The Ribbon

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

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The Immune System, Environmental Factors, And Cancer

Where research has suggested a possible relationship, BCERF critical evaluations address the possibility that exposure to some pesticides may affect the immune system, which may play a role in defenses against some cancers. As in any area of emerging science, there are different perspectives; in this case there are varying ideas as to the relative importance of the immune system in the development of cancers. ***The Ribbon*** *asked two researchers from the College of Veterinary Medicine at Cornell to offer background and perspective on questions of the immune system, environmental factors, and cancer.*

Perspective: Environmental Factors and a Balanced Immune System: A Key to Cell-Mediated Immunity, Cancer Resistance and Allergy

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The Importance of Effective Immune Surveillance

The tragedy of AIDS has raised awareness of the key role of proper immune function in cancer avoidance/ tumor resistance. With HIV infection, the development of AIDS and reduced 'host surveillance capacity' (the ability of our immune systems to screen for anything inappropriate), there has been the dramatic increase in the incidence of an otherwise relatively rare tumor, Kaposi's sarcoma. Such an association underscores the existence of two fundamental processes, either of which can lead to an increased likelihood of cancer: 1) an increase in the rate at which cancer cells emerge within the individual or 2) a decrease in the capacity of the immune system to identify and eliminate cancer cells that occur in each of us. Clearly, changes on either end of this delicate immune surveillance-cancer balance could result in the emergence of cancer. Some chemicals exist that will operate through one or the other of these processes, but chemicals like the fungal toxin, aflatoxin, can increase cancer cell formation while simultaneously decreasing the capacity of the immune system to fight cancer. For this reason, it has become important to identify not only environmental toxins which might represent mutagen/carcinogens, but also chemicals which

are immunotoxic and could depress host resistance to cancer.

The Race Between Immune Response and Cancer Cell Proliferation

In some cases, individuals may succumb to cancer by the immune system failing to recognize the tumor as inappropriate (a genetic blind spot for the tumor) and never mobilizing against it. However, it appears that in a majority of instances, tumor resistance becomes a "race" between the capacity of the tumor cells to replicate and spread versus the rate at which tumor-fighting immune cells can be produced. Elegant experiments in animals have shown that by giving the immune system a two week head start in the "race" against cancer, tumor burdens large enough to kill 1,000 animals can be successfully destroyed in a single animal. Therefore, the first issue regarding effective immune surveillance is to ensure that environmental factors do not impair the capacity of the immune system to compete well in the proliferation race with tumor cells or affect recognition of a tumor.

The Changing Landscape of Immunotoxicology

Historically, the search for immunotoxins during the 1980s and early '90s focused on chemicals which caused wholesale destruction of the immune system. Fortunately, such chemicals are relatively rare and can usually be readily identified. However, more recent efforts have centered on environmental factors which may produce subtle, yet equally problematic changes to the immune system. These chemicals can cause functional immune imbalances which do not produce total immunosuppression, but rather cause shifts in host responses such that our defense against certain diseases are, nevertheless, compromised. Since major losses of immune cells or complete destruction of immune organs usually does not occur in these instances, the identification of such immunotoxins is relatively challenging.

The Basis of Immune Balance

The issue of immune balance was first recognized in 1986 when researchers like Dr. Tim Mosman and colleagues provided evidence explaining why the immune system is able to tailor responses to fit the particular disease challenge at hand (Mosman et al., 1986). For a long time immunologists marveled at the fact that most immune responses mobilize the particular sectors of the immune system best suited to fight the disease (i.e. certain types of antibodies are produced when needed and cancer fighting killer lymphocytes are preferentially generated when appropriate). But little was known about the possible controls over such directed responses. These researchers showed that specific types of T lymphocytes (called type 1 or type 2 helper cells) directed immune responses toward fighting cancer and viral infections or toward fighting certain bacterial and parasitic infectious challenges. In part, this explains why we mobilize precise types of responses which are needed to overcome specific disease challenges. Clearly, a healthy balance of type 1 and type 2 response capacity is necessary if we are to overcome the full spectrum of disease challenges over the course of a lifetime. The flip side of the issue is that an imbalance in type 1 versus type 2 T helper function could produce a major gap in disease resistance including resistance to cancer. Additionally, some forms of imbalance could increase the likelihood of immune-mediated diseases such as autoimmunity, allergy and asthma.

