



## CANCER PREVENTION & RESEARCH INSTITUTE OF TEXAS

Award ID:  
RP180275

Project Title:  
Targeting Stromal ERalpha for Cervical Cancer Therapy

Award Mechanism:  
Individual Investigator

Principal Investigator:  
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Entity:  
University of Houston

### Lay Summary:

Cervical cancer is the fourth most frequent cancer and the fourth leading cause of cancer death in women worldwide. Texas' mortality rate of cervical cancer is 1.2-fold higher than the national average. It contrasts with that the mortality rate of all cancers is lower by 10% in Texas than the US. There has been little progress in the treatment of cervical cancer over the past decade, which accounts for the high cervical cancer mortality at present. Approximately 500,000 women contract the disease and 250,000 people die from it every year. It is clearly demonstrated that human papillomavirus (HPV) is required for the development of cervical cancer. Current HPV vaccines thus hold promise in preventing cervical cancer. However, due to high costs of the vaccines and the requirement of infrastructure for cervical cancer screening (i.e., the Pap test), the preventive methods are not readily available to women in developing countries and medically underserved US population. Most cervical cancers occur in those women. Evidence indicates that, in addition to HPV, other cofactors are required for cervical cancer. We are particularly interested in estrogen because its signaling can be easily targeted. In a mouse model, both HPV and estrogen are required for the development of cervical cancer. We demonstrated that estrogen receptor alpha (ERa) was required for the development of estrogen-induced cervical cancer in the same mouse model. ERa is expressed in cancer epithelial cells and surrounding stromal cells. Our preliminary results suggest that ERa expressed in stromal cells rather than cancer cells is required for the growth of cervical cancer. In this application, we propose to determine roles of epithelial and stromal ERa in cervical cancer. We anticipate that our proposed studies using mouse models will not only reveal a novel mechanism of ERa, but also facilitate the development of first targeted therapy for cervical cancer.