congenital anomalies
INTRODUCTION

Alaska has diverse cultures, severe temperatures, vast landscapes, and sparse local populations, which can affect public health. The U.S. Census 2000 reported that Alaska had 626,932 residents, 119,241 of whom were Alaska Native (AN). In this work, AN includes any people indigenous to the Western Hemisphere: Alaska Native, Native mixed, Aleut, Eskimo, Canadian Eskimo and Indian, and American Indian. Many of these ANs are dispersed throughout federally recognized tribal villages. Solid waste management (SWM) is severely deficient in many of these remote villages, comparable to what is found in developing countries (1,2). Over 95 percent of AN villages use open dumpsites for solid waste disposal. An open dumpsite is a solid waste site that is not maintained, is unlined, contains uncovered wastes, and with generally no marked perimeter and open access (1).

The Alaska Native Tribal Health Consortium (ANTHC) ranks dumpsites based on hazard point factors that include: dumpsite waste contents, average rainfall, distance to drinking water aquifer and domestic water source, site drainage, potential to create leachate at site, accessibility and exposure to the public and vectors, frequency of burning, and degree of public concern over the site (2). Of the 177 villages that have been scored by ANTHC, 70 percent have been scored as high hazard, 25 percent as moderate hazard, and 5 percent as low hazard. Without access to alternatives, all waste that is generated in villages is discarded at the dumpsite, including unknown quantities of hazardous waste from households and industry. The Alaska Department of Environmental Conservation (ADEC) reports that the dumpsites include household waste, as well as some commercial, construction and demolition waste (3). Open dumping can present an environmental and health threat through water and soil contamination, disease transmission, fire danger, and injury to site salvagers (4). In an attempt to reduce waste volume and visual blight, dump fires are set, or non-separated wastes are burned in metal containers (i.e. “burn boxes”), in approximately 75 percent of villages (5).

In 1997, over 45 percent of Alaska villages did not have running water in their homes, and inhabitants must haul their wastewater in “honeybuckets”, because the tundra makes the construction of latrines impractical (6). Human wastes are often discarded at, or near, open dumps, thereby increasing risks of exposure to pathogens when disposing of trash (6-8). Villagers hauling their solid wastes, site salvagers, children, and household pets frequent dumpsites. Many ANs have subsistence diets and there are concerns about contaminants getting into food and water supplies (9, 10).

Increased risks of adverse pregnancy outcomes have been reported near some individual landfill sites and in multi-site studies (11-15). However, these studies have never been performed in rural Alaska. Fetal and neonatal deaths and congenital anomalies (along with other reproductive outcomes) are considered to be sensitive indicators of potential health threats from environmental hazards (14,16-22). These outcomes were selected because they are associated with reproductive health (22) and are readily available through records.

Alaska has a lower neonatal mortality rate compared to the rest of the United States, but
the AN rate is higher than the rest of Alaska (23). The AN fetal mortality rate is lower than the rate of other Alaskans, but the congenital anomaly rate is higher for ANs (23). No study has examined differences in mortality rates and congenital anomalies for Natives in relation to potential hazards from open dumpsites.

Fetal and neonatal mortality rates and the incidence of congenital anomalies were selected to evaluate potential environmental hazards posed by these dumpsites. This was a population-based study that utilized a retrospective cohort design for the years 1997-2001. The purpose of the study was to determine if women living in villages with open dumpsites that have higher hazard rankings would have a higher incidence of these adverse pregnancy outcomes than women living in villages with sites that have lower hazard rankings. We also examined whether the three factors that make up the majority of a dumpsite score (dumpsite contents, distance to drinking water aquifer, and site drainage) were individually associated with the adverse pregnancy outcomes.

MATERIAL AND METHODS

Birth records were obtained from the Alaska Bureau of Vital Statistics for all births to women living in federally recognized Native villages for the period 1997-2001. It is estimated that over 97 percent of births and 99 percent of deaths are entered electronically (24). Eligible pregnancies were those coded as lasting at least 20 weeks, resulting in singleton births, whose mothers’ residences were listed as Native villages. Additionally, the women had to reside in villages for which there existed an evaluation of the hazard potential of the community dumpsite(s). There were 10,360 births in 197 villages determined to be eligible. Birth outcomes of interest included: fetal death (defined in Alaska as occurring in a pregnancy where the last menstrual date subtracted from the date of delivery is at least 20 weeks (23), neonatal death (death to live-born infants less than 28 days of age), and congenital anomalies. Neonatal deaths that were successfully linked with birth files were included (greater than 98 percent of deaths were linked with birth records by the Alaska Bureau of Vital Statistics). Only when a birth and a death both occurred within the state of Alaska, and were entered electronically, was it possible to link records. Neonatal deaths were coded to the ICD-9 (international classification of diseases, ninth revision) for deaths in 1997 and 1998, and to the ICD-10 thereafter. Neonatal deaths resulting from unintentional injuries were excluded. Cause of death for fetal deaths is not specified on Alaskan fetal death certificates, so fetal deaths that resulted from maternal trauma and injuries could not be excluded. Observable congenital anomalies are coded into 22 categories (including no defects and other defects) in Alaskan birth records. For analytic purposes, these anomalies were placed into five broader categories according to anatomic similarity. Central nervous system anomalies included: anencephalus, spina bifida or meningocele, hydrocephalus, microcephalus, and other central nervous system defects. Circulatory and respiratory anomalies included all heart malformations and other circulatory or respiratory defects. Gastrointestinal anomalies included: rectal atresia/stenosis, tracheo-esophageal fistula/esophageal atresia, omphalocele/gastroschisis, and other gastrointestinal defects. Urogenital anomalies include
malformed genitalia, renal agenesis, and other urogenital anomalies. Musculoskeletal and integumental anomalies include cleft lip and palate, polydactyly/syndactyly/adactyly, clubfoot, diaphragmatic hernia, and other musculoskeletal or integumental defects. Anomalies classified as chromosomal were not examined, unless other defects were indicated on the birth record.

Exposure information was the hazard ranking of the dumpsite of the village indicated on the birth certificate as the mother’s residence. Exposure information was ecological, because it was not known that women experienced all, or any part, of their pregnancies in the village of their residence. Dump scores were categorized by ANTHC into high, intermediate, and low hazard potential to health and environment. During the time period of the study, 159 of the villages’ dumpsites had been scored, with 17 other sites being ranked as either: high, intermediate, or low hazard. Because of small numbers, intermediate and low hazard ranked villages were collapsed into a lower hazard category. An additional 21 sites were ranked on the same hazard point factors as above, using data from the Central Council of Tlingit and Haida Indian Tribes of Alaska (CCTHITA) solid waste management database.

Site contents, the distance of the site to a drinking water aquifer, and site drainage are the factors that weighed most heavily in calculated hazard rankings (2). The ranking system scored dumpsites that contained greater than two percent hazardous waste highest on content factors, sites that contained special waste moderate scores, while sites that contained only municipal waste did not affect the overall hazard ranking. Sites that were less than 50 feet from a drinking water aquifer score maximum points in that category, sites within 51 to 600 feet of an aquifer were given moderate scores, while sites that were greater than 600 feet from aquifers did not affect scores. Site drainage that increased the likelihood of ground or surface water contamination was considered a high factor, sites that had moderate drainage, limited ponding, or neutral effects, were considered moderate factors. When dumpsite drainage contributed to the protection of ground or surface water, no points were added to the overall hazard ranking for the site drainage category (2). These factors were used as additional exposure information for the pregnancies occurring in the 180 villages that had detailed hazard point factor rankings.

Covariate information was obtained from birth records and included gender, parity, adequacy of prenatal care, smoking status, alcohol intake, race, mother’s age, and mother’s education, year of birth, gestation, and birth weight.

More information on the mothers’ residence villages was obtained from the state of Alaska Community Database (25). This included average family household size and income, percent of population Native, percent of population in poverty, the population of the village in the year 2000, and the land area of the village in square miles. Additionally, information was gathered on whether the whole village had piped water, part of the village had piped water, or none of the homes in the village were plumbed. Villages were also categorized into those that were isolated with restricted healthcare options, villages with qualified emergency care centers, and regional centers with a qualified acute care facility. Distributions
of the number and percent of births for each covariate in lower and higher potential hazard villages were calculated to determine if these covariates were approximately equally distributed across each exposure category (Table I). The same distributions were calculated for the subset of pregnancies that occurred in villages with specific exposure information on dump-site contents, distance from a drinking water aquifer, and site drainage (Table II). Per agreement with the Alaska Bureau of Vital Statistics, cells in tables with less than five observations are not displayed.

Data analysis
Chi-square tests were used to determine if the distribution of covariates was homogeneous across exposure groups. Fisher’s exact tests were used when expected cell counts were less than five. These tests were performed with all data, and then again with the subset of data with detailed hazard potential information.

Crude incidence rate ratios (RR) and 95 percent confidence intervals (95% CI) were calculated for the effect of hazard ranking on each separate birth outcome and on all deaths combined and all congenital anomalies combined. Fetal deaths were excluded from the analyses for neonatal deaths, and neonatal deaths were excluded from analyses of fetal deaths. We tested homogeneity of the rate ratio across strata of year of incidence and confirmed the assumption of homogeneity by examining a plot of standardized residuals (26).

Poisson regression (26) was used to model the natural log of the incidence rates of deaths and congenital anomalies as linear combinations of the risk factors and covariates. Adjusted incidence rate ratios and 95 percent confidence intervals were calculated to quantify the relationships between the hazard potential of villages and occurrences of all deaths, fetal and neonatal deaths, all congenital anomalies, five anatomic categories of defects, other defects, and multiple defects. Multiple anomalies were counted under each outcome and counted only once for the outcome of all congenital anomalies combined.

Information on gender (female versus male), parity (one or two previous pregnancies, three or more previous pregnancies, and unknown), adequacy of prenatal care, smoking status (did not smoke, smoked during pregnancy, and unknown), alcohol intake (did not drink, drank during pregnancy, and unknown), race (Caucasian, unknown and other, and Native), mother’s age (under 20, 20 through 39, and 40 or more years), mother’s education (less than 11 years, 12 years, and more than 12 years), gestation and birth weight (in one variable), year of birth, village healthcare options (restricted, qualified emergency care center, and qualified acute care facility), and village water hookup (no households plumbed, some household plumbed, and no households plumbed) were used in models. The Kessner Index (27) is a classification scheme of adequacy of prenatal care that accounts for the gestational month in which prenatal care began and the number of prenatal visits with respect to the length of gestation. The Kessner Index was used to define the adequacy of prenatal care into categories of adequate, intermediate, inadequate, and unknown. The gestation/birth weight variable was classified into four categories: term birth, not low birth weight; preterm birth, not low birth weight; preterm birth, low birth weight; and insufficient information/missing value. Preterm birth was defined as gestation lasting...
less than 37 weeks, and low birth weight was defined as less than 2500 grams.

All analyses were performed again, examining the subset of data that had complete information for hazard point factors. The three factors weighted the most in calculating dump scores were used individually as predictors for each outcome of interest. Villages that received the maximum score on site contents were categorized as “high” and contrasted against those that received the median score (categorized as moderate). Only two villages representing 56 pregnancies received a “low” score on contents, so these pregnancies were excluded from analyses. In the next series of models, distance to a drinking water aquifer was categorized as within 50 feet, 50 to 600 feet, and greater than 600 feet. In the last series of models, dumpsites receiving maximum scores for site drainage were categorized as “high”, those receiving median scores were “moderate”, and those receiving no points were “low”.

RESULTS

Chi-square tests revealed that the distributions of gender and year of birth were roughly equal between exposure levels (Table I). There were wide disparities ($p < 0.001$) in the distribution of parity, quality of prenatal care, cigarette and alcohol use, race, maternal age, maternal education level, birth weight and gestation, type of healthcare, and water hookup to households (Table I). Mothers in villages with low hazard ranked dumpsites tended to have had fewer previous pregnancies, better prenatal care, smoked and drank less frequently, were more frequently Caucasian, completed more years of education, had fewer low birth weight and/or preterm births, more often had access to acute care medical facilities, and were more likely to have households in their villages completely plumbed, compared to mothers from villages with higher hazard ranked dumpsites. Other village level covariates are not displayed, as they were not used in any analyses, because, after adjusting for other factors listed on the birth certificate, these factors did not add any additional information to the model.

When examining the subset of villages with detailed hazard information, chi-square tests revealed the distributions of gender, quality of prenatal care, reported cigarette use during pregnancy, and year of birth, were roughly equal between exposure levels (Table II). Parity ($p = 0.03$), alcohol use ($p = 0.04$), maternal education ($p = 0.05$), and birth weight and gestation ($p = 0.03$) were less evenly distributed. There were wide disparities ($p < 0.001$) in the distribution of race, maternal age, type of healthcare, and water hookup to households (Table II). Mothers from villages with high hazard ranked dumpsites were more often Native, more likely to have qualified emergency care facilities rather than acute care hospital facilities in their villages, and more likely to have households either all plumbed, or not plumbed in their villages, than the mothers from villages with low hazard ranked dumpsites.

As the incidences of all outcomes were homogeneous throughout the study period, incidence rates for the entire study period are presented (Table III). The overall mortality rate (fetal and neonatal deaths combined) for the 10 360 infants born between 1997 and 2001 and included in the analyses, was 7.7
Table I. The distribution and chi-square results of individual maternal and village level characteristics across villages with lower and higher hazard potential dumpsites, Alaska, 1997-2001.

<table>
<thead>
<tr>
<th>Covariates</th>
<th>Lower hazard dumpsite</th>
<th>Higher hazard dumpsite</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 5774)</td>
<td>(n = 4586)</td>
</tr>
<tr>
<td></td>
<td>Number</td>
<td>Percent</td>
</tr>
<tr>
<td>Maternal characteristics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender of infant</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>3031</td>
<td>52.5</td>
</tr>
<tr>
<td>Female</td>
<td>2743</td>
<td>47.5</td>
</tr>
<tr>
<td>Parity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 or 2 previous pregnancies</td>
<td>2327</td>
<td>40.3</td>
</tr>
<tr>
<td>0 previous pregnancies</td>
<td>1432</td>
<td>24.8</td>
</tr>
<tr>
<td>3 or more previous pregnancies</td>
<td>1998</td>
<td>34.6</td>
</tr>
<tr>
<td>Missing</td>
<td>17</td>
<td>0.3</td>
</tr>
<tr>
<td>Quality of prenatal care</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adequate</td>
<td>3278</td>
<td>56.8</td>
</tr>
<tr>
<td>Intermediate</td>
<td>1665</td>
<td>28.8</td>
</tr>
<tr>
<td>Inadequate</td>
<td>633</td>
<td>11.0</td>
</tr>
<tr>
<td>Missing</td>
<td>198</td>
<td>3.4</td>
</tr>
<tr>
<td>Cigarette use during pregnancy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Did not report smoking</td>
<td>4189</td>
<td>72.5</td>
</tr>
<tr>
<td>Reported smoking</td>
<td>1560</td>
<td>27.0</td>
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<tr>
<td>Missing</td>
<td>25</td>
<td>0.4</td>
</tr>
<tr>
<td>Alcohol use during pregnancy</td>
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<td></td>
</tr>
<tr>
<td>Did not report drinking</td>
<td>5418</td>
<td>93.8</td>
</tr>
<tr>
<td>Reported drinking</td>
<td>316</td>
<td>5.5</td>
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<tr>
<td>Missing</td>
<td>40</td>
<td>0.7</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
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<tr>
<td>Caucasian</td>
<td>2329</td>
<td>40.3</td>
</tr>
<tr>
<td>Not reported or other</td>
<td>240</td>
<td>4.2</td>
</tr>
<tr>
<td>Alaska Native</td>
<td>3205</td>
<td>55.5</td>
</tr>
<tr>
<td>Maternal Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20 years</td>
<td>777</td>
<td>13.5</td>
</tr>
<tr>
<td>20-39 years</td>
<td>4837</td>
<td>83.8</td>
</tr>
<tr>
<td>40+ years</td>
<td>160</td>
<td>2.8</td>
</tr>
<tr>
<td>Maternal education</td>
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</tr>
<tr>
<td>More than 12 years</td>
<td>1808</td>
<td>31.3</td>
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<tr>
<td>12 years</td>
<td>2697</td>
<td>46.7</td>
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<tr>
<td>Less than 12 years</td>
<td>1115</td>
<td>19.3</td>
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<tr>
<td>Missing</td>
<td>154</td>
<td>2.7</td>
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<tr>
<td>Birth weight and gestation</td>
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<td></td>
</tr>
<tr>
<td>Not low birth weight/term birth</td>
<td>5319</td>
<td>92.2</td>
</tr>
<tr>
<td>Not low birth weight/preterm birth</td>
<td>236</td>
<td>4.1</td>
</tr>
<tr>
<td>Low birth weight/term birth</td>
<td>54</td>
<td>0.9</td>
</tr>
<tr>
<td>Low birth weight/preterm birth</td>
<td>140</td>
<td>2.4</td>
</tr>
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<td>20</td>
<td>0.3</td>
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<td>Year of birth</td>
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<td>1997</td>
<td>1154</td>
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<tr>
<td>2001</td>
<td>1151</td>
<td>19.9</td>
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<tr>
<td>Village level characteristics</td>
<td></td>
<td></td>
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<tr>
<td>Available healthcare in village</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Qualified acute care facility</td>
<td>4303</td>
<td>74.5</td>
</tr>
<tr>
<td>Qualified emergency care center</td>
<td>405</td>
<td>7.0</td>
</tr>
<tr>
<td>Restricted healthcare options</td>
<td>1066</td>
<td>18.5</td>
</tr>
<tr>
<td>Piped water to households in village</td>
<td>3861</td>
<td>66.9</td>
</tr>
<tr>
<td>All households plumbed</td>
<td>1509</td>
<td>26.1</td>
</tr>
<tr>
<td>Some households plumbed</td>
<td>404</td>
<td>7.0</td>
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<tr>
<td>No households plumbed</td>
<td></td>
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</table>
Table II. The distribution and chi-square results of individual maternal and village level characteristics across the subset of villages with detailed hazard score information with lower and higher hazard potential dumpsites, Alaska, 1997-2001.

