DES Action® VOICE

We identify, educate, empower and advocate for DES-exposed individuals

Inside

A Focus on Diethylstilbestrol

SPRING 2019 #160

New Study on Estrogen Metabolism Offers Clues to DES Role in Cancer

Even though the increased risk of breast and vaginal/cervical cancer in DES Daughters has been long established, researchers continue to explore precisely how prenatal exposure to DES can cause cancer.

Researchers already believed it possible that the slightly increased risk of breast cancer might be connected to higher levels of infertility among DES Daughters. In general, risk of breast cancer decreases the more time a woman spends during pregnancy and breastfeeding. Therefore, higher infertility rates and subsequently lower rates of pregnancy and breastfeeding might be at least part of the reason breast cancer risk is higher in DES Daughters, as a collateral or confounding risk. But this is only one possibility, and researchers have suspected other factors are likely involved as well.

Possible Factor Found

New preliminary findings suggest another possible factor after researchers compared estrogen metabolism in a small group of DES Daughters and unexposed women. It appears that exposure to DES before birth affects how estrogen is metabolized in the body, at least based on observations in women after menopause. These differences are ones that already had been previously connected to higher cancer risk in other research.

> In general, DES Daughters had higher levels of overall estrogens, estradiol and estrone than unexposed women.

The study also found that DES Daughters appear to have slightly higher levels of estrogen concentrations, including estradiol (the most potent of the body's estrogens), than unexposed women.

Measuring Estrogen

The study, led by National Institutes of Health researchers Rebecca Troisi, ScD, and Robert Hoover, PhD, was published in October 2018 in the journal *Cancer Epidemiology, Biomarkers and Prevention.*

The researchers took blood samples from 60 postmenopausal women, 40 of whom were DES Daughters and 20 of whom were not exposed prenatally to DES. All of the women were white, and their average age was 61 years.

The women were all participating in the NCI's Combined DES Cohort Study and had never used any hormone supplements/medications or had a cancer diagnosis.

Among the exposed women, 36 had a history of vaginal epithelial changes, tissue changes in the inner lining of the vagina that have previously been associated with high doses of DES exposure during gestation.

The researchers measured the participating women's blood levels of the major estrogens estradiol and

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Did DES Cause Thyroid Problems?

The thyroid is one of the most powerful organs in the endocrine system—and one of the peskiest ones in terms of causing problems in women throughout their lifetimes. It's therefore very reasonable to wonder if the effects of prenatal exposure to DES—an unfortunately powerful synthetic hormone in its own right—might increase the likelihood of thyroid issues.

Research in this area is frustratingly thin, partly because it's so difficult to study the interactions of hormones across a lifetime, given all the other chemical exposures people have from their environment during everyday life. It's also challenging because of physiological differences in thyroid functioning that already naturally occur across different people.

Still, it's an area scientists

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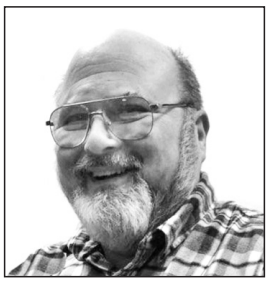
DES Action Launches Men's Discussion/Support Group



Dan Rosenfield

I am Dan W. Rosenfield, and I am the volunteer moderator of the new DES Action Men's Support Group. I am a 71-year-old DES victim, as verified multiple times by my mother, who carried the burden of guilt over my exposure to that concoction over the many years of her and my post-natal life.

My first tangible experience with the effects of DES occurred when I was 16, when one of the hallmarks of prenatal DES exposure, an epididymal cyst, was discovered and treated (needle aspiration). It was removed surgically when I was 17.

Now, in retirement from technical education and technology support services, as I examine my early life, I am beginning to understand many "not quite right" things that went on in my body and mind as I grew up. And I am beginning to better comprehend and understand what's going on inside of my body and mind because I joined DES Action USA.

Sharing Our Experiences Privately

Over the years, and continuing today, a variety of symptoms have appeared that, only within the past year or so, appear almost certainly to be linked to my prenatal exposure to DES. I will tell you my experiences with them as we share our stories and advice in the private group. I, as your moderator, do not want to influence your identification of your own symptoms and feelings about them. We encourage you to communicate, candidly, information about your DES-triggered symptoms, from those you consider to be highly "intimate" to those that are just annoying or so subtle as to be just a vague feeling.

