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Conditional BRCA2 Switch in Human Cells to Study Tumor Progression

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The BRCA2 (Breast Cancer Susceptibility 2) gene is a caretaker of genome integrity. Germline mutations in BRCA2 predispose to a high risk for ovarian and breast cancer. The BRCA2 protein plays an important role in repair of DNA double-strand breaks (DSBs) by homologous recombination. BRCA2 dysfunction results in genome instability including chromosome aberrations and an abnormal number of centrosomes. Centrosome amplification is a hallmark of tumors from BRCA2 mutation carriers and may be responsible for the origin of chromosome missegregation at mitosis and DNA aneuploidy found in these tumors. However, it remains an open question how BRCA2 regulates centrosome duplication and the consequences for tumor initiation and progression. Here, we present the generation of an isogenic inducible BRCA2 human cell line providing a model to study acute loss of the BRCA2 protein. We plan to measure centrosome number and how BRCA2 regulates the centrosome duplication checkpoint with the end goal of understanding tumor initiation and maintenance in BRCA2 mutation carriers.

Biography:

Dr. Jimenez-Sainz Judit is a Postdoctoral Associate in the Jensen group in Therapeutic Radiology Department. Her current research involves biochemical and cellular-based approaches to prevent and unmask the initial steps in breast and ovarian cancer formation. She received her phd from Universidad de Valencia, Spain and UCL, London. She is a member of ECUSA, NYAS and WISAY and she strongly believes in supporting new generation of students in STEM.