conferenceseries.com

J Cancer Sci Ther 2018, Volume 10
DOI: 10.4172/1948-5956-C1-124

Effect of prolactin receptor antagonist in different cancer models

Alkharusi A
Sultan Qaboos University, Oman

Increased levels of prolactin (Prl) receptors have been found in endocrine dependent cancers e.g. breast and prostate cancer as well as in ovarian cancer and glioblastomas. Prl stimulates cell growth by activating the intracellular JAK-STAT pathway. A new line of research in the last years suggests that human cytomegalovirus (CMV) infection can also contribute to several human malignancies including glioblastoma brain tumor and ovarian cancer. A supportive study showed that CMV DNA was present in 50% of fresh ovarian carcinoma tissue samples (39 samples). It was suggested that, human CMV is able to create a more malignant phenotype of tumor cells by the action of its regulatory proteins and non-coding RNA, which will affect their proliferation, invasion of other tissues, survival and other cellular properties. Furthermore, prolactin receptor expression and circulating prolactin levels have been shown to be higher among women with ovarian cancer vs. benign condition or healthy control. Our preliminary studies showed that an experimental CMV infection of glioblastoma and ovarian cancer cell lines activates Prl and Prl signals. We have generated a high affinity Prl receptor antagonist that can be recombinantly produced in E. coli. This antagonist prevents dimerization of the receptor and consists of around 200 amino acids (similar molecular weight as Prl. Using cell based assays in glioblastoma cell lines we found that Prl receptor antagonists reduce STAT activation resulting in cellular growth retardation. An interesting (unpublished) finding is that the Prl system appears to be correlated to the activity of CMV virus in glioblastoma and ovarian cancer. This might open a new therapeutic angel of combining anti-viral treatment and PRLRA along with anti-cancer agents.

akharusi@squ.edu.om