The basis of type 1 versus type 2 T helper function is linked to the production of immune hormones known as cytokines, lymphokines or interleukins. For example, type 1-associated responses are characterized by the production of interleukins-2 and -12 and interferon-gamma. These cytokines are important in the long-term defense against cancer mediated by cytotoxic T lymphocytes. Cytotoxic T lymphocytes are able to attack and lyse cancer cells with considerable specificity and precision. Therefore, they are clearly important in overall resistance to cancer. Additionally, interleukin-2 aids other immune cells such as Natural Killer cells in their fight against tumor cells while interferon-gamma can help to arm macrophages for the production of anti-tumor factors such as nitric oxide. Natural Killer cells and macrophages participate in our front-line defense against emerging cancers and can attack tumor cells within a matter of hours. Therefore, they aid in fighting the very earliest stages in tumor formation and complement the action of cytotoxic T lymphocytes which require days or even weeks to be produced. In contrast, type 2-associated responses involve the suppression of interferon-gamma production and an elevated production of interleukins -4, -5 and -10. In particular, interleukins -4 and -10 can promote IgE-mediated allergic responses (IgE stands for immunoglobulin E, an anitbody that causes histamine release from mast cells) as well as some forms of asthma. These types of responses do little to fight cancer and to the extent that they might represent the predominant response of an unbalanced immune system, the unsuited immune response could allow tumors to progress unabated. In reality, few responses are purely type 1 or type 2, but clearly, the balance of these functional capacities in an individual could influence the quality of the tumor fighting potential as well as the risk of allergic disease and/or asthma.

Chemicals Altering Immune Balance

With those fundamental immunology principles in mind, recent research in immunotoxicology has emphasized a search for environmental factors which might produce T helper (Th) lymphocyte functional imbalances. Among chemicals thought to reduce protection afforded by cell-mediated immunity (type 1) with an increased risk of autoimmunity and allergic disease (type 2) are the heavy metals. In particular, exposure to lead (Pb) and mercury (Hg) appear to represent significant health risks. In the case of mercury, it appears to cause a shift in the host response in favor of Th2 (T helper type 2) responses with concomitant increases in the incidence of certain autoimmune disease. (Prigent, et al., 1995).

A healthy balance of immune response capabilities is important for effective resistance to the full spectrum of diseases (viral, bacterial, parasitic, neoplastic). Exposure to the heavy metal, Pb, at inappropriate concentrations can cause a disruption in immune function. The shift in functional capacities (toward Th2 responses) is likely to reduce tumor immunity while concomitantly increasing the risk of allergic disease and asthma. In an integrated consideration of cancer risk, it is important to identify and reduce problematic exposure to environmental risk factors which would reduce immune resistance to cancer.

While lead has been a focus of childhood behavioral and learning problems, the effect of low-to-moderate levels of lead on the immune system is emerging as an equally serious concern. This topic was recently covered in a review by Dr. MaryJane Selgrade and colleagues (Selgrade et al., 1997). Lead has been shown, in rodents, to cause a shift in Th1/Th2 function. Dr. David Lawrence and other researchers at SUNY-Albany demonstrated that lead can alter the differentiation of T helper cells producing a reduction in those Th1 cells responsible for fighting viral infections and cancer (Heo et al., 1996, 1998). Our own laboratory at Cornell extended these observations to show that exposure to levels of lead which do not affect adult pregnant rats nevertheless cause persistent changes in the immune system of their developing daughters (Miller et al., 1998; Chen et al., 1999). This suggests, as is already suspected for lead-induced-neuro-behavioral toxicity, that embryos and immature offspring may be particularly susceptible to lead-induced immunotoxicity.

