



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

Environmental Chemicals and Breast Cancer Risk Why is There Concern?

There has been growing interest in whether environmental factors, including exposures to certain chemicals or changes in lifestyle, may increase the risk of breast cancer. This fact sheet will discuss research linking environmental chemicals and the risk of breast cancer. This will include exposures of concern in the home and workplace, and chemicals known to cause mammary (breast tumors) in laboratory animals. The fact sheet will also discuss new emerging data on how exposures to certain chemicals early in life may affect breast development and breast cancer risk, as well as new work identifying important gene-environmental interactions. Current challenges and new avenues of research also will be discussed.

Established risk factors only partially explain breast cancer risk

Risk factors consistently associated with a higher breast cancer risk are called “established” risk factors. Established risk factors include getting older, having regular menstrual periods earlier, going through menopause later in life, having a first child late in life, not having any children, having a mother or sister with breast cancer, past exposure of breasts to ionizing radiation, or having certain types of benign breast disease. But these factors explain only about 25 to 50% of breast cancer cases (Madigan et al., JNCI vol. 87, pp. 1681-1685, 1987; Rockhill et al., Am. J. Epidemiol., vol. 147, pp. 826-833, 1998).

Geography, migration and rates of breast cancer: Are there environmental links?

Breast cancer rates vary widely in different parts of the world. Rates are the highest in North America, Northern Europe and Australia. Breast cancer rates are much lower in Japan, China, Africa and India (IARC, GLOBECAN, 2000). It is not clear why there are geographical differences in breast cancer rates. Differences in age of childbearing, diet, lifestyle and exposure to environmental chemicals have been offered as possible explanations. Studies of breast cancer rates of Japanese women who migrate to the US suggest an environmental influence on the risk of breast

cancer. Within one or two generations the breast cancer rates of descendants of Japanese women migrating to the US increase, and become similar to the higher breast cancer rates of western women (Shimizu et al., Br. J. Cancer, vol. 63, pp. 963-966, 1991). Results of studies on twins in Scandinavia also suggest that a woman’s environment plays a significant role in determining her breast cancer risk. In this study inherited factors accounted for about 27% of breast cancer risk, suggesting that environmental factors play a major role in determining the risk of breast cancer (Lichtenstein, N. Engl. J. Med., vol. 343, pp. 78-85, 2000).

How can you be exposed to environmental chemicals?

We are exposed to thousands of naturally occurring and synthetic chemicals over a lifetime. Many chemicals are essential for life and are beneficial, while exposures to other chemicals can be harmful and affect our health. There are many ways our bodies can be exposed to chemicals. This includes exposure in the air we breathe, in the food and beverages we eat, and by contact with our skin. Fetuses can be exposed to chemicals that cross the placenta during pregnancy. Some environmental contaminants can pass from a mother’s body to an infant through breast milk. Certain chemicals can be stored in the fat of fish or animals, becoming more concentrated as they pass up the food chain. These chemicals can be stored in the body for a long time.



Other chemicals may be broken down and are quickly eliminated from the body. Some chemicals first need to be “activated” by enzymes in the body to become cancer-causing chemicals (carcinogens). Other chemicals pose no cancer risk, while others may act as beneficial “anti-cancer” agents. It is impossible to make generalizations about environmental chemicals. Each chemical has a unique pattern in the way it is handled by the body, and has a different potential for whether or not it can contribute to breast cancer risk.

Are there concerns about chemicals in the home and workplace?

We can be exposed to a variety of synthetic chemicals in many different settings, including in our homes and workplaces. Some chemical exposures in the workplace have been associated with a higher risk of breast cancer (See Table 1). More research is needed to help identify the chemicals of concern for different workplace situations. There are relatively few studies of women in the workplace (most occupational studies of cancer risk have been done on men). There is a need for better quality studies to give us better answers. Many of the studies done so far had very limited data on exposure to specific chemicals, and usually only small groups of women were followed for a limited time period.

