DES Action VOICE

Inside

A FOCUS ON DIETHYLSTILBESTROL

SPRING 2007 #112

No Specific Tumor or Gene Mutation Associated with Increased Risk for Breast Cancer in DES Daughters

"In Utero Exposure to Diethylstilbestrol (DES) Does Not Increase Genomic Instability in Normal or Neoplastic Breast Epithelium," Pamela S. Larson, et al., *Cancer*, Volume 107, Issue 9, 1 November 2006.

Reviewed by Kari Christianson

Since the publication last year of research confirming an increased risk for DES Daughters over the age of 40 developing breast cancer (reported in Voice 110), DES Action has received a number of questions about whether there is a specific breast cancer tumor linked with this increased risk. This is a very good question for DES Daughters, considering a specific type of cancer, clear cell adenocarcinoma (CCA), is associated with the lifelong need for gynecologic screening for this vaginal or cervical cancer. We now have an answer.

A first report concerning genetic characteristics of breast tissue from DES Daughters has been released. Researchers, using the medical records and tissue samples of breast cancer from some of the participants of the NCI DES Follow-up Study, reviewed the pathology reports. **They investigated whether or not breast tissue from these DES Daughters exhibited any of the genetic abnormalities that characterize other DES-associated tumors, like clear cell adenocarcinoma of the vagina or cervix.**

Headed by Pamela S. Larson, Ph.D., Department of Pathology and Laboratory Medicine at Boston University Medical Center, the team of researchers ". . . investigated DNA from normal, hyperplastic and malignant breast epithelium in DES-exposed and unexposed women. We expected that the breast tumors, and even the premalignant or normal-appearing tissue, arising in DES daughters might exhibit global or chromosome-specific increases in genotoxicities reported in other DES-associated tumors, specifically MI (micro-satellite instability) and AI (allele imbalance, also known as loss of heterozygosity [LOH])."

These researchers found no genetic differences between the breast cancer tumors of DES Daughters and unexposed women. There was an absence of the micro-satellite instability (MI) that is found in vaginal clear cell adenocarcinoma. This finding confirms "that MI is unusual in human breast cancers" and suggests "that **prenatal DES exposure does not affect DNA mismatch repair mechanisms in the breast**. Similarly, the equivalent amounts of AI (allele imbalance or LOH) seen in the breast tissue,

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> "...prenatal DES exposure may increase the growth of any cells which develop into breast cancer."

DES Dose Amounts Critical In Studying Effects

Developmental Exposure to Estrogenic Compounds and Obesity, Retha Newbold, et al., Birth Defects Research (Part A) (2005).

Reviewed by Pat Cody

This study found that mice exposed prenatally to *low* doses of DES developed an increase in body weight, associated with an increase in body fat. On the other hand, *high* doses of DES "caused a decrease in the offsprings' adult weight."

At the February meeting of the American Association for the Advancement of Science, some researchers working on endocrine disrupters similar to DES, that interfere with processing of hormones, also found weight increases in laboratory animals. Such disrupters include tributyltin and bisphenol A.

Scientist Bruce Blumberg, Ph.D., Director, School of Biological Sciences, University of California Irvine, reported that tributyltin

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MISSION STATEMENT

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Tumor *from page 1*

regardless of exposure, differs from findings in animal models and in vitro systems. Therefore, in utero DES exposure effects, if any, may be tissue, timing and/or species specific. . . ."

The authors conclude that, ". . . combined with the absence of genetic differences detected between groups, younger age of diagnosis (in the exposed group) is also consistent with potential effects of DES on human breast carcinogenesis being mediated by enhanced proliferation."

In consumer-friendlier language this means that there is no specific malignant tumor type associated with the increased risk for breast cancer among DES Daughters over the age of 40. Rather, prenatal DES exposure may increase the growth of any cells which develop into breast cancer. It is this potential for a more rapid growth rate that results in the increased risk for and incidence of breast cancer at an earlier age for DES Daughters. DES VOICE

DES Dose Amounts *from page 1*

disrupted genetic interactions that regulate fat-cell activity in animals. "Exposure to tributyltin is increasing the number of fat cells, so the individual will get fatter faster as these cells produce more of the hormones that say 'feed me." He found that the exposed animals remain predisposed to obesity for life.

