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Ph.D. DISSERTATION DEFENSE

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Title:	PFOS Disrupts Membrane Signaling and Epithelial Integrity in Fallopian Tube Cells
Chairperson:	Dr. Marcin Iwanicki, Department of Chemistry and Chemical Biology
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ABSTRACT

Perfluorooctane sulfonic acid (PFOS) is an industrial chemical that has been widely used for decades in products such as stain repellents, firefighting foams, and food packaging. Its chemically inert and highly stable nature prevents it from breaking down efficiently in the environment or in human tissues, leading to significant bioaccumulation. As a result of this persistence, PFOS is widely detected in human serum. Growing evidence links PFOS exposure to health problems such as infertility, pregnancy complications, and certain cancers, but how it affects the reproductive system at the cellular level remains poorly understood.

In this thesis, I investigated how PFOS affects human fallopian tube epithelial cells — a cell type critical for fertility, as it supports egg fertilization and early embryo development. These cells are also increasingly recognized as the origin of one of the most aggressive forms of ovarian cancer. I found that even short-term exposure to high doses of PFOS causes these cells to change shape, stop proliferating, lose adhesion, and weaken their epithelial integrity — all functions essential for healthy reproductive physiology.

Through gene expression analysis, I discovered that PFOS activates stress-related signaling pathways, including those involving the KRAS oncogene, while reducing the expression of genes responsible for maintaining lipid and cholesterol homeostasis. Strikingly, blocking one of these stress pathways (the MEK/ERK pathway) or supplementing cells with additional cholesterol reversed several PFOS-induced defects, suggesting that the cell membrane plays a central role in how PFOS affects cellular physiology.

To test this hypothesis, I examined the physical properties of the cell membrane and found that PFOS increases membrane fluidity and disorder — changes that can disrupt normal signaling and weaken cellular structural integrity. Together, these findings support a model in which PFOS disturbs the plasma membrane, triggering stress-response pathways and leading to loss of adhesion and impaired epithelial barrier function.

This work advances our understanding of how PFOS affects cells in the female reproductive system and its potential contribution to infertility and ovarian cancer risk. It also highlights the cell membrane as a potential target for preventing or mitigating PFOS-related toxicity.