Several groups that need further evaluation because of potential exposures to known or potential carcinogens include those employed in the chemical and pharmaceutical industries, laboratory and biomedical workers, cosmetologists and hair dressers, workers in semi-conductor, printing and textile dyeing industries, airline personnel, health care workers, and metal plate workers (Aaronson and Howe, *JOEM*, vol. 36, pp. 1174-1179, 1994; Cantor et al., *JOEM*, vol. 37, pp. 336-348, 1995; Habel et al., *JOEM*, vol. 37, pp. 349-356, 1995; LaBreche, vol. II., Sect. 4, <http://www.breast.cancer.ca>, posted 2001).

Of recent interest is whether breast cancer risk may be indirectly affected in night-shift workers exposed to “light at night” which may affect melatonin synthesis (Steven and Rea, *Cancer Causes Control*, vol. 12, 279-287, 2001). Scientists are exploring whether changes in melatonin levels may affect levels of estrogen and breast cancer risk (Davis et al., *JNCI*, vol. 93, pp. 1557-1562, 2001; Hansen et al., *Epidemiology*, vol. 12, pp. 74-7, 2001; Schernhammer et al., *JNCI*, vol. 93, pp. 1563-1568, 2001).

Table 1. Examples of Chemical Exposures in the Workplace Associated with Some Evidence of a Higher Breast Cancer Risk

Acid mists
Benzene
Carbon tetrachloride
Ethylene oxide
Formaldehyde
Lead oxide
Methylene chloride
Styrene

References:

Blair and Kazerouni, *Cancer Causes & Control*, vol. 8, pp. 473-490, 1997
Cantor et al., *JOEM*, vol. 37, pp. 336-348, 1995
Goldberg and Lebreche, *Occup. Environ. Med.*, vol. 53, pp. 145-156, 1996
Hansen, *Am. J. Ind. Med.*, vol. 36, pp. 43-7, 1999
Norman et al., *Int. J. Epidemiol.*, vol. 24, pp. 276-284, 1995
Spiritas et al., *Br. J. Ind. Med.*, vol. 48, pp. 515-530, 1991

Researchers are also interested in measuring chemicals women may be exposed to every day at home. Researchers on Long Island, New York and on Cape Cod, Massachusetts are measuring levels of environmental chemicals in the homes of women with and without breast cancer (see Long Island Breast Cancer Study Project’s website <http://epi.grants.cancer.gov/LIBCSP/>; Cape Cod Breast Cancer Study, Rudel et al., *J. Air Waste Manage. Assoc.* vol. 51, pp. 499-513, 2001). Such studies may help identify the types of chemicals in the home that may be linked to a higher risk of breast cancer. It is important to characterize the types of chemicals found in the home environment. These studies help to identify sources and patterns of exposure, and prioritize chemicals that need further study.

Why is there concern that pesticides may affect breast cancer risk?

There has been concern about exposure to pesticides because of their widespread use in agriculture for crop and livestock protection, for public health in controlling disease-bearing insects, for pest control in homes, schools,



workplaces, gardens, and in recreational areas such as parks and athletic fields. Currently, there are about 865 pesticide active ingredients registered with the Environmental Protection Agency (EPA), and thousands of products containing these chemicals singly or in combination.

Much of the concern about whether pesticides affect breast cancer risk stems from observations of higher rates of cancer in male workers with high exposures to pesticides. There are higher rates of some cancers in male farm workers, including lip and skin cancer, non-Hodgkin's lymphoma, and cancer of the stomach, brain and prostate (Blair and Zahm, *Environ. Health Perspect.* vol. 103 [Suppl. 8], pp. 205-208, 1995). Some of these cancers are due to excessive exposure to UV radiation from the sun (lip and skin cancer). There are many types of exposures on the farm that may affect cancer risk, including exposures to pesticides, solvents, fuel exhaust, and toxins (called mycotoxins) from molds that form in stored crops. While some scientists have found higher cancer rates in farmers exposed to certain pesticides, other studies have not supported an association. An ongoing, large-scale study that will help provide better answers to whether specific chemicals used in agriculture affect cancer risk is the "Agricultural Health Study" (for more information go to <http://www.aghealth.org/index.html>).

