ABSTRACT
Heterotrimeric G-protein–coupled receptors (GPCRs) are the largest family of cell surface receptors, accounting for more than 1% of the human genome. GPCRs transduce extracellular signals from environmental factors, including odorants and tastants, and affect cellular physiology, and even gene expression profiles in cells. The function of GPCR56 is controversial. In contrast to normal tissues, the expression of GPCR56 in cancer cells is relatively high, inferring GPCR56 as the potential carcinogenesis factor. But in melanin tumour cells, an opposite result was reported. Overexpression of GPR56 inhibits melanoma tumor growth and metastasis, and reducing GPCR56 promotes tumor progression. GPCR56 was 4 times down regulated in leukemia model cells, K562 cell, as compared with clinical samples. We propose that the expression of GPCR56 is either against the Leukemia development or is leading cell differentiation. In both cases, the over-expression of GPCR56 may benefit leukemia patients. To define the function of GPCR56, over-expression and gene knock down of GPCR56 in K562 cells were studied. The growth of K562 cells was not affected by the expression of GPCR56 and promegakaryocytic phenotype were increased. To further investigate the function of GPCR56, the expression of cluster of differentiation markers (CD markers) of erythroblast (β-globin, γ-globin), granulocyte (CD13, CD33), monocyte (CD14, CD68), megakaryocyte (CD41, CD61) were detected by RT-PCR. Among these, γ-globin, CD33, CD41 and CD61 were up-regulated, β-globin, CD13, CD14 and CD18 were not affected. Put these together, GPCR56 may lead K562 cells or participate in the pathway to megakaryocyte.

Keywords: Heterotrimeric G-protein–coupled receptors; K562 cell; megakaryocyte