

## RESEARCH COMMENTARY

# Environmental Chemicals and the Risk of Diabetes and Gestational Diabetes

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

*Since the late 1990s research studies have explored whether exposure to environmental chemicals affects the risk of diabetes.*

A recent review by Dr. David Carpenter, professor at the Institute for Health and Environment at the University of Albany, traces the evidence and possible role of various environmental chemicals in the etiology of diabetes (1). In Dr. Carpenter's review, he relates that while earlier studies focused on exploring linkages between environmental contaminants such as dioxin and polychlorinated biphenyls (PCBs) and the risk of diabetes, in more recent years evidence has accumulated suggesting that various persistent chlorinated pesticides may be related to diabetes. In addition, data from the Agricultural Health Study support a role for other less persistent pesticides (such as triazines and organophosphates) in the risk of diabetes in farm workers who are pesticides applicators (5), as well as linkages to gestational diabetes in their spouses (6). This *Research Commentary* will review the emerging evidence and the data gaps that need to be filled to answer the important public health question of whether general or occupational exposures to environmental chemicals affect the risk of diabetes.

The studies that support a role for persistent organochlorines and diabetes risk include studies published by several different groups of investigators that analyzed data from the 1999-2002 National Health and Nutrition Examination Survey (NHANES) (2-3), and a Hispanic version of this survey (4). The NHANES studies, conducted by the Centers for Disease Control and Prevention (CDC), collect health status data and analyze blood and urine samples for a variety of environmental contaminants. Both the general US population and ethnic subgroups, including

Hispanic populations, have been included in these surveys. These surveys suggested that self-reported diabetes was related to a variety of persistent organic pollutants (POPs), including dioxins, PCBs, and pesticides such as DDT and a metabolite of chlordane called oxychlordane (2, 3, 4). The strengths of these studies include that researchers assessed health risks in a cross-section of the American population and blood levels of the various chemicals were measured to approximate past exposures. Disadvantages of these studies include that the diabetes was "self-reported" rather than obtaining diagnosis information from medical records.

As these studies have emerged, questions still remain about their interpretation. It has been hard to determine if any of these associations are truly "causal." Did exposure to the specific chemicals measured in the NHANES and earlier studies cause the diabetes in the subjects studied, or are there alternative explanations? For instance, as has been hypothesized in Carpenter's review (1), many of the

persistent organochlorines are fat-soluble and the primary source of exposure is from animal fat (meat and dairy) and fatty fish. Therefore, it is possible that the particular chemical that was measured may be a surrogate for another chemical that was not measured, but that may occur in the same food source. Many fat-soluble chemicals tend to co-migrate as they travel up the food chain. Hence, it has been very difficult to determine the role of individual POPs as causal factors in inducing diabetes (1).

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Other questions include, do the chemicals act directly to affect diabetes risk, or do they affect other risk factors for diabetes? For example, do the chemicals play a role in the onset of obesity? The answers to these questions are still being explored. There are other trends that do not have an explanation. The blood levels of many of the POPs have been dropping in Americans in the last 20 years (most were banned by the late 1970s), and yet the incidence of diabetes has been increasing in the American population during the same time period. This is the opposite of what would be predicted if organochlorines alone were the driving force for causing diabetes in the last two decades. While studies done to date do suggest that certain persistent chlorinated chemicals may play some role in determining diabetes risk, more work is needed to evaluate a wider range of environmental chemicals.

The NHANES studies provided evidence that exposures to chlorinated chemicals from common sources, e.g. contaminated food, could affect diabetes risk in the US population. But, few studies have determined if occupational exposures to these and other chemicals affect the risk of diabetes. And no studies have explored whether chemical exposures could affect the risk of gestational diabetes in women. Some of these questions have been explored by investigators from the Agricultural Health Study, a long-term, large-scale study of how pesticide use affects a variety of health endpoints in pesticide applicators and their family members (subjects are from Iowa and North Carolina; see <http://aghealth.nci.nih.gov/>).

Unlike the NHANES studies, blood levels of the pesticides were not measured in the Agricultural Health Study. Rather, detailed questionnaires approximated past use of many different types of pesticides, including major classes of pesticides (insecticides, herbicides, and fungicides). The team of researchers, led by Dr. Dale Sandler, an epidemiologist at the National Institute of Environmental Health Sciences (NIEHS), obtained information on lifetime exposure to pesticides and self-reported diabetes self-reported in 33,457 licensed pesticide applicators (5). The researchers looked at two measures of pesticide use: whether the applicators reported ever using the pesticide and cumulative lifetime days of use.

