My research work is focused on the role of estrogens, arsenic, calcium and β2M in cell proliferation, cytotoxicity and disease using MCF-7 and N2A as in vitro models.The first part of the thesis deals with the role of CHIP1 in 17-α-ethinyl estradiol induced MCF-7 breast cancer cell proliferation and survival. 17α-ethinyl estradiol (17αEE) is a semisynthetic alkylated estrogen used as estrogenic component in oral contraceptives. We found that 17αEE modulates the cellular levels of CHIP1, PARP1, p-AKTS473 and HSP 90 in a time dependent manner. CHIP1 is an important tumor suppressor protein which function as E3 ubiquitin ligase for proteins involved in normal and cancer cell physiology. But regulation of cellular levels of CHIP1 is poorly understood. We have discovered that 17αEE induced CHIP1 modulation is inhibited by ER-α antagonist ICI 164384. Knockdown of CHIP1 induces cell proliferation in MCF-7 cells. We also discovered that CHIP1 interacts with PARP1 using co-immunoprecipitation. The second part of the thesis deals with arsenic-estradiol interactions and its implication for cell proliferation and migration in MCF-7 breast cancer cells. Briefly, low doses of arsenic trioxide induce “scratch” wound healing in MCF-7 cells similar to the effect of estradiol which induces wound healing in MCF-7 cells over a wide range of physiological and pharmacological concentrations. 17β-estradiol (E2) inhibits the low dose arsenic trioxide induced scratch wound healing in MCF-7 cells. We discovered that arsenic binds to estradiol resulting in inhibition of both estradiol and arsenic induced scratch wound healing. The third part of the thesis deals with 17β-estradiol (E2) and calcium binding by β2M, which is a minor subunit of the MHC1 molecule. For the first time, we have discovered and reported that β2M binds to calcium, leading to formation of micro aggregates that form pre-amyloid fibrils, using biophysical techniques like resonance rayleigh scattering (RRS), isothermal calorimetery (ITC) and Thioflavin T (ThT) binding. Similarly for the first time we have demonstrated that β2M interacts with estradiol using difference absorption spectroscopy (DAS) and surface plasmon resonance (SPR). Binding of β2M with estrogen inhibits the cytotoxicity of β2M oligomers to N2a cells.