

Environmental Fact Sheet



Birth Defect Research for Children

DES

What is DES?

Diethylstilbestrol (DES) is a synthetic (man-made) hormone. It is the most potent estrogenic compound known – more powerful than natural estrogen. It is one of the general classes of synthetic chemicals called “endocrine disrupting chemicals (EDCs)” that compete with natural hormones for receptor slots within the body. Studies have shown that the timing of exposure to hormones, and their mimics, may be as important as the dosage level.



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What is it used for?

DES was widely prescribed to approximately 5 million American women from 1938 to 1971 primarily for the prevention of miscarriages. DES was also prescribed for symptoms associated with menopause, menstrual disorders, postpartum breast engorgement, primary ovarian failure, “morning after” contraception, and chemotherapy of advanced breast cancer and advanced prostate cancer. More commonly, until 1980, DES was used in the general food supply with pellets implanted in beef, swine, and poultry to promote growth.

Adverse Human Health Effects:

The development and functioning of our bodies are governed by the endocrine system. The endocrine system produces hormones, or chemical messengers, that move through the bloodstream, carrying signals that not only govern sex and reproduction, but also coordinate organs and tissues that work in concert to keep the body functioning properly. DES has a great affinity for estrogen receptors and displaces natural estrogen from its own receptor sites.

Reproductive Effects:

Studies conducted since the 1940s have shown that DES damages the reproductive systems of those exposed in the womb, and increases the risk for cancer, infertility, and a wide range of serious reproductive tract disorders. The disorders for DES daughters include increased risk of pregnancy outside the womb, clear cell cancer of the vagina or cervix, miscarriage and pre-term labor.

Studies also indicate that exposure to DES may increase risk autoimmune disorders and diseases (Vingerhoets AJ, et al: Eur J Obstet Gynecol Reprod Biol 1998 Apr;77(2):205-9

Severity of reproductive abnormalities caused by prenatal exposure appears to vary dependent upon the time and duration of exposure during the first half of pregnancy. Development of reproductive structures such as the upper vagina, cervix, uterus, and fallopian tubes have common embryological (Mullerian) origin that undergoes maximal development during the third and fourth months of pregnancy, and are dependent upon maternal estrogen for their development. Recent research has demonstrated that prenatal DES exposure at this critical time of Mullerian development may permanently alter the structure and function of estrogen receptors rendering them permanently incapable of responding appropriately to natural estrogen in later life. This would explain why some DES daughters do not respond to estrogen therapy for uterine and cervix lining disorders. While DES exposure outside the Mullerian window diminishes reproductive effects, the possibility of developmental impacts to other body systems could be caused by exposure at other formative times during pregnancy.

Much less research has been done on the effect of DES on sons. One study reported that 15 percent of the men whose mothers had taken DES during pregnancy reported genital malformations at three times the rate of unexposed men. The malformations include non-cancerous cysts and varicose veins of the penis, small and undescended testicles, abnormally small penis, and abnormal opening of the penis.

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Although lower sperm counts and more abnormal sperm were also reported, evidence is inconclusive that DES impairs male fertility. More data are needed.

Carcinogenic potential:

The cancer causing ability of DES appears to be its endocrine disrupting effect and its long half-life in the body. Women given DES during pregnancy have an increased risk of breast cancer. DES daughters have increased risk of clear cell cancer of the vagina or cervix.

Fact Sheet by:

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