This online discussion group is open to any DES-exposed men (Sons and Grandsons) who are current DES Action USA members. It is a private discussion/support group, and the information you discuss will not be shared beyond the group.

If you know a DES Son or Grandson who might like to join the free men's group, have him email karen@desaction.org. She will complete the membership for you.

DES VOICE

Renew Your Membership

It's easier than ever to renew your membership. Just log into the site using the email you registered with and your password. If you don't remember your password, you can reset it.

If you no longer use the email you signed up with, email our Community Manager, Karen Calechman, at karen@desaction.org. She will set a temporary password for you.

Thank you for supporting DES Action USA with your membership.

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The mission of DES Action USA is to identify, educate, empower and advocate for DES-exposed individuals.

Contacts

Sister Organizations

Australia

DES Action Australia, Inc.
PO Box 282
Camberwell 3124 Vic. Australia
info@desaction.org.au
www.desaction.org.au

DES Action NSW
14 Edmundson Close
Thornleigh NSW, 2120, Australia
C_devine@bigpond.net.au
www.desnsw.blogspot.com

France

Reseau DES France
1052 rue de la Ferme de Carboue
40000 Mont de Marsan, France
reseaudesfrance@wanadoo.fr
www.des-france.org/accueil/index.php

The Netherlands

DES Centrum
Postbox 1173
3860 BD Nijkerk
voorlichting@descentrum.nl
www.descentrum.nl

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178 Columbus Avenue #237182
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(800) DES-9288
(800) 337-9288
Email: info@desaction.org

Editorial Director and Social Media Manager..... Tara Haelle

Community Manager..... Karen Calechman

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President .....Suzanne B. Robotti

Digital Director .....Deirdre Wyeth

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New Study Explores Risk of Male Birth Defect from Endocrine Disruptors

Scientists have been studying how prenatal exposure to endocrine-disrupting chemicals such as DES might contribute to male birth defects, but a recent review of the evidence shows how much more work needs to be done. The review paper, conducted by researchers at Dartmouth's Geisel School of Medicine, was published in the journal *Current Environmental Health Reports* in December 2018. (The authors had no links to pharmaceutical or chemical companies.)

The review paper focused on a specific male birth defect called hypospadias, in which the urethra (the opening for urine and semen) does not sit at the tip of the penis but instead somewhere else on the head or anywhere along the penile shaft. It's one of the most common birth defects, affecting an estimated one out of 200-300 newborn boys, and can usually be corrected with surgery.

Almost a third of hypospadias cases have known genetic mutation causes, but about 70% of cases don't have an easily identifiable cause. DES is one of several chemicals already linked to hypospadias in past research. Several studies, including ones from the US, France and the Netherlands, have found a substantially higher risk of hypospadias in DES Sons, and the French study found a higher risk in DES Grandsons as well.

Given how male sex organs develop, it makes sense that DES and other endocrine-disrupting chemicals could interfere with healthy penis development. For the first nine weeks of pregnancy, no difference in male and female genitals can be seen in fetal development. Then boys' sex organs form from eight-14 weeks gestation, driven largely by the hormone androgen, and are fully formed by 16 weeks.

Since researchers have already found that mutations in genes related to androgen play a role in hypospadias, they have explored whether exposure to other compounds during that eight-16-week period might also play a role.

A Meta-Analysis of Studies

In the new paper, the researchers looked through several medical research databases for all studies related to hypospadias and environmental exposure to endocrine-disrupting chemicals. They found 37 studies, excluding animal studies and those related to occupational exposures.

The studies included in the review found an increased risk of hypospadias associated with prenatal exposure to proton pump inhibitors, steroids, paroxetine, valproic acid and progestin when used for fertility, though not many studies on these existed overall.

Meanwhile, prenatal exposure to corticosteroids, ondansetron, loratadine, antihistamines and tricyclic antidepressants showed no statistically significant increased risk. Among selective serotonin reuptake inhibitors, another common group of antidepressants, an increased risk was seen only with paroxetine (brand name Paxil), but it was based on only nine cases, so the researchers noted the need for more research into antidepressants.

Though a slightly increased risk showed up with venlafaxine (brand name Effexor), it was borderline statistically significant. In other words, it was on the edge of possibly being coincidence instead of a real link.

Three studies found no link to use of hormonal contraceptives (including those with progestin), but one study did find an increased risk. Similarly, two studies found an increased risk with clomiphene, and one found no increased risk.

