The Ribbon

BCERF

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

Special Issue on Early Exposures and Breast Cancer Risk

In this issue of *The Ribbon* we bring readers three articles addressing various aspects of current research on early exposures to environmental chemicals and breast cancer risk. The first two authors, Dr. Suzanne Fenton of the U.S. Environmental Protection Agency and Dr. Suzanne Snedeker of BCERF, highlight recent laboratory animal and human epidemiological work documenting biological effects of these exposures, as well as the presence of these compounds in the bodies of the study populations. Reeve Chace, part of the Mount Sinai School of Medicine Breast Cancer and Environment Research Center demonstrates a complementary aspect of the research being done at her Center: providing the girls involved in their early exposure studies with opportunities to take action to reduce risk *now*.

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Early Life Exposures to Environmental Compounds: Lessons Learned from Animal Models

By Suzanne E. Fenton, Ph.D., Reproductive Toxicology Division, U.S. Environmental Protection Agency

For nearly 20 years, the search has been on for environmental factors, specifically industrial chemicals, associated with the rising numbers of diagnoses of breast cancer in American women. There have been many attempts to isolate a specific class of compounds in our environment or even in the homes of women diagnosed with breast cancer that would help understand the exposures that either cause or make certain women more susceptible to the disease. Very little has been determined from these costly studies. Yet, judging from many studies in rodent models of human disease the reason may be related to the amount and timing of environmental exposures in comparison to the time of disease. In other words, the effect(s) of an environmental exposure may have begun during childhood or around the time of puberty, yet breast cancer typically manifests itself decades later.

The paradox of "hormesis"

Environmental chemicals that disrupt the normal signals that take place between a hormone and its receptor, or alter the amount of time or the concentration at which a hormone is active in the body, are commonly called endocrine disrupting compounds or EDCs. In recent years, we have learned a fair amount about the effects EDCs can have on the development of many male and female reproductive tissues. Most of the studies have employed high, non-environmental exposures of EDCs to demonstrate these effects. A series of rodent studies with bisphenol A, commonly used as an epoxy resin and in polycarbonate plastics and PVC pipes, demonstrated that this mildly estrogenic compound had effects in multiple reproductive tissues at low doses that were not repeatable at high levels of the compound. We now know that EDCs may have variable and sometimes dissimilar effects on reproductive tissues at high vs. low (more environmentally relevant) exposures. This paradox is called "hormesis." Hormesis, as defined by Wikipedia (http://en.wikipedia.org/wiki/Hormesis), is "the term for generally-favorable biological responses to low exposures to toxins and other stressors. A pollutant or toxin showing hormesis thus has the opposite effect in small doses than in large doses." The mammary gland is one reproductive tissue in which hormesis has been proposed. Therefore, many of the toxicology studies that are performed in rodent models of human disease must perform a dose response of the EDC in question to categorize the high and low dose effects and at which exposure level(s) they are adverse (significantly affect development, growth, or survival of the next generation).

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Table 1.

Environmental compounds proven to alter mammary gland (MG) development following early life exposure.

Compound	Rodent Model Tested	Accelerated MG Development	Delayed MG Development	Reported Putative Long-term Consequences	Reference(s)
Atrazine	Rat		X	MG Tumors Lactation Transgenerational Effects	Rayner et al., 2004. *Toxicol Appl Pharmacol* Fukamachi et al., 2004. *Cancer Sci* Rayner et al., 2005. *Toxicol Sci* Ueda et al., 2005. *Cancer Sci* Enoch et al., 2007. *Environ Health Perspect*
Bisphenol A	Mouse & Rat	X	X	Neoplasia Leukemia MG Tumors Development effects limited to early exposure	Colerangle & Roy, 1997. *J Steroid Biochem & Mol Biol* Markey et al., 2001. *Biol Reprod* Huff, 2001. *Odontology* Nikaido et al., 2004. *Reprod Toxicol* Munoz de-Toro et al., 2005. *Endocrinology Yin et al., 2006. Ind J Exp Biol* Murray et al., 2006 *Reprod Toxicol* Vandenberg et al., 2007. *Endocrinology*
Cadmium	Rat	X		Lactation	Johnson et al., 2003. *Nature Med* Ohrvik et al., 2006. *Toxicol Lett*
Dieldrin	Rat		X	Lactation	Tarrafet et al., 2003. *Food Addit Contam*
Dioxin or TCDD	Rat		X	MG Tumors Prenatal exposure needed for effects Lactation	Fenton et al., 2002. *Toxicol Sci* Brown et al., 1998. *Carcinogenesis* Lewis et al., 2001. *Toxicol Sci* Vorderstrasse et al., 2004. *Toxicol Sci* Desaulniers et al., 2001. *Environ Health Perspect*
Dietylstilbesterol (DES)	Rat & Mouse	X		Spontaneous MG tumors	Too many to list – date back to 1950's
Genestein, Zearalenone, Resveritrol	Rat	X	X	MG Tumors? Effects depend on time and route of exposure	Hilakivi-Clarke et al., 1999. *Oncol Rep* Lemartiniere, 2000. *Am J Clin Nutr* Delclos et al. 2001. *Reprod Toxicol* Padilla-Banks et al., 2006 *Endocrinology*