Editorial Comment from Pat Cody

DES offspring fit into the *high dose* category. A report published 16 years ago using data from the ongoing DESAD study of DES Daughters is titled "Increased Risk of Profound Weight Loss among Women Exposed to Diethylstilbestrol in Utero." It was published in *Behavioral and Neural Biology* 55, 1991.

When we asked Dr. Blumberg why the response to dose differs, he wrote that "the spectrum of receptors that is activated at high doses is different from that activated at low doses.

As you know, DES is a very potent synthetic estrogen. At low doses, I would expect that it acts predominantly on the estrogen receptors (alpha and beta). The estrogen receptors are saturated at relative low doses, therefore, the high dose effects of DES are likely to be mediated through additional receptors."

Please note: what is being discussed here is research in progress, with no firm conclusions possible yet. See the article, below, with another take on information from the same meeting. DES VOICE

DES and Body Size Research

By Fran Howell

A growing group of scientists studying obesity find themselves focusing on endocrine disruptors – those chemicals that interfere with the way our bodies process hormones. So it is with interest that we are following discussions about whether prenatal exposure to some chemicals can potentially make adults fat.

In February several researchers presented their findings at the annual meeting of the American Association for the Advancement of Science (AAAS). Their hypothesis (and please remember that's all it is now) is that exposure to endocrine disruptors before birth can predispose an individual to a battle with weight in adulthood.

At the National Institute of Environmental Health Sciences, developmental biologist Retha Newbold is in the forefront of this new thinking. She has dedicated her career to working on DES research and has seen that developmental exposure is responsible for overweight adult mice.

Newbold did experiments controlling for both diet and exercise. What happened was that DES-exposed mice grew fat, and stayed that way, even when put on diets and made to exercise.

This led Newbold to conclude that early developmental exposure to *low* doses of DES cause a body to produce more fat cells. She found the mice exposed developmentally to low doses of DES tend to grow larger fat cells than those in unexposed mice. The fat cells mostly concentrate in the abdomen of adult DES-exposed mice. But interestingly, Newbold also saw that *high* developmental doses of DES caused a decrease in the mouse offspring's adult body weight.

To find out what the situation is for human DES Daughters and Sons, the on-going National Cancer Institute DES Follow-up Study sent tape measures with its recent questionnaires. Researchers will assess weight ratios and body size to see fat distribution.

"I'll be excited to learn the results from this epidemiology study," says Newbold. "Scientists are just recognizing that fat cells (adipocytes) are endocrine organs, and we know that developmental exposure to DES upsets endocrine systems, so it is reasonable to expect that the regulation and response of fat cells may be affected. This effect could result in obesity, or the opposite effect," she says.

It is important to remember that obesity is seen in many cases where DES exposure is clearly not a factor. Multiple variables are at play, including genetics, exercise and diet. But with so many endocrine disruptors (that act like estrogens) in our environment, like chemicals in plastics, it is fascinating to watch science in progress — to see if they might in some way be involved with weight. DES VOICE

Focus On Environmental Contaminants to Improve Reproductive Health

By Pat Cody

"Environmental Challenges to Reproductive Health and Fertility" was the subject of a conference organized in late January by the University of California Medical Center and the Collaborative on Health and the Environment (CHE), of which DES Action is a member. Scientists in charge wanted to bring attention to a growing body of evidence that even low-level contaminants such as pesticides, herbicides, and chemicals in plastic containers can affect prenatal development.

Participants in the discussions pointed out that human male reproductive health in Scandinavian countries, where most of the research has been done, is deteriorating. They cited lower sperm count, abnormal sperm, and increases in the rates of undescended testes and hypospadias. They speculated that prenatal exposures to contaminants is probably the cause.

Other effects were reported by Dr. Laura Fenster of the California Department of Health Services. She said that exposures to workplace and environmental chemicals may cause preterm delivery, low birth weights and stillbirths. She told the conference that "Another study of births in upper Manhattan concluded that residential exposure to the insecticides chorpyrifos and diazinon reduced birth weight." Once the EPA banned these products for indoor use, birth weight and length improved immediately.

Patricia A. Hunt, a geneticist at Washington State University, told of her research with pregnant mice exposed to low doses of bisphenol A, an industrial compound (see VOICE Spring 2006 for a detailed description). Forty percent of the female fetuses of these mice were chromosomally abnormal. This means the grandchildren of the mother mouse exposed to BPA will be affected.