Other Potential T-helper-disrupting Chemicals

While Pb and the other heavy metals have been implicated in Th disfunction, it is likely that they are not the only environmental factors of concern when it comes to Th problems. Some of the factors currently under examination for T helper imbalance-producing immunotoxicity are UV radiation and various air pollutants (e.g. ozone, sulfur dioxide, diesel exhaust particles). However, in the case of inhaled gases such as ozone and diesel exhaust particles, it is not clear whether the potential enhancement of respiratory allergy results from a purely local effect in the airways or from more systemic changes. It is clear that exposure to ozone, diesel particles or the hydrocarbons from diesel exhaust is likely to result in greater histamine release and/or increased local inflammation in allergic subjects (Molifino et al., 1991; Muranaka et al., 1986; Takenaka et al., 1995). Additionally, some evidence suggests that exposure to diesel exhaust particles can produce enhanced Th2 activity beyond the area of local allergen exposure (e.g. extending to include at a minimum, upper body areas) (Fujimaki et al., 1994). Therefore, it is likely that the list of environmental factors known to produce Th imbalances will increase within the next few years.

Differential Risk

The fact that embryos are susceptible to immune changes at lead dosages which are safe for adults points to another issue in immunotoxicology and in toxicology in general. That is the fact that differential risk exists within the human population. In the case of Pb, there is a clear

age-related risk difference. Developmental windows of hyper-vulnerability are likely to exist for certain toxicant exposures. Additionally, the genetic background of an individual can influence whether or not a low-level toxicant exposure has problematic health implications. Therefore, a longer term goal of the immune-cancer risk research is to identify those segments of the population at greatest health risk from real-life exposures.

This extends beyond overt immunotoxicity studies to the topic of aging and cancer resistance. For example, historically it has been immunological dogma that Th1 function declines with aging in a reciprocal manner to the age-related increase in cancer incidence within the human population. However, from a preventative medicine perspective, it is probable that the lifetime course of chemical exposures (including dietary intake) can influence whether this age-related loss of protection against cancer actually occurs. Therefore, as more attention is paid to environmental influences on Th function, there are opportunities for improved resistance to cancer across the entire age spectrum.

Summary

Several types of environmental chemicals (beyond just the heavy metals) have the capacity to alter T helper cell balance. The disruption of appropriate Th balance is an environmental health change which appears to explain, at least in part, the recent rise in childhood asthma. Clearly, problematic environmental exposures which would increase the risk of asthma while reducing immune protection against cancer would be of significant public health concern. With the new directions undertaken in immunotoxicology, it is hoped that Th-disrupting chemicals can be identified, potentially problematic exposures avoided and the health risk to humans and wild-life, thereby, reduced.

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Perspective: The Role of the Immune System in Breast Cancer

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The Immune Response: A System for Fighting Infection

The immune system evolved to respond in an aggressive but controlled way to dangerous infectious organisms (pathogens). The essence of the response is that components of an innate defense system identify invading pathogens such as bacteria, viruses, fungi and parasites as foreign and establish an environment in which immune cells known as lymphocytes can respond specifically to distinct molecular components (called antigens) of the pathogen and thereby target the pathogen itself for destruction. Sometimes the innate defense system succeeds in halting the invasion before there is any real need for the lymphocytes to be involved, but usually exposure to a pathogen results in a lymphocyte response.