The amount of increased risk for these drugs ranged from two to 11 times higher odds. If one in 250 newborns is typically born with hypospadias, these increased odds would mean two to 11 newborns exposed to one of these drugs out of 250 would have hypospadias.

The amount of risk varied by drug, and there was not enough research to nail down specific, reliable risks for each one. In fact, not enough research existed to be sure of any link to hypospadias for these drugs—more needs to be done, unfortunately.

Studies also looked for possible risks from phthalates (sometimes used in personal care products and other household products), pesticides and other pollutants. One small study from Korea found a weak link between hypospadias and children's urine levels of two different phthalates, but phthalates concentrations vary in children over time and don't necessarily reflect exposure from mothers during pregnancy. In fact, no increased risk of hypospadias was seen corresponding with phthalates levels in the children's mothers' urine in that study.

A small increased risk (less than one extra case per 500 newborns) was seen with two pollutants, hexachlorobenzene and p,p′-DDE.

In some cases, the results were contradictory. For example, medium and low exposure levels to the pesticide atrazine were linked to a slightly higher risk of hypospadias, but exposure to high levels of atrazine actually decreased risk of hypospadias. And another atrazine study found no increased risk at all.

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New Study on Estrogen and Metabolism

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estrone. During metabolism of estradiol and estrone, changes occur at different positions on the molecules that are labeled pathways 2, 4 and 16, resulting in estrogen metabolites.

"Recent observations in humans show that breast cancer risk is related to specific metabolic pathways, with greater metabolism in the 2 pathway showing protection" from breast cancer, Dr. Troisi and her colleagues wrote. So they wanted to better understand how estrogen is metabolized in those exposed to DES versus those unexposed and whether the pathways differed between the two groups.

The researchers focused on these pathways and on total estrogen levels in the women. They also measured ratios of pathways, such as how much activity occurs along pathway 2 relative to pathway 16, written as path 2:path 16.

In general, DES Daughters had higher levels of overall estrogens and of estradiol and estrone than unexposed women.

Other Differences As Well

The authors identified other differences between the groups too: Less metabolic activity occurred in DES Daughters along pathway 2 relative to total estrogens, parent estrogens and pathway 16.

In other words, a lower proportion of metabolism occurred in DES-exposed women along pathway 2 than in exposed women. Pathway 2 is the one associated with lower cancer risk.

At the same time, in DES Daughters, pathway 16 accounted for a higher proportion of metabolic activity relative to total metabolites measured in the women.

When comparing exposed versus unexposed women, the researchers made adjustments to their calculations to take into account existing differences among the women in terms of age, years passed since menopause, body mass index (BMI), number of children and recent alcohol use. These adjustments did not change the findings.

It's worth noting that the DES Daughters involved in this small study were exposed to high doses of DES in the womb, so the findings in this study may not apply evenly across all women with prenatal exposure to DES.

Still, the pattern of relatively lower levels of pathway 2 metabolites compared to higher pathway 16 levels "has been associated with an increased risk of breast cancer in postmenopausal women."

It will require a larger study to understand how big the effect is of these estrogen metabolism differences.

"This area of research has great potential to improve our understanding of the biological mechanisms associated with endocrine disruption [interference with the body's hormone activity] in humans during the prenatal developmental period," Dr. Troisi and her colleagues concluded.

DOI: 10.1158/1055-9965.EPI-18-0135

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have been interested in exploring, relying on long-term observational studies of people and on experimental studies involving animals.

Looking at Endocrine Disruptors

Much of the research in this area has focused not on DES in particular but on the possible effects on fetal development of endocrine-disrupting chemicals in general.

For example, a 2012 study in the journal *Molecular and Cellular Endocrinology* noted that "there is now reasonably firm evidence that PCBs have thyroid-disrupting effects, and there is emerging evidence that also phthalates, bisphenol A, brominated flame retardants and perfluorinated chemicals may have thyroid disrupting properties" (doi: 10.1016/j.mce.2011.09.005).

The dosage of exposure plays an important role in what effects might occur and how strong they are, but DES is a very potent estrogen on its own. If these other endocrine-disrupting chemicals are likely to have a disruptive effect on the thyroid, it's likely that DES could too.

Other studies focusing specifically on DES tend to involve lab animals, especially zebrafish and rats. These studies have not been conclusive, however. Most appear to show at least some effect from DES exposure on thyroid function.

In some cases, the effect appears lasting, such as in one zebrafish study. Another study in rats found increased thyroid function as a result of prenatal DES exposure. In yet another, however, the thyroid appeared able to recover from the effects and still resume normal function.