Another participant, Linda Birnbaum from the EPA, reminded colleagues that, "Animal studies give us much of our knowledge. When a particular environmental toxicant causes multiple effects in multiple species, people are highly likely to be susceptible."

The goal of the conference was to bring together medical, public health professionals, researchers and environmental activists to work out public policies for improving reproductive health and learn how research can influence clinical care. That is a goal that DES Action has pursued for many years! DES VOICE

Breast Cancer Treatments in the '70s & Early '80s May Lead to Greater Heart Disease Risk

"Long-Term Risk of Cardiovascular Disease in 10-Year Survivors of Breast Cancer," Maartje J. Hooning, et al., *Journal of the National Cancer Institute*, March 7, 2007.

Reviewed by Fran Howell

Radiation treatments for breast cancer in the 1970s and early '80s may put women at increased risk for congestive heart failure years later. This is an important finding for many DES Mothers who developed breast cancer as a result of their exposure and were treated for it during that time.

According to researcher Maartje J. Hooning, M.D., who is a medical oncologist at Erasmus Medical Center, Netherlands Cancer Institute in Amsterdam, women treated from 1970 thru 1986 with radiation therapy developed heart disease more often than those who underwent surgery alone and at higher rates than women who were never diagnosed with breast cancer.

Among the more than 4,400 women in the group who were 10-year survivors of breast cancer, 942 cases of cardiovascular disease were diagnosed, including congestive heart failure, heart attack and angina.

Hooning and her team calculate the increased risk for heart attacks in women treated with radiation in the 1970s as 2.55 times greater, with a 1.72 times increased risk for congestive heart failure, compared with untreated women. The risk was only slightly lower for those given breast radiation treatments between 1980-1986.

Adding chemotherapy to the protocol also increased the heart disease risk, and smokers were found to have an even greater chance of developing heart problems years after their breast cancer treatments.

Pointing out that the benefits of treatment outweigh the risks, Hooning urges breast cancer survivors from the '70s and '80s to reduce further risk factors, such as high blood pressure, high cholesterol levels and smoking.

It is important to note that women undergoing radiation for breast cancer today are considered at much lower risk for heart disease because treatment techniques have been significantly improved. DES VOICE

Study Shows Lower Sperm Count In Men With Mothers Who Frequently Ate Beef While Pregnant

Reviewed by Pat Cody

"Semen quality of fertile US males in relation to their mothers' beef consumption during pregnancy," Shanna Swan, et al., *Human Reproduction,* 2007.

Dr. Swan, whose work focuses on hormone exposure in utero, provides a thoughtful study of importance to every pregnant woman. She and her associates noted that while the FDA banned the use of DES for cattle in 1979, other hormones continue to be used as growth promoters. "Six hormones are now in common use in Canada and USA: the three natural steroids, estradiol, eestosterone and progesterone, and the three synthetic hormones, zeranol (an estrogen), trenbolone acetate (a steroid with androgen and glucocorticoid action) and melengestrol acetate (a potent progestin)....All six hormones can induce increased growth and development of the animal....At slaughter, not all steroids have been metabolized or excreted; measurable levels are, in fact, present in muscle, fat, liver, kidneys and other organs present in meat products....The possible effects on human populations exposed to residues of anabolic sex hormones through meat consumption have never, to our knowledge, been studied. Theoretically, the fetus and the prepubertal child are particularly sensitive to exposure to sex steroids."

Swan and her team identified 387 men from five cities around the country who agreed to participate, and whose mothers were willing to answer questions about their eating habits while pregnant with them.

What did they learn? According to Swan, "the number of beef meals consumed by the mother was significantly and inversely related to her son's sperm concentration." On average, the women ate more than four beef meals a week.

The sons of "high beef consumers" had a sperm concentration 24.3% lower than in men whose mothers ate less beef. They note that sperm concentration was not significantly related to mothers' consumption of other meat or to the man's consumption of any meat. Their conclusion: "These data suggest that maternal beef consumption, and possibly xenobiotics in beef, may alter a man's testicular development in utero and adversely affect his reproductive capacity." Even so, all of the male participants had fathered children.

