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 Nurses' Health Study: A Conversation with Dr. Graham Colditz

Professor of Medicine, Harvard Medical School and Professor in the Department of Epidemiology, Harvard School of Public Health

Can you provide some historical perspective on the Nurses' Health Study?

Our study population began with 121,700 nurses over twenty years ago, and we are still getting a 90% response rate to our surveys. Over time, as the population has aged, there are more cases of breast cancer and other cancers. More numbers of cases gives us the ability to both refine our estimates for relations between lifestyle, environment and cancer, and also allows us to look at subsets of cases. For example in the case of breast cancer, we can start to look at those that are estrogen-receptor positive or negative. The other important piece for a long term study like this is the fact that we can, with repeated measures, start to look at people who change their behaviors over time, and determine whether that modifies their subsequent risk. One of the unique things about Nurses' Health is that we are going back to participants and updating exposure information. That is something that clearly isn't done in a retrospective study where you would normally just be getting one set of exposures measures; some of the other cohort studies have historically just had a baseline questionnaire, and looked at the risk of cancer over the next ten years.

What strengths lie in the cohort study design and what weaknesses?

Clearly the fundamental strength of the cohort or prospective design is that information is collected from participants when they are free from disease. In that setting you avoid the potential for bias in terms of recall of exposure, be it diet or activity or other factors, because you are getting the information from participants when they are free from disease and following them forward.

You could argue that for some outcomes there may be different diagnostic criteria or different attention to looking for the disease in a population like the cohort

of nurses, but for a cancer outcome we are fairly safe. If you were doing a case-control study, you would be able to more rigorously apply diagnostic criteria, but in a study for cancer there is little debate for what is cancer and what is not. At the next level one could ask: are all the women having routine screening, or, are the women with bad diets less likely to be screened, and would that lead to some detection bias? A strength of the Nurses' Health Study in fact has been that because of prior medical training of the women in nursing, they have taken up healthy practices like mammography more rapidly than the population in general. That cuts down on the potential of detection bias. They have also more rapidly given up smoking than women in general. And while some say that this may cause a problem, I think in fact that it is helpful, because it lets us look at the benefits of quitting, which we've done. We're less dominated in our outcomes by smoking-related outcomes. Breast cancer and colon cancer are easier to look at in this setting.

In terms of limitations, like most of the cohort studies we have an underrepresentation of African Americans and other racial/ethnic sub-groups. It's a limitation of most all epidemiological studies. The Nurses' Health Study population is 96% white, and that reflects women who were being trained as registered nurses back in the '50s and '60s.

Who are the nurses in Nurses II and why the emphasis on nurses in general?

Nurses II is a younger cohort, comprised of women who were born between '46 and '64. Again these are registered nurses, and were chosen due to the quality of information we were getting in Nurses I. Part of the efficiency of a study like this is having accurate information, and our sense in setting up the study was that medical training would be advantageous. In addition to recording diets, if we were interested in hormones and other drug exposures the thought was that women who were trained in nursing would clearly pay attention to recording that. Also in terms of reporting their medical conditions, again the thought back in the mid-'70s was that women who were trained as nurses would be better informed about their health conditions than the average women. In the last 30 years or so that has changed, and the public in general is probably now more aware of their diagnoses than was the case in the mid-'70s when the design of this study was being finalized. When you look at media coverage and the way that the doctor-patient relationship has changed over time, there is just far more information out there. Think of *Our Bodies, Our Selves;* the fact that that was written tells you that things were not the same in the past as they are now.

Our knowledge of several possible risk factors in breast cancer development has been greatly strengthened by the Nurses' Health Study. What do you feel are the strongest conclusions? For breast cancer in particular, what do you still hope to find out? Are there areas that haven't been touched upon?

Alcohol and the risk of breast cancer, obesity and post-menopausal breast cancer, and use of postmenopausal hormones and postmenopausal breast cancer, would probably be the ones that are the strongest relationships. The lack of association between past use of oral contraceptives and risk of breast cancer is another clear thing that we documented. Documenting where there isn't a long-term health effect is also very important to have on the table.