Of the 50 pesticides included in the survey, seven pesticides were associated with an increased incidence of diabetes in the applicators (5). This included five insecticides; three were organochlorines that persist in the environment but are no longer in use (aldrin, chlordane, and heptachlor) and two were organophosphate pesticides that are still used in agricultural settings (dichlorvos and trichlorfon). Dichlorvos is used to control insects in barns and on livestock. Trichlorfon was used in the past to control soil insects to protect food crops and parasites on livestock, but currently its major use is to control grubs and other soil insects in ornamental plants used in landscaping, and in

lawns and golf courses (6, 7). This study is one of the first to report that use of trichlorfon is associated with a higher risk of diabetes in occupationally exposed individuals (use was significantly higher in diabetics compared to non-diabetics in the Agricultural Health study subjects). The two herbicides associated with a higher risk of diabetes included alachlor (one of the most highly-used herbicides in the US), and cyanazine, a triazine herbicide that has been phased out of use in recent years.

There is other evidence that organophosphate pesticide exposure is linked to diabetes. There is evidence from animal studies demonstrating that this class of insecticides can change how the sugar glucose is processed, as well as evidence that in pesticide poisonings, glucose is higher in the blood or urine when people were exposed to high levels of certain organophosphate insecticides (as cited in 5). However, the Agricultural Health Study is the first study to show that long-term occupational exposure to organophosphate pesticides may increase the risk of diabetes in applicators. These findings should be confirmed in other occupations that have used organophosphate pesticides for pest control, including structural pest control operators, those working in horticulture and greenhouses, golf-course managers, and other turf pesticide professionals.

What is also remarkable is what this study did not show. Exposure to pesticides that are known to be contaminated with dioxin, such as the phenoxy herbicides 2,4,5-TP, were not associated with a significantly higher risk of diabetes in the Agricultural Health Study applicators (5), which is in contrast to findings from the NHANES studies (2-4) and other earlier studies that did show a relationship between dioxin exposure and diabetes (1). Past use of DDT, an insecticide associated with higher diabetes risk in the NHANES studies (2-4), also was not associated with a higher risk of diabetes in the pesticide applicators enrolled in the Agricultural Health Study (5). The reason for the differences between these outcomes is not known.

Despite the interest in whether environmental chemicals affect the risk of diabetes in the general US population and through occupational exposures from agricultural use in predominantly male applicators, few studies have investigated whether there are chemical linkages to gestational diabetes in women. This gap was addressed in a study of pesticide exposures and gestational diabetes in female spouses of applicators in the Agricultural Health Study (8). Female spouses of applicators enrolled in the study completed questionnaires that included information on past pregnancies, self-reported gestational diabetes, and exposure to pesticides. Information on four types of pesticide exposure were assessed: 1) agricultural exposure (mixing or applying pesticides or repairing application equipment as a part of farm work); 2) indirect exposure (planting, pruning, weeding, picking or harvesting crops); 3) residential use (applying pesticides inside the home or the


garden); or, 4) a report of no exposure. A significantly higher risk of gestational diabetes was associated with seven different pesticides used only in the first use category, agricultural exposures. This included two organophosphate insecticides (diazinon and phorate), one carbamate insecticide (carbofuran), and four herbicides (2,4,5-T, 2,4,5-TP, atrazine, and butylate). There was no increased risk of diabetes in women who were indirectly exposed to pesticides or who were exposed through residential use.

None of the specific pesticides associated with gestational diabetes (8) were the same as the pesticides seen to increase diabetes in the applicators (predominantly male) in the study discussed previously (5). However, in both studies there were pesticides in the same class that were related to diabetes risk. For instance, atrazine is a triazine herbicide related to the risk of gestational diabetes (8). Atrazine is similar in structure to the triazine herbicide cyanazine, which was associated with a higher risk of diabetes in the applicator study (5). Also, in the gestational diabetes study and the applicator study, certain organophosphate pesticides were associated with a higher risk of diabetes, though the specific pesticides were different in the two studies. Unlike the applicator study, the gestational diabetes study did observe an association with pesticides known to be contaminated with dioxin (the herbicides 2,4,5-T and 2,4,5-TP), which is consistent with other studies that have seen a higher risk of diabetes with dioxin exposure (1-4).

