

A51

Ion channels alteration in dilated cardiomyopathy in human heart

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The complex mechanism by which genes are mostly involved in cardiomyopathy (clinical heart failure) is not resolved. Cardiac ion channels are shown as a mixture of homomeric and heteromeric tetramers composed of closely related subunits from homo- or heteromeric assembly of α and β subunits, and underlie the main part of the currents. The aim of this study was to compare the expression of the ion channels and regulatory genes in ventricular muscle between failing and non-failing human heart. We also tested the marker genes of inflammation, fibroblast, neuronal tissues and vascular vessels. Therefore, we have quantified the pore-forming α and auxiliary β subunit-coding mRNAs of I_{Ca} , I_{Na} , I_{to} , I_{Kr} , I_{Ks} , I_{K1} channels and regulatory genes by real-time qPCR (BioTrove) in left ventricle of heart tissues. Coding genes for sodium ion channels have marked differences in failing and control tissues of left ventricular muscle. Our results suggest that coding genes for Na^+/Ca^{2+} exchange current is mostly upregulated in dilated cardiomyopathies patients heart while $Na_v1.5$ is much less. Potassium ion channel α and β subunits exhibit different distribution in the failing ventricle tissues versus control. These findings may provide insight into a mechanism responsible for hearth failure, DCM, HCM and RCM due to several regulatory genes-related up- or down-regulation of sodium, calcium, chloride and potassium ion channels.

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