We are still looking at components of the diet such as folate and glycemic load, and the patterns of postmenopausal hormones. That is an area that has changed fairly dramatically in the past ten years – the shift toward women with a uterus taking estrogen plus progesterone. In the '80s, whereas that might have been recommended, most women who were taking hormones were not doing it. Now it is clear that most are. We also have a new effort underway to look at diet after diagnosis of breast cancer and how that might modify survival. We can talk to women about diet before diagnosis but no one really knows whether they are going to get breast cancer or not. But all the patients here who've got breast cancer come in and ask, "what can I do, what can I eat that will help me survive?" and no one has studied it. We've got nothing to tell them.

In Nurses II, with the younger population, we are recording more details about patterns of lactation, in order to have a more rigorous evaluation of breastfeeding and risk of breast cancer. The level of detail will be all the way to asking when solids were introduced to the infant, since that changes the demand on the body and hormonal exposures for the lactating mother. We hope to be able to refine our understanding of the relations; across many studies there is a strong suggestion that longer lactation leads to lower risk of breast cancer. The first generation of nurses actually didn't do a lot of breastfeeding. Population trends drive in part what we can and cannot look at in terms of breast cancer risk factors.

Nurses II also has the 10 to 14 year old children of the nurses participating in a four-year follow-up looking at

diet, physical activity, weight gain, weight control practices, uptake of cigarette smoking, etc. We are pursuing a range of hypotheses there. It won't be too long until this population moves into the period of having benign breast biopsies and that in itself will be informative.

Considering the evidence that breast cancer risk is likely greatly influenced by exposures and conditions early in life, possibly even before birth, can you comment on the strength of some of these associations and the ways that Nurses' Health is contributing to our knowledge in this area?

We've got lots of hypotheses and because of the way we have conducted our research to date, there isn't a whole lot of direct evidence, most of it is indirect. For example, we know that age at menarche is important, and we know that age at first pregnancy is important, so it's not a great leap of faith to say that things happening between menarche and first pregnancy might also be important. We know that around the world there are many studies that show that height is related to breast cancer and other cancer risk, but not many people have studied the determinants of height. If you look generation by generation, we are on average taller than our grandparents; what else has happened? The number of childhood infections we got was much lower than our grandparents, and our diets have changed. But no one has really dissected what part of diet might be related either to the height change or adult cancer risk change.

With regard to *in utero* exposures, Dmitrios Tricholpoulos proposed a while back that hormone levels that one is exposed to *in utero* might vary according to the mother's age, weight or weight gain during pregnancy. And, given that the breast is sensitive to hormones, maybe there is then genetic damage being done even *in utero* that is a consequence of exposure to estrogens during that early growth phase. Trying to take that hypothesis and test it has been slow. We've been able to ask women how heavy they were at birth, which they usually have to go ask their own mothers (although it seems that women are more likely to know how heavy they were when they were born than men are). At some level there is a fair excitement about the potential role of exposures from conception up to first pregnancy as being important, but the actual amount of information out there is still exceedingly small. It would be wonderful if we could find that some component of diet, be it folate or fiber or something else, during that period is protective against cancer. We can have some impact on what kids are eating in the home, perhaps more easily than a population-wide change by the time that they are, for example 25-50. And, the fact that there are school lunch programs means that if one could get beyond the politics of menu-setting, there is that potential of change outside the home, too.

Some of the reported results of the study with regard to breast cancer have sparked discussion about the study's ability to capture "real" breast cancer risk factors; for example, whether or not there is a connection between dietary fat intake and breast cancer risk. Debated questions here include whether the "low-fat" levels studied in Nurses' Health were low enough to make a difference, and the closely related issue of the significance of animal-based foods in the diet. Can you comment on the dietary fat issue?

When we came up with our first non-finding of the hypothesis testing for a relationship between dietary fat intake and breast cancer risk, people questioned why the finding was null. The fact that we are not now the only ones failing to find a prospective relation – there are eight other studies — to me is some affirmation of our finding. The level of commitment to the hypothesis – not wanting to accept that there is no relation — is somewhat surprising. There is lots and lots of debate that has helped people think through ways of analyzing this issue, but I think that the reality is that adult fat intake is probably not a major issue in terms of breast cancer. One of the notions we have is that the very low fat diet in the US is typically high in carbohydrate and in the US that tends toward simple sugars, and maybe something like the glycemic load is in fact having an adverse effect. There is a counter-balancing, if you will. I think we can look at some of those things, but the bottom line still comes back to the low-fat diet as it's available to most people in the US isn't the solution that a plant-based diet might be. At the moment that's not the way that people are getting to the low fat.