The Immune Response and Cancer

In contrast to pathogens, which are molecularly distinct from the host (the individual harboring the infection or tumor), cells which have become cancerous due to exposure to physical or chemical carcinogens share the great majority of their molecular components with all other cells in the host's body. This presents the first of several major problems faced by the immune system when faced with a cancer — the dangerous tissue is difficult to distinguish from normal tissue. Perhaps most importantly the cancerous cells are unlikely to activate the innate defenses which rely heavily on major differences between pathogen and host to recognize the invading organism. Without an appropriately activated innate system, it is difficult to recruit and activate the lymphocytes, which themselves do possess the ability to recognize the subtle differences between cancerous cells and normal cells. Thus, the immune system seems to play little role in preventing the growth of most cancers. This is clearly illustrated by the fact that common forms of cancer, including breast cancer, do not occur at higher frequency in people with AIDS or other immunodeficiency diseases. On the other hand, cancers associated with viral infections, such as Kaposi's sarcoma and cervical cancer, do occur more commonly in people with immunodeficiencies, reinforcing the role of the immune system in dealing with diseases with an infectious component. Paradoxically, prolonged immune system-promoted inflammation associated with certain chronic infections is also thought to play a role in promoting cancer. Nevertheless, the fact that the prevalence of breast cancer does not differ in immunodeficient versus immunologically responsive individuals can be taken as evidence that: 1) the immune system is not playing a major role in either protecting against or promoting breast cancer, and 2) that there is not an underlying role for an infectious pathogen in this common malignancy.

Vaccination Against Cancer

While the data from studies of breast cancer prevalence in immunodeficient individuals argue that the immune system does not play a major role in controlling this type of tumor, there is reason to believe that the full force of the immune system could be brought to bear on cancer through a vaccination strategy. In general, once lymphocytes have become involved in an immune response, the immune system is armed and ready to respond in a much quicker fashion should the host be exposed again to the same antigen. The immune response thus learns from its early experiences and remembers its past encounters. This fact underlies our ability to vaccinate against certain important infectious diseases. By deliberately exposing individuals to dead or crippled pathogens, or to important antigens from these pathogens, we arm the immune system to be able to deal appropriately with the pathogen if the individual should ever be exposed to it in the future. This approach could be used to prevent breast cancer. Ideally, antigens characteristic of and specific to breast cancers would be used to stimulate an immune response that could then recognize and kill any such cancer should it begin to develop. The challenge here of course is to identify antigens that define the cancer and then to produce them in such a form that they could be introduced into someone to induce a protective immune response. The approach is used in infectious disease research but has to date been successful only in a limited number of instances and it is true to say that despite great investment, many of our most successful vaccines continue to utilize whole dead or crippled pathogens rather than defined antigens. Such a "crude" vaccination approach has also been examined for cancer, in settings where surgically removed tumor cells are reintroduced as vaccine into the patient in a form that is highly immunogenic and more likely to activate

the innate defenses or directly activate lymphocytes. Most success using the vaccination approach for cancer has probably come in melanoma, where defined cancer related antigens and whole tumor preparations have been utilized with varying, but sometimes dramatic success.

A major difference between vaccination to prevent infectious disease and vaccination against cancer is that usually the former is used as a preventative whereas the latter presently is considered as part of a treatment regimen in people who already have disease. Although this vaccine therapy approach has been proven to work, there are several complicating factors not least of which is that as is the case in many cancers, breast cancer development is associated with a general immunosuppression that probably further limits the ability of the host's immune system to respond effectively to the tumor and possibly to vaccination. This immunosuppression is the result of the production by tumor cells of molecules that can directly inhibit or otherwise affect lymphocyte function. Unfortunately, radio and chemo-therapy, which target unregulated, fast-dividing cancer cells, also target the immune system, which is itself comprised of highly regulated rapidly dividing cells. Thus cancer itself, plus common treatments for cancer, can have a deleterious effect on the ability of the host to mount an immune response. Nevertheless, on a more positive note, surgical removal of tumors often is associated with a return of immune function and it would be envisaged that this would present the optimal opportunity for therapeutic vaccination.

Cancer Immunotherapy

In addition to vaccination, less specific immunotherapies against cancers have and continue to be used, sometimes with success. Examples include the administration of dead pathogens or pathogen components as immunostimulants which activate the innate defense system creating an environment in which some damage might be done to the tumor and in which tumor specific lymphocyte responses might be more likely to develop. In an attempt to make these treatments less ill-defined, most recent approaches utilize purified versions of the effector molecules generated by the innate defense system in response to exposure to pathogens.

Many Carcinogens are Immunosuppressants: What's the Connection?

