

AB070. Roles and mechanisms of soybean isoflavones in androgen-independent transformation of prostate cancer

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Abstract: The aims of this study were to investigate the impact of soybean isoflavones (SIFs) on the cellular oxidative stress levels in hormone-sensitive type (LNCap) and hormone-insensitive type (DU145) of prostate cancer, and to elucidate the main components and their roles in the transformation of androgen dependency (AD)/androgen independency (AI). The LNCap and DU145 cells were treated with different concentrations of SIFs monomer (including daidzin, genistin, daidzein, and genistein), and the activities of superoxide dismutase (SOD) and malondialdehyde (MDA) were then measured. The content of reactive oxygen species (ROS) in these two cells was also determined after treated with certain effective monomer. The contents of MDA in the two kinds of cells treated with genistein and daidzein were increased, but the contents of SOD were decreased in a concentration-dependent manner. There was no significant difference between daidzin and genistin. The content of MDA in the DU145 cells was higher than that in the LNCap cells under the same conditions, but the content of SOD was decreased, indicating that the oxidative stress level in DU-145 was higher than LNCap, and the ROS level in DU145 treated with effective component was significantly higher than that in LNCap. Genistein and daidzein in SIFs can affect the apoptosis and proliferation of prostate cancer cells through increasing their oxidative stress levels, thus inhibit the transformation of prostate cancer toward androgen-independent type.

Keywords: Soybean isoflavones (SIFs); androgen-independent; prostate cancer

doi: 10.21037/tau.2017.s070

Cite this abstract as: Xu L, Cao Z, Xue Y, Zhao S, Wang K, Jiang C, Chen Q. Roles and mechanisms of soybean isoflavones in androgen-independent transformation of prostate cancer. *Transl Androl Urol* 2017;6(Suppl 3):AB070. doi: 10.21037/tau.2017.s070

AB071. The role of long non-coding RNAs in sunitinib resistance of renal cancer

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Background: The incidence of renal cell carcinoma (RCC) has been rising throughout the world. Approximately 20% of RCC patients presented with advanced stage disease at the time of diagnosis, and in patients with localized RCC, nearly 30% will develop recurrence and metastasis after tumor resection. Recently, improved comprehension of RCC pathogenesis led to the development of receptor tyrosine kinase (RTK) inhibitors, such as sunitinib, as the mainstay of therapeutic options for advanced RCC patients. Sunitinib is an oral multi-targeted RTK inhibitor, which has potent anti-angiogenic effects and direct anti-tumor activities due to the inhibition of vascular endothelial growth factor receptor (VEGFR), platelet-derived growth factor receptors (PDGFR), stem-cell growth factor receptor (KIT) and FMS-like tyrosine kinase 3 (FLT3). However, 10–20% of advanced RCC patients are inherently refractory to sunitinib therapy, and most of the remaining patients end up with drug resistance and tumor progression after 6–15 months of therapy, resulting in failure of sunitinib to efficiently prolong the survival of RCC patients. Several studies have proposed the activation of compensatory signaling pathways for the acquisition of