Department of Molecular and Cellular Biology

Graduate Seminar MCB*6500

Friday, Jan. 20, 2017 in SSC 1511 @ 12:45 p.m.

presented by:

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A Potential Role for Luman in Castration Resistance in Prostate Cancer

Prostate cancer is the second most prevalent cancer with 1 in 7 men being diagnosed with it in their lifetime. Typical treatment involves testosterone deprivation achieved through surgical or chemical castration; however, most prostate cancers re-emerge after castration as androgen-independent or castration resistant prostate cancer, which is often fatal. Prostate cancer progression and metastasis are primarily driven by overexpression and activation of the androgen receptor. Recently we have found that Luman, a cellular stress response protein involved in the unfolded protein response, is a potential co-activator of the glucocorticoid receptor. In Luman gene knock-out mice, there is a reduced level of circulating glucocorticoids as well as reduced levels of testosterone. These mice have high levels of the glucocorticoid receptor and preliminary data indicates that the androgen receptor mirrors this. Luman has been found to be highly expressed in prostate cancer cell lines and has been implicated in other cancers including breast cancer and certain neuroendocrine cancers. Since the glucocorticoid and androgen receptors belong to a closely-related steroid receptor family, similar in structure and function, we propose that Luman may also be a co-activator of the androgen receptor; which when overexpressed, can activate the androgen receptor signaling in the presence of little or no testosterone, such as in castrated prostate cancer patients.