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Analyzing Molecular Pathways between Maternal Obesity and Risk of Endometrial Cancer in F1 Mice Generations

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The incidence and mortality of endometrial cancer have risen by approximately 1.5% and 3.2% respectively in 5 years; 10,470 deaths and 60,050 new cases are anticipated this year. In recent studies, there have been interactions between obesity and Type I endometrial cancer. During pregnancy, if the viable offspring is nurtured in a maternal obesogenic environment, there's a high chance that they would inherit the malfunctioning mitochondria or deregulated epigenetic signatures. These germline modifications result in passing of defective organelles to offspring gametes and can lead to hyper proliferation characterized by hyperplasia and nuclear atypia.

We aim to determine how maternal obesity has contributed to endometrial cancer in offspring by focusing mainly on mice at 72-week time point. To observe whether maternal diet contributed to higher risk of cancer, the mice were fed a control or high fat high sugar (HFHS) diet. After a few weeks, these mice were dissected, and their uteri were collected. We looked for difference in the expression of proteins that have been associated with cancer such as phospho-Pten, Pten, phospho-AKT and AKT of mice on HFHS diet using Western Blotting techniques. We performed Immunohistochemistry to confirm any pathology seen by Hematoxylin and eosin staining (H&E). A pathologist performed pathologic assessment on the stained slides. We observed that offspring exposed to maternal HFHS diet had increased uterine to body weight ratio. Additionally, H&E staining in all cohorts showed neutral phenotype. Lastly, there was no significant different between phospho-AKT or AKT and Phospho-Pten or Pten levels in mice exposed to HFHS diet. In conclusion, maternal diet or direct exposure to HFHS diet mice did not lead to endometrial cancer in initiation by 72 weeks in wild-type C57B/6J offsprings.