

Talk explores link between endometriosis and the estrogen receptor

By Monica Frazier

A meeting of the NIEHS Receptor Mechanisms Discussion Group Jan. 7 featured a seminar by postdoctoral fellow [Katherine Burns, Ph.D.](#), of the Receptor Biology Group (RBG) on "The Role of Estrogen Receptor Signaling in Endometriosis."

Seminar host [Kenneth Korach, Ph.D.](#), lead researcher in RBG and head of the NIEHS Laboratory of Reproductive and Developmental Toxicology, opened the meeting by discussing the history of the Receptor Mechanisms Discussion Group, established more than thirty years ago, and the contributions Burns has made to RBG through her passion for endometriosis research.

Burns began her presentation by noting some eye-opening facts about endometriosis, which affects approximately 5.5 million women in the U.S. and costs the country some \$22 billion annually. In addition to its prevalence and cost, the need to establish an understanding of endometriosis progression is driven by the lack of clinical biomarkers. Presently, endometriosis can be diagnosed only through laparoscopic surgery.

Burns added that for many years the disease has been thought to be associated with estrogen and possibly estrogen-like chemicals in the environment, but there is little research to prove the connection - something she is trying to change.

Developing an endometriosis disease model

An initial hurdle that Burns overcame was to find a good model for studying endometriosis, a condition where cells from the uterus flourish outside the uterine cavity. A condition, known as retrograde menstruation, allows endometrial debris to exit the uterus through the fallopian tubes. This backward movement of menstrual fluid through the fallopian tubes into the abdominal cavity is the leading hypothesis for the formation of endometriosis lesions.

Interestingly, although Burns noted that retrograde menstruation occurs in greater than 90 percent of women, only about 10 percent develop endometriosis. However, she said, through the development of a disease model, "We may be able to study the early initiation of this disease and potentially understand how and why it is forming in some women and not others."

To study the potential role of estrogen receptors (ER) in the development of endometriosis lesions, Burns used both donor and host mice that have one of the genes for ER alpha or beta knocked-out. From her mouse studies, Burns developed a working endometriosis model, to recapitulate human disease and find lesions that are essentially indistinguishable from human lesions, that illustrates the role of ER alpha and beta in lesion establishment and progression.

Environmental exposures and endometriosis

Now that Burns has a working model of disease initiation and progression, she is beginning to look at how environmental toxicants, such as bisphenol A (BPA), may affect endometriosis. Since BPA and the fluorinated variant of BPA, BPAF, have estrogenic-like activity, Burns suspects they play a role in endometriosis and other reproductive conditions.

Burns' initial experiments show that BPA and BPAF have effects on the uterus similar to doses of estrogen, but the BPAF effect is stronger.

"We predict that BPAF may potentiate the development of endometriosis," Burns commented, noting that she intends to continue to study this relationship in future experiments.

(Monica Frazier, Ph.D., is an Intramural Research Training Award fellow in the NIEHS Mechanisms of Mutation Group.)



Burns, a guest speaker at the January Receptor Mechanisms Discussion Group, explained her research on the estrogen receptor's role in endometriosis to an audience of scientists involved in receptor signaling research. (Photo courtesy of Steve McCaw)



Hong Soon Kang, Ph.D., staff scientist in the NIEHS Cell Biology Group, listened during the question and answer session after Burn's seminar. (Photo courtesy of Steve McCaw)

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