



Environmental Medicine Update

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Environmental Causes of Cancer

Naturopathic physicians are trained to treat the cause, not the symptom, of an illness. When it comes to cancer, how often do doctors stop and try to treat the cause? That, of course, would assume that we know what causes cancer. Cancer is a multifactorial process. Hereditary, immune, and environmental factors play a role, as well as viruses and bacteria. There are over 200 types of cancer, each with multiple causes. We may never know the full range of causative agents or combination of agents, but environmental factors must be considered.

The International Agency for Research on Cancer has identified 415 known or suspected carcinogens, which can be viewed at the website <http://monographs.iarc.fr>. Preventing exposure to individual known carcinogens may in turn prevent cancer. Physicians can learn which environmental exposures are linked to specific cancers in order to help prevent the disease and prevent recurrence. We may not be able to pinpoint the cause of cancer, but we can act on what we know to prevent exposures that contribute to it.

Below is a brief outline as to which toxins are linked directly or indirectly to cancer. This outline will provide a foundation for taking a good environmental exposure history and assist in determining which toxins to test for in patients. Something to consider when addressing environmental causes of cancer is that although everyone is exposed to chemicals on some level every day, not everyone gets cancer. Certain factors play a role in whether or not environmental exposures cause cancer.

Such factors include:

- timing of exposure
- dose of exposure
- duration of exposure
- synergistic effect
- genetic polymorphisms

In the past, researchers and clinicians thought that the dose or duration of the exposure was what determined if

the toxin was carcinogenic. This was driven by cases of acute toxicity due to high-dose exposure to a chemical, and occupational studies wherein a worker exposed for a long period of time to a toxin developed cancer. New research has focused on low-dose exposure, such as through food, and the synergistic effect of being exposed to more than one chemical at a time.¹ Also, the timing of the exposure in a patient's life has proved critical. If exposure occurs during pregnancy, there are higher rates of childhood and adult cancers in those exposed in utero.² Different periods of human development are more susceptible to chemical exposure than others, such as when chemicals pass through the placental barrier and during puberty. A recent study showed that children exposed as neonates and during puberty to endocrine-disrupting chemicals such as pesticides, PCBs, and arsenic had increased risk of prostate cancer as adults. This again outlines the importance of the critical window of exposure.³

Genetic polymorphisms need to be considered when discussing why some people exposed to chemicals get cancer and others don't. Genetic polymorphism is a difference in DNA sequence among individuals groups or populations. Single nucleotide polymorphisms, SNPs, are a single base mutation in DNA. Researchers follow SNPs along the genome to track disease genes. Polymorphisms in enzymes that metabolize environmental toxins play an important role in gene-environment interactions and may contribute to individual susceptibility to cancer risk. Toxins are broken down in the liver during phase 1 detoxification involving the cytochrome P450 enzymes. In phase 2 of liver metabolizing, toxins are further broken down through conjugation reactions. Determining patients' SNPs along with exposure history can help determine cancer risk or perhaps explain cancer cause. Almost every cancer has been linked to at least one genetic polymorphism, and testing for SNPs should be considered in all patients with cancer. Take breast cancer as a model.

Breast Cancer

According to the April 20 issue of the *International Journal of Cancer*, women with a N-acetyltransferase 2 (NAT2) polymorphism have an increased risk of breast cancer; those who were also smokers had a 2.4-fold higher risk of breast cancer.⁴ Alcohol consumption has been linked with increased breast cancer in the past, but the mechanism has not been well understood. New research involving a polymorphism of alcohol dehydrogenase may provide answers. Acetaldehyde, the first and most toxic metabolite of alcohol, is broken down by alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH). Two separate studies have found that women with polymorphisms of these enzymes who drink even small amounts of alcohol have increased risk of breast cancer.^{5,6} Other SNPs to consider in breast cancer are the enzymes that break down estrogens such as cytochrome P450 1B1 and 3A4 and the methylation and glutathione pathway.