And none of these studies can tell us exactly what prenatal DES exposure does to the thyroid in humans. In humans, the only option is observational studies.

In a recent DES Granddaughters study covered in a previous VOICE issue, a higher rate of thyroid issues in general were reported by DES Daughters, but the finding did not reach statistical significance. That is, the researchers could not rule out the finding as coincidental or chance.

The Centers for Disease Control and Prevention and the National Cancer Institute do not list thyroid problems as one of the known major effects of prenatal DES exposure. However, there is enough evidence in animal research and on studies into endocrine-dis-

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Researchers Need to Dig Deeper for Multi-Generational Effects

In the early 1970s, when the devastating damages of DES were first being discovered in the Daughters, very few scientists would have been thinking about possible damage to future generations. But as the damage DES caused continues into the third generation, scientists are wondering how DES was passed into these grandchildren, and how many more generations will be affected.

In a recent letter to the journal *Environmental and Molecular Mutagenesis*, DES Action Executive Director Su Robotti teamed up with independent research funder and the founder of Escher Fund for Autism, Jill Escher, to urge researchers to dig deeply into the "germline" theory, which would explain, and perhaps predict, how exposures to women in pregnancy can affect their grandchildren and on.

Su Robotti

Jill Escher

Cells called a "germline" create eggs in the female fetus (all the eggs a woman has are produced when she is in utero). That germline creates a chain between generations and is embedded even into the following generation. If the pregnant woman is exposed to a chemical, like DES, while those eggs are being developed in the female fetus, and if it interrupts the germline or damages it, the fetus being carried is affected, as is the generation following her.

Emerging evidence, Escher writes, suggests that some conditions that scientists are still trying to understand might have origins in germline mutations—because of medicines given to the grandmother. It's possible DES could be a contributor to these mutations, and only additional intensive research into this question can help scientists learn more about the possible connection.

"The DES disaster presents a paradigmatic question of human germline toxicity, and a unique opportunity to better understand generational impacts of this drug, and also the broader phenomenon of hormone disruption in humans," Escher and Robotti wrote. But even with multiple conditions now linking DES exposure to the third generation, most research is simply trying to match up exposure with conditions without digging deeper into how those intergenerational effects come about.

"For example, the issue of neurodevelopmental outcomes and socio-sexual behavior strikes us as very important and mostly unexplored," Escher and Robotti wrote. "Research must think more broadly about [third generation] pathologies precipitated by DES exposure to also encompass the brain, cognitive ability, behavior, sexuality and other crucial endpoints beyond the standard [paradigm of exploring negative effects on the fetus]."

The letter hopes to inspire more scientists to initiate research into these areas and possibly unlock better understanding to other ways exposures during pregnancy can affect later generations. [DOI: 10.1002/em.22288] DES VOICE

Did DES Cause Thyroid Problems?

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rupting chemicals in general to say it's possible DES exposure could affect thyroid function in DES Daughters, or possibly Sons.

More Research Needed

The problem is that too little research exists to confirm that link or to learn more about what it looks like, including the most likely types of problems people might experience.

What does this mean for the DES community? If you're experiencing unexplained fatigue, weakness, intolerance to cold, muscle aches and cramps, constipation, weight gain or difficulty losing weight, poor appetite or a goiter (enlarged thyroid gland), ask your doctor to check your thyroid. If you've been diagnosed with thyroid problems, be sure to tell your family doctor and your endocrinologist (or any other specialists you have) about your DES exposure. It may not change your diagnosis or treatment, but it might give your physicians a little more insight as they try to manage your condition. DES VOICE

Q&A: Carol Devine of DES Action NSW

Since the effects of DES extend around the world, the DES VOICE spoke with DES Daughter Carol Devine, coordinator of DES Action NSW in New South Wales in Australia. Based in Sydney since 1995, the volunteer-driven DES Action NSW receives no government funding and is independent from the Melbourne-based DES Action Australia (part of Tall Girls Inc.). Australian DES Daughters are also welcome to join DES Action USA to keep up with current DES research and connect regularly with their US counterparts.

Carol Devine

Q: Tell us a bit about the DES Daughter experience in Australia.

