

PKC ϵ -dependent signalling in cardiac differentiation

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The Protein kinase C (PKC) family, composed by 12 different isoforms, plays a pivotal role in many biological contexts such as cell differentiation, proliferation and survival. PKC ϵ has been demonstrated to be relevant for cardio-protection as well as in ischemia-reperfusion injury (Budás et al. 2010). Transgenic mice over-expressing a constitutively active PKC ϵ show concentric hypertrophy (Takeishi et al. 2000) suggesting negative effects of a permanently active PKC ϵ in cardiac cells. Although the effects of PKC ϵ over-expression have been analyzed both from the physiological and morphological points of view, molecular studies of its consequences on early cardiac marker gene expression are still lacking.

On the other side Bone Marrow Mesenchymal Stem Cells (BMMSCs) can be induced to acquire a cardiac fate by treatment with 5-azacytidine (5-AZ) (Wakitani et al. 1995), representing a good *in vitro* model for cardiac differentiation studies.

We addressed the role of *in vivo* PKC ϵ over-expression on early cardiac genes (namely, *nkx2.5* and *gata4*) regulation. Our results suggest a negative role of PKC ϵ , mediated by ERK1/2, on expression of these two genes both *in vivo* and in *ex-vivo* rat BMMSCs, showing that this protein is a fine tuner of precursor cardiac cells.

References

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Key words

PKCepsilon, heart, nkx2.5, gata4, ERKs.