Swan suspects that it isn't the beef itself, but rather the hormones or residuals of other persistent pollutants remaining in the meat after slaughter that are suspect – but this remains to be proven. There is a way to further test the theory. Swan would like to see this study repeated with European men born after 1988, when growth-stimulating drugs were banned from cattle raised there.

In an accompanying editorial "Could Hormone Residues be Involved?," Frederick S. vom Saal writes that "during the years this cohort of fertile men were in utero (median year of birth was 1970), beef cattle in North America (where the majority of study participants were born) were routinely treated with the growth-promoting anabolic steroids. For example, the drug diethylstilbestrol (DES) was widely administered to beef cattle in the USA between 1954 and 1979….There has been a trade dispute over the safety of hormone residues in beef going on for many years, with the European Union opposing importing hormone-treated beef from the USA and Canada." Dr. vom Saal reminds readers that "Women would also be expected to be affected by developmental exposure to xenobiotic hormones; studies relating maternal beef consumption to daughters' incidence of PCOS, age at adrenach/menarche and post-natal growth rate would be predicted to show a significant relationship." DES VOICE

What Next? Be Careful

Editorial Comment from Pat Cody

By now the message is beyond argument: hormone replacement treatment (HRT) can be dangerous. And a further lesson is that we also need to be careful about "alternatives" that some companies are promoting.

One example is Estroven, made by Amerifit Brands, whose ad offers this "natural" support during menopause. The ad states that, "Estroven has no known side effects."

Since no scientific studies have been reported on either short or long term effects of Estroven, side effects cannot be known. Further, an important ingredient in Estroven is black cohosh.

As long ago as October 2003, a case of hepatitis directly linked to black cohosh was reported at the 68th Annual Scientific Meeting of the American College of Gastroenterology. Stanley M. Cohen, M.D., of the University of Chicago, in discussing this case, told WebMD Medical News that, "Black cohosh has the potential for serious side effects." Dr. Cohen noted that the use of black cohosh has increased since news on the link between HRT and breast cancer was reported.

More recently, Cancer Research UK writes that, "In July 2006 the European Medicines Agency said that the labeling of black cohosh would have to carry warnings about liver damage….As well as the risk of liver damage, there are other side effects. Black cohosh may make you feel or be sick. And large doses may cause dizziness, slow heart rate, headaches, joint pain and uterine contractions."

You can read more by GoodSearching black cohosh for the details of this and other articles. DES VOICE

Breast Screening Guidelines — No Easy Answers

By Nora Cody

"Screening Mammography in Women 40-49 Years of Age: A Systematic Review for the American College of Physicians," Armstrong, et al., Annals of the American College of Physicians, 2007:146.

New guidelines from the American College of Physicians (ACP) state that women of 40-49 years do not need annual mammograms. They believe that the risks: false positives, unnecessary biopsies, exposure to radiation – outweigh the benefits, since very few women in that age range have risks for breast cancer. The authors make exception for women at risk, such as family history, gene mutation, earlier radiation treatment to the chest - but, significantly for us, do not mention DES exposure (see VOICE Fall 2006).

The American Cancer Society (ACS), which just released its own guidelines, sharply questioned the ACP statement, saying that mammograms help find tumors at their earliest and most treatable stage. For the first time, the ACS has added to its recommendations that *high-risk* women have annual Magnetic Resonance Imaging (MRI) of their breasts.

But a swirl of controversy surrounds that new recommendation – and there are no easy answers for DES Daughters. We know that DES Daughters who are older than age 40 are at increased breast cancer risk. But is that risk considered high enough to put them into the risk category recommended for annual MRIs? That question is still being discussed. At this point the best advice is to talk with your doctor. We hope to provide more information on this in the next *Voice* newsletter. DES VOICE

Fertility Treatments May Affect Sperm Count of Offspring

"Fertility Treatment and Reproductive Health of Male Offspring: A Study of 1,925 Young Men from the General Population," Jensen, et al., *American Journal of Epidemiology*, Vol. 165, No. 5, 2007.

Reviewed by Pat Cody

Here is another study linking male fertility to in utero exposure, in a setting that has not been studied before. Dr. Jensen and colleagues looked at the reproductive health of young Danish men whose mothers had received fertility treatment — usually hormonal.