I fear the majority of DES Daughters in Australia are oblivious to having been DES-exposed and have been unknowingly let down and cheated of their rightful preventive health care. And shockingly, the letdown would be the same for DES Daughters with known exposure. Overall, I'd say the experience for DES Daughters here is isolating, like being kept in the dark. Quite commonly I hear a throwaway remark by doctors, "We don't see many of you anymore." Indeed, such a remark is fraught with questions.

In Australia it is difficult to get an accurate figure on how many women were likely exposed because the specific data on DES-associated cancer (CCA) is flawed by design and mismanagement. The reporting of specifically DES-associated CCA to our "drug watchdog," the Therapeutic Goods Administration (TGA) (equivalent to the US FDA) is not mandatory [the Australian government funds TGA by charging fees to the pharmaceutical industry].

For example, the very first reported case of DES-suspected CCA, a four-year-old girl, was reported in 1972 but not entered into data until 1982. The TGA's figures are static from 1983 to 2001, because 1983 was deemed "the end of reporting." An unnamed doctor apparently phoned the TGA in the early 1980s to advise that reporting is not necessary because clinics have been set up (allegedly to collect CCA case data), which is not true.

Dr. Jules Black, the retired media medical spokesperson for DES Action NSW, reported his DES-exposed patients to the TGA during this period, but later found his reports had disappeared and were never entered into TGA data. TGA Committee Meeting Minutes regarding DES in the 1970s revealed several CCA cases that had gone unreported to the TGA, and at least five children at one children's hospital with likely DES exposure were not investigated.

The Royal Australian and New Zealand College of Obstetricians and Gynecologists (RANZCOG, equivalent to ACOG in the US) estimates Australia has 10,000 DES Daughters, yet, separately, a 2004 medical journal letter estimates Australia has 15,000 DES Mothers in Australia, based on rates of CCA. [Other even less plausible numbers have also been proposed, Carol explained, so it's clear that there is dispute about the "official" numbers.] But based on Australian cancer registry data before 1982 up to 2007, I would estimate 145,000 DES Daughters live in Australia.

Q: How has DES exposure impacted your life over time?

Learning of my DES exposure at age 16 was by chance, when my mother read an article about DES in *Reader's Digest*. The guilt she felt was beyond words. I had vaginal adenosis and, in my childbearing years, four miscarriages, a T-shaped uterus and two premature deliveries (both alive and well today). The latter pregnancy also required cervical stitching, bed rest and medication. Accepting medication was the toughest thing for me. In day-to-day life, my DES exposure feels like a dark cloud over me, with intensified blackening around the time of DES examinations. I should imagine that feeling would ring true with other DES Daughters.

Q: What have been the biggest challenges with DES awareness and advocacy in Australia?

The notion that a publicity campaign about DES would create community anxiety without tangible benefit has been the prevailing catchcry by successive government politicians. TGA expressed a similar sentiment of "not causing alarm" in the 1970s. During the planning stage of the US CDC DES Education Campaign in 2002, we lobbied the Federal Health Minister to consider the CDC's Strategic Plan Draft, and he passed this matter to the TGA. Needless to say, the TGA rejected the US recommendations.

We then organized DES Awareness Weeks to gain media attention. Hundreds of people contacted us, many having just learned of their DES exposure and relaying tragic DES stories. At the Australian Hu-

man Rights Commission in 2011, the Commission agreed that Australians had a fundamental right to be made aware of the possibility of having been DES-exposed, but the Commission had no resources to advocate on our behalf. A 2012 liaison with the Queensland State Senator's Office was similarly ignored.

At the grassroots level, one tragic story epitomizes the long-standing suffering of not knowing about DES and the long-standing pain of guilt due to DES: Two sisters, both of whom suffered classic signs of DES-exposure during their lives, learnt of their exposure for the first time when their mother whispered this on her deathbed.

Q: What problems have you encountered from health agencies in Australia regarding DES awareness?

In 2004 the TGA issued an erroneous alert to remind doctors that DES Daughters should follow the current national cervical screening recommendations (Pap smears every two years) and that all cancers should have been detected by now. This meant a four-year lobbying struggle with politicians and medical authorities, including RANZCOG, to have this misinformation corrected. The rectification was only made possible in 2008 with legal representation, on our behalf, communicating with the TGA.

Since 2014, RANZCOG and Cancer Australia have shared misinformation stating that DES Daughters and DES Mothers should follow the national breast screening recommendation (mammograms every two years). We've so far been told that there are "external influences at play" which change policy and that therefore, under Australian guidelines, DES exposure does not qualify for recommending annual screening.