Table 1. continued

Compound	Rodent Model Tested	Accelerated MG Development	Delayed MG Development	Reported Putative Long-term Consequences	Reference(s)
Nonylphenol	Rat	X		Unknown	Colerangle & Roy, 1996. *Endocrine* Fukamachi et al., 2004. *Cancer Sci* Moon et al., 2007. *J Reprod Devel*
Organochlorine mixtures	Rat		X	Increased MG tumor incidence if carcinogen early in development.	Desaulniers et al., 2004. J Toxicol Environ Health A Desaulniers et al., 2001. Environ Health Perspect
Polychlorinated Biphenyls (PCBs)	Rat	X	X	Dose dependent development and tumor outcomes	Muto et al., 2002. *Toxicology*
PhIP (2-amino-1-methyl-6-phenylimidazol [4,5-b]pyridine)	Rat		X	MG tumors Transgenerational Effects	Ito et al., 1997. *Mutat Res* Snyderwine et al., 1998. *Carcinogenesis* Snyderwine et al., 2002. *Mutat Res*
PFOA	Mouse		X	Lactation	White et al., 2007. *Toxicol Sci*
High Fat diet (PUFA)	Rat & Mouse	X		Increased MG tumor incidence	Walker, 1990. *J Natl Cancer Inst* Hilakivi-Clarke et al, 1997. *Proc Natl Acad Sci USA* Hilakivi-Clarke et al, 1997. *J Natl Cancer Inst* Hilakivi-Clarke et al, 1999. *Nutrition* Uijten et al., 2006. *Reprod Toxicol*
NOTE *– Only positive effects are reported; this does not mean there are no reports in contrast to these.*					

EDCs and precocious development of the "terminal end buds"

There are several other EDCs that have been shown to affect mammary gland growth at either high or low exposure levels. Table 1 summarizes those EDCs that have demonstrated early life effects on the development of the mammary gland (exposures took place before or around the time of birth or around the time of puberty), as well as those shown to have longer lasting effects (into adult life). The effects of these compounds are divided into two groups – either precocious development, that which is faster than normal, or those causing delayed development. The importance of these variant effects of EDCs lies in the amount of time that terminal end buds (TEB) are present in the gland. The TEB are tear-drop shaped structures that are heavily laden with dividing epithelial cells. TEBs are the branching and dividing points in the gland; much like the bulldozer that goes before the paver in making a new highway, these cells plow the way for a new network of epithelial ducts in the developing mammary gland. Much work in rodents, primarily led by the laboratories of Drs. Michael Gould (University of WI-Madison) and Jose and Irma Russo (Fox Chase Comprehensive Cancer Center, Philadelphia, PA), has proven that the TEB are susceptible to chemical carcinogens and the number of TEB exposed to the carcinogen is related to tumor risk (both in terms of numbers of tumors and the timing of tumor development). Precocious development of the TEB has been theorized to

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play a protective role in terms of mammary carcinogenesis. The sooner the TEB develop into adult structures, the sooner they are less susceptible to the effects of other environmental agents. However, in the case of delayed development, the TEB are present in the gland for longer periods of time, leading to increased susceptibility to the effects of environmental carcinogens.

Delayed mammary development also observed

Environmental chemicals that have demonstrated endocrine-related effects in animal models and also alter mammary gland development (precocious or delayed) are shown in Table 1. As described above, some of these compounds have divergent effects depending on the doses used and that is shown for those chemicals having both accelerated or delayed X's. Atrazine, a high use herbicide, is one compound that has been shown to have consistent effects at both high and low doses. Recent work soon to be published in *Environmental Health Perspectives* (http://www.ehponline.org/docs/2006/9612/abstract.html), demonstrates that an exposure to a mixture of atrazine and its biological metabolites for only the last week of pregnancy can have long-lasting effects on the development of the mammary gland. What makes the effect of this mixture interesting is the finding that relatively low doses (only 100 times higher than the levels reported in some ground and surface waters) are able to evoke a delayed development. Other chlorotriazine herbicides break down to the same metabolites as atrazine and therefore this class of herbicides can be generally thought to have similar effects on the mammary gland. Another environmental compound found to dramatically delay mammary development is perfluorooctanioic acid (PFOA), sometimes found in consumer products that are non-stick, grease or water resistant, or weather proof, in addition to its uses as an emulsifier and insulator. A recent report in *Toxicological Sciences* demonstrated that exposure to the compound during pregnancy resulted in halted mammary development in female mouse offspring and delayed lactation (White, et al. [2006]. Gestational PFOA Exposure of Mice is Associated with Altered Mammary Gland Development in Dams and Female Offspring. Toxicological Sciences *96*, 133-144). Although the exposure level used in the study may seem small (5 mg/kg body weight), it was a great deal higher than the exposure levels in the U.S. population, even those in contaminated areas of Ohio and West Virginia.

