Elif Damla Arisan et al., J Cancer Sci Ther 2015, 7:5
http://dx.doi.org/10.4172/1948-5956.S1.044

OMICS International
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International Conference on
Prostate Cancer
June 22-24, 2015 Florida, USA

Roscovitine treatment inhibited cell proliferation and induced apoptosis via modulating activity of Stat3 and Foxo1 under control of Akt in LNCaP prostate cancer cells

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Cyclin dependent kinases (CDKs) are known activators of nuclear hormone receptors; such as androgen receptor (AR) via modulating several cell survival signaling routes. However, novel CDK inhibitors might also prevent cell proliferation and induce apoptosis in different prostate cancer cells regardless of functional AR status. Purine-derived purvalanol and roscovitine are strong apoptotic inducers, caused cell cycle arrest through competing ATP binding sites of several CDKs and disrupt cyclin/CDK complex formation. However, their mechanistic action in prostate cancer cells is not fully understood. In this study, we found that, exposure of LNCaP and DU145 prostate cancer cells to purvalanol (20 mM) and roscovitine (30 mM) for 24 h induced apoptosis compared to untreated prostate cancer cells. According to immunoblotting results, both CDK inhibitors downregulated Stat 3 and caused dephosphorylation of Stat3 in cytoplasmic lysates. In addition, we found that although roscovitine was less effective on Stat3-Foxo1 proteins interaction due to upstream Akt phosphorylation compared to purvalanol treated samples, Stat3-AR binding affinity was decreased in LNCaP cells. Therefore we concluded that while roscovitine were modulating Akt phosphorylation and altered Foxo nuclear transport, it also affected Stat expression profile to induce cell cycle arrest which caused cell death in prostate cancer cells.

Biography

Elif Damla Arisan has completed her PhD from Sabanci University at 2009. She is working in Istanbul Kultur University as Assoc. Prof. since 2013. She has published 31 papers in reputed journals and was a member of the organizing committees of 4 international congresses in tissue engineering, nanomedicine, polyamines and cancer field.

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