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A2A and A3, Adenosine receptors mRNA are overexpressed in an experimental animal model of myocardial infarction

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Background: Adenosine, a purine nucleoside and a "retaliatory metabolite" in ischemia, is ubiquitous in the body, and increases 100-fold during ischemia. Its biological actions are mediated by four adenosine receptors (ARs): A1 and A3, coupled to Gi/o, and the high-affinity A2A and low-affinity A2B, coupled to Gs. Because A1R and A3R are distributed mainly in myocardial cells and A2 are on coronary vascular smooth cells in the heart, adenosine may substantially modulate cardiac function as a whole.

Aim: To determine possible myocardial alterations in the expression of ARs, in an experimental animal model of myocardial infarction (MI).

Materials and Methods: Left ventricular (LV) tissue was collected from male adult minipigs with MI (n=5), induced by permanent surgical legation of the left anterior descending coronary artery and from 5 healthy pigs. mRNA expression of A1R, A2AR, A2BR,A3R was determined by semi-quantitative RT-PCR in tissue sampled collected from border (BZ) and remote zones (RZ) of infarcted area.

Results: Transmural infarction affected about 15% of the LV wall mass. After 4 weeks, mRNA expression was higher in infarct regions than in control for A1R (controls=2.0±1.0, BZ=2.4±0.4, RZ=1.2±0.1), A2AR (controls=0.6±0.3, BZ=1.9±0.2, RZ=1.3±0.04 p=0.002, p=0.04, controls vs. BZ and RZ), A2BR (controls=1.1±0.5, BZ=1.2±0.2, RZ=0.5±0.04) and A3R (controls=0.2±0.07, BZ=2.4±0.7, RZ=0.7±0.07, p=0.006, p=0.002, controls vs. BZ and RZ).

Conclusion: All adenosine receptors, and expecially A2A and A3, are overexpressed in the BZ of MI, consistently with an adaptative retaliatory anti-ischemic adenosinergic changes of post-infarcted heart.