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王建祥课题组在Leukemia期刊上发表"PIG7, transactivated by AML1..."

作者: <u>admin</u> 浏览次数: 发布日期: 2011-11-09 09:25:21

我重点实验室王建祥课题组在在Leukemia期刊发表文章"PIG7, transactivated by AML1, promotes apoptosis and differentiation of leukemia cells with AML1-ETO fusion gene."

Abstract

AML1, the potent transcription factor in hematopoiesis, is antagonized by AML1-ETO in t(8;21) leukemia. Our previous study showed that the differentiation and apoptosis of Kasumi-1 induced by sodium phenylbutyrate (PB), were accompanied by significant upregulation of PIG7 and AML1b (one of the AML1 isoforms). Here, we further investigated the relationship between AML1b and PIG7, also the effects of PIG7 on leukemia cells. The results demonstrated that exogenous AML1b could upregulate PIG7 expression in HEK-293 and CV-1 cells in sequence-specific and dosage-dependent manners, and this effect was antagonized by AML1-ETO. The specific AML1-binding site required for p53-induced gene 7 (PIG7) transactivation was located between nucleotides -1511 and -1503 in the PIG7 promoter. Overexpression of PIG7 could induce the apoptosis and differentiation of Kasumi-1 and SKNO-1 cells, but showed less effect on NB4 cells directly. Moreover, ectopic expression of PIG7 could sensitize these cell lines to PB or all-trans retinoic acid, respectively, which could then be abrogated by downregulation of PIG7 expression. Furthermore, the primary acute myeloid leukemia cells showed similar response to the ectopic expression of PIG7. In conclusion, PIG7 could be transactivated by AML1, which subsequently induces differentiation and apoptosis of leukemia cells, especially those with AML1-ETO fusion gene. Leukemia advance online publication.

Pubmed链接:

http://dx.doi.org/10.1038/leu.2011.178



2011奖学金申请通知 2011.11.15学术活动:Clark.K. Colton教 授





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