Are breast cancer rates higher in women living or working on a farm?

There are very few studies that have evaluated whether farm women have a higher risk of breast cancer (Blair and Zahm, *Environ. Health Perspect.*, vol. 103 [Suppl. 8], pp. 205-208, 1995). In a study of North Carolina farm women, overall breast cancer rates were lower in women who lived or worked on a farm compared to women who did not work or live on a farm. It has been suggested that these farm women may have lifestyles or risk factors that could have reduced their risk of breast cancer (later age at menarche, earlier age at first birth, higher number of pregnancies, less likely to smoke or drink alcohol, higher level of exercise). However in this study, one group of farm women who did not wear protective clothing or gloves when applying pesticides had a two-fold higher risk of breast cancer compared to women who did take proper precautions. The results of this small study suggest that breast cancer risk may be increased in some farm women with high exposures

to pesticides. This study illustrates the importance of reducing exposures to pesticides in workplace situations (Duell et al., *Epidemiology*, vol. 11, pp. 523-531, 2000).

Do organochlorine pesticides affect breast cancer risk in women?

Organochlorine pesticides were used extensively during and after WWII because of their long-lasting effects in controlling insects. Most were banned during the 1970s and 1980s in the US, Canada and Europe because of human health and ecological concerns. Some examples of organochlorine pesticides include: DDT (used in mosquito control and agriculture), dieldrin (used to control termites and other soil insects), chlordane and heptachlor (used to control termites and fire ants), lindane (currently used in agriculture and in anti-lice shampoos), beta-hexachlorocyclohexane (by-product of lindane manufacture), and hexachlorobenzene (fungicide used to prevent mold on crops). These long-lasting chemicals concentrate as they pass up the food chain and are stored in the body fat of animals, fish and humans. Some are endocrine disruptors that affect reproduction in wildlife, especially birds and reptiles. While there are links to some types of cancers (for instance, several organochlorines induce liver or thyroid tumors in laboratory animals), effects on breast cancer risk in humans have been studied only recently.

The organochlorine pesticide that has been studied the most extensively is the insecticide DDT. Over time, DDT breaks down in the environment to a very long-lasting chemical called DDE. Early reports suggested that women with high levels of DDE in their blood or fat had a higher risk of breast cancer. However, the majority of the more recent, well-controlled studies have not been able to confirm these findings. Most of these studies have looked at breast cancer risk in white women living in North America and Europe. These studies of western women have not shown a higher risk of breast cancer in those with higher levels of DDT or DDE. Other populations, including different ethnic groups, have not been studied as well. The results from several studies suggest that breast cancer risk may be higher in African American women who have higher body levels of DDE. We don't have clear answers of whether breast cancer risk is higher for women who live in less industrialized tropical countries that still use DDT against mosquitoes for malaria control. More studies are needed to explore these areas.



For many of the other organochlorines, we have very limited data from human studies. Breast cancer risk was higher in Danish women with high blood levels of dieldrin, but the few studies done on American women have not confirmed this finding. For dieldrin, and other organochlorine pesticides, there are too few studies in women to make a conclusion of whether or not body levels are associated with breast cancer risk (Snedeker, *Environ. Health Perspect.*, vol. 109 [Suppl. 1], pp. 35-47, 2001).

What can we learn from animal studies?

