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Prolactin activation of JAK2/STAT3 signaling pathway through GHR in NSCLC

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It is known that PRL could lead breast and prostate cancer progressions. Recent reports point out the concentration of serum PRL in Non Small Cell Lung Cancer (NSCLC) patients are significantly higher than that in healthy people. The patients with higher concentration of serum PRL have lower survival rate than that in patients with normal level of serum PRL. PRL receptor (PRLR) expression level, however, is not related to patients' survival rate. It is unclear whether PRL promotes lung cancer progression. Meanwhile, the action mechanism of PRL in the proliferation of lung cancer is still unknown. Cells were seeded in 96-well plates with complete medium. After 24 h of plating, cells were serum starved for 16 h and then treated with PRL. MTT assay was applied for cell proliferation. The protein levels of JAK2/STAT3 and VEGF were determined by western blot. The mRNA level was measured by quantitative real time polymerase chain reaction. Our data show that PRL promoted cell proliferation in NSCLC cells. Expressed growth hormone receptor (GHR), not PRLR, was observed in all NSCLC cell lines. Increased expressions of p-JAK2 and p-STAT3 were found in cells treated with PRL. Treatment of PRL also increased STAT3-regulated downstream gene VEGF protein and mRNA expressions. In contrast, the protein expression of p-JAK2 was decreased after inhibition of GHR. PRL activates GHR downstream signaling pathway of JAK2/STAT3. Activated STAT3 is translocated to nucleus and increases downstream gene VEGF transcription activity. PRL promotes cell proliferation and VEGF expression in NSCLC cells.

Biography

Ms. Jou-Chun Chou is a Ph.D. student in the Department of Life Sciences, National Chung Hsing University, Taichung, Taiwan, ROC. Her major is to study the effect and action mechanism of prolactin on the proliferation of human non-small cell lung cancer under the advices of Dr. H. Lin and Dr. P. S. Wang.

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