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Characterization of the carbonic anhydrase 3 gene promoter and the mechanism of *Evi1* mediated repression

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Ecotropic Viral Integration site-1 (*EVI-I*) is a transcriptional repressor protein, its expression contributes to acute leukemia and transforms Rat1 fibroblasts cells. Previous microarray studies confirmed that *EVI-I* is either directly or indirectly regulates transcription of other genes in Rat1 fibroblasts cells and one of these genes is carbonic anhydrase 3 (*caIII*) which interestingly repressed its expression. In order to understand the mechanism by which *EVI-I* repress the expression of *caIII*, the rat *caIII* promoter region was identified and then serious deletions were conducted. The *caIII* promoter reporter assay showed that sp1 which located at Δ280 *caIII* promoter region play a major role in regulation of *caIII* in Rat1 fibroblast cells. Srf, ets and oct which are located at Δ137 *caIII* promoter activity act as potential transcriptional regulation factors may due to combinational activities of all of these three in Rat1 fibroblast cell. For further investigation about the role of these transcription factors on *caIII* promoter, site direct mutagenesis created with -280 (srf, oct and ets) and reporter assay demonstrates loss of *caIII* repression as well, which suggesting potential transcription factor located in this area. In order to locate this transcription factor, different deletions created on -280 on *caIII* gene promoter and reporter assay showed loss of *caIII* repression at -260 location suggesting that potential c/ebp-e transcription factor may co-operate to activate -280 promoter. These findings might suggest the basis for the development of a novel therapeutic strategy for the treatment of leukemia's and solid tumors where *EVI-I* is over-expressed.

Biography

Alaa Saleh is a currently pursuing PhD at Glasgow Caledonian University specializing in Molecular Biology.

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