sorted by cultured smooth muscle and nonmuscle cells. J. Cell Sci. 110: 839–846.

Nag A.C., Cheng M. 1981. Adult mammalian cardiac muscle cells in culture. Tissue Cell. 13: 515–523.

Otey C.A., Carpen O. 2004. Alpha-actinin revised: A fresh look at an old player. Cell Motil. Cytoskeleton. 58: 104–111.

Perrin B.J., Ervasti J.M. 2010. The actin gene family: function follows isoform. Cytoskeleton Hoboken. 6 : 630–634.

Ruzicka D.L., Schwartz R.J. 1988. Sequential activation of alpha-actin genes during avian cardiogenesis: vascular smooth muscle alpha-actin gene transcripts mark the onset of cardiomyocyte differentiation. J. Cell Biol. 107: 2575–2586.

Schaub M.C., Hefti M.A., Harder B.A., Eppenberger H.M. 1997. Various hypertrophic stimuli induce distinct phenotypes in cardiomyocytes. J. Mol. Med. 75: 901–920.

Van Bilsen M., Chien K.R. 1993. Growth and hypertrophy of the heart: Toward an understanding of cardiac specific and inducible gene expression. Cardiovasc. Res. 27: 1140–1149.

Vandekerckhove J., Bugaisky G., Buckingham M. 1986. Simultaneous expression of skeletal muscle and heart actin proteins in various striated muscle tissues and cells. J. Biol. Chem. 261: 1838–1843.

Von Arx P., Bantle S., Soldati T., Perriard J.C. 1995. Dominant negative effect of cytoplasmic actin isoproteins on cardiomyocytes cytoarchitecture and function. J. Cell Biol. 131: 1759–1773.

Wang F., Kovacs M., Hu A., Limouze J., Harvey E.V., Sellers J.R. 2003. Kinetic mechanism of non-muscle myosin-IIB: Functional adaptations for tension generation and maintenance. J. Biol. Chem. 278: 27439–27448.

Winegrad S., Wisnewsky C., Schwartz K. 1990. Effect of thyroid hormone on the accumulation of mRNA for skeletal and cardiac alpha–actin in hearts from normal and hypophysectomized rats. Proc. Nat. Acad. Sci. USA. 87: 2456–2460.

REDISTRIBUTION OF SARCOMERIC MYOSIN AND α-ACTININ IN CARDIOMYOCYTES IN CULTURE UPON THE REARRANGEMENT OF THEIR CONTRACTILE APPARATUS

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Cardiomyocytes in culture undergo reversible rearrangement of their contractile apparatus with conversion of typical myofibrils into structures resembling stress fibers of non-muscle cells. Such a rearrangement is accompanied by the replacement of cardiac actin, the main protein of myofibrils, with its smooth muscle isoform. This study reveals that along with the actin isoform replacement the key structural sarcomeric proteins release from the actin structures and are stored in cell cytoplasm as inclusions non-bound with actin. The data obtained is indicative of the incompatibility of smooth muscle actin with sarcomeric isoforms of these proteins and myofibrillar organization in general.

Keywords: cardiomyocytes in culture, contractile apparatus, myosin, alpha-actinin, actin