Links

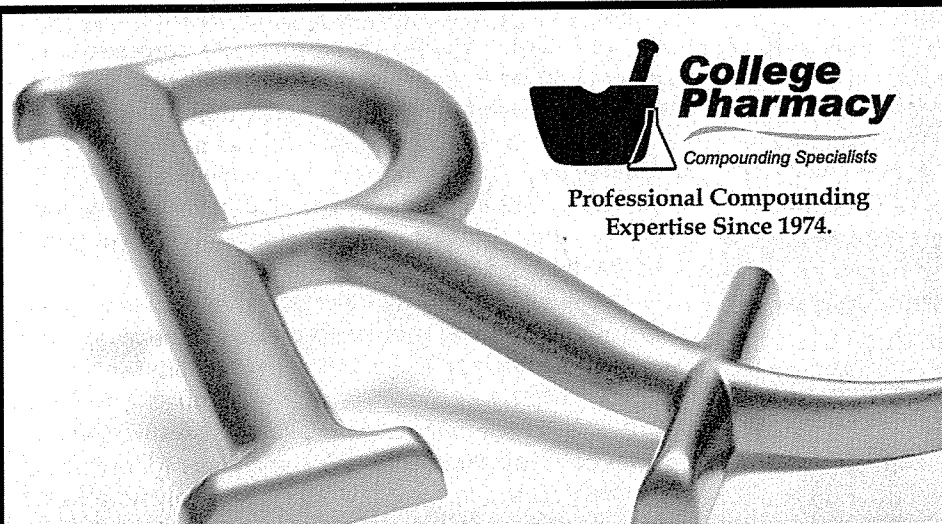
New research is constantly being published linking environmental toxins to specific cancers. Soon even the skeptics won't be able to ignore the evidence. For example, scientists at the University of Massachusetts Lowell and Boston University published an updated scientific review, *Environmental and Occupational Causes of Cancer: New Evidence, 2005–2007*.⁷ The original review was published

in 2006 in the *Journal of Public Policy*.⁸ The updated 2007 report synthesizes the recent peer-reviewed scientific literature and finds compelling new evidence linking cancer with specific exposures, namely:

1. breast cancer from exposure to the pesticide DDT before puberty
2. leukemia from exposure to 1,3-butadiene
3. lung cancer from air pollution
4. gliomas from permanent hair dye
5. non-Hodgkin's lymphoma from exposure to pesticides and solvents
6. prostate cancer from exposure to pesticides and metal working fluids
7. brain cancer from exposure to nonionizing radiation, particularly from mobile phones
8. a range of cancers from exposure to pesticides

History

As more and more evidence links low-dose exposure to common chemicals in the environment, physicians need to evaluate patients for risk. This starts with taking an in-depth environmental exposure history, looking at where a patient grew up or where his mother was living prior to pregnancy. At www.scorecard.org, any zip code can be entered showing the degree of air and water pollution for that area, as well as government Superfund sites. Evaluating places



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of employment, occupations, hobbies, places they have lived, lifestyle, and social factors can provide clues as to what types of toxins patients have been exposed to over the years. For an example of an environmental exposure intake form, please visit www.drmarchese.com and download the form on the "Environmental" page.

Testing

After determining the toxins that the patient has been exposed to, testing can be performed to see which ones are circulating or stored in the body. Pacific Toxicology Lab offers blood and urine testing for various solvents, pesticides, and heavy metals. Numerous other labs offer heavy metal testing through the blood and urine as well. The most common heavy metal test to determine stored metals is a provocative urine test wherein a body-weight dose of a heavy metal chelator is given to a patient and then the urine is collected for 6 hours. This test should not be performed while a patient is undergoing chemotherapy and radiation. The chelator may affect the chemo agent. This type of testing should be performed after conventional oncology care is complete. Testing a patient for genetic polymorphisms in the liver is essential in determining why she may not be clearing toxins from the body or is more susceptible to the carcinogenic effect of the toxin.