There are limitations in the gestational diabetes study. The number of women exposed to the different pesticides evaluated was small, and for this reason, the results of this study need to be confirmed. Further studies on whether there are chemical linkages to gestational diabetes are extremely important. As mentioned in the conclusion of the

gestational diabetes study (8), 4% of women in the US develop gestational diabetes (9) and women with gestational diabetes have a 20-50% chance of developing adult onset diabetes within ten years (10). Hence, finding factors that may contribute to gestational diabetes and subsequent development of adult onset diabetes is important in understanding causes and prevention of diabetes in women.

There are many ongoing studies to determine how environmental chemicals affect diabetes risk, and I recommend Dr. Carpenter's review (1) for one of the best overviews of all the different mechanisms and genes affected by exposures to various chemicals that may play a role in the etiology of diabetes. It is evident that far more mechanisms than changes in glucose metabolism and insulin resistance are affected by chemical exposures. Specific genes and sets of genes can be activated or deactivated by various chemicals, and these actions at a cellular and molecular level may play an important role in the development of diabetes. But, while many genes have been identified in studies evaluating how environmental chemicals affect diabetes risk, the exact mechanisms have yet to be identified.

Given the rise of diabetes in recent years, and its public health consequences, it is very important to establish whether there are linkages to specific chemicals or classes of chemicals. Once established, strategies to decrease exposures to these chemicals in everyday and occupational settings will be important to decrease the risk of this chronic disease. 



This article can be found on our website at:  
<http://envirocancer.cornell.edu/Newsletter/articles/v14diabetes.cfm>

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# Chemicals With Obesogenic Action: An Emerging Area of Concern

By *Barbour S. Warren, Ph.D., Research Associate, BCERF*

Body weight results from the difference between energy intake from food and energy use in physical activity, and basic metabolism (energy used for daily body operations). However, while true, this statement is a simplification. Body weight regulation is quite complex, involving numerous signaling pathways and communication between many body systems/organs. This complexity is shown in animal studies that have defined nine types of causes of overweight and obesity. One new area of concern is that exposure to some chemical toxins both during and after early life development may affect body weight and contribute to obesity. An understanding of these chemicals and the different ways in which they might be acting is not yet well developed. One hypothesis is that exposure to these chemicals may have played a role in the recent and dramatic rise in obesity. This hypothesis is biologically possible but much more study is required before it can be assessed.

## **Obesity**

Obesity (having too much body fat) has been called a “bad player” for a number of reasons. It is a significant risk factor for a number of cancers (including breast cancer), diabetes, cardiovascular disease and arthritis (1). Obesity sneaks up on people gradually, arising from gains of two to four pounds of weight each year (2). Studies in animals have shown nine types of obesity including neural (related to the nervous system), hormonal, drug/toxin, nutritional, environmental, seasonal, genetic, viral, and undetermined (3). Excess body fat is also hard to lose. Our bodies actively protect their fat stores and fat or weight lost is usually largely regained (4). About 72 million adults in the US (one third of the adult population) are currently considered obese.

## **The control of body weight is complex**

How our bodies control their energy stores is very complex and not completely understood (5). The processes involved include the control of appetite, physical activity, basic metabolic rate, and the amount of fat tissue. The coordination of these processes requires organ-to-organ communication between the brain, muscle tissue, liver, stomach, intestines, pancreas, and fat tissues. This coordination uses many signaling agents. These chemical communicators include factors from fat itself, hormones from the liver, stomach, and intestines, and hormones acting in the brain, as well as responses resulting from memory, time of day, and what we see, smell, and taste. This level of complexity provides many targets for disruptive effects.

## **Chemicals with obesogenic actions**

Scientists have recognized the existence of chemical exposures that promote obesity for a long time. Examples include the considerable weight gain seen with long-term use of some antipsychotic (6) and antiepileptic drugs (7), and after nico-

tine withdrawal following stopping smoking (8). Beyond these examples, this obesogenic effect of some chemicals has to some extent been ignored. Drug and chemical safety evaluations have focused on the more common end point of toxicity, weight loss rather than weight gain. This important oversight was pointed out in a recent review of the scientific literature that discussed many examples of weight gain reported in animal toxicity studies (9). Compounds leading to weight gain included organochlorine pesticides, organophosphate pesticides, carbamate pesticides, polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), phthalates, bisphenol A (BPA), heavy metals, and solvents. Although the necessary chemical dose and the extent of weight gain were not examined, these effects are potentially important. Recent studies have begun to evaluate the effect of some of these chemicals on weight gain.