While human studies are given the greatest weight when deciding whether or not a chemical causes cancer, there is little or no information on the cancer-causing potential of most chemicals in people. Much of the information on chemicals and cancer risk comes from carefully controlled laboratory animal studies called “cancer bioassays.” Animal studies are used by federal agencies to identify the hazard and to estimate the cancer risk to humans. These studies are important to help predict cancer risk when human studies are unavailable. In a cancer bioassay, male and female animals from two species (usually mice and rats) are exposed for most of their lives to a range of levels (doses) of the chemical. This approach is intended to maximize the likelihood of detecting cancer-causing chemicals. Before the EPA allows a pesticide to be registered for use, the primary manufacturer (registrant) must submit the results of cancer bioassays conducted in laboratory animals. The EPA can ask for additional studies to be conducted when a pesticide is reviewed for reregistration (for more information on how EPA assesses the health risks of pesticides see <http://www.epa.gov/pesticides/citizens/riskassess.htm>).

Animal studies are also conducted by the National Toxicology Program, a federal agency that screens a variety of chemicals for their cancer-causing potential (see <http://ntp-server.niehs.nih.gov>). Of the 509 chemicals tested by this agency, 42 chemicals were found to cause breast tumors (called mammary gland tumors) in laboratory animals. There is a wide range of different types of chemicals that cause breast tumors in laboratory animals. Some examples include several pharmaceutical drugs, chemical solvents and flame retardants; a variety of chemicals used in the manufacturing of dyes, rubber, vinyl and polyurethane foams; a sterilizing agent for medical instruments; a food additive; several fumigants

and pesticides; a metal used in microelectronics; a mycotoxin produced by molds; and a gasoline additive (See Table 2). Some of these chemicals are still used and produced (for instance, methylene chloride). Others are no longer manufactured or limits have been placed on the maximum exposure allowed in workplaces. This includes the herbicide sulfallate (registration cancelled by EPA), the soil fumigant 1,2-dibromo-3-chloropropane (banned by EPA), 1,2-dibromoethane used as a soil fumigant and as an anti-knock compound in gasoline (uses have been limited and maximum exposure standards have been set in the workplace), and ethylene oxide (limits placed on workplace exposures).

There is concern because some of these chemicals are environmentally persistent pollutants. One example is the soil fumigant 1,2-dibromo-3-chloropropane. Low levels of this persistent pesticide are still detected in well water in California and other states, even though all uses were banned by the EPA between 1979 and 1985 (CA EPA, EH95-06, 1995). Researchers from Finland reported that a chemical found in chlorinated drinking water called “MX” can cause a variety of cancers in laboratory animals, including mammary tumors in rats (Komulainen et al., *JNCI*, vol. 89, pp. 848-856, 1997). Researchers are monitoring levels and trying to find ways to reduce the level of this disinfection byproduct in drinking water supplies (Wright et al., *Environ. Health Perspect.*, vol. 110, pp. 157-164, 2002).

There are few studies available on the human breast cancer risk of the chemicals that are known to cause mammary tumors in laboratory animals. Ethylene oxide is an example of one of the few chemicals where we have evidence of a moderately higher breast cancer risk in women exposed to this chemical and evidence of mammary tumors in laboratory animals (Norman et al., *Int. J. Epidemiol.* vol. 24, pp. 276-284, 1995). Ethylene oxide is used to sterilize medical instruments. For other chemicals, species differences may influence the interpretation of results. Reserpine is an anti-hypertension drug known to increase levels of prolactin (Lee et al., *JAMA*, vol. 235, pp. 2316-2317, 1976), a hormone which plays a strong role in inducing breast tumors in rodents, but plays less of a role in human breast cancer. This species difference may explain why reserpine has been shown to be a mammary carcinogen in rodent bioassays, but there is not strong evidence that it affects breast cancer risk in women taking



**Table 2. National Toxicology Program's Cancer Bioassays
Examples of Chemicals that Cause Breast Tumors in Laboratory Animals**

Chemical solvents

Benzene
1,1-Dichloroethane
1,2-Dichloropropane
Methylene chloride
Nitromethane (also used in rocket and engine fuels)
1,2,3-Trichloropropane

Chemicals used or formed in the manufacturing of dyes

C.I. acid red 114
C.I. basic red 9
2,4-Diaminotoluene
3,3'-Dimethylbenzidine dihydrochloride
3,3'-Dimethoxybenzidine dihydrochloride
2,4-Dinitrotoluene
Hydrazobenzene
o-Nitrotoluene
o-Toluidine hydrochloride

