IJAE
Vol. 118, n. 2 (Supplement): 97, 2013
ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY

PKCε-dependent signalling in cardiac differentiation

Galli D[1,2], Gobbi G[1,2,3], Carubbi C[1], Di Marcantonio D[1], Masselli E[1], Mirandola P[1,2,3] and Vitale M[1,2,3]

[1] Department of Biomedical, Biotechnological and Translational Sciences (S.Bi.BI.T.) Unit of Anatomy, Istology and Embriology, University of Parma, 43126 Parma, Italy
[2] Interdepartmental Center of Molecular and Translational Oncology (COMT), University of Parma, 43126 Parma, Italy
[3] Center for Sport & Exercise Medicine – SEM, University of Parma, 43126 Parma, Italy

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. PKCe has been demonstrated to be relevant for cardio-protection as well as in ischemia-reperfusion injury (Budas et al. 2010). Transgenic mice over-expressing a constitutively active PKCε show concentric hypertrophy (Takeishi et al. 2000) suggesting negative effects of a permanently active PKCε in cardiac cells. Although the effects of PKCε 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* PKCε over-expression on early cardiac genes (namely, *nkx2.5* and *gata4)* regulation. Our results suggest a negative role of PKCε, 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

[1] Budas et al. (2010) Mitochondrial import of PKCepsilon is mediated by HSP90: a role in cardioprotection from ischaemia and reperfusion injury. Cardiovasc Res. 88: 83-92.
[2] Takeishi et al. (2000) Transgenic overexpression of constitutively active protein kinase C e causes concentric cardiac hypertrophy. Circ Res 86: 1218-1223.
[3] Wakitani et al. (1995) Myogenic cells derived from rat bone marrow mesenchymal stem cells exposed to 5-azacytidine. Muscle Nerve 18: 1417-1426.

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