To me, it's obvious the misdeeds by Australia in managing the DES-exposure problem and the constant misinformation issued to the Australian public on crucial DES exposure health matters are way too consistent to be simply an accident. Frankly, it all smacks of cover-up, a full-on effort to exterminate the DES exposure problem. This then begs the question: Is Australia in fact providing a DES exposure management model conducive to the interests of the pharmaceutical industry which could also benefit other countries subservient to the industry? If so, then shame.

Q: What can the DES-exposed community in the US do to support our Australian counterparts?

DES Action USA is basically our lifeline for information. By continuing membership with DES Action, you indirectly help us here in Australia. With any media reports about DES in Australia, I would welcome DES-exposed women in the US to chime in when appropriate and if media dialogue allows this. Lastly, I ask DES-exposed women in the US: Please don't forget us Down Under!

Q: What is your hope for the future as a DES Daughter?

My hope is for a miraculous turn around—for the Australian government to directly and diligently consult with US experts in the field of DES exposure, as well as engage with DES Action USA and its Australian counterparts, in a way that is both transparent and accountable to the Australian public. DES VOICE

Risk of Male Birth Defect from Endocrine Disruptors

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Interestingly, multiple studies on exposure to DDT, a pesticide now banned in the US, did not find an increased risk, but one study found a slightly smaller risk from insect repellent.

What does all this mean?

Frustratingly, it means we need a lot more research into this area to really understand what kinds of environmental chemicals might really increase risk of hypospadias when the fetus is exposed before birth.

"Among the pharmaceutical agents, sex steroids, DES and progestin fertility treatments have all been associated with hypospadias risk," the researchers concluded. "However, results are not entirely consistent, and in some studies, associations were confined to subgroups," such as an increased risk of only one type of hypospadias (based on where the urethra occurred).

Steroids and valproic acid (an anti-seizure medication used for epilepsy and some psychiatric conditions, such as bipolar disorder) showed some possible risk, but the link was weak. Meanwhile, several common medications haven't been studied at all for hypospadias risk, including beta blockers, antibiotics and diabetes medications.

The authors noted how difficult it is to study risks of these environmental agents, especially since exposure amounts, absorption amounts and the timing of exposure (such specific weeks of gestation) vary across different people and situations.

Plus, most of these chemicals are usually mixed with others, making it hard to separate which substance might be involved or whether it's a specific combination of chemicals that makes a difference.

In addition, most of the studies included small groups of participants, making it difficult to understand risks at the population level. The studies mainly relied on medical records or patient surveys, so the researchers could not measure exact amounts of specific substances.

Regardless, it's clear that the most sensitive time for exposure for this birth defect is during eight-16 weeks of pregnancy, and there is enough research to establish a strong link to DES. DES VOICE

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Connecticut Chapter Archives Move to Smith College

In the early decades of DES Action, the state-level organizations were the driving force. The energy of these groups was extraordinary as they got funding for educational programs and public service campaigns, lobbied for DES Day in various states, supported those who were damaged by DES, and created bonds with their fellow volunteers that lasted a lifetime.

The Connecticut chapter of DES Action was one of the more active groups. The three founders, Sally Esposito, Debra Hyman and Caren Glickson, came together over the past year to curate, organize and donate the historical files of DES Action Connecticut. There was a previous chapter in Connecticut, but the name of the woman who ran the first group has been lost to time. If you know who that person was, please let us know.

The Sophia Smith Archives at Smith College gratefully accepted these documents and honored their work, and the work of all the volunteers in the history of the Connecticut chapter, at a recent lunch at Smith College.

Top left to right: *Kathleen Nutter (Accessions Archivist, Smith College), Carrie Baker (faculty in the Study of Women and Gender at Smith), Beth Myers (Director of Special Collections, Smith College), Debra Hyman (DES Action, CT), Student, Sally Esposito (DES Action, CT), Susan Bell (Author, DES Daughters: Embodied Knowledge and the Transformation of Women's Health Politics, Department Head and Professor of Sociology, Department of Sociology, Drexel University), Caren Glickson (DES Action, CT), Suzanne Robotti (Executive Director, DES Action, USA), Mary Ann Moran (Sally's partner).*
Bottom row left to right: *Jacqueline Luce, PhD (Lecturer in Gender Studies, Mount Holyoke College, Principal Investigator: Gender/Sex/Sexuality and DES Exposure), a student, a student, Maureen Callahan (Sophia Smith Archivist, Smith College).*