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PLC-beta 1 regulates the expression of miR-210 during mithramycin-mediated erythroid differentiation in K562 cells

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PLC-beta 1 (PLCβ1) inhibits erythroid differentiation induced by mithramycin (MTH) by targeting miR-210 expression. MicroRNA-210 (miR-210) has been reported to be upregulated in various types of human malignancy suggesting that it has an important role in tumorigenesis. Inhibition of miR-210 affects the erythroid differentiation pathway and it occurs to a greater extent in MTH-treated cells. In this paper we have analyzed the effect of MTH on human K562 cells differentiation. Overexpression of PLCβ1 suppresses the differentiation of K562 elicited by MTH as demonstrated by the absence of γ-globin expression. Inhibition of PLCβ1 expression is capable to promote the differentiation process leading to a recovery of γ-globin gene even in the absence of MTH. Our experimental evidences suggest that PLCβ1 signalling regulates erythropoiesis through miR-210. Indeed overexpression of PLCβ1 leads to a decrease of miR-210 expression after MTH treatment. Moreover miR-210 is up-regulated through both proliferation and differentiation events when PLCβ1 expression is down-regulated. Therefore we suggest a novel role for PLCβ1 in regulating miR-210 and our data hint at the fact that, in human K562 erythroleukemia cells, the modulation of PLCβ1 expression is able to exert an impairment of normal erythropoiesis as assessed by γ-globin expression.

Reference

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Key words

phospholipase Cβ1, erythropoiesis, K562, miR-210, γ-globin,