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ΔNP63α AND YB1: A FUNCTIONAL INTERACTION CONTROLLING CELL PROLIFERATION AND MOTILITY

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P63, cold shock domain protein, PI3K signaling, cell proliferation, cell migration

TP63, a member of the p53 gene family, can be expressed as six different protein isoforms. ΔNp63α is the predominantly expressed p63 isoform in squamous epithelia and is restricted to cells with proliferative potential while it is downregulated in cells undergoing terminal differentiation. ΔNp63α plays a critical role in the morphogenesis of organs/tissues developing by epithelial-mesenchimal interactions such as the epidermis, teeth, hair and glands. Herein, we present evidences that the Y box-binding protein-1 (YB-1), a member of the cold shock domain superfamily of proteins, interacts with ΔNp63α but not with the smaller ΔNp63γ isoform. YB-1 is a transcriptional/translational factor involved in cell proliferation, migration and transformation but the molecular mechanisms governing the involvement of YB-1 in these processes are still unclear.YB-1 shuttles between the nucleus and cytoplasm playing different and even conflicting functions according to its sub-cellular localization. We present data showing that enforced expression of ΔNp63α induces YB-1 nuclear localization and accumulation. In the nuclear compartment ΔNp63α and YB-1 activates the expression of the catalytic subunit of the PI3K kinase (PI3KCA) thereby inducing Akt1 phosphorylation and activation of a pro-survival pathway. Furthermore, our data suggest a role for ΔNp63α in the control of cell motility and adhesion through interaction with YB-1. We also present data showing that ΔNp63α and YB1 colocalize in proliferative cells of epithelia. Our data implicate the existence of a ΔNp63α-dependent mechanism governing cell proliferation and migration through the physical association between ΔNp63α and YB-1, a newly isolated p63 molecular partner.