Many remaining questions require study

Even though the number of EDCs known to alter mammary gland development is growing, the long-term consequences of the exposures early in life have not been as easy or quick to determine. Only a small handful of EDCs have been investigated for their early life effects and chemically-induced or spontaneous tumor formation. Further, only a small number of EDCs known to affect mammary gland development in the offspring have been investigated for the effects of those underdeveloped glands on future generations. The compounds for which adverse long-term outcomes have been described are listed in Table 1. Also, those chemicals for which effects are mediated via prenatal or neonatal exposure and are not repeatable if exposure occurs later in life are noted.

The amount of information that has been gained over the last ten years on the effects of EDCs on the mammary gland is impressive. However, very little can be concluded about exactly which classes of compounds epidemiologists could prioritize in search of candidates for breast cancer risk. There are several 2006 and 2007 references listed in Table 1 that bring even more information to the table about low dose effects and new compounds that may influence mammary gland development.

One problem hampering this field of research is the difficulty in getting funding for studies on the mechanisms used by environmental toxicants or food components in altering mammary gland and potential tumor development. The National Institute of Environmental Health Sciences (NIEHS) has recently posted a request for research proposals that would further address the role of developmental exposure on adverse mammary gland outcomes. The work funded by these grants and those funded in the Breast Cancer and the Environment Research Centers will help to expand our knowledge of environmental influences on breast cancer risk.

New NIEHS Grant Program Information

Department of Health and Human Services, National Institutes of Health

In Utero Exposure to Bioactive Food Components and Mammary Cancer Risk (R01)

http://grants.nih.gov/grants/guide/pa-files/PA-07-178.html

The information in this article has been subjected to review by the National Health and Environmental Effects Research Laboratory of the U.S. Environmental Protection Agency. Approval does not signify that the contents reflect the views of the Agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

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This article can be found on our website at:
http://envirocancer.cornell.edu/newsletter/article/v12animal.cfm

Environmental Estrogens: Affects on Puberty and Cancer Risk

By Suzanne Snedeker, Ph.D., BCERF Associate Director for Translational Research

The breast is unique because most of its development occurs after birth, during the pre-pubertal years through puberty. Researchers are interested in how early life exposures to chemicals, especially those that may affect breast development, may ultimately affect the cancer process. The papers discussed below illustrate how a multi-disciplinary approach is needed to fully understand to what extent young girls are exposed to chemicals of concern, how animal models can be used to tease out whether such exposures influence how the mammary gland develops, and if early exposures to certain environmental chemicals can lead to changes that ultimately affect breast cancer risk later in life.

Studying chemical exposures in children

Although we have known for over seventy years that the environmental chemical bisphenol A (BPA) is an environmental estrogen (Dodds and Lawson, 1938), until recently we have known very little about levels in people. Since this chemical is processed by the body relatively quickly and excreted, levels in the urine are used as biological marker (biomarker) of recent exposure. Less than three dozen papers are available on urinary levels of BPA in humans (most published since 2000), and only a few of these studies document urinary levels of young children during the period of time when breast tissue is starting to develop.

One of these studies was recently published in the January 2007 issue of *Environmental Health Perspectives*, entitled "Pilot study of urinary biomarkers of phytoestrogens, phthalates, and phenols in girls," by a consortium of three Centers devoted to determining if early environmental exposures affect the development of puberty in girls (Wolff et al., 2007). These NIH-funded centers, called the Breast Cancer and the Environment Research Centers (BCERC), will also determine if early exposure to environmental stressors can affect subsequent breast tumor development in laboratory animal models (see **http://envirocancer.cornell.edu/Research/BCERC/** for an overview of these Centers).

BCERC pilot study focus

This pilot study analyzed urinary samples from 90 girls (average age, seven years old) of different ethnic backgrounds from New York City, Cincinnati, and the San Francisco Bay area (30 samples from each location) for a variety of environmental chemicals. The chemicals of interest included both synthetic chemicals and naturally occurring chemicals (phytoestrogens). This study's most basic question was this: of the target environmental compounds these researchers would like to follow in young girls because they may affect pubertal development, which ones can be detected with current methods, and how do levels detected compare to any known values for girls of similar ages? Secondly, would these preliminary results show any variation in levels of the chemicals by geographic location, ethnicity, or one marker of puberty, weight for height (as measured by body mass index)?

Three classes of chemicals were monitored:

- six phytoestrogens (plant estrogens);
- ten phthalates and metabolites of phthalates (found in plastics, clear food wraps, personal care products, and cosmetics);
- nine phenols including BPA (found in polycarbonate sports and baby bottles, epoxy resins used in food can linings, dental sealants, and plastics used in CDs/DVDs, goggles/headlamps), and other phenolic chemicals found in sunscreens, pesticides, anti-bacterials, and detergents.