Chemicals used in the manufacturing of rubber, vinyl, polyurethane foams or neoprene

Benzene (rubber manufacturing)
1,3-Butadiene (rubber manufacturing)
Chloroprene (neoprene manufacturing)
2,4-Diaminotoluene (polyethylene manufacturing)
1,2-Dichloroethane (vinyl chloride manufacturing)
Glycidol (vinyl manufacturing)
o-Nitrotoluene (rubber manufacturing)
2,4-2,6-Toluene diisocyanate (polyethylene foam manufacturing)

Chemical intermediates

Ethylene oxide (anti-freeze products)
Isoprene (formed during ethylene production)

Flame retardants

2,2-Bis(bromomethyl)-1,3-propanediol
2,3-Dibromo-1-propanol

References:

Dunnick et al., Carcinogenesis, vol. 16, pp. 173-179, 1995
Bennett and Davis, Environ. Mol. Mutagen., vol 39, pp. 150-157, 2002
NTP, 9th Report on Carcinogens, 2000
NTP, Chemicals associated with site-specific tumor induction in the mammary gland
<<http://ntp-server.niehs.nih.gov/htdocs/Sites/MAMM.HTML>> cited June, 2002.

Food additive

Methyleugenol (flavoring)

Fumigants and pesticides

Clonitralid (molluscicide)
1,2-Dibromoethane (also called ethylene dibromide)
1,2-Dibromo-3-chloropropane (soil fumigant)
1,2-Dichloroethane (soil/grain fumigant)
1,2-Dichloropropane (soil/grain fumigant)
Dichlorvos (insecticide)
Sulfallate (herbicide)

Gasoline additives

Benzene
1,2-Dibromoethane (lead scavenger)
1,2-Dichloroethane (lead scavenger)

Microelectronics

Indium phosphide (used in semiconductors)

Mycotoxin

Ochratoxin A (toxin produced by molds)

Pharmaceutical drugs

Acronycine (anti-cancer drug)
Cytembena (cytostatic drug)
Furosemide (diuretic)
Hydrazobenzene (used in making phenylbutazone, an antiarthritic drug)
Isophosphamide (anti-cancer drug)
Nitrofurazone (anti-bacterial agent)
Phenesterin (anti-cancer drug)
Procarbazine hydrochloride (anti-cancer drug)
Reserpine (anti-hypertension drug)

Sterilizing agent for medical instruments

Ethylene oxide

Research chemical

5-Nitroacenaphthenol

Riot control / Tear gas

2-Chloroacetophenone



this drug (Curb et al., *Hypertension*, vol. 4, pp. 307-311, 1982; Horwitz and Feinstein, *Arch. Intern. Med.*, vol. 145, pp. 1873-1875, 1985; Laska et al., *Lancet*, vol. 2, pp. 296-300, 1975; Mack et al., *N. Engl. J. Med.*, vol. 292, pp. 1366-1371, 1975).

Several polycyclic aromatic hydrocarbons (PAHs) have been identified as potent mammary carcinogens in animal studies. In the environment, PAHs are formed during the burning of fossil fuels (gasoline, coal, wood, oil), when tobacco is burned or when meats or fatty fish are charbroiled. While workplace exposures to mixtures of PAHs increases the risk of lung cancer, most studies have not shown a higher risk of breast cancer. One limitation of some of the workplace studies has been the small size of the studies. One larger study is evaluating whether exposures to PAHs affect breast cancer risk in women from Long Island, NY. (For more information on PAHs see the BCERF Fact Sheet #41 on *Polycyclic Aromatic Hydrocarbons and Breast Cancer Risk*)

Do endocrine-disrupting chemicals affect breast cancer risk?

