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K562 cell proliferation is modulated by PLCβ1 through a PKCα-mediated pathway

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Phospholipase C β1 (PLCβ1) is known to play an important role in cell proliferation. Previous studies reported aninvolvement of PLCβ1 in G0-G1/S transition and G2/M progression in Friend murine erythroleukemia cells (FELC). However,little has been found about its role in human models. Here, we used K562 cell line as human homologous of FELC inorder to investigate the possible key regulatory role of PLCβ1 during cell proliferation of this humancell line. Our studies on the effects of the overexpression of both these isoforms showed a specific and positive connection between cyclinD3 and PLCβ1 in K562 cells, which led to a prolonged S phase of the cell cycle and a delay in cell proliferation. In order to shed light on this mechanism, we decided to study the possible involvement of protein kinases C (PKC), known to be direct targets of PLC signaling and important regulators of cell proliferation. Our data showed a peculiar decrease of PKCα levels in cells overexpressing PLCβ1. Moreover, when we silenced PKCα, by RNAi technique, in order to mimic the effects of PLCβ1, we caused the same upregulation of cyclin D3 levels and the same decrease of cell proliferation found in PLCβ1-overexpressing cells. The key features emerging from our studies in K562 cells is that PLCβ1 targets cyclin D3, likely through a PKCα-mediated-pathway, and that, as a downstream effect of its activity, K562 cells undergo an accumulation in the S phase of the cell cycle.

References

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Key words
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