All of these 25 chemicals have been identified as having some capacity to disrupt hormones, especially estrogens or androgens.

To do this study, researchers collaborated with the Centers for Disease Control and Prevention (CDC), which has laboratory facilities with the capability of analyzing blood and urine samples for biomarkers of chemical exposures. The CDC launched a national biomonitoring program to track levels of environmental chemicals in the U.S. population in 2001. The CDC biomonitoring program is one of the few sources of information on current levels of environmental chemicals in the general U.S. population.

BCERC pilot study results

Researchers conducting this puberty study found that 18 out of the 25 chemicals studied were detectable in the 95% of the urine samples. The chemical with the highest median values (the median is where half the values are above and half below) in each class of chemicals was enterolactone for phytoestrogens, monoethylphthalate (MEP) for the phthalates, and a benzophenone-3, a component of sunscreens, for the phenols. This is one of

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the few studies that have reported levels of BPA in young girls; the levels for BPA in this puberty study were similar to values reported by the CDC in their 2005 biomonitoring study. Both the phytoestrogen enterolactone, and the environmental estrogen BPA tended to be higher in girls that were not obese (less than the 85th percentile for body mass index); no explanation was given for these trends.

While there was some variation in levels of several phthalates with ethnicity and geographic location, it is difficult to interpret the significance of these findings because questionnaire data from this study have not been fully analyzed yet. Questionnaire data may provide information on possible sources of exposure, including the girls' diet and use of personal care products.

Animal studies of BPA observe pre-cancerous changes

In the same issue of *Environmental Health Perspectives*, a study from the laboratory of Drs. Ana Soto and Carlos Sonnenschein evaluated whether pubertal exposures to BPA in female rats affected the development of the mammary gland and looked to see if any pre-cancerous changes occurred as the BPA-treated animals got older (Durando et al., 2007) . The most striking result from this study was that early, pubertal exposures to BPA (for rats, exposure started at 50 days of age) resulted in a higher percentage of the BPA-exposed animals having pre-cancerous lesions in the mammary gland. These lesions, called hyperplasias, were not observed until the BPA-treated animals were 180 days of age (young adults rats). The size of the treatment groups in this study were small, and the animals were only followed for six months, so further research is needed to determine if these pre-cancerous lesions persist as the animals age. While there is some indication in these studies that BPA may make the animals more sensitive to other chemicals known to be mammary carcinogens, the animals were not followed for a sufficient length of time to make strong conclusions. But, since cancer is a process, the finding of a higher number of mammary hyperplasias with pubertal BPA exposures would support the need to determine how early exposures to environmental chemicals may "imprint" the mammary gland and affect the cancer process.

In support of this thesis are studies from the laboratories of Drs. Gail Prins and Shuk-Mei Ho (Ho et al., 2006) which show that early exposures to BPA during prostate gland development induces prostate hyperplasias in male rats. These changes appear to be tied to a gene-induced change in DNA-methylation patterns, which may be an important factor in the development of prostate tumors as these BPA-treated animals age. This evidence of an imprinting effect with early life exposures to BPA during mammary and prostate development indicates that both glands have critical windows of susceptibility, and that early exposure to environmental estrogens may affect the cancer process.

Determining the sources of exposure

Other studies are needed to more fully characterize routes of exposure to chemicals like BPA during critical times of mammary and prostate gland development. One highly quoted study was conducted by food regulatory authorities in Singapore (Onn et al., 2005). BPA was detected in 19 out of 28 brands of polycarbonate baby bottles tested. Calculated daily intakes of BPA for infants from the baby bottles were below the maximum limit recommended by the Environmental Protection Agency (EPA) of 0.05 milligrams/kilogram body weight per day. The European Union's (EU) tolerable daily intake for BPA is 0.01 mg/kg/day, and while one Norwegian study found that BPA leached out of polycarbonate baby bottles after dish-washing, boiling and brushing, the levels did not exceed the EU limits (Brede et al., 2003).

In one the few studies on children in daycare centers, preschoolers' exposure to BPA appeared to be primarily through food (Wilson et al., 2003). A more recent study on exposures of preschool children to BPA at home and in daycare facilities reported similar results. BPA was detected in 68% of the children's liquid food and in 83% of the solid food samples, and in virtually all hand swipe samples taken from the children. Overall, 99% of the BPA exposure was through the dietary intake route (Wilson et al., 2007). Research from other laboratories suggest that most of the transfer of BPA from epoxy resins (used to line food cans) to the food occurs during the high-heat canning process (Goodson et al., 2004; Kang et al., 2006; Sajiki et al., 2007).

To what extent BPA exposure is from consuming canned food versus other sources in older children is not known. Few published studies have evaluated leaching from polycarbonate sports bottles or from landfills that contain degrading polycarbonate plastics that may leach BPA. BPA has also been found in paperboard food containers, and paper towels made from recycled products (Ozaki et al., 2004; Vinggaard et al., 2000). Carbonless paper, photosensitive fax paper, and various photographic inks may be sources for BPA that makes its way from recycled office paper products to recycled paper board used for food containers.