## **Laboratory studies of obesogenic chemicals**

Only a few chemicals with obesogenic action have been studied so far. Chemicals studied in some detail include: BPA, a building block for the synthesis of plastics; tributyltin, a pesticide added to marine paints; diethylstilbestrol (DES), a synthetic estrogen; and nonylphenol, a wastewater pollutant. Exposure to all of these compounds is common.

The ability of chemicals to promote obesity can be studied several ways. One direct but not definitive method is to examine the ability of chemicals to cause the formation of adipocytes (fat cells). Body fat tissue can be formed in two ways: by the creation of new fat cells and by the storage of more fat in existing and new fat cells. A number of cell lines have been developed for these evaluations (10). These cells are considered preadipocytes, or fat cells at an early stage of development. Differentiation or conversion of these early fat cells to mature fat cells is dramatic. It involves a single round of cell division, a change in cellular shape, and the accumulation of fat in the cells. This is not a definitive examination as it only examines one stage in the pathway to obesity (the formation and filling of fat cells). Tributyltin (11), BPA (12), nonylphenols (13), and phthalates (14) have all shown the ability to cause the formation of mature fat cells in this assay.

Studies in whole animals have also been used to evaluate a chemical's obesogenic potential. In these studies, animals are fed a chemical over a long period of time while their weight is monitored and compared to control animals that have not received the chemical. This is a reliable test since it involves an entire animal, but sometimes effects on animal and humans do not agree. In contrast to what would have been predicted from the fat cell studies, animals fed tributyltin (15), BPA (16), nonylphenols (17), and phthalates (17) all lost weight. These results best support the idea that although these compounds can cause the generation of fat cells, they do not have this activity in whole adult animals.

Effects of these chemicals on the growth and development of animals have also been studied. This is especially important during the time of development of fat tissue. Two critical periods of fat tissue development are before birth and shortly after birth (10, 18). For studies of this type, pregnant animals are exposed to the chemical or their offspring are given the chemical just after birth. These studies have identified several chemicals that can lead to adult obesity following exposure during these critical developmental periods. Compounds with this effect include tributyltin (11, 19), BPA (20), and DES (21, 22). The effects of tributyltin and DES are best understood, and these compounds have been shown to interact directly with some of the cell pathways involved in fat development.

### Human epidemiological studies


Epidemiological studies in humans have largely looked for associations between the levels of environmental chemicals within the body and obesity.

Two studies have used results from the National Health and Nutrition Examination Survey, which examined the levels of phthalates in 5149 participants (23, 24). Phthalates are chemicals mainly used as additives to plastics to increase their flexibility but are also found as additives to cosmetics, soaps, pesticides, paints and lubricants. They are known to be antiandrogenic (counteracting the effects of male hormones). Both of these studies examined associations between the levels of six phthalate breakdown products in urine and waist circumference (a measure of obesity) in men. They found an association between the phthalate levels and waist circumference. These studies were in agreement for all but one of the metabolites examined. One of these studies also examined another measure of the men's body fat: body mass index (BMI). Similar effects of the metabolites were seen with BMI and waist circumference. Only one of the metabolites was active in women and girls. In addition, one of the metabolites that was inactive in men was linked to a decrease in BMI in girls. The phthalates are broken down in the body more quickly than some other chemicals; this raises the question of whether these studies were examining changing exposure levels. However, exposure to these compounds has been found by other researchers to be fairly regular, so that levels of phthalates in the body remain fairly constant (25). Much more study is needed to determine if these results show a cause and effect association between phthalates and obesity.

One study has examined dichlorodiphenyl-dichloroethylene, DDE, a highly stable metabolite of the once commonly used organochlorine pesticide DDT, to see whether there was an effect of prenatal exposures to DDE on later obesity (26). Earlier studies had measured the DDE levels in fish-eating women living in the area around Lake Michigan; 213 adult daughters of these women were assembled and their DDE exposure while they themselves were developing in the womb was estimated from an earlier study of their mothers. The daughters' weights and levels of body fat (BMI) were measured to see if there was an association between body fatness and estimated DDE levels during the daughters' development. The authors concluded that there was a link between the mothers' DDE levels and the daughters' body fat and that this link provided compelling evidence that other similar chemicals

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A recent step in the **Estrogen Connection Project** was to gather input from young women, as well as from many contacts in the advocacy community, on how to most effectively develop small, convenient-to-carry, guidelines for use while shopping for cosmetics and personal care products. Input was collected from Long Island High School students who attended the LI Forum, Margaret Roberts (Program Coordinator, CRAAB!), Kathy Curtis and Bobbi Chase Wilding (JustGreen Partnership). Pictured here is the resulting market guide, which we are currently distributing. 

