

# Fetal exposure to radiation increases risk of testicular cancer

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Male fetuses of mothers that are exposed to radiation during early pregnancy may have an increased chance of developing testicular cancer, according to a study in mice at The University of Texas MD Anderson Cancer Center. The article was published today in *PLoS ONE*.

The study is the first to find an environmental cause for testicular germ cell tumors, the most common cancer in young [Caucasian men](#).

"This discovery launches a major shift in the current research model, placing DNA-damaging agents in the forefront as likely mediators of testicular cancer induction," said corresponding author Gunapala Shetty, Ph.D., assistant professor in MD Anderson's Department of Experimental Radiation Oncology.

## Increasing incidence, few answers

According to the [American Cancer Society](#), more than 8,500 new cases of testicular cancer are diagnosed each year in the United States. During the past 50 years, the incidence has tripled in young Caucasian men throughout the world.

"This increase and the characteristics of [germ cell tumors](#) strongly suggest that [fetal exposure](#) to an environmental agent is responsible," Shetty said. "However, the identification of any agent producing increases in testicular cancer has eluded scientists."

Endocrine disruptors, chemicals that alter the endocrine – or hormonal – system, have been widely suggested as the cause of testicular cancer, but there has been no proof. Fetuses are especially vulnerable to even small amounts of the substances, which are known to cause developmental and cognitive issues.

## **Radiation induces cancer, 2 endocrine disrupters don't**

This study began as an examination of endocrine disruptors as a possible cause of testicular cancer. Researchers separately tested two such substances, the estrogen diethylstilbestrol (DES) and the antiandrogen flutamide.

The endocrine disruptors were introduced into a mouse strain with a high spontaneous incidence of testicular cancer, which should make them more sensitive to cancer caused by environmental agents. But the results showed no increase in testicular cancer.

However, when researchers gave modest doses of radiation, which is a DNA-damaging agent, to female mice in the middle of their pregnancies, all the male offspring developed testicular cancer, compared to 45 percent of mice not exposed to radiation. In addition, the tumors were more aggressive and had more sites of origin.

### **Next steps**

This study suggests that DNA-damaging agents, rather than endocrine disruptors, should be examined as a factor in the increased prevalence of testicular cancer.

"Although radiation exposure of pregnant females has been declining

and is unlikely to be responsible for this increase, we intend to follow this up with studies of DNA-damaging chemicals found in cigarette smoke and air pollution, to which exposures of pregnant women have been increasing," said study senior author Marvin Meistrich, Ph.D., professor in MD Anderson's Department of Experimental [Radiation Oncology](#).

This study opens the door to possibilities for wide-ranging investigation, and researchers agree much work remains to be done.

"A second class of DNA-damaging agents that we intend to study is chemotherapy drugs like cyclophosphamide, which are used to treat pregnant women with breast cancer," Shetty said. "Studies at MD Anderson of the children of these women did not show increases in birth or developmental defects. However, we need to test these agents in our animal model since [testicular cancer](#) usually does not appear until early adulthood."

Provided by University of Texas M. D. Anderson Cancer Center

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