With regard to exposures that may occur during the puberty years, Duty and colleagues (Duty et al., 2005) reported that the use of personal care products predicts urinary concentrations of some phthalate monoesters (many types of phthalates are metabolized and found as monoesters in the urine). While this study was done in men, the study results did support a relationship between use of personal care products (cologne or aftershave) and urinary levels of monoethyl phthalate. Similar studies

characterizing personal care product use (fragrances, hair and body care, nail polish) and exposure to phthalate monoesters in girls are needed.

Further multi-disciplinary research needed

When the full results of the early life exposure-pubertal study from the three Centers become available, it may be one of the first studies to not only characterize pubertal urinary levels of environmental agents and probable sources, but to also give us important data on whether these contaminants, dietary factors, or body size are influencing the onset of breast development and other hallmarks of puberty in young girls.

Studies confirming the Soto lab's findings of a higher incidence of pre-cancerous hyperplastic lesions in the mammary gland after low level pubertal exposures to BPA need to be confirmed by other laboratories. Other studies are needed to determine if this effect is due to an inherent characteristic of BPA, or if equivalent doses of other environmental estrogens also are able to imprint on the developing mammary gland and cause pre-cancerous lesions. Ultimately, studies will need to go beyond showing visual changes (lesions), and need to document whether early exposures to BPA or other environmental chemicals affects genes related to the cancer process.

Real-life exposures to environmental agents are not to limited to one chemical, but are more likely to result from low-level exposures to several or even dozens of environmental estrogens. While exposure studies have documented levels below current EPA and EU limits for single chemicals like BPA, a "risk cup" assessment of how additive effects to multiple environmental estrogens affects puberty, including the onset of breast development, is important. Knowing how environmental stressors affect early breast development needs to be studied at many levels. This includes determining actual levels in urine and current sources of exposure in different ethnic populations, to deciphering the biological basis of any changes that affect puberty or cancer risk at the structural and molecular level.

This article can be found on our website at:
http://envirocancer.cornell.edu/newsletter/article/vl2estrogens.cfm

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News from the Web

1 Fact Sheets

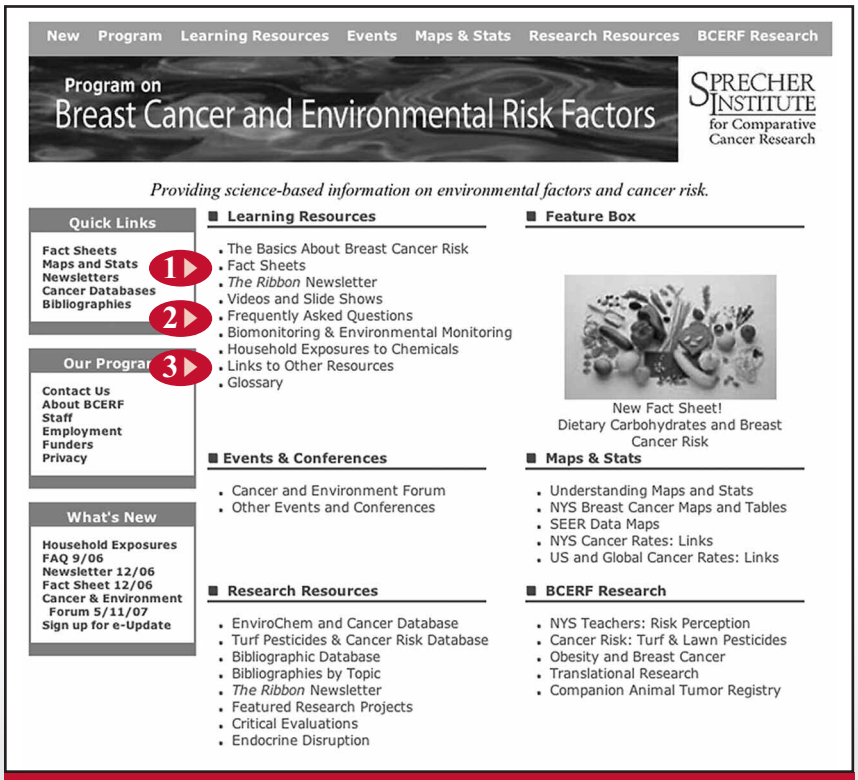
Do Dietary Carbohydrates Affect Breast Cancer Risk? BCERF's newest fact sheet (#55) examines carbohydrates and how their metabolism may affect breast cancer risk. The fact sheet is available on the web at http://envirocancer.cornell.edu/factsheet/diet/fs55.carbohydrates.cfm

2 FAQ

The Frequently Asked Questions (FAQ) section includes new questions about sunscreen and produce from markets outside the United States. On the web at http://envirocancer.cornell.edu/learning/faq/qa.cfm

3 Household Exposure to Chemicals

Chemical exposure in the home is an expanding area of cancer risk research. The new Household Exposures section on our website explains some basic information about this topic, outlines current and future research directions, and links to more information. Learn more at http://envirocancer.cornell.edu/learning/household/exposures.cfm



http://envirocancer.cornell.edu is BCERF's home on the web.