<table>
<thead>
<tr>
<th>Covariates</th>
<th>Lower hazard dumpsite (n = 966)</th>
<th>Higher hazard dumpsite (n = 4462)</th>
<th>p-value</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Percent</td>
<td></td>
</tr>
<tr>
<td>Gender of infant</td>
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<td>0.85</td>
</tr>
<tr>
<td>Male</td>
<td>503</td>
<td>52.1</td>
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</tr>
<tr>
<td>Female</td>
<td>463</td>
<td>47.9</td>
<td></td>
</tr>
<tr>
<td>Parity</td>
<td></td>
<td></td>
<td>0.03</td>
</tr>
<tr>
<td>1 or 2 previous pregnancies</td>
<td>366</td>
<td>37.9</td>
<td></td>
</tr>
<tr>
<td>0 previous pregnancies</td>
<td>198</td>
<td>20.5</td>
<td></td>
</tr>
<tr>
<td>3 or more previous pregnancies</td>
<td>402</td>
<td>41.6</td>
<td></td>
</tr>
<tr>
<td>Missing *</td>
<td>20</td>
<td>0.4</td>
<td></td>
</tr>
<tr>
<td>Quality of prenatal care</td>
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<td>0.16</td>
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<td>Adequate</td>
<td>358</td>
<td>37.1</td>
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<tr>
<td>Intermediate</td>
<td>402</td>
<td>41.6</td>
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</tr>
<tr>
<td>Inadequate</td>
<td>172</td>
<td>17.8</td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>34</td>
<td>3.5</td>
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<tr>
<td>Cigarette use during pregnancy</td>
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<td></td>
<td>0.18</td>
</tr>
<tr>
<td>Did not report smoking</td>
<td>631</td>
<td>65.3</td>
<td></td>
</tr>
<tr>
<td>Reported smoking</td>
<td>329</td>
<td>34.1</td>
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</tr>
<tr>
<td>Missing</td>
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<td>0.6</td>
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<td>Alcohol use during pregnancy</td>
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<td>Did not report drinking</td>
<td>901</td>
<td>93.3</td>
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</tr>
<tr>
<td>Reported drinking</td>
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<td>5.6</td>
<td></td>
</tr>
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<td>Missing</td>
<td>11</td>
<td>1.1</td>
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</tr>
<tr>
<td>Race</td>
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<td></td>
<td>0.001</td>
</tr>
<tr>
<td>Caucasian</td>
<td>117</td>
<td>12.1</td>
<td></td>
</tr>
<tr>
<td>Not reported or other</td>
<td>8</td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>Alaska Native</td>
<td>841</td>
<td>87.1</td>
<td></td>
</tr>
<tr>
<td>Maternal Age</td>
<td></td>
<td></td>
<td>0.65</td>
</tr>
<tr>
<td>&lt;20 years</td>
<td>153</td>
<td>15.8</td>
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</tr>
<tr>
<td>20-39 years</td>
<td>788</td>
<td>81.6</td>
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</tr>
<tr>
<td>40+ years</td>
<td>25</td>
<td>2.6</td>
<td></td>
</tr>
<tr>
<td>Maternal education</td>
<td></td>
<td></td>
<td>0.05</td>
</tr>
<tr>
<td>More than 12 years</td>
<td>119</td>
<td>12.3</td>
<td></td>
</tr>
<tr>
<td>12 years</td>
<td>570</td>
<td>59.0</td>
<td></td>
</tr>
<tr>
<td>Less than 12 years</td>
<td>240</td>
<td>24.8</td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>37</td>
<td>3.8</td>
<td></td>
</tr>
<tr>
<td>Birth weight and gestation</td>
<td></td>
<td></td>
<td>0.03</td>
</tr>
<tr>
<td>Not low birth weight/term birth</td>
<td>897</td>
<td>92.9</td>
<td></td>
</tr>
<tr>
<td>Not low birth weight/preterm birth</td>
<td>37</td>
<td>3.8</td>
<td></td>
</tr>
<tr>
<td>Low birth weight/term birth</td>
<td>9</td>
<td>0.9</td>
<td></td>
</tr>
<tr>
<td>Low birth weight/preterm birth</td>
<td>21</td>
<td>2.2</td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>23</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Year of birth</td>
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<td></td>
<td>0.49</td>
</tr>
<tr>
<td>1997</td>
<td>194</td>
<td>20.1</td>
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</tr>
<tr>
<td>1998</td>
<td>209</td>
<td>21.6</td>
<td></td>
</tr>
<tr>
<td>1999</td>
<td>177</td>
<td>18.3</td>
<td></td>
</tr>
<tr>
<td>2000</td>
<td>186</td>
<td>19.3</td>
<td></td>
</tr>
<tr>
<td>2001</td>
<td>200</td>
<td>20.7</td>
<td></td>
</tr>
<tr>
<td>Village level characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Available healthcare in village</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Qualified acute care facility</td>
<td>118</td>
<td>12.2</td>
<td></td>
</tr>
<tr>
<td>Qualified emergency care center</td>
<td>89</td>
<td>9.2</td>
<td></td>
</tr>
<tr>
<td>Restricted healthcare options</td>
<td>759</td>
<td>78.6</td>
<td></td>
</tr>
<tr>
<td>Piped water to households in village</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>All households plumbed</td>
<td>126</td>
<td>13.0</td>
<td></td>
</tr>
<tr>
<td>Some households plumbed</td>
<td>548</td>
<td>56.7</td>
<td></td>
</tr>
<tr>
<td>No households plumbed</td>
<td>292</td>
<td>30.2</td>
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* Cell value less than 5
Table III. Incidence rates of neonatal and fetal deaths and congenital anomalies, in 197 Alaska Native villages, 1997-2001

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Incidence (per 1000 births)</th>
<th>Number of pregnancies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deaths</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>7.7</td>
<td>10360</td>
</tr>
<tr>
<td>Fetal</td>
<td>4.6</td>
<td>10327</td>
</tr>
<tr>
<td>Neonatal</td>
<td>3.2</td>
<td>10313</td>
</tr>
<tr>
<td>Congenital Anomalies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>25.2</td>
<td>10360</td>
</tr>
<tr>
<td>Central nervous system</td>
<td>1.4</td>
<td>10360</td>
</tr>
<tr>
<td>Circulatory/respiratory</td>
<td>2.2</td>
<td>10360</td>
</tr>
<tr>
<td>Urogenital</td>
<td>2.2</td>
<td>10360</td>
</tr>
<tr>
<td>Musculoskeletal/integumental</td>
<td>7.9</td>
<td>10360</td>
</tr>
<tr>
<td>Other</td>
<td>11.9</td>
<td>10360</td>
</tr>
<tr>
<td>Multiple</td>
<td>1.8</td>
<td>10360</td>
</tr>
</tbody>
</table>

Table IV. Crude and adjusted rate ratios and 95% confidence intervals describing the relationships between lower and higher hazard exposure categories and incidence of fetal and neonatal death and congenital anomalies, Alaska, 1997-2001.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Outcome present</th>
<th>Number</th>
<th>Rate per 1000</th>
<th>Rate ratio crude (95% CI)</th>
<th>Rate ratio adjusted † (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deaths</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All deaths (n = 10360)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower hazard dumpsite</td>
<td>44</td>
<td>5774</td>
<td>7.6</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Higher hazard dumpsite</td>
<td>36</td>
<td>4586</td>
<td>7.9</td>
<td>1.03 (0.66, 1.60)</td>
<td>0.65 (0.34, 1.27)</td>
</tr>
<tr>
<td>Fetal (n = 10327)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower hazard dumpsite</td>
<td>28</td>
<td>5758</td>
<td>4.9</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Higher hazard dumpsite</td>
<td>19</td>
<td>4569</td>
<td>4.2</td>
<td>0.85 (0.48, 1.53)</td>
<td>0.75 (0.28, 1.99)</td>
</tr>
<tr>
<td>Neonatal (n = 10313)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower hazard dumpsite</td>
<td>16</td>
<td>5746</td>
<td>2.8</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Higher hazard dumpsite</td>
<td>17</td>
<td>4567</td>
<td>3.7</td>
<td>1.34 (0.68, 2.65)</td>
<td>0.55 (0.22, 1.38)</td>
</tr>
<tr>
<td>Congenital Anomalies</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All (n = 10360)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower hazard dumpsite</td>
<td>142</td>
<td>5774</td>
<td>24.6</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Higher hazard dumpsite</td>
<td>119</td>
<td>4586</td>
<td>26.0</td>
<td>1.06 (0.83, 1.35)</td>
<td>1.37 (0.92, 2.04)</td>
</tr>
<tr>
<td>Central nervous system</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower hazard dumpsite</td>
<td>6</td>
<td>5774</td>
<td>1.0</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Higher hazard dumpsite</td>
<td>9</td>
<td>4586</td>
<td>2.0</td>
<td>1.89 (0.67, 5.31)</td>
<td>2.36 (0.37, 14.71)</td>
</tr>
<tr>
<td>Circulatory/respiratory</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower hazard dumpsite</td>
<td>12</td>
<td>5774</td>
<td>2.1</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Higher hazard dumpsite</td>
<td>11</td>
<td>4586</td>
<td>2.4</td>
<td>1.17 (0.51, 2.64)</td>
<td>1.42 (0.39, 5.42)</td>
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<td>Gastrointestinal</td>
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<td>6</td>
<td>5774</td>
<td>1.3</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Higher hazard dumpsite</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urogenital</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower hazard dumpsite</td>
<td>10</td>
<td>5774</td>
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<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Higher hazard dumpsite</td>
<td>13</td>
<td>4586</td>
<td>2.8</td>
<td>1.64 (0.72, 3.73)</td>
<td>2.71 (0.67, 10.95)</td>
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<tr>
<td>Musculoskeletal/integumental</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower hazard dumpsite</td>
<td>41</td>
<td>5774</td>
<td>7.1</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Higher hazard dumpsite</td>
<td>41</td>
<td>4586</td>
<td>8.9</td>
<td>1.26 (0.81, 1.94)</td>
<td>1.61 (0.79, 3.29)</td>
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<tr>
<td>Other</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower hazard dumpsite</td>
<td>71</td>
<td>5774</td>
<td>12.3</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Higher hazard dumpsite</td>
<td>52</td>
<td>4586</td>
<td>11.3</td>
<td>0.92 (0.65, 1.32)</td>
<td>1.38 (0.77, 2.39)</td>
</tr>
<tr>
<td>Multiple</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower hazard dumpsite</td>
<td>9</td>
<td>5774</td>
<td>1.6</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
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<td>10</td>
<td>4586</td>
<td>2.2</td>
<td>1.40 (0.57, 3.44)</td>
<td>1.33 (0.34, 5.20)</td>
</tr>
</tbody>
</table>

† Adjusted for gender, interpregnancy interval, parity, adequacy of prenatal care, smoking status, alcohol intake, race, mother’s age and education, healthcare options, piped water, and missing values.

* Cell value less than 5
### Table V. Crude and adjusted rate ratios and 95% confidence intervals describing the relationships between site contents, distance from site to drinking water aquifers, and site drainage with incidence of fetal and neonatal death and congenital anomalies, Alaska, 1997-2001.

<table>
<thead>
<tr>
<th>Models</th>
<th>Outcome</th>
<th>Number</th>
<th>Rate ( \text{per 1000} )</th>
<th>Rate ratio crude (95% CI)</th>
<th>Rate ratio adjusted</th>
<th>Rate ratio adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Downstream</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>present</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All deaths</td>
<td>(n = 5428)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site contents (moderate)</td>
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<td>5214</td>
<td>7.3</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site contents (high)</td>
<td>9</td>
<td>12.7</td>
<td>1.74 (0.42, 7.20)</td>
<td>2.04 (0.48, 8.57)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance to aquifer &gt;600 ft</td>
<td>26</td>
<td>3892</td>
<td>6.7</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance to aquifer 50-600 ft</td>
<td>9</td>
<td>1011</td>
<td>8.9</td>
<td>1.33 (0.62, 2.84)</td>
<td>1.28 (0.59, 2.79)</td>
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</tr>
<tr>
<td>Distance to aquifer &lt;50 ft</td>
<td>5</td>
<td>525</td>
<td>9.5</td>
<td>1.43 (0.55, 3.71)</td>
<td>1.09 (0.41, 2.93)</td>
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</tr>
<tr>
<td>Site drainage (low)</td>
<td>12</td>
<td>1606</td>
<td>7.5</td>
<td>1.05 (0.21, 5.35)</td>
<td>1.07 (0.21, 5.35)</td>
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</tr>
<tr>
<td>Site drainage (moderate)</td>
<td>12</td>
<td>1606</td>
<td>7.5</td>
<td>1.05 (0.21, 5.35)</td>
<td>1.07 (0.21, 5.35)</td>
<td></td>
</tr>
<tr>
<td>Site drainage (high)</td>
<td>12</td>
<td>1606</td>
<td>7.5</td>
<td>1.05 (0.21, 5.35)</td>
<td>1.07 (0.21, 5.35)</td>
<td></td>
</tr>
<tr>
<td>Fetal</td>
<td>Site contents (moderate)</td>
<td>20</td>
<td>5196</td>
<td>3.9</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Site contents (high)</td>
<td>173</td>
<td>12.7</td>
<td>1.74 (0.42, 7.20)</td>
<td>2.04 (0.48, 8.57)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance to aquifer &gt;600 ft</td>
<td>15</td>
<td>3881</td>
<td>3.9</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance to aquifer 50-600 ft</td>
<td>5</td>
<td>1011</td>
<td>8.9</td>
<td>1.33 (0.62, 2.84)</td>
<td>1.28 (0.59, 2.79)</td>
<td></td>
</tr>
<tr>
<td>Distance to aquifer &lt;50 ft</td>
<td>173</td>
<td>7.1</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site drainage (low)</td>
<td>12</td>
<td>1606</td>
<td>7.5</td>
<td>1.05 (0.21, 5.35)</td>
<td>1.07 (0.21, 5.35)</td>
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</tr>
<tr>
<td>Site drainage (moderate)</td>
<td>12</td>
<td>1606</td>
<td>7.5</td>
<td>1.05 (0.21, 5.35)</td>
<td>1.07 (0.21, 5.35)</td>
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</tr>
<tr>
<td>Site drainage (high)</td>
<td>12</td>
<td>1606</td>
<td>7.5</td>
<td>1.05 (0.21, 5.35)</td>
<td>1.07 (0.21, 5.35)</td>
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<tr>
<td>Neonatal</td>
<td>Site contents (moderate)</td>
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<td>5194</td>
<td>3.5</td>
<td>1.00</td>
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<td>6.4</td>
<td>1.65 (0.22, 12.33)</td>
<td>3.08 (0.38, 25.27)</td>
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<tr>
<td>Distance to aquifer &gt;600 ft</td>
<td>11</td>
<td>3877</td>
<td>2.8</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance to aquifer 50-600 ft</td>
<td>11</td>
<td>3877</td>
<td>2.8</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance to aquifer &lt;50 ft</td>
<td>173</td>
<td>7.1</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site drainage (low)</td>
<td>12</td>
<td>1606</td>
<td>7.5</td>
<td>1.05 (0.21, 5.35)</td>
<td>1.07 (0.21, 5.35)</td>
<td></td>
</tr>
<tr>
<td>Site drainage (moderate)</td>
<td>12</td>
<td>1606</td>
<td>7.5</td>
<td>1.05 (0.21, 5.35)</td>
<td>1.07 (0.21, 5.35)</td>
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</tr>
<tr>
<td>Site drainage (high)</td>
<td>12</td>
<td>1606</td>
<td>7.5</td>
<td>1.05 (0.21, 5.35)</td>
<td>1.07 (0.21, 5.35)</td>
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</tr>
<tr>
<td>Congenital Anomalies (n = 5367)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Musculoskeletal/integumental</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site contents (moderate)</td>
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<td>5214</td>
<td>8.6</td>
<td>1.00</td>
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<td></td>
</tr>
<tr>
<td>Site contents (high)</td>
<td>173</td>
<td>6.3</td>
<td>0.73 (0.10, 5.32)</td>
<td>0.75 (0.10, 5.47)</td>
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</tr>
<tr>
<td>Distance to aquifer &gt;600 ft</td>
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<td>3892</td>
<td>8.2</td>
<td>1.00</td>
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<td></td>
</tr>
<tr>
<td>Distance to aquifer 50-600 ft</td>
<td>8</td>
<td>1011</td>
<td>7.9</td>
<td>0.96 (0.44, 2.09)</td>
<td>1.01 (0.46, 2.21)</td>
<td></td>
</tr>
<tr>
<td>Distance to aquifer &lt;50 ft</td>
<td>173</td>
<td>7.6</td>
<td>0.96 (0.44, 2.09)</td>
<td>1.01 (0.46, 2.21)</td>
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</tr>
<tr>
<td>Site drainage (low)</td>
<td>5</td>
<td>281</td>
<td>17.8</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site drainage (moderate)</td>
<td>27</td>
<td>3541</td>
<td>7.6</td>
<td>0.43 (0.17, 1.11)</td>
<td>0.39 (0.14, 1.08)</td>
<td></td>
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<tr>
<td>Site drainage (high)</td>
<td>14</td>
<td>1606</td>
<td>8.7</td>
<td>0.49 (0.18, 1.36)</td>
<td>0.45 (0.15, 1.33)</td>
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</tr>
<tr>
<td>Other</td>
<td>Site contents (moderate)</td>
<td>50</td>
<td>5214</td>
<td>9.6</td>
<td>1.00</td>
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<tr>
<td>Site contents (high)</td>
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<td>6.4</td>
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<td>0.75 (0.10, 5.47)</td>
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<tr>
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<td>3892</td>
<td>10.3</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance to aquifer 50-600 ft</td>
<td>12</td>
<td>1011</td>
<td>11.9</td>
<td>1.15 (0.61, 2.20)</td>
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</tr>
<tr>
<td>Distance to aquifer &lt;50 ft</td>
<td>173</td>
<td>7.6</td>
<td>0.74 (0.27, 2.07)</td>
<td>0.72 (0.26, 2.02)</td>
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<tr>
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<td>17.8</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site drainage (moderate)</td>
<td>28</td>
<td>3541</td>
<td>7.9</td>
<td>0.74 (0.23, 2.44)</td>
<td>0.72 (0.22, 2.38)</td>
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<tr>
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<td>15.6</td>
<td>1.46 (0.44, 4.83)</td>
<td>1.43 (0.43, 4.77)</td>
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† Adjusted for gender, interpregnancy interval, parity, adequacy of prenatal care, smoking status, alcohol intake, race, mother’s age and education, healthcare options, piped water, and missing values.

a Cell value less than 5

* p < 0.001

CI = Confidence interval
ft = feet (1 foot = 0.3048 metres)
per 1000 births, while the rate for fetal deaths was 4.6 and neonatal deaths was 3.2 per 1000 births. The incidence rate for all congenital anomalies combined was 25.2 per 1000 births. The category “other” defects had the highest rate, with 11.9 per 1,000 births, where musculoskeletal/integumental defects had the highest incidence of a specific anomaly (7.9 per 1000). Incidence rates for other defects ranged from 1.4 to 2.2 per 1000 births.

Crude estimates revealed that mothers residing in villages with higher hazard rankings were at no detectable increased risk for all deaths combined, fetal deaths, or neonatal deaths, compared to mothers residing in villages with lower hazard rankings (Table IV). Adjusted estimates failed to detect significant differences at the 5 percent level of confidence between exposure groups. However, effect estimates were in a positive direction for combined congenital anomalies, central nervous system anomalies, circulatory and respiratory anomalies, urogenital anomalies musculoskeletal and integumental anomalies, other defects, and multiple defects (Table IV). The outcome group including all anomalies (RR = 1.37, 95% CI: 0.92, 2.02) had the narrowest confidence intervals, embracing fewer values below 1.00 than other outcomes.

There were 5428 births in villages with detailed exposure rankings. Crude estimates did not detect any significant difference in death rates for infants born to mothers in villages with high hazard site contents versus infants born to mothers residing in villages with moderate hazard site contents (Table V). Adjusted estimates also failed to detect significant differences for the two exposure categories for all deaths (RR = 2.04; 95% CI: 0.73, 1.80) fetal deaths (RR = 3.08; 95% CI: 0.38, 25.27), and neonatal deaths (RR = 3.67; 95% CI: 0.45, 29.86). Neither crude, nor adjusted estimates detected a significant difference in incidences for all deaths, fetal deaths, or neonatal deaths, when using the distance of the dumpsite from the drinking water aquifer as a predictor. In fact, estimates for the moderately exposed group were higher than estimates for the more heavily exposed group for all deaths and fetal deaths (Table V). Crude models detected no significant differences in incidence of death across low, moderate, and high site drainage exposure groups when predicting deaths. Adjusted estimates were close to unity for all deaths and fetal deaths, and in a protective direction for neonatal deaths.

When examining congenital anomalies, there were only 5367 births in villages with detailed exposure information available for analyses. Only models examining risks for musculoskeletal and integumental defects and other defects converged. Crude estimates predicting musculoskeletal and integumental defects did not demonstrate any differences when examining site contents, dumpsite distance to drinking water aquifer, or site drainage (Table V). Likewise, adjusted estimates did not differ across exposure groups for dumpsite contents, distance to a drinking water aquifer, or site drainage. In fact, when comparing moderate hazard site drainage to low hazard site drainage, it was found to be somewhat protective against defects (RR = 0.39; 95% CI: 0.14, 1.08). For the analysis containing only other defects, the crude rate ratio revealed infants born to mothers residing in villages with high hazard site contents had an increased rate for other defects (RR = 3.96; 95% CI: 1.70, 5.32), compared to
infants born to mothers residing in villages with moderate hazard site contents. Adjusted estimates also detected an increased rate (RR = 4.27; 95% CI: 1.76, 10.36) for high hazard site contents, compared to the referent category. Neither crude, nor adjusted rate ratios for other defects detected a significant difference with respect to dumpsite distance to a drinking water aquifer and site drainage.