## What You Can Do Now

**Learn** more about product ingredients

**Use** the Environmental Working Group's Database to

**Look Up** ingredients in products you use  
<http://cosmeticdatabase.com/>



**Watch** videos on the BCERF web site  
<http://envirocancer.cornell.edu/research/endocrine/videos/>

**Read** product labels and

**Make** choices to  
**Avoid** these Environmental Estrogens:



Parabens	Placental Extracts	UV Screens
Butylparaben	Placental	Benzophenone-1 or -2
Ethylparaben	Extract(s)	Homosalate
Isobutylparaben		Octinoxate
Methylparaben		Oxybenzone
Propylparaben		Sulisobenzene

Videos are also on **YouTube™**  
<http://www.youtube.com>

**Use** the search word "enviroestrogen"  
**Share** with your friends & family



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# BCERF Outreach Efforts Continue with Emergency Responders

## In print:

We have created an educational poster entitled *Protection is Prevention* outlining some pertinent information from our *BCERF Alert for Women Firefighters* brochure. We have printed 100 copies to be distributed throughout New York State. An article about the *Alert* and the poster has been submitted for the April issue of *The Volunteer Firefighter*.

## In person:

Chris Batman-Mize began distribution of the *Protection is Prevention* posters both through the mail and in person during the last week of February and continuing in March. She traveled to several regions around the state including Long Island and the Albany area, casting a wide net in both rural and urban areas.

## On the Web:

The *Protection is Prevention* poster was posted to the Fire Service Women of New York State (FSWNYS) website in addition to the *Alert*.

➤ [http://www.fswnys.org/fswnys\\_news.html](http://www.fswnys.org/fswnys_news.html)

## On the BCERF website:

HTML English version of the brochure

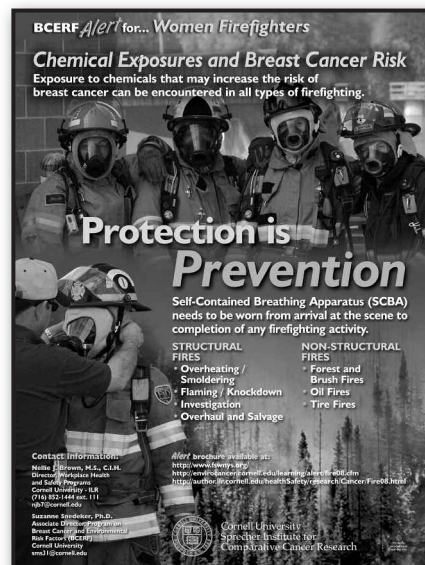
➤ <http://envirocancer.cornell.edu/learning/alert/fire08.cfm>


HTML Spanish version of the brochure

➤ [http://envirocancer.cornell.edu/learning/alert/fire08\\_espagnol.cfm](http://envirocancer.cornell.edu/learning/alert/fire08_espagnol.cfm)

Outreach efforts for the *Alert* have reached 100,000 people throughout New York State. The brochure has been posted on electronic list serves directed to:

- All individuals enrolled in state fire training courses
- New York State Fire Chiefs
- Fireman's Association of the State of New York (FASNY)
- New York State county fire coordinators



Finally, Dr. Suzanne Snedeker and Nellie Brown, Director of Workplace Health and Safety Programs, ILR Extension, will travel to the 2009 Conference of the International Association of Women in Fire & Emergency Services taking place in Omaha, Nebraska in May. They will present a workshop entitled “Breast Cancer Risk of Women Firefighters.” The workshop will include an overview of the factors that affect the risk of breast cancer, the different types of fire scenarios that can result in exposures to chemicals that may affect breast cancer risk, and hands-on case studies on use of protective equipment. 


## Chemicals With Obesogenic Action: An Emerging Area of Concern *continued from page 5*

should also be studied.