Another example is in the area of organochlorines in blood samples of participants; Nurses' Health found no significant differences in blood levels of chemicals tested between women with and without breast cancer. Questions here include whether the blood is the appropriate site to test this question and whether timing of exposure is critical. Is Nurses' Health pursuing the questions of exposure to environmental chemicals further?

The findings come from nurses' blood collected in 1989 and values there do not predict subsequent risk. I think part of the problem in studying this has to do with which environmental contaminant we are dealing with; some

of them are across the board going down and for some there are still pockets in the country where there may be some substantial exposure. But the sense we have is that again, this is not an area that has been as rewarding at explaining breast cancer as thought when the initial hypothesis was set forth. We have another updated analysis that is under review at the moment that is basically showing the same results. That is as far as we have gone with breast cancer. But yes, because we collected it in 1989, we have blood levels in middle-aged and older women, and again, one might ask, is it your exposures in adolescence that matters? These are issues that, across the board, are very hard to disentangle.

What do results mean to the individual woman? We are learning more about genetic predisposition to breast cancer. Is genetic information collected in this study? How can knowledge gained from Nurses' Health contribute to useful information for women of varied genetic backgrounds?

Epidemiology estimates population average risks and is very good at predicting the future burden of disease in the population. If we know that 30% of the population is currently smoking cigarettes, and their age, we can predict how many cases of lung cancer there will be, how many heart attacks there will be. But we are very poor at predicting which of the smokers actually will get lung cancer, which of them will get a heart attack. But we can get very close to the exact right number of cases. Even with breast cancer, I have seen it said and I in fact would agree, that like smoking and lung cancer, we know what causes breast cancer, it's just we cannot predict at the individual level who will get breast cancer. The milieu of female hormones clearly drives breast cancer risk. We can contrast the rates in China and Asia with the US, and when you take into account the different reproductive patterns, you explain more than 50% of the difference between rates in China and rates in the US. What I'm saying comes back to the fact that the results of epidemiologic studies are definitely applicable to the population, be it one thousand women or ten thousand women. What they mean to the one woman in ten thousand trying to interpret them is harder. We say drinking two alcoholic drinks a day increases risk of breast cancer by 50%. But if an individual woman cuts down her alcohol, she hasn't cut down on her risk by 50%. If a thousand women cut down their alcohol then the number of cases of breast cancer in that thousand women will be reduced accordingly. But most of them weren't going to get breast cancer anyway, and most of them still won't get breast cancer when they've stopped drinking. The challenge is how you move from the population level to the individual level and I actually don't think that genetics is going to help there. Like all these dietary exposures, there is going to be a myriad collection of genetic factors that predispose to cancer, apart from the very high risk BRCA1 that affects 5% of breast cancer. Otherwise I actually don't think that all the genetic detail is going to help very much.

One can take epidemiologic findings and say that they have a clear implication for the whole population; for example, if we say low folate in the diet increases risk of colon cancer. Then, if the FDA recommends an increase in folate fortification to the food supply, that is going to lift the folate levels in the whole population, so the population level finding is translated to a population level change. What the translation is for individuals gets to be a whole lot harder unless it's something that we can see everyone changing. I would rather we frame messages like, "everyone should increase their level of physical activity," than "if you walk for half an hour a day you have lowered your risk of disease A, B, or C." The latter unfortunately implies that if you walk for half an hour, you won't get the disease. We're not promising that, we are talking about lowering the chances. We're trying to do a better job talking about risk from the academic end.

Have the ongoing results of this study changed your own thoughts one way or the other with regard to how much an individual can do to actually prevent cancer? How about we as a society; what are the barriers to risk reduction on a large scale in the US?

Both the results and trying to teach about cancer prevention have definitely pushed me to think more and more about the ways we can translate our findings to societal level changes. As we talk about increasing physical activity we start to ask, what are the barriers to people being out there walking? Is it violence on the streets? Is it cars running you over? Why are our towns designed so that you can't go to the store unless you've got a car? Seeing our results and thinking about how they translate back to behavior changes helped me start to think about the level at which we have to think about a change and what the barriers might be.

