



Cornell University Program on Breast Cancer and Environmental Risk Factors in New York State (BCERF)

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FACT SHEET #41

Polycyclic Aromatic Hydrocarbons and Breast Cancer Risk

Polycyclic aromatic hydrocarbons (PAHs) are a group of over 200 different chemicals formed when coal, wood, gasoline, oil, tobacco or other organic materials are burned. They can be formed in food when fish or meats are charbroiled. Several PAHs have been identified as chemicals that can cause mammary (breast) cancer in laboratory rats and mice. Whether PAHs found in the environment affect breast cancer risk in humans is an active area of research. Workplace exposure to mixtures containing PAHs and other chemicals has been associated with a higher risk of other types of cancers. This fact sheet provides information about cancer risk of PAHs, sources of exposure, ways to minimize exposure, applicable regulations and ongoing research.

What are polycyclic aromatic hydrocarbons (PAHs)?

The term, *polycyclic aromatic hydrocarbons* or PAHs, refers to a group of over 200 chemicals made up of varying numbers of carbon and hydrogen atoms connected in ring-like forms. PAHs are environmental pollutants that may be found in air attached to dust particles, in soil, stream sediment, water and food. They are found in nature in coal and crude oil and in emissions from forest fires and volcanoes. Most PAHs entering the environment are formed unintentionally during burning (coal, oil, wood, gasoline, garbage, tobacco and other organic material) or in certain industrial processes. PAHs usually occur as mixtures of two or more PAHs and other chemicals.

Do PAHs cause breast cancer in laboratory animals?

Researchers have reported that female rats receiving a single injection of the synthetic PAH called

7,12-dimethylbenz[a]anthracene (DMBA) developed mammary (breast) tumors. Several other purified PAHs are well known breast carcinogens in laboratory animals. The PAH, *benzo[a]pyrene*, caused breast cancer in rats when injected into breast tissue or when high doses were fed to rats and to mice over a long period of time. One study compared the ability of *benzo[a]pyrene* and two other PAHs to cause cancer in rats when injected into breast tissue. Using the same dose for each, *benzo[a]pyrene* had marginal ability to induce breast tumors, while *DMBA* caused a higher number of animals to develop breast tumors. A third PAH tested, *dibenzo[a,l]pyrene*, was identified as having the highest breast cancer potency of all PAHs tested so far.

Do PAHs cause breast cancer in humans?

It is not known whether PAHs affect breast cancer risk in humans. Studies done to date are not conclusive. Some of the research on PAHs and breast cancer in humans is summarized below.



- **Cigarette Smoking:** While cigarette smoking is a major source of exposure to PAHs, most studies have not seen a higher risk of breast cancer in women who smoke.
- **Workplace studies:** A small number of studies have been conducted involving women with work exposure to PAHs. None have shown an increased breast cancer risk. These studies involved a small number of women, and larger studies are needed to further evaluate PAH exposures and breast cancer risk in the workplace.
- **Markers of exposure:** More recent studies have begun to evaluate whether measurable markers (indicators) of exposure to PAHs are associated with breast cancer risk. Once PAHs enter the body, they go to most internal organs, especially those containing fat. Most PAHs are converted in the body to non-toxic materials and leave the body in a few days in urine and bowel movements. Some become activated and bind to genetic material (DNA) forming *PAH-DNA adducts*. PAH-DNA adducts are markers of PAH exposure. The formation of DNA adducts is thought to be necessary for the development of cancer.

One study compared PAH-DNA adduct levels in 100 women with breast cancer to 105 women without breast cancer. Twice as many of the women with breast cancer had high PAH-DNA adduct levels in their breast tumors compared to the levels in breast tissue of women without breast cancer. However, the average levels of DNA-PAH adducts was similar in the normal breast tissue of women with breast cancer and in the women without the disease. So, while PAH-DNA adducts may be formed in greater numbers and accumulate in breast tumors, breast cancer risk cannot be predicted from levels of PAH-DNA adducts in normal breast tissue. The role of

PAHs in the development and progression of breast cancer is still under investigation.

- **Genetic differences:** Studies are also underway to evaluate the risk of breast cancer due to differences in genes (called genetic polymorphism). Certain genes affect the way the body activates or breaks down PAHs. One gene of interest is called *CYP1A1*. In a study of 216 women with breast cancer and 282 women without breast cancer, women with different types of the *CYP1A1* gene (polymorphism of *CYP1A1*) who also smoked cigarettes had a higher risk of breast cancer. A long-term national study, the Nurses Health Study, found no overall increase in risk of breast cancer in women with different types of *CYP1A1*. The results suggested that women with one type of *CYP1A1* who had begun cigarette smoking before the age of 18 years were at increased risk of breast cancer. However, this finding involved only eleven women with breast cancer, which is a very small proportion of the total 466 breast cancer cases in the study. While not conclusive, these findings should be investigated in further studies.

