

Environmental toxicants causing ovarian disease across generations

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Washington State University researchers have found that ovarian disease can result from exposures to a wide range of environmental chemicals and be inherited by future generations.

WSU reproductive biologist Michael Skinner and his laboratory colleagues looked at how a fungicide, pesticide, plastic, dioxin and hydrocarbon mixtures affected a gestating rat's <u>progeny</u> for multiple generations. They saw subsequent generations inherit ovarian disease by "epigenetic transgenerational inheritance." <u>Epigenetics</u> regulates how genes are turned on and off in tissues and cells. Three generations were affected, showing fewer ovarian follicles—the source of eggs—and increased polycystic ovarian disease.

The findings suggest ancestral environmental exposures and epigenetics may be a significant added factor in the development of ovarian disease, says Skinner.

"What your great grandmother was exposed to when she was pregnant may promote ovarian disease in you and you're going to pass it on to your grandchildren," he says. "Ovarian disease has been increasing over the past few decades to effect more than 10 percent of the human female population and environmental epigenetics may provide a reason for this increase."

The research appears in the current issue of the online journal PLoS ONE. It marks the first time researchers have shown that a number of



different classes of environmental toxicants can promote the epigenetic inheritance of ovarian disease across multiple generations.

Research by Skinner and colleagues published earlier this year in PLoS ONE showed jet fuel, <u>dioxin</u>, plastics, and the pesticides DEET and permethrin can also promote epigenetic inheritance of disease in young adults across generations.

The work is a departure from traditional studies on several fronts. Where most genetic work looks at genes as the ultimate arbiters of inheritance, Skinner's lab has repeatedly shown the impact of the environmental epigenetics on how those genes are regulated. The field is already changing how one might look at toxicology, public health and biology in general.

The new study, says Skinner, provides a proof of concept that ancestral environmental exposures and environmental epigenetics promote ovarian disease and can be used to further diagnose exposure to toxicants and their subsequent health impacts. It also opens the door to using epigenetic molecular markers to diagnose ovarian disease before it occurs so new therapies could be developed.

In a broader sense, the study shows how epigenetics can have a significant role in disease development and life itself.

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