Many carcinogens, at least as used experimentally, are immunosuppressive. Thus it seems reasonable to question whether suppression of the immune system is a major factor leading to the development of tumors in response to environmental exposure to carcinogens. The argument against this would again be that immunodeficient individuals seem no more prone than the rest of the population to developing tumors against environmental carcinogens.

"We Need to Know"

Ad Hoc Discussion Group

"Learning Together"

Mark Your Calendars!!!

The next Ad Hoc Discussion Group meeting
Wednesday, October 13, 1999

New York Hospital—Cornell Medical Center
Staff Annex 2 Building
21 Blooomingdale Road
White Plains, NY 10605

11:00am to 4:00pm.

Ad Hoc Discussion Group meetings are open to any and all stakeholders to come together to discuss issues related to breast cancer and environmental risk factors.

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

FACT SHEETS

Single copies available at no cost. For multiple copies please contact BCERF (address below).

General Information on Breast Cancer

__*FS # 3*—Understanding Breast Cancer Rates

__*FS # 5*—The Biology of Breast Cancer

__*FS # 6*—Tumor Supressor Genes - Guardians of Our Cells

__*FS # 9*—Estrogen - What is the Relationship?

__*FS #10*—Estrogen - What Factors Affect a Woman's Exposure to Estrogen?

Diet and Lifestyle

__*FS # 8*—Childhood Life Events

__*FS #13*—Alcohol

__*FS #18*—Fruits and Vegetables

__*FS #19*—Exercise

__*FS #27*—Dietary Fat

__*FS* #29—Breast Feeding

CRITICAL EVALUATIONS OF PESTICIDES AND BREAST CANCER

Critical Evaluations are available on the BCERF web page as portable document files (pdf), and can be accessed on the BCERF web site (see address below).

If you would like to order a hard copy please indicate below and send your check payable to Cornell University for **$3.00 each**, to cover the cost of reproduction and mailing.

__#1 2,4-D
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__#8 Atrazine
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Pesticides and Breast Cancer Risks

__*FS # 2*—DDT, DDE and the Risk of Breast Cancer

__*FS #11*—An Evaluation of Chlordane

__*FS #12*—An Evaluation of Heptachlor

__*FS #14*—An Evaluation of 2,4-D

__*FS #15*—An Evaluation of Lindane

__*FS #16*—An Evaluation of Simazine

__*FS #17*—An Evaluation of Cyanazine

__*FS #20*—An Evaluation of Dichlorvos

__*FS #23*—An Evaluation of Atrazine

__*FS #26*—An Evaluation of Chlorpyrifos

__*FS #28*—An Evaluation of Diazinon

Pesticide-Related Issues

__*FS # 4*—Reducing Pesticide Exposure in the Home and Garden: Alternatives and Proper and Legal Use Resource Sheet

__*FS #7A*—Reducing Potential Cancer Risks from Drinking Water--*Part I: Contaminant Sources and Drinking Water Standards*

__*FS #7B*—Reducing Potential Cancer Risks from Drinking Water--*Part II: Home Water Treatment Options*

__*FS #21*—Avoiding Exposure to Household Pesticides: Protective Clothing

__*FS #22*—Safe Use and Storage of Hazardous Household Products

__*FS #24*—Consumer Concerns About Pesticides in Food

__*FS #25*—Pesticide Residue Monitoring and Food Safety

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Sandra Steingraber, Ph.D.

Visiting Assistant Professor, Cornell University

BCERF is extremely pleased to welcome Sandra Steingraber, Ph.D., to Cornell; she will be in residence here from July 1999 through September 2000. Ecologist, poet, and cancer survivor, Sandra is the author of *Living Downstream: An Ecologist Looks at Cancer and the Environment,* as well as *Post-Diagnosis*, a volume of poetry, and co-author of work on ecology and human rights in Africa, *The Spoils of Famine*. She received her doctorate in biology from the University of Michigan, and master's degree in English from Illinois State University. She has taught biology at Columbia College, Chicago, and has held visiting fellowships at the University of Illinois, Radcliffe/Harvard University, and Northwestern University.

