Serum amyloid A (SAA)-mediated tumorigenesis in triple negative breast cancer (TNBC)



Conceptual framework for the triplet of obesity, immunity, and $\ensuremath{\mathsf{TNBC}}$

SAA promotes **③** TNBC tumorigenesis via the SAA-TLR2-CXCL8 axis, integrating with **①** obesity-derived SAA and **②** SAA-mediated immunogenicity. The outcomes will offer the SAA-TLR2-CXCL8 axis as a molecular mechanism for health disparities observed in obesity and TNBC, contributing to better overall survival and therapeutic options in patients with obesity and TNBC susceptibility.

Targeting CXCR2 as a driver for obesity-derived progression of ovarian cancer (OC)



Schematic representation of CXCR2-mediated signaling to link obesity and ovarian cancer

CXCR2-mediated signaling could be a driver for obesityderived progression of ovarian cancer by crosstalk between CXCR2 in adipose tissues and CXCR2-specific chemokines CXCL1 and CXCL8 in ovarian cancer cells; this would enhance the inflammatory burden and accelerate disease progression, leading to high mortality of ovarian cancer