Two studies examining PCBs and BMI found opposite results. One small study (53 participants from Quebec, Canada) reported no association between PCB levels in the blood plasma and BMI (27). In contrast, a larger study of a different population (335 Akwesasne Mohawks from New York State) reported an association between PCBs and BMI (28). Interpretation of these results is difficult, as PCBs tend to accumulate in fat tissue. Plasma PCB levels could merely result from the size of the participants' fat reservoirs for the PCBs and have nothing to do with fat generation itself.

## Conclusions

Results to date have shown that some chemicals have actions that can promote obesity. However, the research in this area is very limited. Needed research in this area should first ask: which compounds have obesogenic activity, what are the critical periods of exposure for different compounds, and what levels of exposure are required to produce this effect? While some have suggested that exposure to chemicals with obesogenic activity has contributed to the increase in obesity seen in the US, understanding of

this effect is in its very infancy, and it is far too early to conclusively evaluate this hypothesis. 

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# BCERF Program Affected by Cuts to NYS Budget: Environmental Chemical Part of Program Faces Closure

## *Dear Friends and Colleagues,*

*As you are all aware, a fiscal crisis has hit our nation and New York State (NYS) especially hard. It has also hit our Program on Breast Cancer and Environmental Risk Factors (BCERF) here at Cornell University.*

The 2009-10 Executive Budget for NYS released by Governor Paterson in mid-December 2008 did not include funding to continue the support of the BCERF program. At that time we were informed by our Office of Government Affairs in Albany that the likelihood of our funding being restored to the NYS budget under current fiscal conditions was near zero. Our state funding had been supporting our research and education programs on the breast cancer risk of environmental chemicals in the home and workplace.

Our college and department are also severely fiscally challenged, and there are no funds available to provide us bridge funds to allow us time to pursue securing funds from other granting sources.

With this scenario, Dean Michael Kotlikoff in consultation with our department chair and director, Dr. Rodney Page, advised us that the portion of our program that had been funded through the NYS budget through a contract with the NYS Department of Health, must face closure.

What does this mean? It means that while we have been working hard to finish education and research projects currently funded under our state contract, after March 31, 2009 we will not have funding to support the continuation of these programs. It also means not having funding to continue many of our staff, including senior staff (both myself and Carmi Orenstein, our Assistant Director), other staff, and a number of part-time collaborators being supported with through our NYS funding.

While several state legislators in the NYS Assembly and Senate have recently advocated for the restoration of our funding, as of March 20, 2009, we do not know if funding has a chance of being restored. Hence, we must continue with planned closure procedures.

### **After April 1, 2009 we will no longer have funding to support the following activities:**

- Regional Cancer and Environment Forums
- Talks, presentations and workshops on environmental chemicals
- Quarterly newsletter, *The Ribbon*
- Critical evaluations on cancer risks of chemicals
- Our website at <http://envirocancer.cornell.edu>
- On-line cancer risk databases (e.g. the Turf Pesticide

and Cancer Risk Database)

- Fact sheets on environmental chemicals
- Videos on environmental estrogens in everyday products
- BCERF *Briefs* on endocrine disrupting chemicals
- BCERF *Alerts* on chemical exposures and cancer risks in the workplace
- Outreach with workplace audiences, including teachers and women firefighters
- Much of our risk communication research and outreach with pesticide applicators

Please note, that the work in the BCERF program on obesity prevention and breast cancer risk reduction, “Small Steps are Easier Together,” will continue. That work is funded by a grant from the US Department of Agriculture, and will be continued by our colleagues Dr. Carol Devine, Dr. Barbour Warren, and Ms. Mary Maley.

Our state funding for our contract entitled “Breast Cancer and Environmental Risk Factors in New York State” is unique. It allowed us to pursue many unique projects, to respond to requests for information on cancer risks of chemicals from many corners of our state, and develop innovative educational programming, and pursue translational research on cutting-edge issues to address the breast cancer risk of chemicals in the home and workplace.

I have been truly blessed to work with so many gifted staff, and with so many people in our state and beyond, on crucial issues concerning the cancer risks of environmental chemicals. We value the partnerships we have built with breast cancer and environmental organizations. You have been our source of inspiration, support, and our colleagues.

With sincere thanks for the opportunity to be a part of a wonderful program that made “sense out of the science” on the breast cancer risk of environmental chemicals, I am

*Very sincerely yours,*  
Suzanne Snedeker, Ph.D.  
Associate Director  
Principal Investigator for the NY State contract on Breast Cancer and Environmental Risk Factors in New York State

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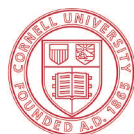
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