DISCUSSION

This work failed to detect a significant increase in the rates of fetal, or neonatal deaths, or all deaths combined, in infants whose birth certificates indicated that their mothers resided in villages with higher hazard dumpsites, compared to villages with lower hazard sites. Although 95 percent confidence intervals included unity, adjusted estimates suggested a protective effect associated with residence in high hazard villages.

Aside from gastrointestinal defects, adjusted estimates for all categories of congenital anomalies were in a positive direction. However, all 95 percent confidence intervals included values below one. Estimates were slightly higher than, or within range of, results reported in studies with positive results for birth defects (11-15). This study had a much smaller sample size than these other studies with positive results, and the 95 percent confidence intervals for the largest category, which contained all defects combined, included the fewest values below one. This indicates that a study using a larger sample size may be able to detect meaningful associations between dumpsite hazard potential and the incidence of congenital anomalies as significant.

In an attempt to define exposure more finely, hazard factors that had the most influence on overall dumpsite scores were used as individual predictors. Defining these models resulted in a nearly 50 percent reduction in sample size. Only models predicting all deaths, fetal deaths, neonatal deaths, musculoskeletal and integumental defects, and other defects, had enough cases in each level of exposure for the statistical models to converge. There was a four-fold increase in the rate of other congenital defects in infants born to mothers residing in villages with dumpsites with high hazard dumpsite contents, compared to mothers residing in villages with moderate hazard dumpsite contents. Although confidence intervals included one, high hazard dumpsite contents also indicated an elevated rate for fetal and neonatal deaths (individually and combined). No patterns could be detected in any models using distance of dumpsites to drinking water aquifers and site drainage. This could be because of no actual effect, a small sample size, or because some AN villages prefer to use rain catchment systems, snowmelt, or untreated river water to obtain drinking water, and other villages test and treat water regularly.

The largest obstacle encountered with this study is the definition of exposure. It is not known what proportion of their pregnancies women spent in their village of residence indicated on the birth record. Although the dumpsites were ranked in generally the same time period as the study period, the quality of the dumpsites could conceivably have been labile during each pregnancy.
The available level of village healthcare was adjusted for in the analyses, but information on covariates, such as underlying health conditions and occupational exposures, were not. Covariates were distributed differently between exposure levels. Other potentially confounding factors not measured may have been distributed differently. These covariates could have differed between exposure groups. Another concern is that studies performed in other states have found that birth record information often does not correlate with the patients’ medical records (28,29). Although this analysis has not been performed with Alaskan records, results need to be interpreted cautiously.

Information on congenital anomalies was gathered from birth records. This method reduced complications associated with linking records in different databases. Additionally, birth defects occurring in stillborn infants may be less likely to be reported to the Alaska Birth Defects Registry (ABDR) (30), so using birth certificate information might find more cases with respect to defects in fetal deaths. However, only birth defects apparent within the first few days following birth are entered on the birth certificate. The overall congenital anomaly rate in our study was 25.2 per 1,000 births (excluding chromosomal abnormalities). However, the rate of children with at least one major congenital anomaly for 1996-2000 was 52.6 per 1000 births as reported by the ABDR. The ABDR rate includes chromosomal defects, but, as these defects were relatively uncommon, it is still approximately twice the rate of the birth record rate in this study. Defects are categorized into fairly broad categories on birth records, rather than coded to the ICD-10, which can make comparability between studies difficult. Since the inception of the ABDR in 1996, reporting rates and early case ascertainment have improved, so further studies may increase power by using the registry rather than birth records when defining cases.

Conclusions
Future studies examining potential health effects associated with open dumpsites in AN villages should include exposure measurements that are more precise. Studies with crude exposure definitions sometimes provide information indicating potential risks, but misclassification bias is inherent. Several contaminants identified in, and proximal to, individual dumpsites (arsenic, lead, methyl mercury, and several petroleum hydrocarbons) are associated with negative birth outcomes (14,31,32). In examining health effects associated with environmental exposures from hazardous waste sites, it is always preferable, though rarely possible, to identify direct pathways of exposure. Reproductive outcomes can be sensitive indicators of environmental insults, as the reproductive system often fails before other systems (14,19,33). Other outcomes of interest could include chromosomal abnormalities or DNA adducts. Sparse populations and long incubation periods make the study of cancers difficult, although this is an outcome of great interest to ANs.

Although a substantial number of studies have been conducted, risks to health from dumpsites are difficult to quantify. Exposure information is often poorly defined, and effects of exposure to low levels of environmental contaminants are not easy to quantify, either singly or jointly. The most important aspect
of this study is that it is the first to attempt to characterize the relationship of infant deaths and congenital anomalies to residents of AN villages with respect to open dumpsites.

Acknowledgements
We would like to acknowledge Phillip Mitchell of the Alaska Bureau of Vital Statistics (ABVS) for providing birth record data, the Central Council of Tlingit and Haida Indian Tribes of Alaska and Alaska Native Tribal Health Consortium for exposure information, and Zender Environmental Science and Planning Services.

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Philip H. Kass, DVM, PhD
Department of Population Health and Reproduction
University of California, Davis
One Shields Ave,
Davis, CA 95616
USA
Email: phkass@ucdavis.edu
Body of Evidence: 
Reproductive Health and the Environment

Introduction
A growing number of health care providers report a rise in patients seeking treatment for infertility and other reproductive health issues (Luoma, 2005; Giudice 2006). Current research suggests that reproductive health may be supported or compromised by several factors including age, genetics, nutritional health, stress, and pharmaceutical use (Schettler et al., 1999). Mounting scientific evidence also suggests that chemicals and contaminants commonly found in the human environment, drinking water, and food supply can influence human fertility and reproduction, though in complex, often poorly understood ways. Several recent scientific discoveries about the effects of environmental pollutants on human reproductive health merit the attention and concern of health care providers treating patients with reproductive health issues (Giudice et al., 2005).

This bulletin provides an introductory overview of what role environmental exposures might play in fertility and reproductive health, with a special focus on evidence drawn from Alaska. This bulletin also provides resources and opportunities for further information about how clinicians can respond.

Health Outcomes of Potential Concern
Reproductive health outcomes with environmental links have been found in females and males of all ages. These issues are not only of concern to health care providers in gynecology or obstetrics, who may have patients struggling to conceive, but also, to pediatricians observing young children with malformed genitalia or early-onset or delayed menarche.

A host of symptoms or conditions are linked or suspected to be linked to environmental exposures. These include, among females (see Buck et al., 2006; Giudice et al., 2005):

- Premature ovarian failure
- Malformed reproductive organs
- Early or delayed menarche
- Infertility or compromised fertility
- Recurrent pregnancy loss
- Inability to carry baby to term, pregnancy compromise, birth defects, congenital abnormalities, and low birth weight
- Damage to fetal reproductive organs
- Premature menopause
- Uterine fibroids

Among males, key trends in reproductive health include (see Hauser 2006; Giudice et al., 2005):

- Undescended testes or malformed reproductive organs (e.g., cryptorchidism and hypospadias).
- Compromised sperm shape and quality, including issues of mobility, motility, and genetic integrity.
- Testicular dysgenesis syndrome, or TDS, is a suspected cluster of effects (undescended or malformed reproductive organs, testicular cancer, and decreased sperm quality) with a hypothesized common fetal origin (Skakkebæk et al., 2001).

Furthermore, after tracking a study group of Massachusetts men for twenty years, scientists observed an overall decline in testosterone levels that were not attributable to aging or lifestyle (Travison et al., 2007), further supporting the possibility that environmental exposures are affecting male reproductive health. More generally, there are also rising rates of diseases or conditions known to affect human fertility in which environmental exposures might also play some role in the etiology, including endometriosis, and ovarian, cervical, and testicular cancer (Giudice et al., 2005).

Sources of Environmental Exposures
Alaska Native peoples and others living in the circumpolar region bear a disproportionate burden of environmental contaminants (AMAP 1997; 2004; Berner 1999; Chary 2000). Several known or suspected reproductive toxicants, such as polychlorinated biphenyls (PCBs) and pesticides, have been detected in the Alaskan environment and food system, as well as in the blood of some Alaska Native people (Berner 1999; Carpenter et al., 2005;...
Middaugh et al., 2000) and other circumpolar populations (AMAP 1997).

These chemicals and contaminants originate from both local and remote sources. Many chemicals and heavy metals are produced or released into the environment by industries located in the Lower 48 states and throughout the world, but because they are slow to degrade, and can travel vast distances in air and water currents, they tend to condense, concentrate, and then persist in the cold, northern climate. As a result, the circumpolar Arctic region has become a hemispheric sink for many pollutants and chemicals that may pose reproductive risks (AMAP 2004; Tenenbaum 1998). Of particular concern are pesticides, which are used elsewhere but accumulate in Alaska due to global fate and transport from southern latitudes.

Local industries and formerly-used defense sites are also current sources of exposure to heavy metals (e.g., lead and mercury) and industrial chemicals (e.g., PCBs) in Alaska (Button 2002). Thus, PCB exposures may result not just from global transport, but from formerly-used defense sites in Alaska as well (Carpenter et al., 2005).

These persistent pollutants enter the food chain, where they concentrate in the fatty tissues of predator species. As a result, many of these pollutants are found in the traditional subsistence foods that sustain a significant portion of the Alaskan population (AMAP 2004; Berner 1999; Chary 2000; Middaugh et al., 2000). Though a route of exposure, traditional foods remain the best source of sustenance for Alaska Native people as they are both nutritionally rich and culturally essential. Therefore, it is imperative that health care providers support local, national, and international initiatives to eliminate the use of chemicals known to accumulate in foods.

Humans also are exposed by inhaling air or drinking water that is contaminated by these pollutants. Such exposures can occur at home or on the job, outdoors as well as indoors, as many chemicals can collect in household air and dust (Rudel et al., 2003). In some cases, exposures can occur when substances containing reproductive toxicants are absorbed into the skin, such as phthalates (pronounced ‘thal-lates’), which are commonly added to self-care and beauty products (Rudel et al., 2003).

While researchers document these compounds in air, water, soil, foods, and consumer products, research also confirms that these substances enter the body. Ongoing population monitoring conducted by the U.S. Centers for Disease Control and Prevention (CDC) reports that the average American carries detectable levels of various reproductive toxicants that are also present in the environment (US CDC 2005). In general, many chemicals and pollutants, because of their chemical-physical characteristics, either build up in human systems or are ubiquitous so that, even if excreted by the body, constant re-exposure maintains elevated body levels.

Many chemical compounds are detected at levels known or suspected to affect the reproductive system. As scientists learn more about low-level effects of chemical exposures and the unique vulnerabilities of some individuals, the levels of current human burdens increasingly have flagged the attention of researchers and health care providers (Giudice et al., 2005). More recently, the effects of low-level exposures have garnered the attention of policy-makers. In many instances, however, there remain data gaps about whether and how low-level exposures affect the function of the human body (National Research Council 2006) and the reproductive system.

Researchers have not studied many compounds for their reproductive effects. A recent tabulation found that less than 10% of the 100,000 chemicals currently registered for commercial use have been studied for their human health effects, and only a fraction of those have been tested for how they affect reproductive health (Davis and Webster, 2002).

**Timing of Exposures**

The timing of exposure strongly influences the observed outcomes. Exposures to pesticides such as alachlor, atrazine, and diazinon, for example, were found to compromise sperm quality of human adult males (Swan et al., 2003b). Similarly, new research suggests links between men’s exposures to a particular phthalate and lower sperm concentration and decreased mobility (Hauser et al., 2006). Researchers also have found that exposures encountered in utero can shape the subsequent reproductive health of adults. Clinicians and scientists now know that the placenta does not shield the developing fetus from chemicals or pollutants the mother encounters (Colborn et al., 1996). For example, some conditions and disease of the reproductive tract have origins in exposures encountered during particular periods of fetal development (Giudice et al., 2005).

The effect of in utero exposures was first seen among the children of women who were, beginning in the 1940s, prescribed DES, a synthetic estrogen, to prevent miscarriage. Though not apparent at first, as exposed children reached puberty, a pattern of reproductive health problems emerged. Follow-up research has confirmed a host of outcomes in both the male and female children of women who took DES, including rare reproductive cancers and malformed reproductive organs, menstrual irregularities, and infertility or sub-fertility, i.e., difficulty conceiving (Schrager and Potter 2004).

Today, DES serves as one model for how exposures during critical periods of fetal development can affect reproductive health in later life stages.

A body of evidence now demonstrates that many chemicals humans routinely encounter in the environment can mimic or interact with the endocrine system, and like DES, can affect the fertility and reproductive health of offspring, though through other biological mechanisms (Colborn et al., 1996; McLachlan 2001). Synthetic or hu-
man-made substances can exhibit estrogenic, androgenic, or anti-estrogenic and anti-androgenic properties. That is, they can act like hormones, or block normal hormone function, which in turn, affects reproductive development (Colborn et al., 1996).

So called ‘endocrine disruption’ of human reproduction by environmental chemicals has been demonstrated in animals, but more recently in humans as well. A 2003 study published in The Lancet found that women with higher in utero exposures to DDT— a pesticide banned from use in the US, but still used in other countries and present in circumpolar ecosystems (van Oostdam et al., 2004; Simonetti et al., 2001)—took longer to conceive once they reached reproductive maturity (Cohn et al., 2003). Researchers are also compiling evidence that phthalates encountered in utero affect adult male fertility later in life (Latini et al., 2006).

Environmental chemicals also interfere with the fertility of subsequent generations by altering how genes direct key biological processes. A recent study published in Science found that when pregnant female mice were exposed to two commonly used pesticides, vinclozolin and methoxychlor, the exposures compromised the fertility of male offspring in three subsequent generations (Anway et al., 2005). This is the first study to find multi-generational effects. Although the study was performed on mice and involved exposures that are higher than humans might encounter, it is biologically plausible that similar trends might be seen among humans since genetic processes are known to be reasonably similar in both species (Anway et al., 2005).

Accumulating Evidence and Continued Research

Though research is on-going, reproductive environmental health researchers and clinicians agree there is already a substantial body of evidence implicating low-level exposures as a likely contributor to trends currently seen in reproductive outcomes (Giudice et al., 2005).

Supporting evidence comes from a wide variety of sources. Wildlife studies associate exposures to increased rates of malformed reproductive organs, infertility, and overall population declines (Colborn et al., 1996; Guillette and Moore 2006). In humans, the most compelling evidence comes from observations of reproductive outcomes following acute, high-level, occupational exposures (Colborn et al., 1996). For example, over thirty years ago, researchers demonstrated decreased fertility in male farm workers due to regular, occupational exposures to DBCP, an agricultural fumigant (Whorton et al., 1977; Slutsky et al., 1999).

Currently, researchers investigate what reproductive risks are posed by routine, low-level exposures to environmental chemicals. In addition, researchers also examine the effects of multiple, simultaneous exposures, which better account for the reality that humans encounter reproductive toxicants in complex mixtures (Denham et al., 2005). When substances interact, they may increase or mediate toxicological effects in unforeseen ways. Compounds deemed high priority for research on reproductive effects include (Giudice et al., 2005):

- Commonly-used pesticides
- Chemical additives (used in production of plastics, e.g., Bisphenol A)
- Perfluorinated compounds (used in production of stain-resistant, water-repellant, or non-stick surfaces)
- Chemical additives to beauty and self-care products
- Polybrominated diphenyl ethers (PBDEs) (used as flame retardants in household upholstery and electronics).

Many of these substances are what scientists call ‘emerging concerns.’ They comprise a new class of persistent, bioaccumulative toxicants that have been studied far less than organochlorines such as PCBs and the pesticide, DDT. Research has already suggested some of these compounds are reproductive toxicants. For example, recent research found decreased sperm counts in male offspring of pregnant rats exposed to a single, low-dose of a ubiquitous brominated flame retardant. These effects were seen at levels already detected in samples of US women’s breast milk (Kuriyama et al., 2005).

Though ongoing research focuses on the reproductive toxicity of these emergent chemicals, new scientific evidence also advances our understanding of the reproductive concerns posed by PCBs, DDT, and other organochlorine exposures. Thus, these too, remain a priority concern as scientists continue to identify significant reproductive outcomes, how they affect the reproductive system, and at what levels.

New Research in Alaska

Though scientific research about how environmental factors affect human reproductive health in Alaska is limited, important trends can be inferred from studies conducted within the state.

For example, researchers recently documented a high rate of reproductive organ and testosterone-related problems among male, Sitka black-tailed deer on Kodiak Island. The study, published in Environmental Health Perspectives, argues that this unusual pattern is attributable to in utero exposures, when pregnant females ingested foods containing hormonally-active agents (Veeramachaneni et al., 2006).

Also in 2006, a team of UAA scientists examined whether exposure to perchlorate, which has many industrial and military applications, has reproductive effects on...
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fish. Scientists suspect exposures are occurring in Alaska through imported produce and milk and possibly through seepage from military sites where munitions are stored, many of which have yet to be tested (O’Malley 2006). The UAA perchlorate-exposed fish exhibited altered patterns of sexual development and courtship behaviors. Also, exposed females developed both female and male reproductive organs and were capable of producing both eggs and sperm. This is the first study to demonstrate that perchlorate exhibits androgenic properties and impacts reproductive health (Bernhardt et al., 2006).

The first human studies of environmental exposures and reproductive health outcomes were published last year in The American Journal of Epidemiology and The International Journal of Circumpolar Health. Researchers examined the association between reproductive outcomes and open dumps located near many Alaska Native villages. They found that women from villages with open dump sites that were considered most hazardous delivered babies who weighed less, were too small for their gestational age, were born too early, or had higher rates of some birth defects than did women living in villages near less dangerous dump sites (Gilbreath and Kass 2006a; 2006b).

Though we know little about what exposure or combination of exposures contributed to these outcomes, or exactly how women were exposed, this research spotlights the role exposures could play in reproductive outcomes experienced by women entering your clinic. It also suggests that some exposures relevant to reproductive health outcomes stem from local, rather than only global sources.

Relevant epidemiological studies of reproductive health outcomes among other circumpolar populations are ongoing (e.g., Northern Contaminants Program, Canada; Project INUENDO in Greenland, Sweden, Ukraine, and Poland) as is research with populations who eat traditional, subsistence foods. One recent study conducted among the Mohawk of New York and Canada, who also rely on subsistence foods, suggests exposure to certain types of PCB congeners and lead was associated with changes in the timing of girls’ menarche (Denham et al., 2005). As new studies are published, they will contribute to our developing understanding of how environmental factors may be influencing human reproduction and fertility in Alaska.

What Health Care Providers Can Do

1. Request and organize grand rounds or workshops on reproductive environmental health.
2. Share case studies at medical meetings.
4. Routinely collect environmental and occupational histories. For more information and suggestions about conducting an environmental history, consult Schettler, Solomon, Valenti, and Huddle’s Generations at Risk: Reproductive Health and the Environment (1999), which includes a primer for clinicians (Chapter 10).
5. Learn more about exposure sources and reproductive toxicity of chemicals and environmental pollutants:


   The National Library of Medicine’s Hazardous Substances Data Bank (toxnet.nlm.nih.gov)

To investigate potential exposure outcomes common in Alaska, clinicians also could reference:

   The Toxics Release Inventory maintained by the Environmental Protection Agency, which reports major sources of toxics by zip code (www.epa.gov/tri).

   Arctic Health, a resource portal maintained by University of Alaska Anchorage and the National Library of Medicine (www.arctichealth.org).

   Arctic Monitoring and Assessment Programme (www.amap.no).

