Inhibition of Gqα Signaling by Expression of G11α Pseudogene in Huangqi Induced Human Leukemia K562 Cells

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ABSTRACT
Pseudogenes have long been marked as ''junk'' DNA, however, more evidence showed that some of them may have been involved in cell function. We have identified G11α pseudogene expression in the huangqi treated K562 cells while it was not detected in hemin and HMBA treated cells. The activation of G11α pseudogene expression by huangqi was confirmed by reporter assay. Meanwhile, huangqi also induced β-globin gene expression, a marker of erythrocyte differentiation. Since the erythrocyte and megakaryocyte differentiation share the same progenitor, we hypothesize the G11α pseudogene participates in the inhibition of K562 cells megakaryocyte differentiation and hence promotes erythrocyte formation and as well as globin gene expression. Gqα was reported to involve in megakaryocyte differentiation and is induced by PMA. To test our hypothesis, the K562 cells were transiently transfected with G11α pseudogene and Gqα minigene as control and PMA were added to the culture. The RT-PCR showed CD41 and CD61, markers of megakaryocyte differentiation, were upregulated in the presence of PMA. The two CD markers were reduced in both G11α pseudogene and Gqα minigene transfected cells compared with empty vector transfected cells. In conclusion, our results showed that huangqi prompted β-globin gene expression by the induction of G11α pseudogene expression. The authors also demonstrated the inhibitory function of G11α pseudogene on Gqα and the inhibitory function may apply to the treatment of Gqα-mediated signaling disorders, e.g. myocardial hypertrophy and Uveal melanoma.

Keywords: Gq/11 alpha, K562 cell, CD41, CD61

REFERENCES