Think of breastfeeding, and my analogy there would be changes made with regard to smoking. Say breastfeeding has a proven impact on breast cancer risk, with higher breastfeeding lowering risk (and the same applying to ovarian cancer). If we actually applied an

OSHA-type regulation to something like breastfeeding we can say that in fact employers have to provide lactation facilities for women upon return to work because the disease burden from not breastfeeding is so high. You can do the arithmetic and show that, but what are the issues? In this country women return to work early after childbirth compared to Europe, where they probably get six to nine months off, which in itself would facilitate longer breastfeeding. When a woman returns to work and wants to continue breastfeeding, she will need facilities to be able to pump. Many employers would consider that a distraction, but in fact the cost effectiveness data show that an infant who is fed breast milk has fewer infections, which means the mother is going to take less time off work to take her kid to the doctor. Changing the workplace to facilitate breastfeeding would be wonderful; we changed the workplace around tobacco exposure because your smoking would influence my health; your breastfeeding is not going to influence my health, so it's harder to get the externalities all lined up. But one needs to ask, if we could change the workplace around the health impact of passive smoking, which is small but detectable, why can't we do the same around breastfeeding?

Dietary change is complicated by all the marketing forces that span so many different levels, be it the junk food advertising that's aimed at kids when they are watching TV, through to the way food is packaged. Then, in the inner cities, access to supermarkets and fresh fruits and vegetables becomes an issue that has a real impact on having population-wide changes. In Boston the mayor has sponsored farmer's markets in every neighborhood during the summer, so maybe that has an impact on fruit and vegetable consumption in the summer. But what happens to the other nine months of the year in Boston, where you can go four or five miles and a couple of bus changes to get to the supermarket? You can get to some of these structural barriers pretty quickly when you start to think about changes in diet. The literature on tobacco uptake is informative. The adult message that is put out by the industry is that everyone makes his or her own informed choice about smoking. But rather it is probably a learned behavior, with a variety of forces and influences, including what is deemed to be culturally appropriate. Our diet follows that same model, with a variety of forces and influences. We must identify them and harness them to promote wellness.

Research Commentary

Using Comparative Cancer Models for Breast Cancer Research

Human, canine and murine BRCA1 genes: sequence comparison among species. Szabo CI, Wagner LA, Francisco LV, Roach JC, Argonza R, King MC, Ostrander EA. (Fred Hutchinson Cancer Research Center and University of Washington, Seattle) Human Molecular Genetics 5:1289-1298, 1996.

The role of comparative cancer research in the effort to reduce the morbidity and mortality of cancer in humans is evolving rapidly. Rodent models of human cancer are essential for investigation of numerous *in vivo* issues. In most cases, however, data from rodent studies must be extrapolated to humans with caution and some technologies are not easily studied in small animal models. Investigation of cancer in companion animals (dogs and cats) offers significant advantages to the research community because of similarity in cancer etiology, repeated access to normal and tumor tissue during a study and clinically relevant treatment outcomes. Many academic and pharmaceutical cancer research institutions are now supporting investigations in companion animal cancer as one component in a complementary array of preclinical models. Such investigations are clearly done to advance human health but animal health has advanced substantially as a result.

Breast cancer is the **most** common malignancy in female dogs (estimated incidence = 175/100,000 dogs at risk but risk in sexually intact dogs is approximately 260/100,000). Canine breast cancer is similar to breast cancer in women in several ways: 1) 95% of cancer arises from the glandular epithelial tissue, 2) approximately 35-45% of cancers express estrogen and/or progesterone receptors and such expression represents a favorable outcome, and 3) aggressive clinical behavior occurs in approximately 25% of dogs with breast cancer including regional and widespread metastases.

Based on the report of Szabo, et al, cited above, canine breast cancer is now known to be genetically similar

Table 1: Similarity between the human BRCA1 gene and the canine and murine BRCA1 sequences.

