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Osteoglycin post-transcriptional regulation by miR-155 induces cellular architecture changes in H9c2 cardiomyoblasts

Grasieli de Oliveira[a], Paula Paccielli Freire[a], Ana Carolina Mieko Omoto[a], Sarah Santiloni Cury[a], César Seigi Fuziwara[b], Edna Teruko Kimura[b], Maeli Dal-Pai-Silva[a], Robson Francisco Carvalho[a]

[a]Department of Morphology, Institute of Biosciences of Botucatu, São Paulo State University, Botucatu, São Paulo, Brazil

[b]Department of Cell and Developmental Biology, Institute of Biomedical Sciences, University of São Paulo, São Paulo, Brazil

Corresponding author: Robson Francisco Carvalho Email: robson.carvalho@unesp.br

Abstract

Several studies have demonstrated dysregulated cardiac microRNAs (miRNAs) following cardiac stress and development of cardiac hypertrophy and failure. miRNAs are also differentially expressed in the inflammation that occurs in heart failure and, among these inflammatory-related miRNAs, the miR-155 has been implicated in the regulation of cardiac hypertrophy. Despite these data showing the role of miRNA-155 in cardiomyocyte hypertrophy under a hypertrophic stimulus, it is also important to understand the endogenous regulation of this miRNA without a hypertrophic stimulus to fully appreciate its function in this cell type. The first aim of the present study was to determine whether, without a hypertrophic stimulus, miR-155 overexpression induces H9c2 cardiac cells hypertrophy *in vitro*. The second objective was to determine whether osteoglycin (Ogn), a key regulator of heart mass in rats, mice, and humans, is post-transcriptionally regulated by miR-155 with a potential role in inducing H9c2 cells hypertrophy. Here, we show that, without a hypertrophic stimulus, miR-155 significantly repressed Ogn protein levels, but induce neither alteration in morphological phenotype nor in the expression of the molecular markers that fully characterize pathological hypertrophy of H9c2 cells. However, most importantly, Ogn silencing in H9c2 cells mimicked the effects of miR-155 overexpression in inducing cellular architecture changes that were characterized by a transition of the cell shape from fusiform to rounded. This is a new role of the post-transcriptional regulation of Ogn by miR-155 in the maintenance of the cardiac cell morphology in physiological and pathological conditions.

Keywords

Mimecan, cardiac cell morphology, cardiac pathological hypertrophy