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Connexin 26 expression in mammalian cardiomyocytes

[1] Francesco Bianchi - [2] Enza Polizzi - [2] Antonietta R.M. Sabbatini - [3] Silvia Del Ry - [3] Manuela Ciabati - [1] Stefania Moscato - [1] Letizia Mattii

[1] University of Pisa, Department of Clinical and Experimental Medicine, Pisa, Italia - [2] University of Pisa, Dept. of Surgical, Medical, Molecular Biology and Critical Care, Pisa, Italia – [3] CNR Pisa, Biochemistry and Molecular Biology Laboratory, Institute of Clinical Physiology, Pisa, Italia

Connexins (Cxs) are a family of membrane-spanning proteins named according to their molecular weight. They have been known to form membrane channels mediating cell-cell communication, which play an essential role in the propagation of electrical activity throughout the heart. So far, expression of seven isoforms, namely Cx30.2, Cx37, Cx40, Cx43, Cx45, Cx46 and Cx57, have been found in cardiac myocytes (1,2). Cx26 has been described in a number of tissues but not yet in the heart, and its mutations are frequently associated with deafness and skin diseases (3,4). To our knowledge, the expression of Cx26 also in human, pig, rat and mouse cardiomyocytes has been demonstrated for the first time in the present study. Interestingly, this Cx was found as scattered throughout cell cytoplasm but not at level of the intercalated disks where the other cardiac Cxs are mainly located. Furthermore, in cardiomyocytes of a pig model of left ventricular dysfunction (LVD), Cx26 expression was modulated and dipyridamole treatment, which was previously demonstrated to have a protective action on left ventricular function (5), was associated to an increased Cx26 expression. Dipyridamole induced the same effect in cardiac rat cell line H9c2. For our study, paraffin embedded sections of human auricle, pig ventricle, mouse whole heart and H9c2 cells were used. Several methods were employed to test the expression of Cx26. In particular, different immunohistochemical and molecular biology techniques were performed by using two types of primary anti-Cx26 antibodies to ascertain the specificity of cardiomyocyte immunopositivity for Cx26 avoiding analysis-dependent artifacts.

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Keywords

Connexin 26; connexin 43; cardiomyocytes; human; pig; mouse; immunohistochemistry; dipyridamole.

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