Out of a group of 1,925 volunteers, 47 had mothers who reported receiving such treatments. Those men had a 45% lower sperm count, fewer active normal sperm, smaller testes, and lower testosterone levels. The authors conclude that these findings raise concern about possible late effects of fertility treatment and that larger scale studies of children born after fertility treatment should be performed. DES VOICE

HOW YOU CAN HELP US HELP YOU

Doctor Referrals

Did you know we get more requests for doctor referrals here at DES Action than any other request? Hundreds of them!

But sadly, for many parts of the country we have no names to give out. There must be many more doctors (and other health care providers) out there but they aren't listed.

If you met a DES-exposed individual on the street and the topic came up, would you recommend your doctor? If so, please let us know.

All we need is a name and contact information. Email **desaction@columbus.rr.com** or call **800-337-9288**. Don't keep it to yourself. Feel good about helping someone today!

Your Voice

Do you have a story to tell? The **Your Voice** column in our *Voice* newsletter gives members an opportunity to write about how they are living good lives in spite of, and with, DES exposure.

Now it's your turn! Your Voice feature editor Ann Giblin is collecting stories about how DES exposure was the spark that ignited your active participation in an activity or career, "for the common good." It would be great to hear Your Voice telling that story. Please contact Ann at **ann@WinterlakeAssoc.com** with your ideas. Provide just a sentence or two—or even better (!)—your 500 to 1,000 word article as soon as possible.

GoodSearch

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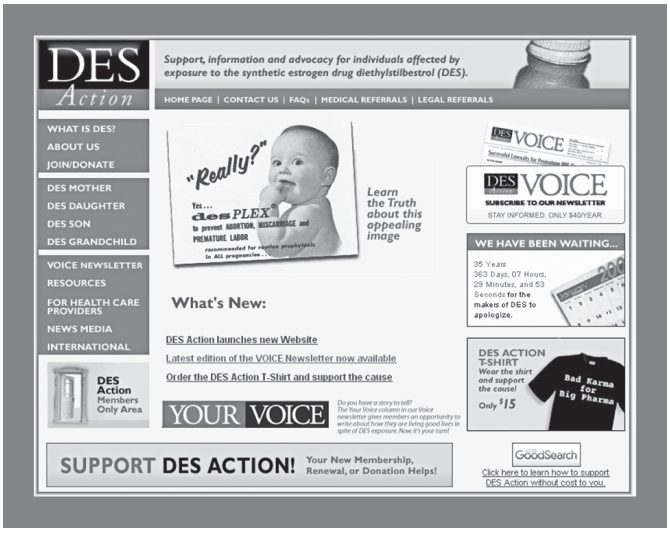

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DES Screenplay Accepted by Hamptons International Film Festival

By Fran Howell

DES Daughter Caitlin McCarthy dreams big. After learning of her DES exposure in 2005, she researched and wrote a scientific drama about the origins of DES and how it came to be the world's first major drug disaster.

Wonder Drug moved a step closer to reality when it was selected for the Hamptons International Film Festival's 7th Annual Screenwriters' Lab (www.hamptonsfilmfest.org) co-sponsored by the prestigious Alfred P. Sloan Foundation.

Held in April, the Lab paired emerging screenwriters with established writers who conducted one-on-one mentoring sessions. McCarthy worked on her script with Tom Gilroy (*Spring Forward*, *Location*) and Joshua Marston (*Bus To Queens*, *Maria Full of Grace*).

McCarthy is now fine-tuning *Wonder Drug*, using suggestions from her mentors. "This is all so incredibly exciting and I learned so much," she says.

But there is more for McCarthy than simply learning scriptwriting techniques. Throughout the year she will be assisted in making contacts with various friends of the acclaimed Hamptons International Film Festival, who include industry producers, agents, and movie development executives. The goal is to help McCarthy get her movie made by creating a networking support structure that taps into the professional film industry. These are invaluable contacts for an up and coming screenwriter.

"I have very good feelings about this. I want everyone in the DES community to know I wrote *Wonder Drug* for all of us. Won't it be terrific to have our issues taken seriously?"

McCarthy says she used great care in writing *Wonder Drug*, and Sir Ralph Dodds, son of DES creator Sir Charles Dodds, served as script consultant. Her intent is to raise public awareness of DES with a movie shown in theaters around the country — and around the world. If anyone can do it, McCarthy can! We congratulate her for being accepted into the Hamptons Screenwriter's Lab and look forward to seeing *Wonder Drug* some time soon.