Hormones and growth factors act as chemical messengers in the body. Certain hormones and growth factors are important in normal growth and functioning of the breast, but they also can have a role in the cancer process. Examples of these hormones include estrogen, progestins, prolactin, and growth hormone (Nandi et al., *Proc. Natl. Acad. Sci. USA*, vol. 92, pp. 3650-3657, 1995; Russo and Russo, *JNCI Monograph*, vol. 27, pp. 17-37, 2000). Examples of growth factors include insulin-like growth factors (Kleinberg et al., *Br. J. Cancer Res. Treat.*, vol. 47, pp. 201-208, 1998), and members of the epidermal growth factor family. In many cases these chemical messengers affect the rate of cell division in the breast, or they may work with hormones to help support the growth of breast tumor cells.

Breast cancer takes many years to develop – often up to 30 or more years – because of the many changes that must occur before a normal cell becomes a cancerous cell that divides out of control. Scientists are concerned that some environmental chemicals can either mimic the effects of hormones or growth factors, or affect how fast the body makes or breaks down these hormones. Through these actions an environmental chemical could affect the delicate balance that controls cell division. More than half of all breast tumors depend on estrogen for growth. Chemicals

that mimic the effect of estrogen may play a role in supporting the growth of estrogen-dependent breast tumors. For example, preliminary research suggests that occupational exposure to the environmental estrogen 4-octylphenol is associated with a higher risk of breast cancer (Aschengrau, et al., *Am. J. Ind. Med.*, vol. 34, pp. 6-14, 1998).

In addition to concerns about how environmental estrogens may affect breast cancer risk, there also is evidence that these “xenoestrogens” can affect reproduction in wildlife and possibly in humans (Crisp et al., *Environ. Health. Perspect.*, vol. 106 [Suppl. 1], pp. 11-56, 1998; McLachlan, *Endocrine Rev.*, vol. 22, pp. 319-341, 2001). Because of these concerns, the US Congress passed the Food Quality Protection Act in 1996. This legislation mandates that all pesticide active ingredients be tested for their estrogen-mimicking and other hormone disrupting effects. EPA is currently validating the screening tests that will be used. After these screening tests are validated, EPA expects to test more than 865 pesticide active ingredients and about 150 high volume industrial chemicals for endocrine-disrupting effects (for more information go to <http://www.epa.gov/scipoly/oscpendo/>).

Do chemical exposures early in life affect breast cancer risk?

Childhood and adolescence are critical periods of breast development. Exposures to cancer-causing chemicals when the breast is developing may affect breast cancer risk later in life. Studies have shown that the developing mammary glands (breast tissue) of young rats and mice have bud-like structures composed of rapidly dividing cells. These dividing immature breast cells are more susceptible to the damaging effects of cancer-causing chemicals. During pregnancy breast cells undergo changes making them more mature. Mature breast cells appear to be more resistant to the effects of carcinogens, and can more easily repair damage caused by cancer-causing chemicals. (See BCERF Fact Sheet #8 on *Childhood Life Events and the Risk of Breast Cancer*)

In utero exposures to estrogenic chemicals may increase breast cancer risk. A drug that acts like estrogen, called diethylstilbestrol (DES), was prescribed to pregnant women from the mid-1940s to 1970s, to prevent spontaneous abortions. Women who were treated with DES during



pregnancy have a moderately higher breast cancer risk (Calle et al., *Am J. Epidemiol.*, vol. 144, pp. 645-652, 1996; Colton, et al., *JAMA*, vol. 269, pp. 2096-2100, 1993; Greenberg et al., *N. Engl. J. Med.*, vol. 311, 1393-1398, 1984). DES can also cause mammary (breast) tumors in mice (IARC, Suppl. 7, 1987). This is one of the reasons researchers are interested in whether early exposures to chemicals in the womb affects breast cancer risk later in life.

