ARC '16

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http://dx.doi.org/10.5339/qfarc.2016.HBPP3022

# Inhibition of p90 Ribosomal S6 Kinase Attenuates Cell Migration and Proliferation of the Human Lung Adenocarcinoma through Phospho-GSK-3β and Osteopontin

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## Background

Lung cancer is the second most common cancer in both men and women and it is the leading cause of cancer deaths worldwide. Lung cancer can be divided into two broad categories: non-small cell lung cancer (NSCLC), which consists of about 85% of all lung cancers and small cell lung cancer (SCLC), which account for 15% of all lung cancers. The evolution of lung cancer is a multistep process involving genetic and epigenetic alterations. Standard treatment therapies such as radiation therapy, chemotherapy and surgery has reached a plateau phase. As a result, much work has centered on identifying the molecular targets involved in the tumor cell proliferation, survival and metastasis in effort to identify novel therapeutic approaches. p90 ribosomal S6 kinase (p90RSK) constitutes a family of serine/threonine kinases that have been shown to be involved in cell proliferation of various malignancies via direct or indirect effects on the cell-cycle machinery.

## Objectives

To investigate the role of p90RSK in lung adenocarcinomas and whether the inhibition of p90RSK diminishes cancer progression. Moreover, we investigated the involvement of glycogen synthase kinase-3β (GSK-3β) and osteopontin (OPN) in the p90 RSK induced lung adenocarcinoma progression.

**Cite this article as:** Abdulrahman N, Jaballah M, Poomakkoth N, Riaz S, Abdelaziz S, Issa A, Mraiche F. (2016). Inhibition of p90 Ribosomal S6 Kinase Attenuates Cell Migration and Proliferation of the Human Lung Adenocarcinoma through Phospho-GSK-3β and Osteopontin. Qatar Foundation Annual Research Conference Proceedings 2016: HBPP3022 http://dx.doi.org/10.5339/qfarc.2016.HBPP3022.

## Methods

p90RSK, OPN, GSK-3β protein expression were examined in the A549 human lung adenocarcinoma cell line in the presence and absence of BI-D1870 (BID), a p90RSK inhibitor. Gene expression of anti-apoptotic and pro-apoptotic markers namely Bcl2 and Bax, respectively, were studied by reverse transcription polymerase chain reaction. In addition, the A549 lung adenocarcinoma cell line was characterized for cell proliferation using the MTT assay and cell migration using the scratch migration assay.

## Results

Our study revealed that the treatment of the A549 lung adenocarcinoma cell line with BID resulted in a significant reduction in protein expression of p90RSK 1 ($69.32 \pm 12.41\%$ of control; $P < 0.05$). The inhibition of p90RSK also showed a significant suppression of cell proliferation ($54.3 \pm 6.73\%$ of control; P