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Friday, May 11, 2007 • 10:00am - 3:00pm • Hilton Garden Inn, Ithaca, NY

BCERF

Spring Regional Cancer and Environment Forum

Speakers will include:

Dr. Cheryl Siegel Scott, U.S. Environmental Protection Agency
Trichloroethylene Cancer Epidemiology: A Consideration of Select Issues

Dr. Julia G. Brody, Silent Spring Institute
Environmental Pollutants and Breast Cancer on Cape Cod and Beyond

For more information or to RSVP, please contact Carmi Orenstein • (607)255-1185 or cso1@cornell.edu

Growing Up Healthy in East Harlem and the Bronx, New York

By Reeve Chace, M.P.H., Research Coordinator
The Community Outreach and Translation Core
Mount Sinai School of Medicine Breast Cancer and the Environment Research Center

The Hip Hop Dance Party. The Central Park Scavenger Hunt. Cells, Genes, and Protein Machines.

These may not sounds like the typical components of a research study, but they are integral to the success of the *Growing Up Healthy* study at the Mount Sinai School of Medicine's Center for Children's Environmental Health and Disease Prevention Research in New York City.

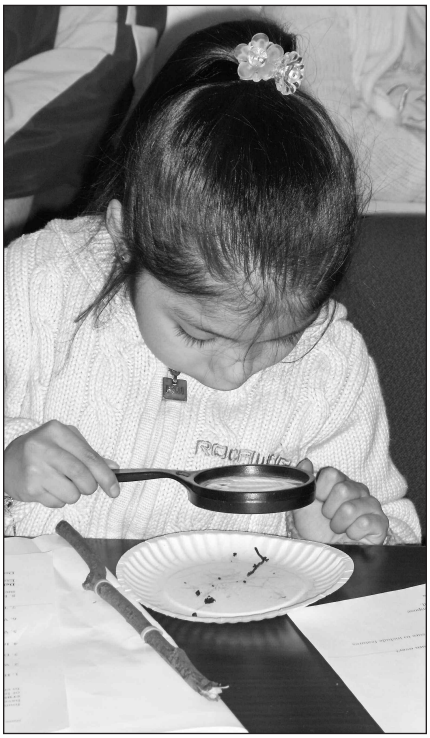
Children who enroll in *Growing Up Healthy*, a longitudinal study of environmental exposures during puberty, are invited to these activities on a regular basis in an effort to keep them involved with the study while educating them about the environmental health issues the study is intended to address.

The activities are planned and created by the Community Outreach and Translation Core (COTC), directed by Dr. Luz Claudio and managed by Reeve Chace, of the Mount Sinai School of Medicine. In addition to invitations to events, the COTC mails fact sheets, health bulletins, study updates, and special materials to participants' homes to keep study members involved in the research. *The Growing Up Healthy* study (GUH) is one of three epidemiological studies being conducted by the multi-site Breast Cancer and the Environment Research Centers, jointly funded by NCI and the NIEHS.

Community-based Collaborations

The goal of the COTC is two-fold. First, the aim is to engage children and their parents in the activities of the COTC for the duration of the five-year study. Participants only need to see study staff once per year, so the COTC aims to bridge the distance between study visits and maintain interest in the study by offering participants unique opportunities for fun and education. Second, the COTC aims to instill in study participants the sense that they are our partners in the scientific process.

Most participants in the *Growing Up Healthy* Study live in low-income, minority communities in East Harlem and the Bronx, NY. A key to engaging this population and building their trust has been our partnership with community-based organizations that already provide services to residents of these communities. For example, the COTC has partnered with the City Parks Foundation, an organization dedicated to increasing environmental awareness among New York City children, to bring its hands-on science programs to *Growing Up Healthy* enrollees. During the February school vacation of last year, the City Parks educators came to the Mount Sinai COTC laboratory bearing bugs and magnifying glasses for a series of workshops entitled *Make Your Own Nature Journal*. The educators encouraged the *Growing Up Healthy* kids to take a closer look at the creatures they can find in the park right outside their doors, and helped them increase their powers of observation by asking kids to record their observations in a home-made Nature Journal.



Another successful partnership was formed with Amy Jordan, who founded the organization Sweet Enuff to raise awareness of diabetes through dance. Jordan, who herself suffers from Type I diabetes, helped the COTC organize a highly successful Hip Hop Dance Party, held on Martin Luther King Day at the Julia de Burgos Cultural Center in East Harlem, a well-known community center. Jordan heads up a volunteer troupe of teenage dancers she calls Youth Ambassadors, who began the program by performing a

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dance routine. They then engaged the kids in an interactive discussion about healthy versus unhealthy foods. Finally, the Youth Ambassadors taught the kids a series of new dance routines, reminding everyone that exercise can be fun.