   Alaska Native Traditional Knowledge and Native Foods Database, run by the Alaska Native Science Commission, the Institute of Social and Economic Research (ISER) and the University of Alaska Anchorage. (www.nativescience.org/html/arctic_contaminants.html)


   “The Collaborative on Health and the Environment (CHE) is a national, non-partisan partnership of individuals and organizations concerned with the effects of environmental contaminants on human and ecosystem health. CHE seeks to raise the level of scientific and public dialogue about the role of environmental contaminants and other

1 Hazard rankings of open dump sites determined by The Alaska Native Tribal Health Consortium.
environmental factors in many of the common diseases, disorders, and conditions of our time. CHE promotes interdisciplinary discussions, sharing of scientific evidence, outreach and education, and an agenda to bring about systemic change in improving environmental public health. Underlying all of CHE’s activities is a commitment to strong, uncompromised science. CHE believes truth emerges through discussion and that civility among CHE partners is a condition of honest dialogue and learning.” For more information visit: www.healthandenvironment.org

CHE-Alaska formed as a regional group in December 2005 following the Alaska Conference on Health and the Environment and invites participation from health care professionals, researchers, health-affected individuals and patient groups, students, advocacy organizations, and any other individual or group concerned about protecting the health of current and future generations from environmental harm. For more information, please visit: www.akaction.org

Additional Resources for Health Care Providers


American College of Preventive Medicine Environmental Health Resource Center http://www.acpm.org/education/environmentalhealth.htm

Association of Occupational and Environmental Health Clinics
1010 Vermont Avenue, NW, Suite 513
Washington, DC 20005
Website: http://www.aoeoc.org/
Phone: (888) 347-AOEC (2632)


Project INUENDO—Human Fertility at Risk from Biopersistent Organochlorines in the Environment. A research project supported by the European Commission. Examines couple fertility, semen quality and neonatal studies of reproductive hormone profiles among four populations in Greenland, Sweden, Poland and Ukraine. Website: http://www.inuendo.dk/

Physicians for Social Responsibility
Conducts trainings and workshops on environmental health for health care practitioners. Also, publishes ‘tool kits,’ pocket-sized, quick reference charts, and fact sheets on environmental health issues. Website: /www.psr.org
Also, for more resources, see Greater Boston Physicians for Social Responsibility at http://psr.igc.org

University of California at San Francisco, National Center of Excellence in Women’s Health, Program on Reproductive Health and the Environment. Website: www.ucsf.edu/coe/prhe.html


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This publication authored by: **Rebecca Gasior Altman**, Doctoral Candidate, Department of Sociology, Brown University

This publication has been peer-reviewed by:

**Dr. Birgit Lenger**, ND (Natural Health Center, LLC), Alaska Community Action on Toxics; **Ruth A. Etzel**, MD, PhD (George Washington University School of Public Health and Health Services); **Pamela K. Miller**, MEN, Vi Waghiyi, and Coleen Keane (Alaska Community Action on Toxics); **Ted Schettler**, MD., MPH (Science and Environmental Health Network), (Alaska Community Action on Toxics); **Rachel Morello-Frosch**, PhD, MPH (Brown University), Phil Brown, PhD (Brown University), Laura Senier, MA, MPH (Brown University), and the members of the Contested Illnesses Research Group (Brown University). The author thanks these individuals for their time and assistance.
Alaska Community Action on Toxics (ACAT) is a statewide environmental health and justice organization established in 1997. Our mission is to assure justice by advocating for environmental and community health. *We believe that everyone has the right to clean air, clean water, and toxic-free food.*

We help communities implement effective strategies to limit their exposure to toxic substances and to protect and restore the ecosystems that sustain them and their way of life.

We work to eliminate the production and release of harmful chemicals by industry and military sources, ensure the public’s right to know, achieve policies based on the precautionary principle, and support the rights of Indigenous peoples.

The Collaborative on Health and the Environment (CHE) is a national non-partisan partnership of individuals and organizations concerned with the role of the environment in human and ecosystem health. CHE seeks to raise the level of scientific and public dialogue about the role of environmental contaminants and other environmental factors in many of the common diseases, disorders, and conditions of our time. CHE promotes interdisciplinary discussions, sharing of scientific evidence, outreach and education, and an agenda to bring about systemic change in improving environmental public health. Underlying all of CHE’s activities is a commitment to strong, uncompromised science. We believe that the truth emerges through discussion and that civility among CHE partners is a condition of honest dialogue and learning. Visit us at: [www.healthandenvironment.org](http://www.healthandenvironment.org).

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Diabetes: The Role of Persistent Toxic Chemicals in this Complex Disease

While diabetes is rarely considered to be an environmentally-induced disease, there is increasingly strong evidence that environmental exposure contributes to the incidence of both Type I and Type II diabetes. Type I diabetes, which usually has onset in childhood, is an autoimmune disease with loss of the pancreatic beta cells and secondary lack of insulin. Type II diabetes, which usually has adult onset, is associated with a cellular resistance to the actions of insulin (Zimmet et al., 2001).

A study of the U.S. Air Force personnel who were exposed to dioxin-contaminated Agent Orange on Vietnam during the war found a highly significant relationship between exposure to dioxin and onset and severity of diabetes in those individuals with the greatest exposure (Henrikson et al., 1997). This prompted a committee of the National Academy of Sciences’ Institute of Medicine (IOM, 2000) to conclude that there was suggestive evidence of an association between dioxin exposure and diabetes.

Pesatori et al., 1998 and Bertazzi et al., 1998 found elevated diabetes in individuals exposed to dioxins at Seveso, Italy. A similar conclusion was drawn by Vena et al., 1998 from a study of phenoxyacid herbicides and chlorophenol production workers exposed to dioxins.

Cramer et al., 2000 studied a population of individuals exposed to dioxin from a Superfund site, and demonstrated that plasma insulin concentrations were significantly higher in individuals with elevated dioxin levels. The scientists concluded that high serum dioxin levels are associated with insulin resistance.

Longnecker et al., 2001 studied 2,245 pregnant women, 44 of whom had diabetes. The mean serum PCB level in the women with diabetes (3.77 ppb) was 30% higher than the controls (2.79 ppb). The relationship of PCB levels to the adjusted odds ratio (OR) for diabetes was linear. Taking PCB levels < 2.50 ppb to have an odds ratio of 1.0, the OR was 2.9 for PCB levels 2.50-3.75, 4.4 for PCB levels 3.75-5.00 and 5.1 for PCB levels greater than 5.0. All values were statistically significant, showing that increased exposure is associated clearly with increased risk.

Strong support for this relationship is also found in the Fierens et al., 2003 population-based study which showed that, after adjustment for age and other variables, that the total toxic equivalency factor (TEF) and 12-marker PCB concentrations were 62% and 39% higher, respectively than in controls. The ORs were 5.1 (95% confidence interval (CI) = 1.18-21.7) for dioxins, 13.3 (95% CI = 3.31-53.2) for coplanar PCBs and 7.6 (95% CI = 1.58-36.3) for 12 marker PCBs.

Vasiliu et al., 2006 investigated a Michigan cohort that had elevated exposure to polybrominated biphenyls (PBBs), and found a significant risk of diabetes with PCB concentrations (OR 2.33, 95% CI = 1.25-4.34), but not with PBB concentrations.

The strongest evidence for the relationship between PCB and other organochlorine exposure and diabetes has come from use of the US National Health and Examination Survey data. Lee et al., 2006 studied PCB 153, two dioxin congeners and three pesticide levels in relation to risk of diabetes in 2,016 adults. For PCB 153, the OR for individuals with serum levels in the greater than 90th percentile was 6.8 (95% CI = 3.0-15.5), and the p for trend was <0.001 with increasing concentration. Porta, 2006 stated: “Another striking finding in Lee and co-workers’ study is that there was no association between obesity and diabetes in individuals with non-detectable levels of persistent organic pollutants. Obesity was a risk factor..."
for diabetes only if people had blood concentrations of these pollutants above a certain level. This finding might imply that virtually all the risk of diabetes conferred by obesity is attributable to persistent organic pollutants, and that obesity is only a vehicle for such chemicals. This possibility is shocking.

Rylander et al., 2005 also reported a significantly elevated risk of diabetes in relation to concentrations of PCB 153 in Swedish fisherman and their wives.

Everett et al., 2007 independently analyzed similar data to that used by Lee et al., 2006 in determining diabetes risk for exposure levels of PCB 126 in 2,090 persons. PCB 126 levels >83.8 pg/g showed an OR for diabetes of 3.68 (95% CI=2.09-6.49) as compared to PCB 126 levels < 31.2 pg/g. The results of Lee et al., 2006 and Everett et al., 2007 are of particular interest because PCB 153 is not active at the dioxin receptor, whereas PCB 126 is. Therefore it may be that both dioxin-like and non-dioxin-like PCBs increase risk of diabetes.

In a follow-up study, Lee et al., 2007 found that the relationship with diabetes was much greater for the sum of four dioxin-like PCB congeners that it was for the sum of three dioxins, the sum of three furans, the sum of five non-dioxin-like congeners or the sum of four organochlorine pesticides.

Our research team, Codru et al., 2007, has recently reported that in a Native American population that is not particularly highly exposed to PCBs and pesticides, there is a significant relationship between tertiles of PCBs, hexachlorobenzene and DDE and risk of diabetes, identified either on the basis of having been diagnosed by a physician or by having a fasting blood glucose level of 125 mg/ml or higher. For total PCBs, the chance that an individual would have diabetes if their PCB levels were in the top tertile was 3.9 fold greater than that for individuals whose PCBs levels were in the bottom tertile after adjustment for gender, age, body mass index and smoking. For the two pesticides, the risks were even more elevated. However for mirex, a different organochlorine pesticide, there was no significant elevation in risk of diabetes with exposure. When individual PCB congeners were studied, the risk associated with PCB 74 (a congener most associated with consumption of contaminated fish) was 4.9 fold for the highest to lowest tertile.

Animal studies are consistent with the evidence that PCB and dioxin exposure increases risk of diabetes. Nishizume et al., 1995 showed that rats given Kanechlor-400 (a Japanese equivalent to Aroclor) showed depressed insulin sensitivity which increased with the duration of PCB exposure, as well as disturbed glucose and lipid metabolism and elevated serum lipids. Stahl, 1995 reported that dioxin alters enzyme activity related to glucose metabolism in rat liver cells. Several older studies have demonstrated morphological changes in the structure of the pancreatic beta cells upon PCB exposure (Kimbrough et al., 1972; Wassermann et al., 1975). Boll et al., 1998 demonstrated that gluconeogenic enzymes in rat liver are altered upon PCB exposure.

**CONCLUSION**

Diabetes is a serious, life-threatening disease that is increasing in frequency. It is usually ascribed to obesity and inactivity, along with genetic susceptibility. However, the growing body of evidence strongly indicates that exposure to persistent organic pollutants is important, and indeed may be a very important factor, in elevating risk for diabetes.

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**Cited References**


This publication authored by:

**Dr. David O. Carpenter, Director**
Institute for Health and Environment
School of Public Health
University at Albany, SUNY
Professor: Environmental Health Sciences
and Biomedical Sciences
Alaska Community Action on Toxics (ACAT) is a statewide environmental health and justice organization established in 1997. Our mission is to assure justice by advocating for environmental and community health. We believe that everyone has the right to clean air, clean water, and toxic-free food.

We help communities implement effective strategies to limit their exposure to toxic substances and to protect and restore the ecosystems that sustain them and their way of life.

We work to eliminate the production and release of harmful chemicals by industry and military sources, ensure the public’s right to know, achieve policies based on the precautionary principle, and support the rights of Indigenous peoples.

The Collaborative on Health and the Environment - Alaska

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Benzene

**What Is Benzene?**

Benzene is a colorless liquid with a sweet odor. It evaporates into the air very quickly and dissolves slightly in water. It is highly flammable and is formed from both natural processes and human activities.

Benzene is a natural part of crude oil, gasoline, and cigarette smoke. Natural sources of benzene also include volcanoes and forest fires.

Industrial use includes adding benzene with other chemicals to make plastics, resins, and nylon and other synthetic fibers.

Benzene is also used to make some types of rubber, lubricants, dyes, detergents, drugs, and pesticides.1

**How Are We Exposed?**

We are exposed to benzene in the following ways:

- Outdoor air contains low levels of benzene from tobacco smoke, automobile service stations, exhaust from motor vehicles, and industrial emissions.
- Air around hazardous waste sites or gas stations contains higher levels of benzene.
- Vapors (or gases) from products that contain benzene, such as glues, paints, furniture wax, and detergents, can also be a source of exposure.
- Working in industries that make or use benzene may also result in exposure.

**Symptoms & Health Outcomes**

- Breathing high levels of benzene can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, unconsciousness, and even death.2,3
- Eating or drinking foods containing high levels of benzene can cause vomiting, irritation of the stomach, dizziness, sleepiness, convulsions, rapid heart rate, and even death.4,5
- Long-term exposure to benzene has a major effect on the blood. Benzene causes harmful effects on bone marrow and can decrease red blood cells leading to anemia.6 It can also cause excessive bleeding and can affect the immune system, increasing the chance for infection.7,8,9
- Breathing high levels of benzene for many months has been associated with irregular menstrual periods and a decrease in the size of ovaries.10 It is not known whether benzene affects fertility in men.
- Benzene has been determined to be carcinogenic.11 Long-term exposure to high levels of benzene in the air can cause leukemia, particularly acute myelogenous leukemia (AML), a cancer of the blood-forming organs.12,13
- Benzene can pass from the mother's blood to a fetus.14 Animal studies have shown low birth weights, delayed bone formation, and bone marrow damage when pregnant animals breathed benzene.15
Follow Up Action

Several tests can show if a patient has been exposed to benzene:
- Conduct a test to measure benzene in the breath; this test must be done shortly after exposure.
- Benzene can also be measured in the blood; however, since benzene disappears rapidly from the blood, this test is only useful for recent exposures.

If you think your patient may been exposed to benzene:
- Refer patient to a physician.
- Chronic exposure can be addressed by treating the symptoms and informing patients about ways to limit future exposure to benzene (see “Reducing Your Exposure” section below).

Reducing Your Exposure

You can prevent or minimize exposure to benzene in the following ways:
- Whenever possible, limit contact with gasoline and cigarette smoke. Families are encouraged not to smoke in the home, in enclosed environments, or near children.
- Be aware of fumes from gasoline and from products that contain benzene such as paints, glues, furniture wax, and some detergents. When using these products, make sure that you are in a well ventilated area, such as outdoors or next to an open window or vent.
- Avoid idling vehicles in front of school doors or vent intakes.
- Keep the door between the garage (or other area where gasoline is stored) and the home shut tightly to decrease the inhalation of benzene from gasoline.

**Bisphenol-A (BPA)**

**What is Bisphenol-A?**
Bisphenol-A (BPA) is a high-volume production chemical used to make epoxy resin and polycarbonate plastic products, including some kinds of water bottles, baby bottles, and food storage and heating containers. It is also used in the lining of metal food cans and in dental sealants, and is an additive to certain plastics used in children’s toys. The chemical was first developed as a synthetic estrogen and was later polymerized to produce polycarbonate. Bisphenol-A mimics estrogen activity and is known as an “endocrine disruptor,” a chemical that interferes with the hormonal system in animals and humans and contributes to adverse health effects. New scientific evidence is emerging that demonstrates that exposure to bisphenol-A may affect multiple organs and systems within the bodies of animals and people.

**How Are We Exposed to Bisphenol-A?**
Humans are exposed to bisphenol-A on a daily basis through consumption of food and beverages contaminated with bisphenol-A, as well as environmental contamination. Polycarbonate plastic can become unstable over time and with use, allowing bisphenol-A to leach into material in contact with the plastic. Additionally, bisphenol-A is now found nearly everywhere in the environment and commonly found in dust particles, surface water and drinking water, as over 6 billion pounds are produced worldwide each year and the production of bisphenol-A releases approximately 2 hundred thousand pounds of the chemical into the atmosphere annually.

**Bisphenol-A in Our Bodies**
A recent study by scientists from the U.S. Centers for Disease Control and Prevention found that 92.6% of Americans now carry bisphenol-A in their urine at an average level of 2.6 µg/L. Although the United States Environmental Protection Agency (EPA) considers exposure to 50 µg/kg/day of bisphenol-A safe, this standard was set in 1993 and is based on studies from the 1980s. In a review of scientific literature on BPA conducted in 2005, researchers found numerous studies indicate a wide range of health effects from exposure to bisphenol-A at much lower doses (as low as 2 parts per billion in some studies) than considered “safe” by the EPA. In August 2007, over 30 scientific experts on bisphenol-A, known as the Chapel Hill panel, published a consensus statement in the peer-reviewed journal *Reproductive Toxicology*, stating significant evidence indicates adverse health effects occur in animals at levels within the range of exposure that is typical for humans living in developed countries.

In 2007, the Canadian Government began several studies of the effects of bisphenol-A on human health and the environment. They found that newborns and infants up to 18 months of age were exposed to levels of BPA similar to exposure levels that showed
effects in laboratory animals.\textsuperscript{11}

More recently, the US National Toxicology Program (NTP) Center for the Evaluation of Risks to Human Reproduction (CERHR) released the \textit{NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Bisphenol-A} which contributed significantly to the discussion of bisphenol-A’s health effects. This report identified evidence from animal studies which raised “some concern” that current exposure levels to bisphenol-A to human infants, fetuses, and children may result in developmental changes to the prostate gland and brain.\textsuperscript{12} Due to the panel’s concern about the effects of bisphenol-A on children under six, the NTP has teamed up with the CDC to investigate further the effects on bisphenol-A on young children, infants, and neonates.\textsuperscript{13}

\textbf{WHAT DOES EXPOSURE TO BISPHENOL-A MEAN FOR OUR HEALTH?}

While the majority of research on bisphenol-A has been conducted on animals and cell cultures, there is strong evidence that similar effects occur in humans. The Chapel Hill panel concluded in August 2007, “Based on existing data we are confident … the similar effects observed in wildlife and laboratory animals exposed to bisphenol-A predict that similar effects are also occurring in humans.” Moreover, research on estrogenic compounds all over the world is finding consistently that “animal studies of the effects of estrogenic substances are highly predictive of human impacts.”\textsuperscript{14}

Recent research has linked bisphenol-A to the following health effects:

\textbf{Endocrine disruption:}
As early as 1936, bisphenol-A was shown to be an environmental estrogen. Compared with natural estrogen, bisphenol-A is a less potent activator of the classic estrogen receptor, but in recent years it has been recognized that “BPA is equipotent with estradiol in its ability to activate responses via recently discovered estrogen receptors associated with the cell membrane,” as found in several studies on cell culture and laboratory animals. In addition to being shown to attach to estrogen receptors, evidence suggests that bisphenol-A also can change endogenous hormone synthesis, hormone metabolism and hormone concentrations in blood. Exposure to bisphenol-A has been shown to cause changes in tissue enzymes and hormone receptors as well as interacting with other hormone-response systems.

\textbf{Recurrent miscarriage:}
Researchers found that women with a history of recurrent miscarriage had average blood serum levels of bisphenol-A at 2.59 ng/ml, more than three times higher than women with successful pregnancies,\textsuperscript{15} a finding predicted by previous animal studies.\textsuperscript{16}

\textbf{Altered mammary gland development:}
In a laboratory study, mammary gland development was significantly altered in mice exposed to 250 ng BPA/kg body weight/day of bisphenol-A,\textsuperscript{17} which is 2000 times lower than the EPA’s safety standard for bisphenol-A (0.5 mg/kg/day). Scientists suggest that this study’s implications for human health include increased susceptibility to breast cancer after perinatal exposure to bisphenol-A.

Low doses of bisphenol-A have also been shown to decrease the effectiveness of chemotherapeutic agents used for breast cancer.\textsuperscript{18}

\textbf{Prostate cancer:}
Research using cell cultures showed that a concentration of bisphenol-A of 1 nM made
prostate cancer cells less responsive to the hormone treatment used to control prostatic adenocarcinomas into remission.\textsuperscript{19} Whether this cell culture impact also occurs in people is uncertain, but the concentration is lower than the average level of bisphenol-A found in Americans, as reported in 2008.\textsuperscript{7}

**Altered brain development and behavior:** Scientists found that bisphenol-A exposure in the womb changes sexual differentiation of the brain and behavior in rats at only 30 µg/kg/day,\textsuperscript{20} lower than the dose considered safe by the EPA.\textsuperscript{8} For some behaviors tested, results suggest that bisphenol-A exposure was linked to both demasculinization of males and defeminization of females.

