

## Effect of dipyridamole on gap junctions regulation in diseased myocardium

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Gap junctions (GJ) mediate electrical coupling between cardiac myocytes, allowing the spreading of the electrical wave responsible for synchronized contraction [1]. GJ function can be regulated by modulation of connexon densities on membranes, connexin (Cx) phosphorylation, trafficking and degradation. Recent studies showed that adenosine involves Cx43 turn-over in A<sub>1</sub> receptor-dependent manner [2], and dipyridamole increases GJ coupling and amount of Cx43 in endothelial cells [3].

As the abnormalities in GJ organization and regulation have been implicated in diseased myocardium [1], the aim of the present study was to assess the regional expression of molecules involved in GJ regulation in a model of left ventricular dysfunction (LVD). For this purpose the distribution and quantitative expression of Cx43, its phosphorylated form pS368-Cx43, PKC phosphorylated substrates, RhoA and A receptors, were investigated in experimental models of right ventricular-pacing induced LVD, undergoing concomitant dipyridamole therapy or placebo, and compared with healthy myocardium obtained from sham operated minipigs.

Results demonstrates that an altered pattern of factors involved in Cx43-made GJ regulation is present in ventricular myocardium with left ventricular dysfunction. Moreover, the dipyridamole treatment, that results in an improvement of heart function, seems to act also modulating expression and activation of these factors.

### References

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### Keywords

Cx43, RhoA, PKC, heart disease, dipyridamole.