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Joint Informational Hearing on Breast Cancer and the Environment
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“Overview of: “State of the Evidence: What is the Connection between Chemicals and Breast Cancer?””

I am a professor at Tufts Medical School. My main research interest for the last 28 years has been breast cancer. In 1989, together with my research partner Dr. Carlos Sonnenschein, we accidentally discovered that some laboratory plastic ware leached chemicals that mimicked the female hormone estradiol, causing breast cells to proliferate. Since then, we have been investigating the health effects, including breast cancer, of environmental chemicals that mimic estrogen. I am going to provide an overview of the State of the Evidence Document, which summarizes the scientific research linking chemicals to the development of breast cancer. This peer-reviewed document was initiated by The Breast Cancer Fund and released at the first informational hearing on breast cancer and the environment convened last February by Senator Deborah Ortiz and the Senate Health and Human Services Committee.

My testimony will make the following points: 1) emerging evidence points to the role of environmental chemicals in causing breast cancer, 2) the controversy about epidemiological studies on the link between environmental exposures and breast cancer is due to incorrect design, and 3) animal studies clearly indicate that environmentally relevant doses of these estrogen mimicking chemicals produce measurable negative effects.

Breast cancer is now the most frequent type of cancer in women. During the past half-century, a swift increase of the lifetime risk of breast cancer has been observed in the US. In the 1940s, a woman’s lifetime risk of breast cancer in the US was 1 in 22. Today, the risk is 1 in 8. Breast cancer is also the leading cause of death in women ages 34 to 54. This swift increase cannot be attributed to genetic causation. Yet, the genetic causes of cancer continue to be the main topic of study in breast cancer research. Factors known to increase the risk of breast cancer including reproductive history, genetic factors, alcohol and exercise, account for less than 50% of all cases. I believe it is high time to seriously consider environmental chemicals as the most likely cause of this sudden increase in risk. Unlike genetic causation, searching for
environmental agents may produce evidence that can be used to prevent cancer. The State of the Evidence report summarizes our present knowledge and makes a well-balanced argument linking exposure to environmental chemicals to this increase in breast cancer incidence. This peer-reviewed document brings together, for the first time, several decades worth of research on breast cancer and the environment. I was one of the reviewers of this document and I fully endorse its content.

The increasing risk of breast cancer and other cancers has paralleled the proliferation of synthetic chemicals since World War II. An estimated 85,000 synthetic chemicals are registered in the USA, yet toxicological screening data are available for only 7 percent of these chemicals. Since many of these chemicals are endocrine disruptors, it is immediately apparent that the task of linking synthetic chemicals to breast cancer is going to be daunting. This is because we only know how to study one chemical at a time, and we are instead exposed to complex mixtures of hundreds, if not thousands, of synthetic chemicals.

The most compelling evidence linking chemicals and breast cancer is based on the fact that lifetime exposure to natural estrogen increases the risk of breast cancer, and that the use of hormone-replacement therapy and oral contraceptives also increase the risk. It has recently been proposed that this cumulative risk starts during fetal development. In fact, animal studies showed that exposure to DES during fetal life increases the risk of mammary cancer. Similarly, fetal exposure to dioxins also results in increased risk.

There are strong epidemiological data linking the synthetic estrogen DES and the estrogenic pesticides dieldrin and DDT to breast cancer. Several studies have found significant correlations between exposure to a given chemical and breast cancer, while others did not. It is becoming clear that many studies showing negative results measured exposure at the time of cancer diagnosis. However, we know that causal agents must have acted many years before the cancer was diagnosed. For example, recently published data on the Seveso, Italy dioxin accident measured TCDD dioxin blood levels at the time of the accident in 1976 and correlated it with breast cancer incidence, which occurred decades later. A 10-fold increase in TCDD blood level was associated with a 2.1 increase in risk for breast cancer (95% confidence interval, 1.0-4.6). More recently, at the International Society for Environmental Epidemiology in Vancouver in early August, Cohn et al. reported on a study that examined DDT and DDE levels in blood samples taken between 1959 and 1967. They demonstrated a significantly increased risk of
breast cancer among women with higher levels of DDT (and not DDE), but only among women who were exposed to DDT before age 15.

All women carry persistent pollutants in their bodies. Data by Dr Olea and collaborators show that these chemical mixtures, rather than single chemicals, correlate with breast cancer risk. Indeed, the results from these new studies are very alarming and support the conclusions of the State of Evidence document.

More research is needed to better understand the problem. And it must be a different kind of research. We need to develop adequate methodology to assess the effects of very complex mixtures of chemicals. We need to focus on timing of exposure—critical windows of vulnerability such as fetal life, puberty, pregnancy and menopause. And we need to study ubiquitous chemicals recently found to be endocrine disruptors. For example, very recent data in animals show that environmentally relevant doses of a ubiquitous plastic component, bisphenol A, causes significant effects in the mammary gland of animals exposed during fetal development. Among these changes is an increase in the structures that give rise to mammary cancer.

Negative results that have been obtained using wrong assumptions about when exposure should be measured, or about which marker should be measured, are being used to dismiss the notion that exposure to hormonally active environmental chemicals may be the underlying cause of the present breast cancer epidemic. It is time to stop repeating the same inconclusive experiments that measure exposure at the time of diagnosis. Animal studies suggest that we should look, instead, at exposures during fetal development and puberty.

Pursuing the research that will lead to more precise answers about exposure to complex mixtures and windows of vulnerability will take many long years. Meanwhile, it would be irresponsible to wait until all the evidence is gathered before articulating a preventive policy. It is time to shift the burden of proof from the exposed people to the manufacturers of these chemicals. Governments should articulate a public health policy that protects citizens in the first place, regardless of the economic consequences of the policy. As a physician, I am bound to the “do no harm” oath regarding individual patients. The aim of public policy should also be “do no harm.” As elected officials, you have an immensely important role in formulating policy that will reverse the epidemic. I think that the “State of Evidence” document provides the bases for a rational and effective preventive policy.