Male and female sex cell determination requires lifelong maintenance and protection

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The way in which the sex of an organism is determined may require lifelong maintenance, finds new research from the University of Minnesota. According to the study published today in the journal *Developmental Cell*, sex-specific transcription factors perform lifelong work to maintain sexual determination and protect against reprogramming of cells from one sex to the other.

Previous research at the University of Minnesota's Department of Genetics, Cell Biology, and Development showed sex determination is not permanent. Using a mouse model, researchers found the sex of gonadal cells – those found in the ovaries or testes – require maintenance throughout life. This research also showed loss of a single transcription factor can result in the transformation of male cells into female cells.

"DMRT1 in the testis and FOXL2 in the ovary have been identified as key transcription factors responsible for maintaining sexual differentiation. What we asked in this study was how the cells maintain sexual differentiation and why their sex determination requires continuous protection," said David Zarkower, Ph.D., principal author and director of the Developmental Biology Center at the University of Minnesota.

Zarkower's research team took a closer look at DMRT1 and determined it partners with the male fetal sex determination gene called Sox9 to maintain male sexual determination after birth in a mouse model. Part of that work includes silencing genes normally involved in the female fetal

sex determination process. This discovery indicates lifelong sex determination maintenance requires a process related to prenatal sex determination.

Another notable discovery is DMRT1's ability to limit retinoic acid (RA) signaling, preventing RA from activating genes normally involved in female sex determination and female organ development.

"While RA signaling between cells is absolutely required for sperm production and male fertility, we found that RA also has a dark side. If DMRT1 is not there to act as a guardian of maleness, RA has the potential to activate genes driving male-to-female transdifferentiation," said Zarkower. "This shows cell signaling can transform the identities of the very cells that use it from male to female. We think other cell types may also require similar mechanisms allowing them to use critical signaling molecules without becoming reprogrammed."

Provided by University of Minnesota

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