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Retinoic acid-induced *blr1* expression requires RARÎ±, RXR, and MAPK activation and uses ERK2 but not JNK/SAPK to accelerate cell differentiation

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Summary

Upstream signaling requirements of retinoic acid (RA)-induced *blr1* expression and downstream signaling consequences of *blr1* over-expression in a human myeloid leukemia cell line demonstrate that mitogen-activated protein kinase (MAPK) signaling complexes are involved in both avenues. RA-induced myeloid differentiation and G_1/G_0 growth arrest of HL-60 cells is known to require the activation of the RARÎ± and RXR retinoid receptors, as well as activation of the MAPK, ERK2. Transcriptional activation of the Burkitt's lymphoma receptor 1 (*blr1*) gene occurs early during RA-induced differentiation of HL-60 cells and requires these same three activating processes. The use of retinoid ligands that activate either the RARÎ± or the RXR retinoid receptors

use of retinoid ligands that activate either the RARΙ± or the RXR retinoid receptors revealed that *blr1* mRNA induction was detectable only when both RARΙ± and RXR were activated. Neither the RARΙ± nor RXR selective ligands alone induced expression of *blr1*, but the combination of the two ligands induced the expression of *blr1* to the same extent as RA. The MAPKK (MEK) inhibitor, PD98059, was used to determine whether extracellular signal-regulated kinase (ERK2) activation was necessary for induction of *blr1* mRNA. PD98059 inhibited induced *blr1* mRNA expression, due to RA or activated RARΙ± plus RXR ligands, indicating that ERK2 activation is necessary for *blr1* mRNA expression. Previous studies showed that ectopic expression of *blr1* also caused increased MAPK activation, in particular ERK2, and subsequently accelerated RA-induced differentiation and G_1/G_0 growth arrest. Inhibition of ERK2 activation inhibited differentiation of *blr1* transfectants, suggesting that the accelerated differentiation reflected *blr1*-enhanced ERK2 activation. The present data also demonstrate that ectopic expression of *blr1* increased JNK/SAPK activity, but JNK/SAPK activation was not needed for accelerated RA-induced differentiation and growth arrest. The results show that the signals known to be required for HL-60 differentiation, activated RARΙ±, RXR, and ERK2, are necessary for *blr1* mRNA expression. Downstream consequences of *blr1* over-expression include enhanced MAPK signaling.

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Key words

BLR1; retinoids; leukemic monocytic cell differentiation; MAPK

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