	Canine	Murine
Nucleic Acid Identity	84%	72%
Amino Acid Identity	74%	53%
Amino Acid Similarity	90%	79%
Functionally Important Sites		
Amino terminal	96% AA similarity	90% AA similarity
RING finger motif Carboxy	1 AA difference/40AA	100% identity
terminal	85% AA similarity	85% similarity
Central Portion	70% identity	53% identity

to breast cancer in women. These investigators identified and compared the nucleic acid and protein sequence of the BRCA1 homolog in dogs and mice. In all three species the BRCA1 gene codes for a protein of between 1850-1900 amino acids which may function as a tumor suppressor.

In addition to similarities in general structure, the sites of missense gene alterations known to account for exceptionally high rates of breast cancer in women occur in highly conserved regions of the gene in both the dog and rodent.

It is estimated that 5-10% of breast cancer in women may be attributed to inheritance of mutations in the BRCA1 gene. The role of BRCA1 mutations in dogs is unknown and to date no mutations in the BRCA1 gene have been identified in breast cancer of dogs. However, inheritance of canine breast cancer has been suggested by several studies and identification of canine families with strong heritable tendencies of breast cancer would be valuable. Since pedigree and breeding information is recorded for many purebred dogs and because this is a spontaneously occurring, frequent tumor, investigating the significance of this gene in breast cancer development may be more relevant in dogs than in rodents.

As the canine and feline genome are resolved, many genetically based diseases such as cancer are found to be etiologically similar across species lines. Many tumor suppressor genes and oncogenes in dogs and cats are strongly conserved in the regions where functional mutations occur in human cancer. Furthermore, environmental exposure of carcinogens has been documented to increase the risk of various cancers in dogs. It seems obvious that such a relevant and prevalent resource should be more aggressively incorporated into cancer control programs. Currently, however, this resource is vastly underutilized. Development of tumor registries, tumor depositories, comprehensive companion animal cancer centers and cooperative research endeavors are required to extend the phenomenonology of canine and feline cancer into clinically applicable programs for the investigation of prevention, early diagnosis and control of cancer for all species.

Prepared by:
Dr. R.L. Page, Director
The Comparative Cancer Program at Cornell University

Dr. Page is the Director of the new Comparative Cancer Program at Cornell University. The ***mission*** of the Comparative Cancer Program is to promote and integrate existing strengths in cancer research with other critical components of cancer management such as prevention, early diagnosis, clinical care, education and outreach. In order to achieve this goal the program will develop programmatic linkages **from** basic and applied cancer research laboratories around the campus to the developing efforts in traditional and transgenic animal sciences ultimately **bridging** to clinical investigations at the Veterinary Medical Center.

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FACT SHEETS

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

General Information on Breast Cancer

__# 3—Understanding Breast Cancer Rates

__# 5—The Biology of Breast Cancer

__# 6—Tumor Suppressor Genes - Guardians of Our Cells

__# 9—Estrogen - What is the Relationship?

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

Diet and Lifestyle

__# 1—Phytoestrogens and the Risk of Breast Cancer--*Revision*

__# 8—Childhood Life Events

__#13—Alcohol

__#18—Fruits and Vegetables

__#19—Exercise

__#27—Dietary Fat

__#29—Breast Feeding

__#33—Dairy Foods and the Risk of Breast Cancer

CRITICAL EVALUATIONS OF PESTICIDES AND BREAST CANCER

Critical Evaluations are available on the BCERF web page (see address below) as portable document files (pdf).

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
__#2 Lindane
__#3 Heptachlor and Heptachlor Epoxide
__#4 Chlordane
__#5 Simazine
__#6 Cyanazine
__#7 Dichlorvos
__#8 Atrazine
__#9 Chlorpyrifos
__#10 Diazinon

Pesticides and Breast Cancer Risks

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

__#11—An Evaluation of Chlordane

__#12—An Evaluation of Heptachlor

__#14—An Evaluation of 2,4-D

__#15—An Evaluation of Lindane

__#16—An Evaluation of Simazine

__#17—An Evaluation of Cyanazine

__#20—An Evaluation of Dichlorvos

__#23—An Evaluation of Atrazine

__#26—An Evaluation of Chlorpyrifos

__#28—An Evaluation of Diazinon

__#32—An Evaluation of Alachlor

Pesticide-Related Issues

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

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

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

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

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

__#24—Consumer Concerns About Pesticides in Food

__#25—Pesticide Residue Monitoring and Food Safety

__#30—Resources for Information on the Health Effects of Pesticides and Responding to Pesticide Poisonings

__#31—Integrated Pest Management Around the Home and Garden

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"We Need to Know"

Ad Hoc Discussion Group

"Learning Together"

On March 17, 44 people attended the BCERF Ad Hoc Discussion Group meeting held in the Assembly Parlor at the Capitol Building in Albany.