Results from animal studies have shown that early exposures to some chemicals can have permanent effects on the way the breast develops and its susceptibility to carcinogens. Early exposure to certain environmental chemicals may keep the mammary gland in an immature state for longer periods of time, increasing its susceptibility to carcinogens (Fenton et al., *Toxicol. Sci.*, vol. 67, pp. 63-74, 2002). So, many chemicals may not cause a tumor to develop directly, but they may work in subtle ways to increase breast cancer risk. For instance, in one study female rats were exposed prenatally to an environmental contaminant, a dioxin called TCDD. When these dioxin-exposed rats were older, they were also exposed to a known breast carcinogen called dimethylbenz[*a*]anthracene (DMBA). The female rats pre-treated with dioxin developed more breast tumors than the rats not pre-treated with dioxin. The researchers suggested that the dioxin treatment prenatally changed how the breast tissue developed, keeping the breast in an immature state with a greater number of dividing bud structures for a longer time (Brown et al., *Carcinogenesis*, vol. 19, pp. 1623-1629, 1998). Similarly, results of preliminary studies conducted by EPA researchers have suggested that prenatal treatments with the herbicide atrazine can also help keep rat breast tissue in an immature state for prolonged periods of time (Fenton and Davis, *Toxicol. Sci.*, vol. 66, pp.185, 2002). While the implications for human cancer risk are not yet known, it is important that researchers fully explore the many ways chemicals may affect breast cancer risk.

How can genes influence responses to environmental chemicals?

Many chemicals have to become “activated” in the body to become carcinogens. Some people have differences (also called variations or polymorphisms) in certain genes that control these activation pathways. If a person has a variation in such genes, this may result in more activation and a higher level of the active form of the carcinogen. This may put the person at greater risk for developing certain cancers,

including breast cancer. For example, women with high body levels of environmental chemicals called polychlorinated biphenyls (PCBs) usually do not have a higher risk of breast cancer. However, in one study breast cancer risk was higher in a group of women who had both a high level of PCBs and a variation in an activation gene called *CYP1A1* (Moysich et al., *Cancer Epidemiol. Biomark. Prev.*, vol. 8, pp. 41-44, 1999). This is an example of a “gene-environment interaction.” More research is being done to identify important gene-environment interactions. This will help identify groups of women who may have a higher breast cancer risk if they are exposed to certain chemicals.

What are the challenges and new avenues for research?

There are many challenges that face scientists as they evaluate how breast cancer risk may be affected by exposure to environmental chemicals. Some of the greatest challenges are the complexity of the disease and that it takes many years for most breast cancers to develop. It is very difficult to characterize chemical exposures that occurred 10, 20 or even 30 years before a breast tumor is detected. It is also hard to determine how individual chemicals may affect breast cancer risk when we are exposed to low levels of thousands of chemicals over a lifetime.

What are potential avenues of future research? More studies are needed to explore the wide variety of chemicals that may affect breast cancer risk. For instance, there is interest in whether certain antihistamines and antidepressants affect breast cancer risk. There also is interest in whether environmental chemicals, such as certain phthalates used in plastics, play a role in premature breast development and later risk of breast cancer. We need better tools to identify potential cancer-causing chemicals and better ways to measure exposures to chemicals. New powerful molecular techniques are being developed that may help to identify “molecular” footprints, including identifying chemicals that activate specific cancer genes or that turn off genes that can suppress cancer. While studies are ongoing to screen for and identify breast carcinogens in animal cancer bioassays, new screening techniques are being developed that will allow for more rapid screening of a larger number of chemicals.

More research is needed to identify gene-environmental interactions that may help identify groups of women who may be at higher risk when exposed to certain chemicals, and identify endocrine-disrupting chemicals that can support




the growth of breast tumors. More research is needed not only to define the types of exposures encountered in the workplace and the home, but also to evaluate how exposure during critical periods of breast development may affect cancer risk later in life. A combination of human, animal, and molecular-based studies is needed to address how environmental chemicals may affect the risk of breast and other cancers.

Please note, while some citations are provided in the body of this Fact Sheet, a more extensive bibliography on *Chemicals and the Risk of Breast Cancer*, is available on the BCERF web site: <http://www.cfe.cornell.edu/bcerf/>

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