At doses considered safe by the EPA, bisphenol-A blocked the synapse of estradiol in the hippocampus and prefrontal cortex of rodents and non-human primates.\textsuperscript{21} This signaling pathway may play a critical role in mood and cognition for humans.

**Insulin resistance and cardiovascular diseases:** A recent study in adult mice provided evidence of a link between bisphenol-A exposure and increased risk of type II diabetes, hypertension and dyslipidemia.\textsuperscript{22} In this study, scientists found that chronic exposure to low doses of bisphenol-A leads to insulin resistance in adult mice. Doses used in their experiments were 5 times lower than the dose considered safe by the EPA.\textsuperscript{8}

Based on population-wide exposure data from 2003-2004, scientists examined links between urinary bisphenol-A levels and chronic diseases in adults between the ages of 18 and 74.\textsuperscript{23} They found high urinary levels of bisphenol-A are linked to cardiovascular diagnoses. Adults in this study who had high urinary levels of BPA were also more likely to have diabetes.

**Developmental origins of adult health and disease:** The 2007 “Chapel Hill Bisphenol A Expert Panel Consensus Statement: Integration of Mechanisms, Effects in Animals and Potential to Impact Human Health at Current Levels of Exposure” states that enough evidence exists to suggest that adverse health outcomes may not become apparent until after exposure during critical developmental periods has happened. Especially of concern is that “these developmental effects are irreversible and can occur due to low-dose exposure during brief sensitive periods in development, even though no BPA may be detected when the damage or disease is expressed.”

Disruption of hormone signaling pathways by BPA during critical periods of development also may lead to obesity.\textsuperscript{24}

**Regulations for Bisphenol-A**
Federal regulation of toxic chemicals is an important part of protecting public health. However, according to the Environmental Working Group, “The nation's system of regulations for industrial chemicals like [bisphenol-A] are embodied in the Toxic Substances Control Act, a law passed in 1976, and the only major environmental or public health statute that has never been updated.”\textsuperscript{25} Furthermore, “under this law, companies are not required to test chemicals for safety before they are sold, and are not required to track whether their products end up in people or the environment at unsafe levels.” To date, the U.S. Food and Drug Administration has not performed a standard toxicology study or determined an Acceptable Daily Intake (ADI) for bisphenol-A.\textsuperscript{26}

Policies are currently being considered on a federal level that prohibit the sale of products that contain bisphenol-A.\textsuperscript{27} On March 13, 2009, leaders from the House of Representatives and the Senate announced legislation which would ban the use of
bisphenol-A in all food and beverage containers.\textsuperscript{28}

Several states have also introduced legislation which would limit the sale of bisphenol-A, especially in children's products. In 2008, Maine enacted the Toxic Chemicals in Children's Products Act which will phase out the use of chemicals, including BPA, that have been scientifically proven to cause cancer, reproductive toxicity, developmental toxicity, or endocrine disruption.\textsuperscript{29} In March 2009, Canada became the first nation to restrict the use of bisphenol-A in children's products.\textsuperscript{30}

**Reducing Your Exposure**

You can prevent or minimize exposure to bisphenol-A in the following ways:

- Use glass, stainless steel, or polyethylene bottles (PETE, PET, or \#1; HDPE or \#2; LDPE or \#4) instead of polycarbonate (PC or \#7) bottles.\textsuperscript{24}
- Avoid using polycarbonate containers to store hot foods or to heat foods in microwaves, as bisphenol-A tends to leach faster with higher temperatures.\textsuperscript{25} Use glass or ceramic containers instead.
- Cut back on consumption of canned foods to reduce exposure to bisphenol-A contamination from the interior coating of the container. Also, avoid canned foods with higher fat content, which may have higher levels of bisphenol-A.\textsuperscript{25}
- Before getting dental sealants, check with your dentist about the ingredients in the products they use, as some formulations may leach bisphenol-A.\textsuperscript{25}


Cyanide

WHAT IS CYANIDE?
Cyanide can appear in several forms, such as a liquid, gas, powder, or as crystals/salts. It has a faint, bitter, almond-like odor. It can be found naturally in some foods, plants, and can be in the smoke of burning wood, paper, plastics and manmade fibers.\(^1\)

Cyanide is used in industry, including gold and silver mining, electroplating, production of plastics, manmade fibers, colors and dyes, pesticides, developing photos and as a fumigant to get rid of pests in ships.\(^1\) The use of cyanide in gold mining is controversial. Some states, such as Montana, and some cities have entirely banned all the uses of cyanide in mining.\(^2\)

Cyanide is toxic in small amounts. Cyanide in water and soil is a health hazard to people and animals.\(^2\)

Mining can contaminate surface streams, wetlands, rivers, and groundwater downstream. Cyanide reacts to form other compounds that are toxic and persistent. Fish, plants and other living organisms are sensitive to the effects of cyanide and its associated compounds.

HOW ARE WE EXPOSED?
The three most common ways that people may be exposed to cyanide are:

- **Fire from a burning building:** Inhalating smoke from a building fire is one of the most common ways that people can be poisoned by cyanide. Hydrogen cyanide may be produced during fires when products such as wool, paper, cotton, silk, and plastics are burned.

- **Cyanide-related industry:** People can be exposed to cyanide through breathing air, drinking water, touching soil, or eating foods contaminated with cyanide through industrial operations such as mining. Accidents during the transportation of cyanide for mining may also be a source of exposure.

- **Tobacco use:** Smoking tobacco is a major source of cyanide exposure.\(^3\)

SYMPTOMS & HEALTH PROBLEMS
Cyanide is a poison if a person swallows it in water or food, breathes it in, or contacts it with skin:\(^1\)

- **Swallowing cyanide:** Depending on the amount swallowed, the first signs of cyanide poisoning could be deep breathing and shortness of breath, followed by convulsions (seizures) and loss of consciousness.\(^1\) It can later cause apnea (a condition when a person stops breathing or breathes intermittently).

- **Breathing:** Inhalation of small amounts of cyanide may cause headache, dizziness, weakness, and vomiting. Inhalation of larger amounts may cause gasping, irregular heartbeat, seizures, fainting, and rapid death.\(^1\)

- **Skin contact:** When cyanide comes in contact with skin, the symptoms are the same as when it is inhaled.\(^1\)

- **Long-term health effects of exposure to cyanide:** Survivors of
serious cyanide poisoning may develop heart and brain damage.³

**FOLLOW UP ACTION**
- If you think your patient may have been exposed to cyanide, refer patient to a physician.

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**Reducing Your Exposure**

You can prevent or minimize exposure to cyanide in the following ways:
- Try to quit smoking.
- Avoid breathing in tobacco smoke whenever possible.
- Evacuate a burning building immediately; smoke from burning plastics contains cyanide (and carbon monoxide).
- Do not burn plastics. Cyanide in smoke can arise from the burning of certain plastics.
- Although it may be uncertain how far contaminants may spread around mine sites, it is advisable to have water and traditional food sources close to, downstream or downwind of these sites tested for the possibility of harmful exposures. If you have concerns about the safety of your water or traditional foods, contact ACAT or visit our website at www.akaction.org.

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Environmental Tobacco Smoke (ETS)

**What Is Environmental Tobacco Smoke (ETS)?**
Environmental tobacco smoke (ETS), or secondhand smoke, is released into the air from exhaled tobacco smoke and burning cigarettes, cigars, and pipes. Tobacco smoke contains over 4,000 chemicals, many which are poisonous.¹

There is new evidence that people are exposed to chemicals from tobacco smoke long after a person has smoked in a given indoor area.²,³,⁴,⁵ Thirdhand smoke is a new term for tobacco toxins that remain and persist after a period of active smoking. These chemicals are deposited on surfaces such as tables, furniture, and floors, as well as in dust. Thirdhand smoke also volatizes and contaminates air for days, weeks, and even months after the smoking has ceased.²

**How Are We Exposed?**
- Smoking cigarettes
- Inhaling secondhand smoke
- Smoking indoors, which may increase exposure, especially in poorly ventilated areas⁶
- Inhalation of contaminated indoor air or contact with surfaces contaminated with thirdhand smoke²

**Symptoms & Health Problems**
Children exposed to secondhand smoke may experience the following short-term health effects:⁷
- Upper and lower respiratory infections⁸

**Follow Up Action**
- Refer to the Community Health Aide Manual (CHAM) section, “Stopping Tobacco” for detailed information on helping patients quit smoking. Patients are more likely to quit with the advice and support of their health care providers.⁷
- If the patient needs medications to quit or is pregnant, breastfeeding, or a teenager, refer patient to a physician.

- Otitis media with effusion, or fluid in the middle ear⁹,¹⁰,¹¹
- Sudden infant death syndrome (SIDS)¹²,¹³,¹⁴
- Worsening of asthma⁸,¹⁵,¹⁶,¹⁷
- Bronchitis¹⁸,¹⁹
- Pneumonia¹⁸,¹⁹
- Impaired growth and development²⁰,²¹,²²

Children exposed to secondhand smoke may experience the following long-term health effects:⁷
- Reduced lung function²³,²⁴
- Higher risk of asthma²⁵
- Higher risk of cancer⁸,²⁶

The following health effects have been linked to exposure to secondhand smoke in adult non-smokers:⁷
- Higher risk of lung cancer⁸
- Higher risk of coronary heart disease¹⁸,²⁷
A new study shows that adults who are aware of the harms to children from thirdhand smoke are more likely to have a no-smoking rule in the home.\(^2\) Thus, incorporating the idea of thirdhand smoke into tobacco counseling messages could discourage smoking in the home.

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**REducing Your Exposure**

You can prevent or minimize exposure to tobacco and secondhand smoke in the following ways:

- Try to quit or cut back on smoking to improve your health and the health of your loved ones.
- Avoid smoking around other people to reduce their exposure to secondhand smoke.
- Avoid smoking indoors, especially in areas that are not well ventilated, to reduce secondhand and thirdhand smoke.
- Non-smokers can reduce their exposure to secondhand smoke by avoiding indoor areas where people smoke or by starting a no-smoking rule in their homes or workplaces.
- Parents who smoke should cover their clothes with a clean jacket or shirt before picking up or holding a baby.

Both smokers and nonsmokers may reduce their risk of cancer in the following ways:

- Eat dark berries (such as blueberries and crowberries) and other foods high in flavonoids daily. The National Cancer Institute reports that foods containing flavonoids (particularly onions and apples) may reduce lung cancer risk by nearly 50 percent.\(^2\) Freezing berries is even better, as it bursts the cell membrane and increases the body’s ability to absorb and use these nutrients.
- Eat foods high in lutein, such as leafy greens (including kale and spinach), carrots, corn, red peppers, rosmarinette, tomatoes, potatoes, and red, blue and purple fruits. Foods containing lutein are linked to a lower risk of lung, breast and colon cancers.\(^2\)
- Consider drinking green tea, which contains high amounts of antioxidants and is less processed than black tea.\(^2\) Many studies show that green tea protects against cancer development.\(^30\) Smokers who drink green tea have shown decreased oxidative DNA damage, which is a precursor to cancer.\(^33\) Green tea may also fight cancer in people who have already been diagnosed, for instance by preventing breast cancer from coming back\(^34\) and helping ovarian cancer survivors to live longer.\(^35\) When preparing green tea, steep for at least 5 minutes to make sure that a greater concentration of antioxidants are released, and avoid adding milk or cream which may inactivate the antioxidants.\(^2\)

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Mercury and Gold Mining

**What is Mercury?**
Mercury is a heavy metal that is found naturally in different forms in the environment. Major sources of mercury emissions in the United States come from coal-fired power plants, municipal and medical waste incinerators, and mining operations.\(^1,2\)

The process of gold mining releases large amounts of mercury into the environment which results in mercury contamination. One way that mercury can be released into the environment from gold mining operations is from the use of cyanide to remove gold from the ore in which mercury is found. This process also removes mercury, which is then released into the environment.\(^3\) In the past, mercury itself was commonly used to remove gold, but in the United States this method has largely been replaced by cyanide removal methods (described above), with the exception of some recreational mining.\(^2\) Mercury can also be released into the environment from a number of other mining operations, such as heap leach operations, tailings facilities, waste rock piles, and ore processing methods involving heat.\(^3\)

When mercury is present in water bodies and sediments, bacteria convert it into methylmercury, a compound that builds up through the food chain in fish, marine mammals, and humans.\(^4\)

Mercury (in its elemental form) can also be found in medical equipment such as sphygmomanometers (blood pressure meters), thermometers, and silver-colored dental amalgams used for fillings. Thermostats, fluorescent light bulbs, and some batteries may also contain mercury. These items may contaminate the environment if thrown away improperly in a landfill or incinerator.

**How Are We Exposed?**

**Methylmercury:**
- Methylmercury builds up in muscle tissue, commonly causing exposure to happen from eating large, predatory fish, marine mammals, and wildlife at the top of the aquatic food chain such as polar bears.\(^2,4,5\)
- All species of wild Alaska salmon are very low in mercury levels, so salmon is not a major source of mercury exposure.\(^6\)
- Methylmercury is also passed to fetuses through the placenta and to nursing infants through breastmilk.\(^4\)

**Elemental Mercury:**
- Exposure to elemental mercury usually happens through inhalation of contaminated air near mines, hazardous waste sites, landfills, and other dump sites containing light bulbs, batteries, and medical waste.\(^4\)
- Mine workers may be exposed to mercury by breathing mercury vapor.\(^3\)
- Exposure to elemental mercury may happen in the home and other indoor areas due to broken fluorescent light bulbs.
Elemental mercury is only minimally absorbed after eating contaminated items or through the skin.\(^4\)

**Symptoms & Health Outcomes**
- Mercury is a known mutagen (an agent that changes, or mutates, genetic material) and teratogen (an agent that disrupts fetal or embryonic development) and a suspected carcinogen.\(^2\) While the EPA has not classified elemental mercury as a human carcinogen, based on “inadequate human and animal data,”\(^7\) it has determined that methylmercury is a possible human carcinogen.\(^8\)
- Methylmercury is highly toxic and is linked to a variety of adverse health effects on the central nervous system, including visual and hearing impairment, tremors, and muscle spasms.\(^4\)
- One study found that prenatal methylmercury exposure through mothers’ regular consumption of fish and marine mammals resulted in deficits in language, attention, and memory in children,\(^9\) even after researchers controlled for co-contamination by PCBs.\(^10\) (*Please see the Polychlorinated Biphenyls [PCBs] fact sheet for more details.*)
- There is strong evidence that mercury exposure is linked to diseases such as cerebral palsy; impaired learning, memory, coordination, and attention span; mental retardation; hearing loss; dermatitis; psychiatric disturbances; seizures;\(^11\) and acute bronchitis and pneumonitis.\(^12\)
- Early signs of long-term exposure to elemental mercury may include the following nonspecific symptoms of the central nervous system: insomnia, forgetfulness, loss of appetite, and mild tremor. These symptoms may be misdiagnosed as psychiatric illness.\(^4\)
- The Alaska Mercury Biomonitoring Program has tested over 350 women, including women from 6 villages in the Norton Sound region and Nome. According to their results, none of these women had hair mercury levels higher than the World Health Organization’s guideline of 14 parts per million, which is considered the lowest level linked to adverse health effects on the fetus.\(^13,14\)

**Follow Up Action**
- If you think your patient may have been exposed to mercury, refer patient to a physician.
- Recommend breastfeeding to postpartum patients.\(^4\) In communities at risk of chemical exposures, it is even more important that mothers breastfeed their babies because breast milk reduces the effects of the chemicals on infants and makes them healthier.\(^15,16,17,18\)
- Support eating traditional subsistence foods, unless you have information that indicates mercury or other contamination.
- The Alaska Department of Department of Health and Social Services, Division of Public Health (DPH) has offered the Statewide Maternal Hair Mercury Biomonitoring Program since 2002, which now provides free and confidential testing of mercury levels in hair for all Alaskan women of childbearing age (15-45 years old).\(^13\) Encourage women of child-bearing age to participate in the Alaska Mercury Biomonitoring Program. Information can be found online at [http://www.epi.hss.state.ak.us/eh/biom](http://www.epi.hss.state.ak.us/eh/biom).
**You can prevent or minimize exposure to mercury in the following ways:**

- **Eat fish with lower mercury levels, such as all species of wild Alaska salmon.** The Alaska Department of Department of Health and Social Services, Division of Public Health (DPH) recommends eating as much as you want of all species of wild Alaska salmon, as well as pacific cod, walleye pollock, black rockfish, pacific ocean perch, halibut under 20 pounds, lingcod under 30 inches length, and canned chunk light (not white) tuna.6

- When eating ocean fish, consider eating smaller, younger fish whenever possible, as older fish tend to build up more mercury over time.5 The DPH recommends eating less of the following fish which are known to have high mercury levels: sablefish, halibut larger than 20 pounds, lingcod greater than 30 inches length, rougheye rockfish, yelloweye rockfish, salmon shark, and spiny dogfish. For detailed recommendations, see the DPH full report here: http://www.epi.alaska.gov/bulletins/docs/rr2007_04.pdf or call the DPH Section of Epidemiology at 907-269-8000.6

- **Do not burn products that may contain mercury, such as thermometers, thermostats, fluorescent light bulbs, and batteries.**
  - For instructions on recycling fluorescent light bulbs, please see the Solid Waste Alaska Network (SWAN) fact sheet here: http://www.ccthita-swan.org/pdf/fluorescent.pdf or call 800-344-1432, ext. 7184
  - For information on recycling batteries, please see the Solid Waste Alaska Network (SWAN) webpage on battery recycling here: http://www.ccthita-swan.org/Tutorials/batt_recycle.cfm or call 800-344-1432, ext. 7184

- **For instructions on safe clean-up of broken fluorescent light bulbs, please see the ENERGY STAR fact sheet here:** http://www.energystar.gov/ia/partners/promotions/change_light/downloads/Fact_Sheet_Mercury.pdf or call the ENERGY STAR Hotline at 888-STAR-YES (888-782-7937).

- In the event of spills, do not allow children or teens to play with liquid mercury.

- Although it is uncertain how far contaminants may spread around mine sites, it is advisable to have water and traditional food sources close to, downstream or downwind of these sites tested for the possibility of harmful exposures. If you have concerns about the safety of your water or traditional foods, contact ACAT or visit our website at www.akaction.org.

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Mold

**WHAT IS MOLD?**

- Mold is a type of fungus that grows in moist areas on natural forms of material, including food, fabrics, carpets, upholstery, walls, and ceiling tiles. Mold usually will grow in damp environments such as basements, kitchens, bathrooms, air conditioning units, refrigerators, and potted plants. Sewage leaks can cause toxic molds or "black mold."

**HOW ARE WE EXPOSED?**

- Mold exposure occurs through inhalation and skin contact with affected surfaces.
- Poor indoor ventilation and air quality may cause mold exposures.

**SYMPTOMS & HEALTH PROBLEMS**

- Mold exposure can lead to respiratory symptoms including rhinitis, sneezing, eye irritation, coughing and wheezing.
- Children and adults may experience allergic reactions or worsening of asthma symptoms from mold exposure.
- Mycotoxins are toxic chemicals produced by molds. Exposure to mycotoxins in the air is linked to pulmonary bleeding (bleeding in the lungs), sudden death in some reported infant cases, and organic dust toxic syndrome (ODTS) in some documented adult cases.
- Mycotoxins are also associated with some kinds of cancer.

**FOLLOW UP ACTION**

- If you think your patient may have been exposed to mold, report to Public Health Nurse (PHN) or refer patient to a physician.

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**REDUCING YOUR EXPOSURE**

You can prevent or minimize mold growth and subsequent exposure in the following ways:

- Keep indoor environments dry. Within 24 hours of a leak or flood, clean up all water and remove water-damaged items. Keep indoor humidity levels between 30-40%.
- Properly ventilate indoor environments.

To remove mold growth:

- Wearing protective gloves and a dust mask, clean affected areas with soap and water, then apply a solution of 1 part vinegar and 1 part water, and wipe clean.
- Make sure to ventilate the area by opening windows and turning on ceiling and exhaust fans.
- Toxic mold removal may require professional help.