Avoidance

If history and testing determine that the patient has been exposed to a chemical linked to cancer, clearing toxins from the body should commence with a chelation or cleansing program. Again, it is best to wait until after

conventional oncology is complete to begin this process, as it can be time-consuming and may fatigue the patient. At a minimum, the patient should be educated on avoiding toxins at home, work, and travel. This includes education on air filtration, water filtration, diet, and lifestyle changes.

Resources and Further Information

Books

- Silent Spring*, by Rachel Carson
Our Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival? A Scientific Detective Story, by Theo Colburn
Living Downstream: A Scientist's Personal Investigation of Cancer and the Environment and Having Faith, both by Sandra Steingraber
Consumer's Dictionary of Cosmetic Ingredients, by Ruth Winters
Drop Dead Gorgeous: Protecting Yourself from the Hidden Dangers of Cosmetics, by Kim Erickson

Websites

- My Web page: drmarchese.com
Cornell University Program on Breast Cancer and Environmental Risk Factors: envirocancer.cornell.edu
Environmental Health News: environmentalhealthnews.org
Green Guide for Everyday Living: thegreenguide.com
Health Care without Harm: noharm.org
The Alliance for a Healthy Tomorrow: www.healthytomorrow.org
Northwest Coalition for Alternatives to Pesticides: pesticide.org
Environmental Working Group: ewg.org

Summary

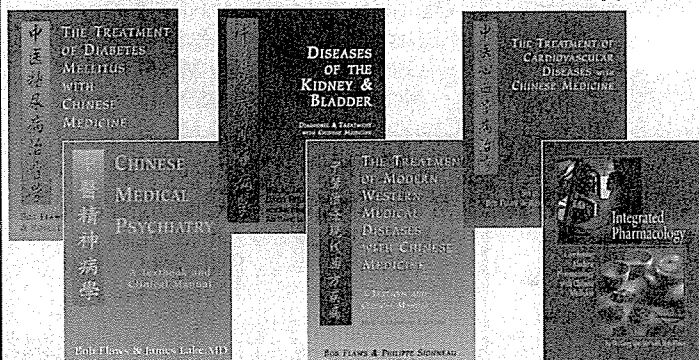
- Cancer arises from a complicated web of multiple causes, and we may never know the full range of agents or combinations of agents.
- We can learn which environmental exposure are linked to specific cancers in order to prevent the disease and prevent recurrence.
- In the end, we must act on what we know to prevent exposures at home, at work, and in the environment that contribute to cancer.

Notes

1. Welshons WV et al. Large effects from small exposures. I. Mechanisms for endocrine-disrupting chemicals with estrogenic activity. *Environ Health Perspect.* 2003;111:94-1006.
2. Anderson LM et al. critical windows of exposure for children's health: cancer in human epidemiological studies and neoplasms in experimental animal models. *Environ Health Perspect.* 2000;108(3):573-594.
3. Prins GS. Endocrine disruptors and prostate cancer risk. *Endocr Relat Cancer.* 2008;15(3):649-656.
4. Hirvonen SP et al. NAT2 slow acetylator genotype as an important modifier of breast cancer risk. *Int J Cancer.* 2005;114(4):579-584.
5. Coutille C et al. Risk factors in alcohol associated breast cancer: alcohol dehydrogenase polymorphisms and estrogens. *Int J Oncol.* 2004;25(4):1127-1132.
6. Seitz HK, Becker P. Alcohol metabolism and cancer risk. *Alcohol Res Health.* 2007;30(1):38-41.
7. Clapp R, Jacobs M, Loechler E. Environmental and occupational causes of cancer: new evidence, 2005-2007. Available at www.sustainableproduction.org.
8. Clapp R, Howe G, Jacobs M. Environmental and occupational causes of cancer revisited. *J Public Health Policy* 2006;27:61-76.

INTEGRATION INFORMATION

Books with an understanding and explanation of both Western & Chinese medical theory. Chinese diagnosis and treatments for common diseases of all types.



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