Participants were eager to discuss the timely information presented on two important New York State projects: the Cancer Surveillance Improvement Initiative and the Pesticide Sales and Use Registry (PSUR).

Director June Fessenden MacDonald's BCERF update included announcement of her own retirement and pleasure working with the group during these last years (see *Letter from the Director* on page 9). She also described the coming expansion of the Community Environmental Health Education projects to take place in the current budget year.

NYS Cancer Surveillance Improvement Initiative

The group was fortunate to have Dr. Mark Baptiste, Director of the Bureau of Chronic Disease Epidemiology and Surveillance at the NYS Department of Health (DOH), come to discuss the cancer mapping project, or, the NYS Cancer Surveillance Improvement Initiative. This project involves four work teams focusing on: cancer registry improvement; cancer outcomes mapping; cancer risk factors mapping, and; risk communication (map interpretation for the public). An advisory council plays an important role in the project, addressing questions such as unit of analysis (for example, is zip code an appropriate unit of analysis?), priorities for the Initiative, and techniques for integrated and meaningful analysis and display of data.

Dr. Baptiste discussed the criteria for success of the Initiative. The analysis and dissemination of results must be scientifically valid, responsive to the needs of the public, and maintain the confidentiality of all New Yorkers. He outlined the time line for the project – many readers will by now have seen the county maps, as well as the sub-county (by zip code) breast cancer incidence maps, which were released in mid-April. Many people have wondered how areas which are described as having "excessive" cancers are determined. Dr. Baptiste described the statistical techniques used to identify excesses that are least likely to be due to chance.

Also important are the Initiative's strategies for health risk communication. He discussed audience identification, the development of fact sheets, and recommendations derived from focus groups. The BCERF Ad Hoc group had questions such as whether there are plans to overlay data, such as that of the DEC, with these incidence maps. Dr. Baptiste said these data sets will be examined, but no definite plans are in place for maps. Another question brought up the problem of length of residence in a particular zip code – with people's migrations we will have issues when looking at environmental exposures. Dr. Baptiste described that this surveillance tool is not a complete source of data on residential histories, but rather can be hypothesis-generating, and that other epidemiological inquiries like the Long Island Breast Cancer Study Project can follow up. Further questions and discussion contributed to what was an overall important interaction on this critical project.

Three Agenda Items on Pesticide Sales and Use Registry (PSUR)

In the first of three agenda items addressing PSUR, Bill Smith of Cornell's Pesticide Management Education program reported on satellite database development relative to PSUR. Cornell's role is to provide technical expertise to the NYS Department of Environmental Conservation (DEC) on PSUR, as well as database design and implementation. Bill outlined major initiatives, such as technology forums, additions to the database, and replacing paper-based labels. He also looked to the future, describing activities with which his program would be involved, and demonstrated possible searches on the database. Several questions from the group focused on the problems members of the public face in obtaining information on inert ingredients.

The presentation of Bob Haggerty, Chief of the Pesticide Reporting Section of the Bureau of Pesticide Management, NYC DEC, kicked off the remainder of the afternoon's discussion of PSUR. He and Bill Smith provided an update on PSUR, as well as touching on related legislation in other states. Bob announced that DEC is moving in one year, but that it will not affect PSUR. He continues statewide education on the requirements of the law, with 4,000 attendees at workshops last November and December. Unfortunately, data quality is still an issue, and additionally, 3,000 entities did not report by February 1. Enforcement actions are being taken.

Audrey Thier of Environmental Advocates began the panel discussion of *PSUR: Perspectives on the First Three Years*. While her organization is pleased with the generation of data so far, she suggested several improvements. These include: active ingredient reporting; translation of gallons and other measures to pounds; measures of homeowner use, and; mandatory electronic reporting. She also noted that farmers use less pesticide than other commercial applicators.