WHAT ARE PESTICIDES?
Pesticides are a group of chemicals made for the purpose of killing or otherwise deterring “pest” species. The word pesticide may refer to insecticides, herbicides, fungicides, or other pest control methods. Because pesticides are made to be toxic, they inherently have adverse health effects on animals and humans—even at low levels—and can stay in the environment after the being applied. There are many different types of pesticides made for different results.

HOW ARE WE EXPOSED?
Because of the many types and uses of pesticides, there are many ways people can be exposed to these chemicals:
- Living or working near areas where pesticides are used can be a risk factor for exposure.
- Wind and rain may move pesticides away from where they were used, causing contamination of groundwater and/or soil.
- Using pesticides in your home or on your land increases your exposure to these chemicals through inhalation, ingestion and absorption through skin.
- Use of insect repellents on the body increases exposure to pesticides, because pesticides can enter the body through the skin.
- Eating contaminated foods can increase exposure, as some pesticides are carried long distances from where they are made and used by air and ocean currents into the north and known to build up in the fats of Arctic animals.
- Farmers and field workers can be exposed to high levels of pesticides.

SYMPTOMS & HEALTH OUTCOMES
The health effects of pesticide exposure can be different based on the specific pesticide and the level of exposure. Different pesticides will result in a range of symptoms. Included here are the symptoms of pesticide exposure for a few of the most common pesticide groups, and specific pesticides within those groups.

Organophosphates (e.g. Malathion):
Organophosphate pesticides are among the most commonly used pesticides.

Prenatal exposure to organophosphates has been linked to abnormal brain development that disrupts cognitive function and memory and persistent abnormal behavior. In humans, prenatal exposure has been linked to abnormal reflexes, delayed development, and an increase in attention deficits and hyperactivity.

Malathion is an organophosphate insecticide commonly used to control insects in homes, outside, and in farming. Malathion is both acutely and chronically toxic to mammals. Studies of malathion in many animals have shown it binds to and is stored in fat. This means malathion may build up in the fats of animals, and people may be exposed by eating these animals. Current research suggests malathion could contribute to the
symptoms of both type 2 diabetes and atherosclerosis, or clogged arteries. Malathion has been found to disrupt lipid metabolism in rats. Rats treated with malathion had significantly increased low density lipoproteins and plasma triglycerides, in a way that suggests it may contribute to atherosclerosis. Malathion and other organophosphate pesticides have been found in breastmilk. Measurement of urinary malathion levels showed that malathion was found more often and at higher levels in children than in adults.16

**Organochlorines (e.g. DDT and Endosulfan):**
Organochlorine pesticides are a family of agricultural pesticides that are known to break down slowly in the environment and accumulate in the fatty tissues of animals. Many studies have linked organochlorine pesticide exposure with consumption of contaminated animals, mostly fish and marine mammals.17

Many organochlorine pesticides are endocrine disruptors. Numerous studies have linked organochlorine pesticide exposures with cancers and other health effects. Exposure to DDT has been linked to pancreatic cancer and non-Hodgkin’s lymphoma.21 Endosulfan is an organochlorine pesticide that is increasingly used worldwide and is already building up in the environment.22 Endosulfan has been shown to compete with estradiol for binding with an estrogen receptor. Studies of toad tadpoles showed that levels of endosulfan found in the environment were enough to delay development, increase mouth and skeletal abnormalities, and increase mortality.23 Endosulfan exposure has been shown to act like estrogen and reduce the levels of testosterone and other hormones in rats.24 Endosulfan exposure has been linked to delayed sexual maturation in boys.25 Endosulfan exposure has been found in placenta samples and umbilical cord blood, suggesting endosulfan in the mothers’ body may enter developing fetuses.29

**Pyrethroids (e.g. Permethrin):**
Man-made pyrethroids are a type of insecticides made to copy the action of the natural plant insecticides called pyrethrins. Pyrethrins have relatively low toxicity for mammals compared to some pesticides. This has prompted the use of pyrethroids as a public health measure to control mosquito vector populations and used widely in farming, as well as for lice and flea treatments.

Although pyrethroids are generally thought to be a relatively low-toxicity pesticide, they are still toxic to humans and produce many adverse health effects. Excessive pyrethroid exposure often results in temporary numbness and tingling in the exposed body part that goes away within several hours. In animals, exposure can cause a slight whole body tremor, twitching of the dorsal muscles, and even death. Permethrin also causes an increase in the metabolic rate which, along with tremor, causes hyperthermia and can lead to death.

There is also evidence that permethrin is an endocrine disrupting chemical, meaning that it can interfere with the hormone system. One study found that permethrin had endocrine disrupting action on human endometrial cells and breast cancer cells. A later study confirmed that permethrin, as well as three other pyrethroids, increased the growth of human breast carcinoma cells. This suggests that pyrethroids may aid in the progression of some cancers. There is other evidence that the metabolic breakdown products of permethrin have greater endocrine disrupting potential than permethrin itself. Exposure to permethrin and cypermethrin (another pyrethroid) as well as organophosphates pesticides reduced healthy sperm concentrations in exposed men. There is evidence that the full range of pyrethroid toxicology is not
fully understood, and that precautions should be taken to limit human exposure.\textsuperscript{33}

**DEET**

N,N′-Diethyl-m-toluamide, also known as DEET, is one of the most commonly used pesticides. In addition to direct exposure from insect repellent application, DEET is a common contaminant in surface waters.\textsuperscript{38}

DEET can enter our bodies through the skin quickly and circulate in our bodies.\textsuperscript{4} “DEET exposures have been associated with death, toxic encephalopathy, acute manic psychosis, seizure, and cardiovascular and dermal toxicities.”\textsuperscript{39} There is evidence that DEET exposure kills brain cells, and that DEET and malathion together may have greater effects regarding destruction of brain tissue.\textsuperscript{40}

These toxic effects are especially prevalent in children, because they absorb DEET more easily through the skin and have a larger ratio of skin surface area to body mass as compared to adults.\textsuperscript{41} Excessive use and over-application of DEET with small children could cause harmful exposures.\textsuperscript{42}

**Carbamates (eg, Carbaryl):**

Carbamate pesticides are usually used in farming or in home lawns and gardens. Carbamate pesticides work by targeting cholinesterase, the enzyme that breaks down acetylcholine, an important neural hormone. In insects, this causes a subsequent build up of acetylcholine, malfunction of the nervous system, and eventually death.\textsuperscript{43} In humans, carbamate pesticides can have a similar effect on the nervous system.

In addition, carbamates seem to have negative effects on steroidal molecule metabolism, which may contribute to some cancers.\textsuperscript{44,45} Carbaryl exposure has been linked to higher risk for non-Hodgkins lymphoma.\textsuperscript{46} A significant increase in kidney and liver tumors was seen in rats exposed to carbaryl in the laboratory.\textsuperscript{47} Rats prenatally exposed to carbamates had impaired development of reflexes.\textsuperscript{48}

**FOLLOW UP ACTION**

- If you think your patient may have been exposed to pesticides, refer patient to a physician.
- Chronic exposure can be addressed by treating the symptoms and informing patients about ways to limit future exposure to pesticides (see “Reducing Your Exposure" section below).
REDUCING YOUR EXPOSURE

You can prevent or minimize exposure to pesticides in the following ways:

- Wash fruits and vegetables, including local berries and greens, before eating to remove traces of pesticides.
- Safe alternatives can be used to solve nearly all pest problems without the need for toxic chemicals. We recommend prevention and use of non-toxic alternatives first. For example:
  - Citronella oil can be used instead of DEET to keep mosquitoes away.
  - Wear long-sleeved shirts made of thick fabric (such as denim or canvas) and wear head nets and other nets to keep mosquitoes from landing on skin.
  - Install screen doors and windows to keep mosquitoes out.
  - Use mousetraps instead of poison bait.
  - Burning of alder branches at fish camp helps to keep them out of the area.
  - Peppering fish during processing and drying can keep flies away.
  - You can prevent pests from entering your home by sealing cracks in windows, doors and baseboards.
  - Storing food properly and cleaning food or beverage spills will help to keep out unwanted pests from your home.
  - Lice combs and mayonnaise or olive oil can be used instead of chemical shampoos to treat head lice.
- If DEET has been used on children, make sure they bathe right away after they come home from the outdoors. DEET should be washed off the skin, especially before reapplication.
- If possible, reduce or eliminate the use of pesticides in and around your home or workplace.
- If you are exposed to pesticides at work, take actions to reduce the amount of pesticide on your clothes and possessions when you go home. Consider leaving contaminated clothes and tools at work or outside, removing shoes and outerwear before entering your home, and bathing at work or soon after you return home.
- Whenever possible, buy organic fruits and vegetables and other organic products to reduce your exposure.
- Consider eating less fat, as some pesticides build up in the fat of animals.
  - Consider removing fat that you see on meat and fish whenever possible.
  - Consider eating leaner meats whenever possible.
  - Consider eating smaller amounts of dairy products; or, if available, choose low fat or nonfat dairy products.


Petroleum Products and Products of Incomplete Combustion

What are Petroleum Products?
Petroleum products come from crude oil, and their by-products contribute to air pollution and global warming. They are used as fuel for automobiles, airplanes, snow machines, ATVs, cooking stoves, generators, furnaces, and more.

How Are We Exposed?
Breathing vapors and exhaust of petroleum products is a major source of exposure. Petroleum products contribute to air pollution, a serious public health problem which kills about 70,000 Americans each year.

Exposure to vapors can occur when filling fuel tanks for vehicles, stoves, or houses. Soil contaminated with petroleum products or fuel spills may also release toxic vapors. If a house or building is located over an area where there has been an oil spill, vapors can enter the building through cracks in the walls and floors, as well as places where pipes enter the building.

People are exposed to exhaust from petroleum products through traffic, ATVs, snow machines, storing vehicles in a garage that is attached to a house, using stoves with no ventilation, and living near petroleum refineries or power plants.

Drinking water contaminated by petroleum products is another source of exposure. When gasoline seeps into soil, it can contaminate groundwater used for drinking. Most chemicals in gasoline and other petroleum products are removed during water treatment, but people who drink untreated water or water from private wells can be exposed.

Other possible sources of exposure to petroleum products include:
- Accidental intake of petroleum products: children may swallow petroleum-based fuels by accident when, for example, they are stored in a familiar container such as a soda bottle;
- Touching petroleum products, soil or water contaminated by petroleum products;
- Swimming in water contaminated by petroleum products;
- Using fuel oils to wash paint or grease from skin or equipment;
- Fixing gasoline or diesel engines;
- Cleaning up fuel spills without proper protective equipment;
- Working in the oil and gas industry;
- Having served in the Persian Gulf War: many veterans have reported exposures to diesel fuel and oil fires;
- Some people also inhale gasoline or diesel fumes in order to become intoxicated. This is known as “huffing” or “sniffing” and is done more often by children and adolescents than adults. According to a 2004 study, gasoline was the most commonly abused volatile substance in the United States.

Symptoms & Health Outcomes
The health effects of exposure to petroleum products can differ based on the specific chemical and the level of exposure.
Different petroleum products will result in a range of symptoms. Included here are the symptoms of exposure for a few of the most commonly used petroleum products.

**Benzene**
Benzene is a petrochemical, meaning it is a refined product of oil. It is present in the environment in water, soil and air. Some of the major sources of benzene are car exhaust, paint, and glues. Cigarette smoke also contains benzene from the burning of tobacco. Exposure to benzene through food, water, and soil is not common, unless there is a local source of contamination. Approximately 95% of exposure to benzene is through inhalation. Acute exposure can cause dizziness, rapid heart rate, tremors and unconsciousness.

Benzene is known to cause cancer. The most significant health risk to low level exposure to benzene is leukemia. However, research suggests that benzene can induce other diseases of the blood, such as anemia, and uncontrolled bleeding. Benzene may also cause reproductive effects such as irregular menstruation and birth defects. (Please see the Benzene fact sheet for more details.)

**Gasoline**
Gasoline is a complex mixture of volatile hydrocarbons derived by distillation from crude petroleum. More than 100 billion gallons of gasoline are used annually in the US. Gasoline combustion is an important contributor to air pollution around us and global warming.
- Severe ingestion can result in central nervous system toxicity, visceral involvement, and inflammation of the lungs which can lead to respiratory arrest.
- Less serious nervous system effects include dizziness and headaches, while more serious effects include coma and the inability to breathe.
- Long-term occupational exposure to gasoline vapors is linked to kidney and nasal cancer. It can also result in damage to the nervous system.
- Chronic exposure to combustion products of gasoline is linked to an increased risk for cancer.

**Diesel Exhaust**
Diesel exhaust is produced by motor boats, heavy equipment, generators, trucks, buses, and trains. These engines release emissions that severely compromise air quality.

Inhaling diesel exhaust particles (DEPs) can induce inflammation of the respiratory tract, and increase the incidence of wheezing, bronchitis, and asthma hospitalization. Allergic and inflammatory response to DEPs may facilitate development of allergies to other irritants. Symptoms of existing asthma or allergic rhinitis (inflammation of the mucus membranes of the nose) may worsen with DEP exposure.
- Exposure to diesel exhaust also may cause throat irritation, headache, dizziness, nausea, tiredness, and coughing.
- DEP exposure can also lead to a decreased immune response to bacterial infection.
- Chronic inhalation of DEPs is linked to an increased incidence of heart and lung diseases.
- There is an increased incidence of lung cancer in those who have chronic or occupational exposure to diesel exhaust.
- Chronic exposure to diesel exhaust is also linked to premature death.

**Kerosene**
Kerosene is used as a fuel in cooking stoves and heaters. Exposure to kerosene fuel and exhaust is linked to several adverse health effects.
Accidental intake of small amounts of kerosene may cause coughing, fever, vomiting, tachypnoea (rapid breathing), constipation, restlessness, drowsiness, abdominal pain, and diarrhea. Intake of large amounts of kerosene may cause convulsions, coma, or even death.

Skin contact with kerosene for short periods may cause itchy, red, sore, or peeling skin.

Using kerosene to clean used gasoline engine oil off skin may facilitate the passage of carcinogens from the oil to the lungs.

Cooking on gas stoves indoors is linked to an increase in asthma and acute respiratory infections in children.

Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic Aromatic Hydrocarbons (PAHs) are a group of over 100 different chemicals that are formed during the incomplete combustion of oil, gas, coal, garbage, wood, tobacco, or charcoal broiled meat. They are also found in asphalt, roofing tar, moth balls, creosote wood preservatives, incense, pesticides, explosives, plastics, skin creams for psoriasis, and anti-dandruff shampoos that contain coal tar.

Engine lubricating oils are known to accumulate carcinogenic PAHs while the engine is running.

Health risks linked to exposure to PAHs include:
- Clogged arteries and heart disease: a high occupational exposure to PAHs is related to a high risk of mortality from heart disease.
- Carcinogenicity: PAH exposure from coal emissions increases the risk damage to DNA and lung cancer. There is a strong link between childhood cancer and prenatal or early postnatal exposure to PAHs.
- PAHs are linked to breast cancer, laryngeal cancer, and leukemia.
- PAHs are toxic to the kidney and are linked to kidney cancer.
- Benzo[a]pyrene is a PAH and toxic to the reproductve system. It is linked to decreased fetal growth and developmental delays.

Carbon Monoxide

Carbon monoxide, or CO, is an odorless, colorless gas which is found in combustion fumes from vehicles, stoves, wood, coal, lanterns, and heating systems. Inhaling CO can lead to sudden illness and death.

Common early signs and symptoms of carbon monoxide poisoning include headache, dizziness, fatigue, lethargy, weakness, drowsiness, nausea, vomiting, chest pain, shortness of breath, anxiousness, impaired judgment, and increased blood pressure, heart rate, and respiratory rate.

Late signs of carbon monoxide poisoning are cherry-red skin, hypotension, poor capillary refill, unconsciousness, seizures, coma, and cardiac arrest.

Exposure to low levels of carbon monoxide over a long period of time can also lead to adverse health outcomes:
- Low birth weight is linked to chronic exposure to CO. Increased exposure to CO during the first trimester of pregnancy is linked to lower birth weight. A recent study provided evidence that exposure to CO reduces fetal growth during all trimesters of pregnancy.
- Cardiovascular and hematological effects: CO has been shown to increase the formation of blood clots. There is also evidence that links CO with an increased risk for a heart attacks, arrhythmias, and an increased risk of readmission to the hospital for heart problems after the first heart attack.
You can prevent or minimize exposure to petroleum products in the following ways:

- **Ventilation is one of the most effective ways to improve indoor air quality.** When possible, open doors, windows, and vents to reduce the level of indoor air pollutants. Cook in well-ventilated areas and clean the chimney so that carbon monoxide, benzene, PAHs, and other chemicals in smoke can escape.

- If possible, use gasoline containers specifically designed to store gasoline, which do not allow gasoline vapors to seep through the plastic, do not contain additional vent holes, and contain spill-proof spouts. Avoid storing gasoline in plastic containers made for other purposes, such as milk jugs or soda bottles.

- Pour gasoline in well-ventilated areas or outdoors. Avoid using around children.

- Store petroleum products out of the reach of children in childproof containers. Also, make sure that they are not stored in food or water containers.

- Store and use petroleum products as far away from your drinking water as possible. Keep petroleum products tightly closed and store them in places where people do not spend much time, such as in a garage or shed. The best storage place is cool, dry, and well-ventilated.

- If you have a fuel storage tank, inspect it for leaks and corrosion.

- When trying to remove oil from hands, use non-toxic, water-based solvents instead of kerosene and benzene whenever possible.

- Wear gloves or use a barrier cream made to protect skin from petroleum products when you are repairing vehicles.

- Wear protective clothing and equipment so that your skin is not exposed to gasoline. If your skin is exposed to gasoline, wash it thoroughly. Wash your hands carefully before eating, smoking, drinking, or using the toilet if you have been handling petroleum products. Wash clothing that has been contaminated with gasoline separate from other clothing.

- Install a carbon monoxide detector in your home and office.

- In houses that were built on fuel contaminated ground, seal the cracks in floors, foundation, and areas around pipes to decrease fuel vapors entering the house from outside.

- Do not let children pump gas into your vehicle. If they are small and stand at the same level as the gas pump, they will inhale more gasoline vapors.

- Avoid leaving ATVs, snow machines, and other vehicles running near homes and other places where people spend time.

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**Respiratory effects:** Higher exposure to carbon monoxide is related to more emergency room admissions for respiratory distress. Exposure to carbon monoxide from air pollution is linked to worsening of acute asthma in children and adults.

**Follow Up Action**

- Acute exposure to petroleum products or carbon monoxide is a medical emergency. If you think your patient may have been exposed to petroleum products or carbon monoxide, refer patient to a physician immediately.

- The Agency for Toxic Substances and Disease Registry provides a more detailed explanation of the medical management of patients exposed to gasoline. Keeping yourself from being exposed to petroleum products while providing patient care is also important. This guide can be found at: www.atsdr.cdc.gov/MHMI/mmg72.html

- Chronic exposure can be addressed by treating the symptoms and informing patients about ways to limit future exposure to petroleum products and carbon monoxide (see "Reducing Your Exposure" section below).

**Reducing Your Exposure**


Wilhelm M, Riz B. 2005. Local variations in CO and particulate air pollution and adverse birth outcomes in Los Angeles County, CA, USA. *Environmental Health Perspectives* 113(9): 1212-1221.