Filling a Need

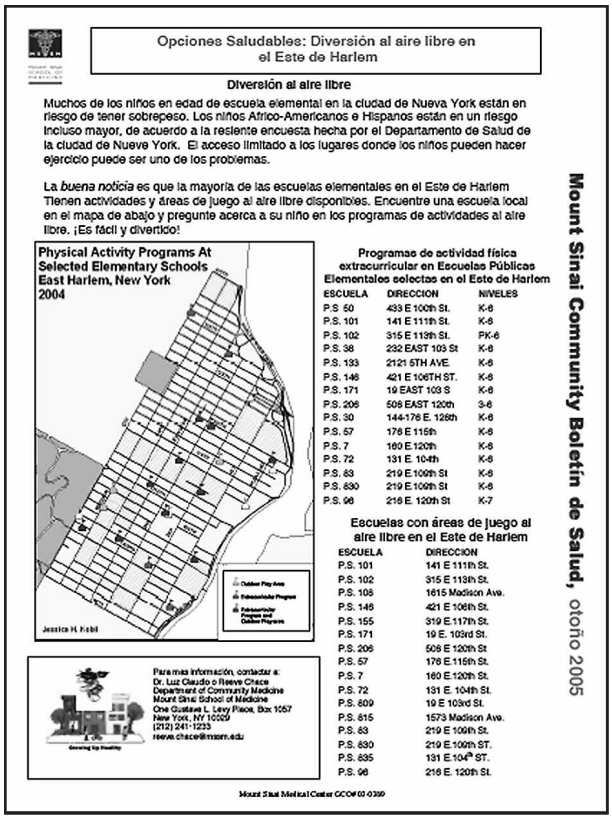
When not partnering with community organizations on health- or science-promoting activities for children, the COTC reaches out to parents and guardians of participants in ways that are intended to fill the needs of parents with young children, especially those who are low-income. For example, during the first week of September, the COTC purchased backpacks through the non-profit organization Kits for Kidz, filled them with basic school supplies such as notebooks, crayons, glue sticks, and pencils, and distributed them to over 100 study participants for our 2006 Back-to-School Backpack Giveaway. Parents were impressed with the quality of the backpacks, and appreciated the time and money saved by not having to do as much back-to-school shopping with their children. The backpack giveaway was a boon to parents, and it had the added bonus of aiding the COTC in its retention efforts, as research staff were able to follow up with participants when they came into the office to pick up their backpacks.

Similarly, just before the December holidays, the COTC held the *Growing Up Healthy* Photo Studio, a week-long event in which we transformed our lab into a "professional" photo studio. We purchased an inexpensive backdrop featuring a snow scene and snowman, took digital portraits of children posed alone or with siblings, parents, or grandparents (or whomever came along), and printed out the photos on the spot with a digital photo printer. Children decorated inexpensive cardboard frames with craft supplies, and then had portraits of themselves in a handmade frame to keep or give as gifts for the holidays. Many parents requested several copies of the photos, and one mother told us that she'd never before had a photograph of her two daughters together. Again, while generating goodwill and providing children and their relatives with a fun holiday activity, the event also gave research staff another opportunity to connect with participants between study visits.

Materials Development

To supplement the activities and events on offer, the COTC also develops and disseminates environmental health materials. Some materials are specifically tailored to study participants, such as the *Kids in Action* newsletter, which is mailed out to the study cohort twice a year. The newsletter updates participants on important *Growing Up Healthy* study news, past COTC events, community resources, and other health information. Other materials, like the COTC Health Bulletins, provide environmental health information that is applicable to both study participants and the community in general. In the summer, we created fact sheets that listed the locations of all the farmers' markets in East Harlem and the Bronx, where most of our study participants live (see fact sheet below). With the help of medical students experienced in GIS mapping, we also created two different maps of East Harlem that showed the locations of public playgrounds and food stores with healthy options available (see map in Spanish, opposite page). Another map, entitled "East Harlem School Zones Are A Junk Food Target" graphically depicted just how close fast food stores are to local public schools.

One of our most popular items has been the credit card-sized, bilingual *Pocket Guide to Plastics*. Initially, we had created a fact sheet called the "Quick Guide to Safe



Opciones Saludables: Diversión al aire libre en el Este de Harlem

Diversión al aire libre

Muchos de los niños en edad de escuela elemental en la ciudad de Nueva York están en riesgo de tener sobrepeso. Los niños Africo-Americanos e Hispanos están en un riesgo incluso mayor, de acuerdo a la reciente encuesta hecha por el Departamento de Salud de la ciudad de Nueva York. El acceso limitado a los lugares donde los niños pueden hacer ejercicio puede ser uno de los problemas.

La *buena noticia* es que la mayoría de las escuelas elementales en el Este de Harlem Tienen actividades y áreas de juego al aire libre disponibles. Encuentre una escuela local en el mapa de abajo y pregunte acerca a su niño en los programas de actividades al aire libre. ¡Es fácil y divertido!

