NSW Big Ideas Oncology Seminar Series

UNSW-USYD

Professor Gail Prins

Tuesday 13th August 2024

Seminar: 16:00-17:00
Networking: 17:00-17.30

Charles Perkins Centre,
Johns Hopkins Dr, Camperdown USYD

Hosts: Prof Vanessa Hayes (USYD)
Prof David Thomas (UNSW)

Scan the QR code or CLICK HERE to RSVP

Virtual option: CLICK HERE to join

Zoom Meeting ID: 938 382 6375
Password: 509617

Professor Gail Prins is the Michael Reese Professor in the Department of Urology, Pathology and Physiology, University of Illinois and co-Director for the Chicago Center for Health and Environment (CACHET). She is best known for her ground-breaking research that led to the connection between Bisphenol-A (BPA), a common plastic additive found in thousands of consumer products, and cancer. She has defined the field of developmental estrogenization (developmental reprogramming) of the prostate gland in response to natural or pharmaceutical estrogens and endocrine disrupting chemicals (EDCs).

Environmental Toxicants Reprogram Prostate Stem Cells & Increase Prostate Carcinogenic Risk

Professor Gail Prins has over 40 years of experience in endocrinology, prostate carcinogenesis and environmental chemical exposures that drive prostate disease with aging. Her laboratory employs molecular, cellular and histologic approaches using animal models, organ and cell cultures and prostasphere/organoid models with animal and human prostate cell. Their findings have determined that several compounds, including BPA, PFAS, and inorganic arsenic (iAs), reprogram normal human prostate SPC populations, leading to increased cancer susceptibility. Of note, the observed alterations in stem cell self-renewal, progenitor cell proliferation and lineage commitment by these compounds are mediated through chemical-specific mechanisms. BPA reprograms the SPC epigenome and transcriptome leading to increased sensitivity of the daughter differentiated cells to estrogen-driven carcinogenesis. Chronic PFAS exposures rewire the prostate SPC metabolome and transcriptome leading to a pre-carcinogenic stem cell fate and enhanced tumor growth. In contrast, low-dose iAs triggers autophagic flux blockade in SPCs which activates the p62-KEAP-NRF2 axis, resulting in direct progenitor cell transformation. Together, our findings document that prostate SPC populations, which express multiple membrane and nuclear receptors, are direct targets for many environmental toxicants that reprogram and transform the cells. Due to the long-lived nature of prostate stem cells, these processes can underpin increased prostate carcinogenic potential over extended time periods.

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