WHAT ARE PHTHALATES?
Phthalates are a group of industrial chemicals that add flexibility and resilience to many consumer products. Of particular concern are di-2-ethylhexyl (DEHP), benzylbutyl phthalate (BzBP), dibutyl phthalate (DBP), and very likely diethyl phthalate (DEP). DEHP and BzBP are primarily used as plasticizers in polyvinyl chloride (PVC)-based plastics, as well as other flexible plastics, and found in tablecloths, furniture, vinyl flooring, shower curtains, garden hoses, inflatable swimming pools, plastic clothing such as raincoats, children’s toys, automobile upholstery and tops, medical tubing, and blood storage bags.1,2 DEP and DBP are used in non-plastic consumer items as fixatives, detergents, lubricating oils, and solvents and can be found in carpets, paints, glue, insect repellents, time release capsules, and personal care products such as soap, shampoo, hair spray, nail polish, deodorants, and fragrances.2-6

PHTHALATES IN OUR BODIES
In a nationwide assessment of the exposure of the U.S. population to phthalates, scientists from the Centers for Disease Control and Prevention (CDC) measured levels of urinary phthalate monoesters (breakdown products of phthalates) in 289 people and found phthalates at unexpectedly high levels in every person tested.7,8 According to this assessment, researchers determined that “from a public health perspective, these data provide evidence that phthalate exposure is both higher and more common than previously suspected.”7 Though not representative of the population due to the small sample size, this study found that women of reproductive age (20 to 40 years) had substantially higher levels of DBP than the rest of the study group.7

In the 2005 CDC study, phthalates were once again widely detected in human urine samples with an increased sample size of over 2,500 Americans.3 Overall, the study indicated that, with the exception of MEP (mono-ethyl phthalate, the urinary metabolite of DEP), children age 6 to 11 years of age excreted higher concentrations of phthalate metabolites than the older age groups.1 In the same report, researchers found that women had higher levels of MEP, MBP (mono-butyl phthalate, a urinary metabolite of DBP), MBzP (mono-benzyl phthalate, the urinary metabolite of BzBP), and three urinary metabolites of DEHP: MEHP (mono-2-ethylhexyl phthalate), MEOHP (mono-(2-ethyl-5-oxohexyl) phthalate), and MEHHP (mono-(2-ethyl-5-hydroxyhexyl) phthalate).1

In a more recent study, the phthalate monoesters MEHHP, MEOHP, and MEP were found to be present in 100% of 90 girls age 6 to 9, and MBP, MBzP, and MEHP were found in over 94%.10
HOW ARE WE EXPOSED TO PHTHALATES?
There are multiple human exposure routes for phthalates including oral, inhalation, ingestion, dermal, and intravenous—through transfusions and other medical devices and procedures.1,2,5,6,8 Phthalates are easily released from the plastic products in which they are used as they are not part of the polymer chain which forms the plastic.6

People are exposed to phthalates through direct contact with consumer products containing phthalates, consumption of contaminated water or food (as the phthalates in plastic packaging may leach into the food they hold), inhalation of contaminated air, and during the manufacturing and disposal of products containing phthalates.2,6,11

Individual phthalates have various routes of exposure:
• The largest source of DEHP exposure for the general population is diet, followed by inhalation of contaminated indoor air.11 Exposures in food result from DEHP accumulating in foods and from the leaching of DEHP while processing, packaging, and storing.11 The single largest use of DEHP is as a plasticizer for poly-vinyl chloride plastic (PVC). DEHP can leach from PVC under certain circumstances, causing direct human exposures.3,4,10
• DEP exposure results mainly from direct use consumer products containing fragrances and personal care products such as shampoos, scents, soap, lotions, and cosmetics, and from inhalation of air containing these chemicals.3,10 Exposure to DEP is also found in products such as toothbrushes, tools, food packaging, insecticides, and aspirin.6
• Exposure to DBP has been linked most commonly to cosmetics, mainly nail polish, but DBP is also found in pharmaceutical coatings, insecticides, and some printing inks.3,4,10
• BzBP is an industrial solvent and found within adhesives, vinyl flooring, sealants, car-care products, and some personal care products such as hair spray.3,4,10

WHAT DOES PHTHALATE EXPOSURE MEAN FOR OUR HEALTH?
Evidence that exposure to phthalates has possible adverse health effects has been building over recent years. Phthalate exposure has been linked to the following health concerns:

Reproductive and Developmental Effects: Phthalate exposure can begin in utero and in one study is strongly associated with a shorter pregnancy duration.12 Another study also found that prenatal phthalate exposure at environmental levels is associated with altered male reproductive development in humans, including shortened anogenital distance (signifying feminization), an increased likelihood of testicular maldescent, small and indistinct scrotum, and smaller penile size.13 Later research documented altered male reproductive hormone levels in baby boys most highly exposed to phthalates in their mother’s breast milk.14 Phthalate exposure has also been linked to lower sperm counts, reduced sperm motility, and damaged sperm in men.15

Respiratory System:
Studies have shown concentrations of phthalates in house dust are associated with asthma and rhinitis in children.16 Exposure to PVC flooring containing
Phthalates has also been linked to increased bronchial obstruction during the first two years. In adult men, exposure to certain phthalates has been linked to reduced lung capacity at magnitudes similar to those observed with tobacco smoke.

**Effects in Laboratory Animals:**
Depending on the exposure level, exposure to DEHP resulted in observed effects on the pituitary, thyroid, thymus, ovaries, testes, lung, kidneys, liver, and blood.

**Regulations for Phthalates**
In the United States, federal regulation of phthalates fall under the Toxic Substances Control Act which was passed in 1976 and has not been updated since. Though phthalates are considered a hazardous waste and are regulated as pollutants when released into the environment in the U.S., they remain basically unregulated in consumer products. Several states have tried and failed to pass laws banning phthalates in certain consumer products. However, in September, 2007 the California State Senate successfully passed a bill that would ban 6 phthalates in children’s toys beginning in 2009. On the global level, the European Union banned the use of six phthalates in children’s toys and products in 2005, and prior to that, fourteen counties had restricted or banned their use.

**Reducing Your Exposure**
Scientific evidence indicates that phthalates readily exit our bodies through urinary metabolites. Although people are continually exposed to phthalates because they are ubiquitous, you can minimize re-exposure to phthalates by:

**Avoid products containing PVC plastic:**
- Avoid the use of PVC/vinyl in building and home remodeling. Use safe alternatives such as those recommended by the Healthy Building Network (www.healthybuilding.net).
- Purchase natural fibers, polyester, or nylon shower curtains instead of vinyl.
- Avoid plastics marked with the #3 symbol; these are likely to contain PVC.
- Check with your children’s toy manufacturers to see if they have pledged to stop using PVC. Toymakers Brio, Chicco, Early Start, Evenflo, Gerber, Lego, Prime Time, Sassy, and Tiny Love have already made the commitment.

**Awareness of Household Products:**
- Educate yourself as to whether the adhesives, caulk, grout, and sealants you use contain phthalates. In the National Institutes of Health’s Household Products Database, you can search for phthalate as an ingredient. It can be found at www.householdproducts.nlm.nih.gov.

**Phthalate-free Personal Care Products:**
- Look at ingredient lists and avoid products listing “fragrance” or phthalates.
- Choose products from companies that have signed the Compact for Safe Cosmetics. For a listing, check out: www.safecosmetics.org.
- Research the ingredients of your personal care products, beyond what is on the ingredient label. Check out the Skin Deep Database at www.ewg.org/reports/skindeep2/index.php for a more complete listing.

**Know What Is In Your Food:**
- Grow and harvest your own food without using pesticides, or purchase organic foods if possible.


19 EPA. 2006. Inventory Update Reporting. Environmental Protection Agency. Available: http://www.epa.gov/oppt/iur/


Polybrominated Diphenyl Ethers (PBDEs)

What Are PBDEs?
Polybrominated diphenyl ethers, or PBDEs, are a class of flame retardant chemicals added to many consumer products found in the home, office, automobiles, and airplanes. Three mixtures used widely—penta-BDE, octa-BDE, and deca-BDE—made up 14%, 6%, and 80% of the 1999 worldwide production, respectively. Usually found in electronics, such as TVs, and used in some furniture foams, fabrics, and kitchen appliances, the industry voluntarily ended production in the United States of the formulations of penta and octa in 2004 after high levels were found in breast milk. However, the deca formulation is still being produced and used mostly in plastic electronics such as televisions and computer casings. It is also used in the upholstery covers of items such as furniture, mattresses, and car seats.

PBDEs are also part of a larger chemical class called polyhalogenated aromatic hydrocarbons (PHAHs) which include other highly toxic chemicals such as polychlorinated biphenyls (PCBs) and dioxins. PBDEs are intrinsically hazardous because of their chemical make-up: (1) they are stable, meaning they stay in the environment and do not break down easily; (2) they are lipophilic, meaning they build up in fatty tissues of living organisms; and (3) they have toxic properties, including the potential to act as endocrine disruptors. Their persistence and fat solubility allow them to both biomagnify and bioaccumulate, meaning they build up in the bodies of animals and humans as they move through the food chain. Although the main component in deca-BDE, BDE-209, has a relatively short half-life in people, animal studies show that the liver breaks down BDE-209 into the more persistent and bioaccumulative formulations known as hepta-BDE, octa-BDE, and nona-BDE.

How Are We Exposed?
There are many ways that humans are exposed to PBDEs, including eating contaminated foods and incidental intake of PBDE-contaminated dust. Because of the chemical make-up of PBDEs, they do not fully attach to the products in which they are used. Deca, in particular, breaks down into more toxic products and volatizes when exposed to ultraviolet (UV) light. It is not entirely clear how deca is being released from products, though some think it might be released from physical abrasion or deterioration of the product. Penta does not need UV light exposure to volatize. PBDEs are released from furniture, electronics and other products when exposed to UV light, causing them to be present in the air we breathe and in dust. Although penta-BDE and octa-BDE are no longer produced in the United States, exposure continues from old computers, furniture, fabrics, and other consumer sources that were made before the discontinuation.

Out of all possible exposure routes, the highest intake of deca-BDE results from household dust. Another major route
of exposure is eating fatty foods such as meat and dairy products, as PBDEs are fat-seeking and build up in the food chain.

Eating fish and marine animals may be another route of exposure because they have been found to contain high levels of deca-BDE. The high levels in marine environments may be due to atmospheric deposition (when contaminants from the air come down to the earth’s surface by rain, snow, falling particles, and the absorption of gas) of deca-BDE as well as sewage discharge into the oceans.

PBDEs IN OUR BODIES
Due to their widespread use, persistence and bioaccumulative properties, PBDEs have been found in humans at high levels. One study showed that 5 percent of American women have levels of PBDEs that are close to the levels linked to reproductive problems in animals, though we do not know if humans are more or less sensitive than animals. PBDEs have been found in mothers’ breast milk and in the blood of mothers and their babies. People of the Arctic may experience an even higher risk due to their traditional diet rich in fat from marine mammals. Concentrations of PBDEs have grown over the years in marine mammals due to atmospheric transport (transported long distances from areas of production and use via air and ocean currents into the north) and bioaccumulation.

WHAT DOES EXPOSURE TO PBDEs MEAN FOR OUR HEALTH?
PBDEs have been linked to many adverse health effects, including:

Developmental Effects:
Studies in rodents suggest that neonatal exposure to PBDEs permanently affects learning and memory functions, impairs motor activity, and is linked to aberrations in spontaneous behavior and hyperactivity that seem to be permanent. However, it is not known what the developmental effects are in people or at what doses they happen.

Reproductive Effects:
PBDEs are mildly estrogenic compounds which have been correlated to cryptorchidism, or undescended testes, in newborn boys, and shown to permanently impair sperm development in rodent studies. Exposure to PBDEs in household dust is linked with lower levels of androgens (male hormones) in adult men. PBDEs have also been associated with delay of puberty in both male and female rodents and changes in sexual development and gender-specific sexual behavior. Exposure to PBDEs has also been linked with low birth weight, birth defects, reduced weight gain during pregnancy, changes in ovary cells, and reduced sperm count. The breakdown products of PBDEs also may inhibit aromatase, an enzyme important in the formation of androgens and estrogens (male and female hormones) and in the skeletal development of both males and females.

Cancer:
One study suggests that in utero exposure to PBDE concentrations is associated with an increased risk of testicular cancer in men. The Agency for Toxic Substances and Disease Registry (ATSDR) lists deca-BDE as a possible human carcinogen based on the development of liver tumors in rats that were given the substance.

Thyroid Problems:
In a study of newborn babies, high PBDE levels in cord blood are associated with decreased levels of thyroid hormones. Corresponding animal studies have also shown that PBDE exposure is linked to decreased circulating concentrations of thyroid hormone and decreased thyroid weight in adult rodent offspring.
REGULATIONS FOR PBDEs
There have been many steps forward in the regulation of PBDEs in recent years, but much more is needed to ensure the safety of public health. Internationally, Sweden was the first to initiate a phase-out of PBDEs in the late 1990s, followed by the European Union—first phasing out penta-BDE and octa-BDE and then deca-BDE in 2006. In 2008, Norway banned the use of deca-BDE in new consumer products, and Canada prohibited the manufacture of all PBDEs and put in place restrictions on the import of certain PBDEs. In the United States, industry voluntarily ended production of penta-BDE and octa-BDE in 2004, but deca-BDE is still allowed to be used in consumer products. Both Washington and Maine are currently discontinuing the use and manufacture of deca-BDE, with other states around the nation aiming for similar legislation.

REDUCING OUR EXPOSURE

You can minimize your exposure to PBDEs by taking the following steps:

PBDE-free Furniture:
- Before purchasing furniture, find out which companies offer PBDE-free products. The following websites can help:
  - Pollution in People: [www.pollutioninpeople.org/safer/products](http://www.pollutioninpeople.org/safer/products)
- Contact the company directly if you cannot figure out if the manufacturer uses PBDEs.
- Choose furniture made with less flammable fabrics like leather, wool and cotton.

PBDEs in Foam Padding:
- Foam items purchased before 2005 are likely to contain PBDEs. Make sure these items are completely covered in fabric with no rips.
- Avoid reupholstering foam furniture.
- Be very careful when removing old carpet. Try to keep your work area separated from the rest of the house and thoroughly clean up the area.

Reduce Your Dust Exposure:
- Wash your hands often to remove dust particles that your hands pick up throughout the day on everything you touch.
- Use a wet rag or cloth while dusting to avoid kicking up the dust in the air.
- If possible, use a vacuum fitted with a HEPA filter. These vacuums can trap smaller particles of dust and will be more likely to remove contaminants from your home.
- Vehicles have been exempt from recent PBDE laws and high levels have been found inside cars. Removing dust with a wet cloth and keeping car seat cushions in good repair will help to reduce your exposure.

PBDE-free Electronics:
- Many companies are beginning to make electronics with alternatives to PBDEs. Certain PBDE-free products are available from Canon, Dell, HP, Intel, Erickson, Apple, Acer, Nokia, Motorola, LG Electronics, and Sony.

Consider Eating Less Fat:
- Consider choosing leaner meat and poultry cuts.
- Consider removing fat that you see on meat and fish whenever possible.
- Choose cooking methods that remove excess fat such as broiling, grilling, and roasting.
1 McDonald TA. 2005. Polybrominated diphenyl ether levels among United States residents: Daily intake and risk of harm to the developing brain and reproductive organs. Integrated Environmental Assessment and Management 1(4):343-354.


Polychlorinated Biphenyls (PCBs)

**WHAT ARE POLYCHLORINATED BI PHENYLS (PCBs)?**

- PCBs, compounds once used as insulators in the electrical industry, are now banned in the United States, Northern Europe and other countries. These compounds are found throughout the environment and in human bodies worldwide.¹,²
- PCBs are considered persistent organic pollutants (POPs) because they do not easily break down and they stay in the environment for many years. POPs are toxic at extremely low levels.
- PCBs were left behind as waste by the military at formerly used defense sites (FUDS) throughout Alaska. PCBs produced throughout the world can travel on wind and ocean currents and build up in wildlife and people of the North/Arctic, a process called global transport.³

**HOW ARE WE EXPOSED?**

PCBs build up in fatty tissue in the body. People are exposed to PCBs through foods such as fish, dairy products, marine mammals, and through breast milk.¹,²,⁴

**SYMPTOMS & HEALTH PROBLEMS**

- Prenatal exposure to PCBs is linked to lower developmental test scores, short-term memory defects, and lower IQ levels in children.¹,²,⁵
- Health effects that may result from high exposures to PCBs include: chloracne, keratoses and pigmentation of the skin; mixed peripheral neuropathy; and gastritis.¹,⁶,⁷
- Long-term and chronic PCBs exposures are also associated with adverse health effects, including: cancer, immune suppression, neurobehavioral problems, endocrine disruption of sex steroid and thyroid function, cardiovascular disease, and diabetes.³,⁸,⁹

**FOLLOW UP ACTION**

- If you think your patient may have been exposed to PCBs, refer patient to a physician and contact ACAT at (907) 222-7714 or info@akaction.org.
- Recommend breastfeeding to postpartum patients.¹ In communities at risk of chemical exposures, it is even more important that mothers breastfeed their babies because breast milk reduces the effects of the chemicals on infants and makes them healthier.¹⁰,¹¹,¹²,¹³
- Support patients’ continued consumption of traditional subsistence foods, unless you have information that indicates PCBs or other contamination.
**Reducing Your Exposure**

You can prevent or minimize exposure to PCBs in the following ways:

- Consider removing fat that you see on meat and fish whenever possible.
- Consider eating leaner meats whenever possible.
- Consider eating less dairy products; or, if available, choose low fat or nonfat dairy products.²
- Although it is uncertain how far contaminants may spread around formerly used defense sites, it is advisable to have water and traditional food sources close to, downstream or downwind of these sites tested for the possibility of harmful exposures. If you have concerns about the safety of your water or traditional foods, contact ACAT or visit our website at [www.akaction.org](http://www.akaction.org).

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Solvents

What Are Solvents?
Solvents are any class of chemicals used to dissolve materials. Usually, solvents are extremely volatile and reactive, meaning they quickly evaporate into their gaseous form and interact with other molecules to form byproducts (or different chemical combinations) quickly.1

Because of their ability to dissolve solids, solvents are often used to clean and sanitize. Solvents are also used industrially for many purposes such as degreasing equipment or cleaning electronics before sale.1

How Are We Exposed?
People can be exposed to solvents in a number of ways:
- The main route of exposure for solvents is breathing it in.
- Household exposures to solvents are fairly common. Solvents are commonly added to many household products such as cleaning supplies and paints.1
- Individuals may also be exposed to solvents in the workplace, while working with solvents.1
- Indirect exposure can occur when solvents added to consumer products—such as paint or gasoline—evaporate into the air.
- Environmental exposures to solvents are also possible, as some solvents persist in the environment due to improper disposal.2 Other solvents such as benzene are released into the air from gasoline exhaust.3

Symptoms & Health Outcomes
There are different health effects of solvent exposure depending on the specific solvent and the length of exposure. Different solvents will result in a range of symptoms. The health effects of some common solvents are included here.

Ammonia
Ammonia is commonly occurring and naturally present in the environment. Exposure to these background levels is not harmful.4 However, important research shows that acute exposure to high concentrations of ammonia and chronic exposure to moderate levels can be harmful or deadly.

At low levels, ammonia inflicts tissue damage to the exposed area, including the nose, throat, and lungs if it is inhaled.5,6 At higher levels, animal studies suggest it may interfere with neurological processes, especially when ingested.7,8 Airborne ammonia can cause irritation of the eyes resulting in inflammation, short-term blindness or altered vision, watering eyes, and corneal abrasions.4

Bleach
Sodium hypochlorite is the active ingredient in chlorine bleach and cleaning products containing bleach. Exposure to sodium hypochlorite irritates skin and mucus membranes, and high concentrations of sodium hypochlorite can cause serious tissue damage.9
In addition to being acutely toxic, sodium hypochlorite can react with other cleaners and organic matter to form dangerous chemicals. Chloroform can be created when bleach contacts organic matter during cleaning\textsuperscript{10} and can be created in chlorinated drinking water as well.\textsuperscript{11} Free chlorine gas can be created when bleach is mixed with acidic solutions.\textsuperscript{12}

Chloroform is a probable human carcinogen according to the US EPA.\textsuperscript{13} Chronic exposure is known to cause depression, lack of concentration, and irritability. Chlorine gas is acutely toxic and can cause irritation to the nose and throat, chest pain, vomiting, and even death.\textsuperscript{14}

**Benzene**

Benzene is a petrochemical, meaning it is a refined product of oil. It is present in the environment in water, soil and air. Some of the major sources of benzene are car exhaust, cigarette smoke, and paint or glues.\textsuperscript{3} Exposure to benzene through food, water, and soil is not common, unless there is a local source of contamination. Approximately 95\% of exposure to benzene is through inhalation.\textsuperscript{15} Acute exposure can cause dizziness, rapid heart rate, tremors and unconsciousness.\textsuperscript{3}

Benzene is known to cause cancer. The most significant health risk to low level exposure to benzene is leukemia.\textsuperscript{15} However, research suggests that benzene can induce other diseases of the blood, such as anemia, and uncontrolled bleeding.\textsuperscript{16} Benzene may also cause reproductive effects such as irregular menstruation and birth defects.\textsuperscript{3} *(Please see the Benzene fact sheet for more details.)*

**Simple Green**

Simple Green contains 2-butoxyethanol, a type of chemicals known as ethylene glycol ethers (EGEs), that are commonly used as solvents both in businesses and industries.