Physical Activity Programs At Selected Elementary Schools East Harlem, New York 2004

Programas de actividad física extracurricular en Escuelas Públicas Elementales selectas en el Este de Harlem

ESCUELA	DIRECCION	NIVELES
P.S. 50	433 E 100th St.	K-6
P.S. 101	141 E 111th St.	K-6
P.S. 102	315 E 113th St.	PK-6
P.S. 38	232 EAST 103 St	K-6
P.S. 133	2121 5TH AVE.	K-6
P.S. 146	421 E 106TH ST.	K-6
P.S. 171	19 EAST 103 S	K-6
P.S. 206	506 EAST 120th	3-6
P.S. 30	144-176 E. 126th	K-6
P.S. 57	178 E 115th	K-6
P.S. 7	160 E.120th	K-6
P.S. 72	131 E. 104th	K-6
P.S. 83	219 E.109th St	K-6
P.S. 830	219 E.109th St	K-6
P.S. 98	216 E. 120th St	K-7

Escuelas con áreas de juego al aire libre en el Este de Harlem

ESCUELA	DIRECCION
P.S. 101	141 E 111th St.
P.S. 102	315 E 113th St.
P.S. 108	1615 Madison Ave.
P.S. 146	421 E 106th St.
P.S. 155	319 E.117th St.
P.S. 171	19 E. 103rd St.
P.S. 206	506 E 120th St
P.S. 57	178 E.115th St.
P.S. 7	160 E.120th St.
P.S. 72	131 E. 104th St.
P.S. 609	19 E 103rd St.
P.S. 615	1573 Madison Ave.
P.S. 83	219 E 109th St.
P.S. 830	219 E.109th ST.
P.S. 835	131 E.104th ST.
P.S. 98	216 E. 120th St.

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Mount Sinai Medical Center GCO# 02-0399

Mount Sinai Community Boletín de Salud, otoño 2005

Plastics" that we planned on mailing to our study participants. But at a Community Advisory Board meeting, a board member suggested that we develop something more long-lasting than a piece of paper, which would most likely be thrown away after one viewing. As a direct result of this suggestion, we came up with a laminated card that easily fits into a wallet, and graphically depicts the recycling symbols of the "safer" plastics on one side and those that should be avoided on the other (see card graphics, right). The card was mailed to participants with the fact sheet, and has also been distributed at health fairs and conferences, like the 3rd Annual Symposium of the Breast Cancer and the Environment Research Center in San Francisco. To date, copies of the cards have been requested by physicians, scientists, public health officials, breast cancer advocacy groups, community members and others nationwide.

Tapping into Local Networks

As the *Growing Up Healthy* study progresses, the COTC plans to continue to tap into community resources and piggyback on already existing public health programs to give our participants the greatest access to all the health promoting resources available to them. Public health education is a collaborative endeavor, and in addition to the programs and activities originating with the COTC, we want to make *Growing Up Healthy* participants aware of and encourage them to take advantage of other programs already existing in the community.

A recent activity demonstrates how the COTC both benefits from and helps disseminate the message of other health agencies. The New York City Department of Health recently partnered with the State Department of Agriculture on the Healthy Bodegas Initiative, a city-wide effort to increase access to healthy foods in low-income neighborhoods by encouraging bodegas to carry new snack packages of sliced apples and baby carrots. In an effort to support this initiative, the COTC ordered 100 of these snack packages to give out at our Hip Hop Dance Party. The distributor was so impressed with the mission of *Growing Up Healthy* that he donated the snacks free of charge; in turn, COTC was able to promote the availability of these new products to the 200 community members who attended the dance party, while simultaneously introducing the children in the study to a wholesome, delicious snack available in their neighborhood food stores.

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Going Forward

As the COTC moves forward through the next four years, we plan to continue to offer participants an ongoing menu of fun and educational activities adapted to the needs of the children in the study as they grow older. We recently began distributing "Young Scientists' Club" membership binders to study participants, where they can keep a library of the fact sheets and newsletters we send them, and can track their study progress using special *Growing Up Healthy* stickers, mailed to them upon completion of each study component. In addition, a comic book is in the works, featuring our specially-designed *Growing Up Healthy* mascot, Enviro-Gurl, renamed Starr of Health by a participant in one of our newsletter contests. Through these efforts, the COTC aims to keep study attrition to a minimum while increasing environmental health literacy in the East Harlem community served by the Mount Sinai Medical Center.

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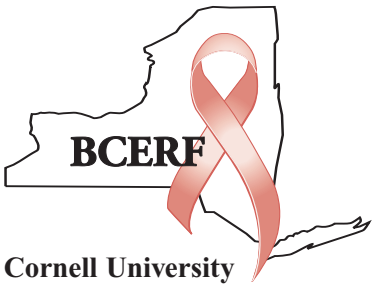
This article can be found on our website at:
http://envirocancer.cornell.edu/newsletter/article/vl2grow.cfm



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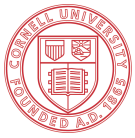

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