In the U.S. Ovarian cancer remains the 5th leading cause of cancer related deaths for females, due enlarge to high rates of recurrence, subsequent chemoresistance, and diagnosis at late stages of the disease. Within the ovarian cancer tumor microenvironment cells are exposed to aberrant mechanical stimuli that trigger adverse cellular responses, termed mechanotransduction. Ascites in the peritoneal cavity mechanically stimulates tumor cells through shear stress. The influence of mechanical shear stress has begun to show evidence of enhanced cancer stem cell marker expression and metastatic potential, research has yet to investigate the influence of the range of forces that are experienced as well as potential pathways of intervention. To address this, OVCAR3 and OVSAHO ovarian cancer cells housed within a combination agarose-collagen type I hydrogel were exposed to 11, 5, and 1 dynes/cm² of shear stress in a custom-built bioreactor.

Ovarian cancer cell lines show significant change in morphology under all levels of shear stress stimulus. Cells exposed to shear stress stimulation also display significantly enhanced proliferation (1.44 fold) and increased chemoresistance to paclitaxel and carboplatin. Under shear stress stimulation, no significant change in cellular death was found when compared to control conditions. Bru seq analysis showed a reduction in mucin 15 regulation (2 fold change in OVCAR3 experiments and 1-fold change in OVSAHO experiments) which was further confirmed through qPCR analysis and western blot. The upregulation of proliferation marker Ki67 and decreased cell death under drug treatment indicates a cancer assistive mechanism from shear stress stimulus and chemoresistance tendencies.