Sandra serves on President Clinton's National Action Plan on Cancer, administered by the US.Department of Health and Human Services. She has been the keynote speaker for conferences on human health and the environment throughout the United States and Canada, and has been invited to lecture at many university campuses, medical schools, and teaching hospitals. She is recognized for her ability to serve as a two-way translator between the cancer research community and the community of women cancer activists.

The highly acclaimed *Living Downstream* presents cancer as a human rights issue. It is the first book to bring together data on toxic releases, made available under right-to-know laws, with newly released data from US cancer registries. In 1997, Sandra was named one of Ms. Magazine's Women of the Year, and in 1998, she received from the Jenifer Altman Foundation the first annual Altman Award "for the inspiring and poetic use of science to elucidate the causes of cancer."

In her time at Cornell, Sandra will turn to the ecology of pregnancy and childbirth, including a detailed discussion of prenatal risk factors for breast cancer. She will look closely at the development of the mammary gland from fetal life through adolescence, pregnancy and menopause. She will also take up the issue of breast milk contamination, as it affects both the health of nursing infant and mothers' own risk for breast cancer. Sandra says that "just as *Living Downstream* was inspired by my own cancer diagnosis, my new book project is a direct result of the joyous birth of daughter, Faith, in September 1998. Motherhood casts a whole new light on the experience of possessing breasts!"

Research Commentary

Human Breast Milk Contamination

Detection of Monocyclic Aromatic Amines, Possible Mammary Carcinogens, in Human Milk

Lillian S. DeBruin, J.B. Pawliszyn, P.D. Josephy. Chemical Research in Toxicology Volume 12, 78-82, January 1999.

The worldwide contamination of human breast milk is a well-kept secret. This is not to say that the topic has not been thoroughly studied. A quick computer search will turn up citations for hundreds of research papers, reviews, and reports. (Type "breast milk contamination" into a medline search and see for yourself.) These publications consistently document the presence of suspected carcinogens—especially fat-seeking, chlorinated organics—in the milk of nursing mothers from Kenya to Kentucky and the Arabian peninsula to the Arctic Circle. Indeed, because breast milk occupies the highest rung on the food chain ladder, it is the most contaminated of

all human foods, with bioaccumulative toxics in mothers' milk reaching levels that far exceed their concentrations in animal-based foods (dairy, fish, eggs, and meat). And yet, in spite of this impressive accumulation of scientific knowledge, we hear almost no public discussion on the issue.

As a breastfeeding mother myself, I am dismayed by the silence. In my experience, childbirth educators, pediatricians, midwives, lactation consultants, and breastfeeding advocates such as La Leche League downplay the pollution of human milk in order to prevent women from choosing the bottle over the breast. But keeping secrets is never a good strategy for advancing public health. Mothers today confront a dismal Hobson's choice: Do we feed our babies the highly contaminated milk from our own bodies (which swarms with disease-fighting immune cells, brain-enhancing sugars, allergy-suppressing proteins, and bactericidal elements)? Or do we opt for a nutritionally inferior but lesser contaminated formula? The conventional wisdom—that the known benefits of breastfeeding in fighting infectious diseases outweigh the long-term, less understood risks of increased chemical exposure—is an untested supposition and a hardly reassuring one. The obvious third choice—that mothers should feed their babies human milk uncontaminated with carcinogens—is currently available to no woman on earth.

Happily, some brave breast cancer activists are beginning the much need public conversation that breastfeeding activists are apparently too fearful to initiate. The connection is a simple one: if toxic chemicals are ubiquitous in breast milk, then they are also present in the breasts of all women, lactating or otherwise, and may be contributing to cellular damage in the breast ducts. Such activists will want to take note of a new study published by researchers at the University of Guelph in Ontario, Canada. This study looks at a class of industrial chemicals that, heretofore, has not received close attention by breast cancer researchers: the aromatic amines.