In humans, long term exposure to EGEs can result in dizziness, lethargy, headaches, and memory loss as well as hematopoietic effects such as decreases in some types of blood cells.\textsuperscript{17,18} Long-term exposure in animals has been linked to damage to DNA, sperm and blood cells.\textsuperscript{19,20,21} Exposure to 2-butoxyethanol is known to cause hemolysis, or destruction of blood cells, and reduce concentrations of hemoglobin in rats,\textsuperscript{22} and research has correlated 2-butoxyethanol exposure to an increase in tumors in rats and mice.\textsuperscript{23}

**Toluene**

Exposure to toluene can happen in many different ways, but the main route of exposure is inhalation from contaminated air. Gasoline, car exhaust, cigarette smoke, and paint or paint thinner are all common sources of toluene.\textsuperscript{24} Using nail polish significantly increases concentrations of toluene in the air.\textsuperscript{25} Acute exposure to toluene has been shown to result in dizziness, headaches,\textsuperscript{26} manual impairment, intoxication, and loss of color recognition.\textsuperscript{27}

In humans, long-term exposure to toluene has been linked to mucus membrane irritation, altered temperature and sound perception, intoxication\textsuperscript{24} and an increase in spontaneous abortions.\textsuperscript{26} It is also linked to dizziness, headaches,\textsuperscript{29} and hearing loss.\textsuperscript{30} In animals, toluene exposure has been linked to lower dopamine and noradrenaline levels, important neural hormones\textsuperscript{31} and an overall decrease in brain weight.\textsuperscript{32}

**Follow Up Action**

- If you think your patient may have been exposed to solvents, refer patient to a physician.
Reducing Our Exposure

You can prevent or minimize exposure to solvents in the following ways:

- Whenever possible, buy products that do not contain harmful solvents. Water-based paints and glues and citrus-based cleaners are safer to use.
- If you have solvents or products containing solvents in your home, carefully label them and store them in a safe place, especially in homes with young children.
- When you use products with solvents, such as paints, cleaners, or nail polish, make sure they are used in well ventilated areas and open windows or use fans to reduce the amount of solvents in the air.
- Never mix cleaning solutions or solvents, as the resulting vapors can be harmful or deadly.
- Gasoline and exhaust are major sources of solvents and other contaminants. Turn off the engines of cars, ATVs, and snow machines when not needed.

Triclosan

What Is Triclosan?
Triclosan is an antibacterial chemical used to kill bacteria in a wide range of consumer products including soaps, deodorants, cosmetics, cleansing lotions, toothpaste, plastics, and fabrics. Antimicrobial pesticides, such as triclosan, are made to destroy or suppress the growth of microorganisms and are regulated by the Environmental Protection Agency. However, when triclosan is used in products intended for contact with the human body (i.e., personal care products such as soaps, antiseptics, and toothpaste) or in food or food wrappers, it is not considered a “pesticide” by U.S. law and is regulated by the Food and Drug Administration. Triclosan is mostly used in consumer products that are disposed of down drains. Wastewater treatment plants do not completely remove triclosan from the water before it is released. Thus, triclosan is thought to be found everywhere throughout the environment and the water cycle. Between 1999 and 2000 the U.S. Geological Survey tested water samples for 95 different chemicals including triclosan, which was among the most often found chemicals and had some of the highest levels.

How Are We Exposed to Triclosan?
Although triclosan was developed more than 30 years ago, over the last 10 years there has been a big increase in consumer products that contain triclosan. Daily exposure to consumer products that contain triclosan (including at least one brand of toothpaste, skin-care products, and other household products) is most likely the main source of exposure for the U.S. population. A number of companies that make toothpaste and soap products with triclosan claim that the active ingredient continues to work for up to 12 hours following use. As a result, consumers are exposed to triclosan long after the few minutes it takes to wash their hands or brush their teeth. Triclosan can be absorbed through the skin, mouth, and nose.

Triclosan In Our Bodies
In an on-going nationwide study of exposure of the U.S. population to environmental chemicals, scientists from the Centers for Disease Control and Prevention measured samples taken from 2003-2004 for triclosan in 2,517 people and found that 74.6% had detectable levels of triclosan in their urine.

In a pilot study of 90 girls ages 6-9 years old, triclosan levels in their urine were detected in 67.8%. Triclosan has also been found in the breast milk and plasma of breast-feeding mothers. In one study that collected and tested the breast milk and plasma of 36 mothers for triclosan, those who used personal care products containing triclosan had higher levels suggesting that consumer products are a main source of exposure.
SYMPTOMS & HEALTH OUTCOMES
In acute toxicological studies, triclosan has been found to have low toxicity to humans and other mammals.\textsuperscript{11} However, many cases of skin irritation and eczematous rash resulting from skin contact with products containing triclosan have been reported.\textsuperscript{12} Long-term chronic exposures to triclosan may result in more serious health outcomes.

Thyroid Disruption:
The thyroid gland regulates development and metabolism. Because triclosan is structurally similar to thyroid hormone and has been shown to build up in the bodies of certain fish, it may disrupt normal growth and development in humans and wildlife. One study showed that triclosan interfered with the thyroid hormone in frogs and affected the timing of metamorphosis in tadpoles with levels commonly found in the environment.\textsuperscript{13}

Allergies:
The overuse of triclosan (and other antibacterial chemicals) may be linked to increased allergies and asthma.\textsuperscript{2}

Follow Up Action
- If you think your patient may have been exposed to triclosan, refer patient to a physician.
- Long-term exposure can be addressed by treating the symptoms and informing patients about ways to limit future exposure to triclosan (see “Reducing Your Exposure” section below).

Products Containing Triclosan

\begin{center}
\textbf{SOAP:} Dial\textsuperscript{®} Liquid Soap; Softsoap\textsuperscript{®} Antibacterial Liquid Hand Soap; Tea Tree Therapy\textsuperscript{™} Liquid Soap; Provon\textsuperscript{®} Soap; Clearasil\textsuperscript{®} Daily Face Wash; Dermatologica \textsuperscript{®} Skin Purifying Wipes; Clean & Clear Oil Free Foaming Facial Cleanser; DermaKleen\textsuperscript{™} Antibacterial Lotion Soap; Naturade Aloe Vera 80\textsuperscript{®} Antibacterial Soap; CVS Antibacterial Soap; pHisoderm Antibacterial Skin Cleanser; Dawn\textsuperscript{®} Complete Antibacterial Dish Liquid; Ajax\textsuperscript{®} Antibacterial Dish Liquid.

\textbf{DENTAL CARE:} Colgate Total\textsuperscript{®}; Breeze\textsuperscript{™} Triclosan Mouthwash; Reach\textsuperscript{®} Antibacterial Toothbrush; Janina Diamond Whitening Toothpaste.

\textbf{COSMETICS:} Supre\textsuperscript{®} Café Bronzer\textsuperscript{™}; TotalSkinCare Makeup Kit; Garden Botanika\textsuperscript{®} Powder Foundation; Mavala Lip Base; Jason Natural Cosmetics; Blemish Cover Stick; Movate\textsuperscript{®} Skin Lifting Cream HQ; Paul Mitchell Detangler Comb; Revlon ColorStay LipSHINE Lipcolor Plus Gloss; Dazzle.

\textbf{DEODORANT:} Old Spice High Endurance Stick Deodorant; Right Guard Sport Deodorant; Queen Helene\textsuperscript{®} Tea Trea Oil Deodorant and Aloe Deodorant; Nature De France Le Stick Natural Stick Deodorant; DeCleor Deodorant Stick; Epoch\textsuperscript{®} Deodorant with Citrisomes; X Air Maximum Strength Deodorant.

\textbf{OTHER PERSONAL CARE PRODUCTS:} Gillette\textsuperscript{®} Complete Skin Care MultiGel Aerosol Shave Gel; Murad Acne Complex\textsuperscript{®} Kit; \textsuperscript{®} Diabet-x\textsuperscript{™} Cream; T.Taio\textsuperscript{™} sponges and wipes, Aveeno Therapeutic Shave Gel.

\textbf{FIRST AID:} SyDERMA\textsuperscript{®} Skin Protectant plus First Aid Antiseptic; Solarcaine\textsuperscript{®} First Aid Medicated Spray; Nexcare\textsuperscript{™} First Aid, Skin Crack Care; First Aid/Burn Cream; HealWell\textsuperscript{®} Night Splint; 11-1X1: Universal Cervical Collar with Microban.

\textbf{KITCHENWARE:} Farberware\textsuperscript{®} Microban Steakknife Set and Cutting Boards; Franklin Machine Products FMP Ice Cream Scoop SZ 20 Microban; Hobart Semi-Automatic Slicer; Chix\textsuperscript{®} Food Service Wipes with Microban; Compact Web Foot\textsuperscript{®} Wet Mop Heads.

\textbf{COMPUTER EQUIPMENT:} Fellowes Cordless Microban Keyboard and Microban Mouse Pad.

\textbf{CLOTHES:} Teva\textsuperscript{®} Sandals; Merrell Shoes; Sabatier Chef’s Apron; Dickies Socks; Biofresh\textsuperscript{®} socks.

\textbf{CHILDRENS TOYS:} Playskool\textsuperscript{®}: Stack ‘n Scoop Whale, Rockin’ Radio, Hourglass, Sounds Around Driver, Roll ‘n Rattle Ball, Animal Sounds Phone, Busy Beads Pal, Pop ‘n Spin Top, Lights ‘n Surprise Laptop.

\textbf{OTHER:} Bionare\textsuperscript{®} Cool Mist Humidifier; Microban\textsuperscript{®} All Weather Reinforced Hose; Thomasville\textsuperscript{®} Furniture; Deciguard AB Ear Plugs; Bauer\textsuperscript{®} 5000 Helmet; Aquatic Whirlpools; Miller
REDUCING OUR EXPOSURE

You can prevent or minimize your exposure to triclosan in the following ways:

- Keep clean without using antimicrobials: Wash hands often and thoroughly. Lather hands for at least 10 to 15 seconds and rinse with warm water. Regular soap lowers the surface tension of water and washes away unwanted bacteria.
- Wash hands often, especially when handling food, before you eat, after you go to the bathroom, or when you are around someone who is sick.
- Dry hands with a clean towel to help remove any germs that did not get washed away.
- Wash children's hands and toys often.
- Since triclosan is almost always found in personal care products, make sure to read all the ingredients when buying these products. Triclosan is also called Microban, Irgasan, Lexol, Ster-Zac, Cloxifenolum, and Biofresh.
- Australian tea tree oil and grapefruit seed extract have natural antimicrobial properties and are included in some soaps.

Uranium

What is Uranium?

- Uranium is a radioactive element found in mineral deposits in certain locations around the world.\(^1\) It is a hard metal that is mined for the production of energy in nuclear power plants and is also used in the production of nuclear weapons. Mineral deposits containing uranium usually range from .002 to .004 percent of the rock material.\(^2\)

- Uranium is used by the military when making nuclear weapons, such as atomic bombs. Nuclear weapons are currently stored in many countries around the world, including the United States, Russia, the United Kingdom, France, and China. Uranium is also the fuel used in nuclear power plants.

- A form of uranium used by the U.S. military in bullets and other weapons is called “depleted uranium,” which means the portion left after enriched uranium is removed from natural uranium. The term “depleted” is misleading because it hints that this form of uranium is safer, but depleted uranium acts the same way in the body as natural uranium. Depleted uranium is a toxic heavy metal and a radioactive hazard to human health and the environment.\(^3,4\) It is used to strengthen munitions and other weapons due to its high density.

- In the case of the planned uranium mine near Elim, Alaska, the proposed method for extracting uranium ore is in-situ leach (ISL) mining. In ISL mining, a series of wells are drilled into the ore deposit. A leaching liquid (a strong acid solution such as sulfuric acid or ammonium-carbonate) is then injected through these wells into the ore, separating the uranium from the ore body. The liquid with uranium is pumped to the surface and piped to a processing plant where the uranium is separated. Most of the leftover solution is recharged with more of the leaching agent and then injected back into the wells. However, a certain amount of this solution is not re-used and becomes waste, which is returned back into the ore body in disposal wells.

How Are We Exposed?

- People can be exposed to uranium through breathing air or drinking water contaminated with uranium, eating food grown in areas contaminated with uranium, working in factories that process uranium, or living near a uranium mine.\(^5\)

- With ISL mining, there is a risk of groundwater contamination. The leaching process not only moves uranium particles, but also releases other contaminants and toxic metals that can contaminate groundwater, including radioactive thorium, radium, radon, lead, cadmium, and arsenic.

- Soldiers currently deployed and other military personnel returning to Alaska may be exposed to depleted uranium.

Symptoms & Health Problems

- The harmful effects of uranium on human health have been linked to its radioactive and heavy metal properties.\(^6\)
Inhaling or ingesting uranium can cause malignant and nonmalignant respiratory diseases, kidney failure, leukemia, and stomach, kidney and soft tissue cancer.\(^2\,5\,7\)

Animal studies suggest that uranium may adversely affect reproduction and harm the developing fetus.\(^1\)

**Follow Up Action**

- If you think your patient may have been exposed to uranium, refer patient to a physician and contact ACAT at (907) 222-7714 or info@akaction.org.

**Reducing Your Exposure**

You can prevent or minimize exposure to uranium in the following ways:

- Keep children from putting hands in their mouths and have them wash their hands often to lower the amount of contaminated soil that could be eaten or swallowed.\(^3\)
- Although it may be uncertain how far contaminants may spread around mine sites, it is advisable to have water and traditional food sources close to, downstream or downwind of these sites tested for the possibility of harmful exposures. If you have concerns about the safety of your water or traditional foods, contact ACAT or visit our website at www.akaction.org.

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Text for Poster: Protecting Our Health in Alaska

**Pesticides**
Insecticides, herbicides, fungicides, and rodenticides (collectively known as “pesticides”) contain chemicals that are toxic to humans and can harm the health of children and adults. Safe alternatives can be used to solve nearly all pest problems without the need for toxic chemicals. We recommend that you try prevention and non-toxic alternatives first. For example, citronella oil can be used instead of DEET to keep mosquitoes away. Burning of alder branches at fish camp helps to keep them out of the area. Pepper ing fish during processing and drying can discourage flies. You can prevent pests from entering your home by sealing cracks in windows, doors and baseboards. Storing food properly and cleaning food or beverage spills will help to keep out unwanted pests from your home. Lice combs and mayonnaise can be used instead of chemical shampoos to treat head lice.

**Keep It Outside**
Remove your shoes outside to prevent tracking of harmful chemicals into your home. If you work with chemicals in your job, be careful not to expose your family to your work clothes and wash them separately.

**Berries and Greens**
Rinse berries and greens with water before you eat them to reduce possible exposure to harmful contaminants that may be in the dust on the outside of the plants.
Manage Waste

Burning Trash in Barrels and Burn Boxes
Lower the amount of waste by reducing, recycling, and re-using materials. Avoid the burning of plastics to prevent exposure to toxic chemicals. The smoke from burning plastics can harm people’s health and may be linked with illnesses such as asthma, increased risk of cancer and diabetes, and reproductive health problems. Some communities have banned plastic bags to reduce waste. For information on these projects, contact ACAT. Make sure that burn barrels and burn boxes are not used near places where people might be exposed to the smoke.

Open Dumpsites
Open dumps are a hazard to health and may have dangerous toxics. Keep children away from dumps and streams that flow from these waste sites. Dump sites should be fenced and properly maintained to prevent animals and people from toxic exposures.

Personal Care Products
Some personal care products may have chemicals that have been linked with harmful health effects. Phthalates are a group of chemicals found in everyday products such as lotions, soaps and cosmetics that have been shown to have reproductive health effects. You can find non-toxic alternatives at www.safecosmetics.org. Some of the ingredients that might cause harm include phthalates, thimerosal, lead acetate, formaldehyde, sodium lauryl sulfate, parabens, and toluene.

Furniture and Electronics
PBDEs (polybrominated diphenyl ethers) are flame-retardant chemicals added to many consumer products, including furniture and electronics such as TVs and computers. PBDEs don’t break down quickly and build up in living organisms including people and wildlife. PBDEs may affect learning, damage reproduction and the thyroid. You can reduce your exposure to PBDEs by choosing PBDE-free products. To find out if a product contains PBDEs, go to www.thegreenguide.com or call the manufacturer. Dust your home often with a wet cloth, and sweep or vacuum to lower exposures to PBDEs in dust particles.

Plastics
Chemicals found in plastic products such as food containers, baby bottles and toys have been linked to health effects including fertility and other reproductive health problems. Using glass, ceramic or wooden products will reduce your exposure to these chemicals. If you must use plastic, avoid plastics marked #7 (these are polycarbonate plastics that contain the chemical bisphenol A). Avoid using plastic wrap and heating plastic containers in the microwave oven.

Household Products
Many cleaning products contain chemical solvents linked to infertility and other health problems. You can buy non-toxic cleaners or make your own using vinegar, baking soda and borax soap. Water-based paints, glues and citrus-based cleaners are safer to use. For suggestions about other safe product recommendations, contact ACAT or visit our website at www.akaction.org.

Clean Hands
Wash hands often to prevent harmful exposures to chemicals and bacteria.
**Mold**
Mold is a fungus that grows in moist areas and is linked to lung bleeding and other problems in babies, allergic reactions and some cancers in adults. Fix water leaks as soon as possible and make sure that you have good air flow to prevent mold growth. To remove mold, spray with a solution of 1 part vinegar and 1 part water, then wipe clean while wearing a dust mask.

**Tobacco Smoke**
Tobacco smoke contains thousands of chemicals, many of which are poisonous. If you quit smoking, it will improve your health and the health of those around you. Do not smoke indoors because it exposes others to secondhand smoke. Children are especially vulnerable to the harmful effects of secondhand smoke.

**Formerly Used Defense Sites (FUDS)**
Many dangerous chemicals have been used by the military and left behind when sites were abandoned. Although it may be uncertain about how far contaminants may spread around formerly used defense sites, it is advisable to have water and traditional food sources close to, downstream or downwind of these sites tested for the possibility of harmful exposures. For more information on possible chemical exposure in these areas, contact ACAT or visit our website at www.akaction.org.

**Persistent Organic Pollutants (POPs)**
Persistent organic pollutants, or POPs, are chemicals that take a long time to break down, they build up in animals and people, and are toxic. Ocean and wind currents transport POPs from thousands of miles away to the north/Arctic, where they enter the food chain—into the soil and plants, insects, fish, wildlife, and people. POPs include many dangerous chemicals such as PCBs, DDT and dioxin. For more information on ways to take action against these chemicals, contact ACAT or visit our website at www.akaction.org.

**Vehicles**
Exhaust from ATVs and other vehicles contains toxic chemicals that may lead to health effects such as respiratory ailments, cancer, heart disease, and damage to the nervous system. Avoid breathing exhaust from vehicles so that you can reduce your exposure to these chemicals. You can reduce exhaust from your vehicle by turning off the engine when the vehicle is stopped for more than a few minutes. Don’t let children handle gasoline, and store fuels safely in well-marked containers out of the reach of children.

**Mining**
Chemicals that are harmful to human health, such as heavy metals, are released into the environment through mining. Although it may be uncertain about how far contaminants may spread around mine sites, it is advisable to have water and traditional food sources close to, downstream or downwind of these sites tested for the possibility of harmful exposures. If you have concerns about the safety of your water or traditional foods, contact ACAT or visit our website at www.akaction.org.