Patrick Hooker, Director of Governmental Relations for the NY Farm Bureau followed, began by agreeing with Ms. Thier about where in the state pesticides are applied and intensity of use. He emphasized that pesticides would not be used if not they were not needed, which is apparent by the expense, time and training required for their use. He also relayed farmers' concern that good products might be pulled from the market. He urged activists to consider the PSUR program relative to resources spent.

Gunther Fishgold of 1-in-9 Long Island Breast Cancer Action Coaltion urged reporting of all pesticide use, and improved research in the possible link between pesticide exposure and breast cancer risk. His group would like to see pesticide use data overlayed with data of cancer incidence in a mapping project.

Pat Voges, the Government Affairs Chairman of the Nassau/Suffolk Landscape Gardeners Association spoke as someone "who represents the people who fill out the forms." He emphasized that until those who illegally apply pesticides are removed from business, the data are not meaningful. He also made the case that many who need to report are not able to do so electronically, such as many single-family businesses.

The panel offered perspectives on these many issues. Audrey Thier suggested some of the possibilities for electronic reporting for those without computers, as are offered in California. Diverse perspectives came together on the issue of illegal applicators, with the group reaching consensus. One action point was that, with guidance, consumers could play a key role by being aware of laws and making sure to only hire certified pesticide applicators.

MARK YOUR CALENDARS!

The next Ad Hoc Discussion Group meeting will take place on Wednesday, June 21, 2000 at the Carriage House, Bayard Cutting Arboretum Oakdale, Long Island

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.

Letter from the Director

I requested this space so I could say goodbye to the many of you I have come to call friend and colleague and to those of you I have not met, but hear from once in awhile. As of July 15 I will be leaving the directorship of BCERF and retiring to Vinalhaven, Maine, an island 12 miles off the coast of Maine in West Penobscot Bay. As the Maine motto says, enjoying "The Way Life Should Be."

During the past five years I have enjoyed getting to know you and will miss those interactions I have had with the many diverse groups and individuals concerned about breast cancer and the environment. I have been privileged to work with very able and dedicated people at Cornell, across New York State, and nationally. Serving as Director of BCERF has been a wonderful, albeit at times trying, experience; a satisfying way to end my academic career. It is always uplifting to hear how someone is using our materials whether breast cancer patient and her/his family, a high school student for a report, a policy maker or a physician half way around the world.

I am grateful for your commitment and support over the years as we struggled into existence and grew to be not only a nationally, but internationally recognized and respected program. BCERF will continue to respond to the need for information and education with our translational research and breast cancer risk reduction efforts. The program will continue to grow and address additional concerns. One new area this year will be pesticides and childhood cancers.

I also want to use this space to provide my successor with a "Welcome Aboard". The incoming BCERF Director, Rodney Dietert, Professor of Immunology and

Toxicology, is no stranger to those of you who have been with the program since that first year. Rod was one of the Cornell founding members of BCERF and has continued to be a strong advocate of the program. In fact, he helped to provide the current home for BCERF in the Institute for Comparative and Environmental Toxicology (ICET) in the Cornell Center for the Environment while serving as director of ICET. He was a member of the BCERF Executive Committee for three years. More recently, he has supported BCERF programming by giving a presentation at an Ad Hoc Discussion Group meeting, reviewing Critical Evaluations and contributing to this newsletter. Rod is a strong proponent of the need to promote interdisciplinary research at Cornell and elsewhere to address complex environmental and health issues. His research interests are in the area of toxics and childhood health and disease.

On a more personal level, Rod grew up in Texas, was educated in North Carolina, and has been a health-effects researcher and teacher at Cornell for the past 23 years. He asked me to relay to all of you that he "is thrilled to formally join the BCERF program as Director and to help continue the efforts that have reached so many people."

I look forward to continued excellence by the BCERF staff in all their endeavors. I thank them for all their hard work and devotion to breast cancer risk reduction. I wish them and you the very best for the future. I have truly enjoyed working with and knowing all of you.

What's New On The Web
http://www.cfe.cornell.edu/bcerf/

The lazy days of summer are coming soon, but far from a restful retreat, we here at the BCERF web are working away diligently. There are going to be many behind-the-scenes changes in the way the BCERF web works, but hopefully all the construction will remain invisible to our visitors. We will still be adding new materials throughout the summer, so visit us soon.

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