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Nuclear DGKα regulates cell cycle progression in K562 cells

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The existence of an independent nuclear inositide pathway distinct from the cytoplasmic one has been demonstrated in different physiological systems and in diseases (1). Phosphatidylinositols (PIs) play an important role in nuclear function regulation and behave differently from their counterparts in the cytoplasm. The autonomous nuclear PI cycle in eukaryotic cells is involved in different regulation processes, from cell proliferation to differentiation and many others (2). At nuclear level an array of kinases and phosphatases can modulate PIs. Among these, Diacylglycerol Kinases (DGKs) are a class of phosphotransferases that phosphorylate diacylglycerol (DAG) and induce the synthesis of phosphatidic acid. We Investigated DGKα localization and function in human erythroleukemia cell line K562. Synchronization experiments at different cell cycle checkpoints showed an important expression of DGKα in the nuclear fraction of this cell model, slightly peaking at G2/M. This suggested that DGKα might have a function in nuclear signaling. In particular, nuclear DGKα expression can modulate cell cycle progression, leading to changes in the phosphorylated status of the Retinoblastoma protein (pRb), thus, regulating G1/S transition: DGKα silencing or downregulation leads to impaired G1/S transition and its overexpression leads to S phase progression. The molecular mechanism by which nuclear DGKα controls pRb phosphorylation and therefore cell cycle regulation in K562 cell line are still unclear. Further studies are needed to better understand the role of DGKα in relation to other pivotal PIs involved in cell cycle regulation in the hematopoietic system.

References

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Keywords

Nuclear lipid signaling, cell cycle, DGK, DAG