Breast cancer is the second leading cause of cancer death among women. Early detection and diagnosis, along with targeted therapies, have drastically improved survival rates. However, this improvement does not extend to patients with metastatic, triple-negative breast cancer (TNBC). TNBC is an aggressive cancer that is estrogen receptor-negative, progesterone receptor-negative, and HER-2 negative. Therefore, TNBC cannot be targeted via hormone therapies or novel molecularly targeted therapies, such as anti-HER2 therapies. Toxic chemotherapeutics, such as Doxorubicin, remain the primary treatment option. However, Doxorubicin exhibits cumulative dose-limiting cardiotoxicity that limits its clinical utility. In order to create and optimize therapeutic options for the treatment of TNBC, a better understanding of these tumors and their drug interactions is necessary. Often, two-dimensional (2D) in vitro models are employed to assess these interactions, but these models do not effectively recapitulate the complexity and multicellular nature of in vivo tumors. Three-dimensional (3D) in vitro models present more physiologically relevant models for assessing the interplay between drug and tumor by attempting to mimic the complexity of the tumor microenvironment.

For this study, the potency of Doxorubicin was determined using conventional cell toxicity assays. Both the SRB assay, which measures the total protein content of cells, and MTT assay, which evaluates the mitochondrial enzymatic activity of cells, were performed. The assays were conducted using classic 96-well flat-bottom plates with drug exposures ranging from 100 to 0.0001 uM Doxorubicin in the human TNBC cell-line, MDA-MB-231-LUC-GFP. They were evaluated following 24, 48, and 72 hours of drug exposure. The potency (IC50 value) was calculated after 72 hours of drug exposure. Cytotoxicity correlated positively with Doxorubicin concentration and length of Doxorubicin exposure.