Why do PAH breast cancer results in humans differ from results in animals?

- Some of the PAHs that cause breast tumors in animals (like DMBA) are synthetic chemicals only used in laboratory experiments. Humans are not exposed to pure, high doses of individual PAH that are used in laboratory animal studies. They are exposed to low levels of mixtures of PAHs and other environmental pollutants.
- Studies have shown that most PAH mixtures are less potent than individual PAHs. Interactions between PAHs and other chemicals in the mixtures may reduce the potential to cause cancer.



- Laboratory animals may be more susceptible to PAHs because of differences in the activation or detoxification of PAHs.
- Age of exposure to PAHs may also be important. Young female rats treated with PAHs when their breasts are developing have a very high risk of forming breast tumors. In contrast, if a PAH is given to an older animal that completed a pregnancy and has mature breasts, the risk of developing breast tumors is much lower. More studies in humans are needed to determine whether exposure to PAHs during puberty when the breast is developing affects breast cancer risk in later years.

Do PAHs cause other cancers?

Several PAHs have caused cancer of the skin, lungs, stomach, liver and bladder in laboratory mice or rats. The PAHs were given to the animals over a long period of time by applying them to the skin, through the diet, in the air, or by injection into their veins. The tumors tended to develop at the site of contact with the PAH, but also at more distant sites.

Exposure to PAHs in the workplace is not due to single PAHs, but to a mixture of several PAHs and other known or possible cancer-causing substances. Breathing mixtures, like cigarette smoke or exposures from coal coke-ovens in the workplace, causes increased deaths from lung cancer. A few occupational studies reported increases in skin cancer after prolonged skin exposure to mixtures of PAHs and other known cancer-causing chemicals. Aluminum smelter and coal coke-oven workers had an increase in urinary bladder cancer, but the increase appeared more likely due to the other known cancer-causing agents in the workplace. Since some individual PAHs and certain PAH mixtures cause cancer in laboratory animals, it is assumed the

increased cancer risks seen in the human studies may be caused in part by PAHs, but the evidence is not conclusive.

How can I be exposed to PAHs?

Everyone has some exposure to PAHs. The main sources of exposure to PAHs for the general public are from breathing PAHs (in tobacco smoke, wood smoke, traffic exhaust and other contaminated air), from eating PAHs formed in charbroiled foods, and from drinking contaminated water. For many people, the greatest exposure to PAH occurs in the workplace from breathing PAHs in the air and from skin contact. Here are some examples:

Air: PAHs are found in tobacco smoke. Smoking increases personal daily PAH exposure by 30-fold. PAH levels are higher in urban areas where there is heavy traffic exhaust and high diesel engine use. Levels are also higher where coal, oil, wood, tires, or agricultural crops are burned. Workplace exposures with higher air levels of PAH include: coal coking, coal tar production, aluminum/iron/steel production, municipal trash incineration, asphalt production and smokehouse operations.

Food: Foods grown in areas with PAH-contaminated soil or air may contain higher levels of PAHs. Charbroiled meat or fish and smoked foods (meats and cheeses) have higher levels of PAHs.

Water: Water can become contaminated with PAHs from runoff in urban areas, waste water from certain industries (e.g. creosote manufacturing, aluminum smelting), or from PAHs carried in the air. The level of PAH in untreated drinking water is approximately five times that of treated municipal drinking water supplies.



Skin contact: Concentrations of PAHs in soil from areas of heavy traffic and automobile exhaust are approximately 50-times higher than in non-polluted areas. Near industrial sources of PAHs, levels in soil may be several thousand times higher. Most PAHs in soil come from PAH in the air. The level varies with the distance from the polluting source. Other important sources for soil contamination include: sludge from sewage treatment plants; sites used for burning coal, wood, or gasoline; and former industrial sites like wood-preserving facilities using creosote. PAH-containing personal care products include certain prescription coal tar body lotions and anti-dandruff shampoos (not over-the-counter lotions or shampoos). Working with creosote or asphalt can increase exposure to PAHs.

PAHs can be broken down in the environment. In the air, PAHs break down by reacting with sunlight and other chemicals over a period of days to weeks. Bacteria can break down PAH in water and in soil after a period of weeks to months. However, certain PAHs can stay in the soil, groundwater (well water) and sediment of rivers and lakes for years.