Aromatic amines have many sources. They have long been used in the manufacture of dyes. They are also used to make plastic foams, pesticides, and pharmaceuticals. They are a byproduct of tobacco smoke. They are added to rubber during vulcanization, and they are used in color photography. They are called amines because the chemicals in this broad class are derived from ammonia. They are therefore distinguished by having both nitrogen and carbon in their molecules. "Aromatic" means that the chemical members of this class all possess a hexagonal ring of carbons (like benzene) rather than a single straight chain. Aromatic amines have already been identified as mammary carcinogens in laboratory rats. Human data is scarcer, but the authors point to some compelling occupational data. Women workers in a Russian dye-making factory, for example, showed excess rates of breast cancer.

In carrying out their study, the authors had to first establish an analytical method for measuring the presence of monocyclic aromatic amines in biological fluids. Indeed, they are the first team of researchers to detect such contaminants in human milk. As the authors themselves note, the implications of such a finding are gravely important. While environmental organochlorine pollutants (pesticides, dioxins, and PCBs) have been identified in human milk for decades, few organochlorine chemicals can, all by themselves, cause breast cancer in animals. Aromatic amines such as o-toluidine, on the other hand, are known to cause ductal carcinomas in rats. Their detection in human milk means that the ductal epithelial cells of the human breast are routinely being exposed to a class of chemicals for which the data on carcinogenicity is overwhelmingly clear.

The experimental methods used in the study were impressive. Detections of aromatic amines could be made by using solid-phase microextraction coupled with gas chromatography/mass spectrometry. The human milk in this study was collected from 31 lactating mothers without occupational exposure to aromatic amines. Some had been breastfeeding for only a few days; other for more than two years. Seven were smokers and 24 were non-smokers.

The results were unequivocal. Monocyclic aromatic amines were detected in milk samples from all mothers, both smokers and non-smokers. These included aniline, o-toluidine, and N-methylaniline. All mothers had aniline in their milk at levels ranging from 0.05 parts per billion to 5.2 parts per billion. (These concentrations are comparable to the average levels of some of the most common organochlorine breast milk contaminants.) The milk of eleven mothers contained both o-toluidine and N-methylaniline. Interestingly, levels of contaminants did not correlate with length of lactation, the fat or protein level of the milk, or with each other. There was no significant difference between smokers and non-smokers. Also, there were no differences in contamination between the milk of first-time mothers and those with more than one child. The authors did not, however, test for an association between mother's age and contaminant levels

The authors end their report by asking some important questions for further study. Unlike organochlorines, aromatic amines have short half-lives and are readily excreted in urine (which is why they are also strongly linked to bladder cancer). Are the levels of these chemicals therefore the result of recent rather than long-ago exposures? (The lack of difference between contaminant levels in uniparous and multiparous mothers suggests such a conclusion and stands in stark contrast to the data on organochlorine levels in breast milk where levels fall dramatically as length of lactation and number of breast-fed children increase). If these exposures are indeed recent and ongoing, what is their ultimate source? Diet? Air? Water?

Breast cancer activists will find in this study renewed reasons to focus on environmental causes of the disease. Nursing mothers will find more reasons for heartache in the obvious question not addressed by this study: what is the effect of aniline-laced breast milk on my child? This is certainly the question in my mind as I watch the mouth of my nine-month-old daughter tug rhythmically and blissfully at my breast, her own rosebud nipples rising and falling with each swallow.

Prepared by Sandra Steingraber, Ph.D., Visiting Assistant Professor, Cornell University Center for the Environment

WHAT'S NEW "ON THE WEB"

http://www.cfe.cornell.edu/bcerf/

Requests for information from the web site has remained near an average of 3,000 requests per month.

The top five requested fact sheets were:

Estrogen and Breast Cancer Risk
Exercise and Breast Cancer Risk
Fruits and Vegetables and the Risk of Breast Cancer
Phytoestrogens and Breast Cancer Risk
Childhood Life Events and Breast Cancer Risk

Topics that received the most requests for pages include the subjects of pesticide and diet.

Marie Stewart, BCERF "Webmaster"

The Ribbon is published by the Cornell Program on Breast Cancer and Environmental Risk Factors in New York State. Comments are welcome; contact the Editor

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