Are there medical tests to see if I've been exposed to PAHs?

There is no medical test generally available in a physician's office to check if you have been exposed to PAHs, or if you are more susceptible to a health effect from PAH exposure. Certain tests are used by researchers and workplace physicians to compare groups of people who have had greater exposure to PAHs to those who have not had the exposure. Tests include measuring PAH-breakdown products in the urine, and testing blood, skin, lung, or other body samples for PAHs or PAH-DNA adducts. There is great variability in results, however, because of individual differences in ability to

activate or break down PAHs, and to repair DNA damage caused by PAHs. While the tests do tell if groups of people were exposed, they do not tell when the exposure occurred, or if exposures were high or low.

What can I do to reduce my exposure to PAHs?

- Don't smoke tobacco products and avoid exposure to tobacco smoke.
- Make sure indoor kerosene heaters, wood-burning stoves and fireplaces, and coal-burning furnaces are working properly and are well vented. Consider using alternatives to burning fossil fuels, such as solar and wind power.
- Minimize exposure to PAH in the workplace by making sure that equipment is operating properly, follow good work practices, and use appropriate personal protective equipment.
- Avoid open burning of garbage, leaves and yard waste, construction materials, tires or the burning of agricultural fields.
- Properly dispose of used motor oils, gasoline, diesel fuel, heating oil, and creosote to prevent soil and water contamination.
- Eat less smoked meats, fish or cheese. When grilling or broiling avoid charring the food or letting the food come in contact with the heat source or flame.
- Use public transportation or car pool to reduce car exhaust.



How are PAHs regulated?

The US Occupational Safety and Health Administration (OSHA) sets the limits for maximum exposures allowed for specific workplace mixtures of PAHs, like coke oven emission. (For more information see OSHA's web site at <http://www.osha-slc.gov/SLTC/coaltarpitchvolatiles/index.html>) The National Institute of Occupational Safety and Health (NIOSH) recommends exposure limits to PAH mixtures in other work sites, such as asphalt paving. The Environmental Protection Agency (EPA) requires that industries report environmental releases of PAH that are greater than one pound.

The New York State Department of Environmental Conservation sets limits for certain mixtures of PAH in water. The maximum level of total PAHs allowed is 0.2 microgram per liter of water (0.2 millionth of a gram in one quart of water).

What kinds of new research are being done?

- NIOSH is conducting research on miners exposed to diesel exhaust and workers in foundries to evaluate the effect of PAH exposures on formation of DNA-adducts and on the function of anti-cancer tumor suppressor genes.
- National Center of Environmental Health is investigating new methods to measure PAH biomarkers and to evaluate exposure to PAHs.
- Breast cancer researchers, funded by the National Cancer Institute (NCI) and the Department of Defense, are studying differences in genes (genetic polymorphisms) that affect the way estrogen and environmental cancer-causing agents (including PAHs) are handled by the body.

- The NCI and National Institute of Environmental Health Sciences are funding the Long Island Breast Cancer Study, which is evaluating whether breast cancer risk is affected by exposure to PAHs or to persistent organochlorine pesticides.

What are future research needs?

- Studies are needed to evaluate whether lifestyle habits in childhood or adulthood associated with PAH exposure (e.g. breathing tobacco smoke or eating charbroiled meats or smoked foods) affect breast cancer.
- Further studies are needed of to see if the levels of PAHs in urine or body tissues can be used to predict the level of PAH exposure that has occurred or the likelihood of developing cancer.
- Additional studies are needed to determine if other individual PAHs or PAH mixtures not yet evaluated cause cancer in animals or humans.
- Studies of workplace PAH exposures should be expanded to include larger numbers of workers and better measures of PAH exposure.
- Confirmation is needed of studies that have shown an increased risk of breast cancer associated with specific gene types (genetic polymorphism).

Conclusions

- PAHs are found widely in the environment as well as in certain workplace situations.
- Several PAHs can cause breast tumors and/or other tumors in laboratory animals.
- In studies conducted to date, women with workplace exposures to PAHs do not have a higher risk of breast cancer.




- Most studies have not found a higher risk of breast cancer in women who smoke, even though tobacco smoke is a major route of exposure to PAHs.
- Whether low levels of PAHs in the environment affect the risk of human breast cancer risk is still under investigation.
- Exposure to mixtures of PAHs and other chemicals increases the risk of other human cancers, such as lung cancer.

An extensive bibliography on *Polyaromatic Hydrocarbons and Breast Cancer Risk* is available on the BCERF web site: <http://www.cfe.cornell.edu/bcerf/>

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