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BIOCHEMICAL MARKERS OF CARDIAC REMODELING

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Introduction: Cardiac remodelling is one of the pathogenic pathways leading to heart failure, which occurs due to ischemic, mechanical and inflammatory damage to cardio myocytes and cardiac interstitium.

Purpose: Detection of biochemical markers of cardiac remodeling in the context of ischemic myocardial injury**.**

Material and methods: Have been analysed 82 bibliographic sources published during the 2000-2019 in the electronic databases Medline, PubMed, Medscape, Hinari and Google Academic, as well as from the Medical Scientific Library of “Nicolae Testemitanu” State University of Medicine and Pharmacy.

Results: The possible mechanisms by which galectin-3 mediates cardiac fibrosis have been explored by a number of groups (Figure 1). Direct evidence has shown that recombinant galectin-3 can convert silent fibroblasts into myofibroblasts and induce cardiac fibroblast proliferation, TGF-β synthesis, collagen production.Was demonstrated that increasing galectin-3 protein expression by the galectin-3 gene promotes collagen I synthesis in HL-I cardiomyocytes, which promotes cardiac decompensation.Others studies revealed that galectin-3 can promote oxidative stress in human cardiac fibroblasts, a novel mechanism of galectin-3-induced cardiac damage. Oxidative stress is a disturbance in the balance between reactive oxygen species (ROS) production and antioxidant detoxification. In patients with HF, oxidative stress occurs in the myocardium and correlates with left ventricular dysfunction.

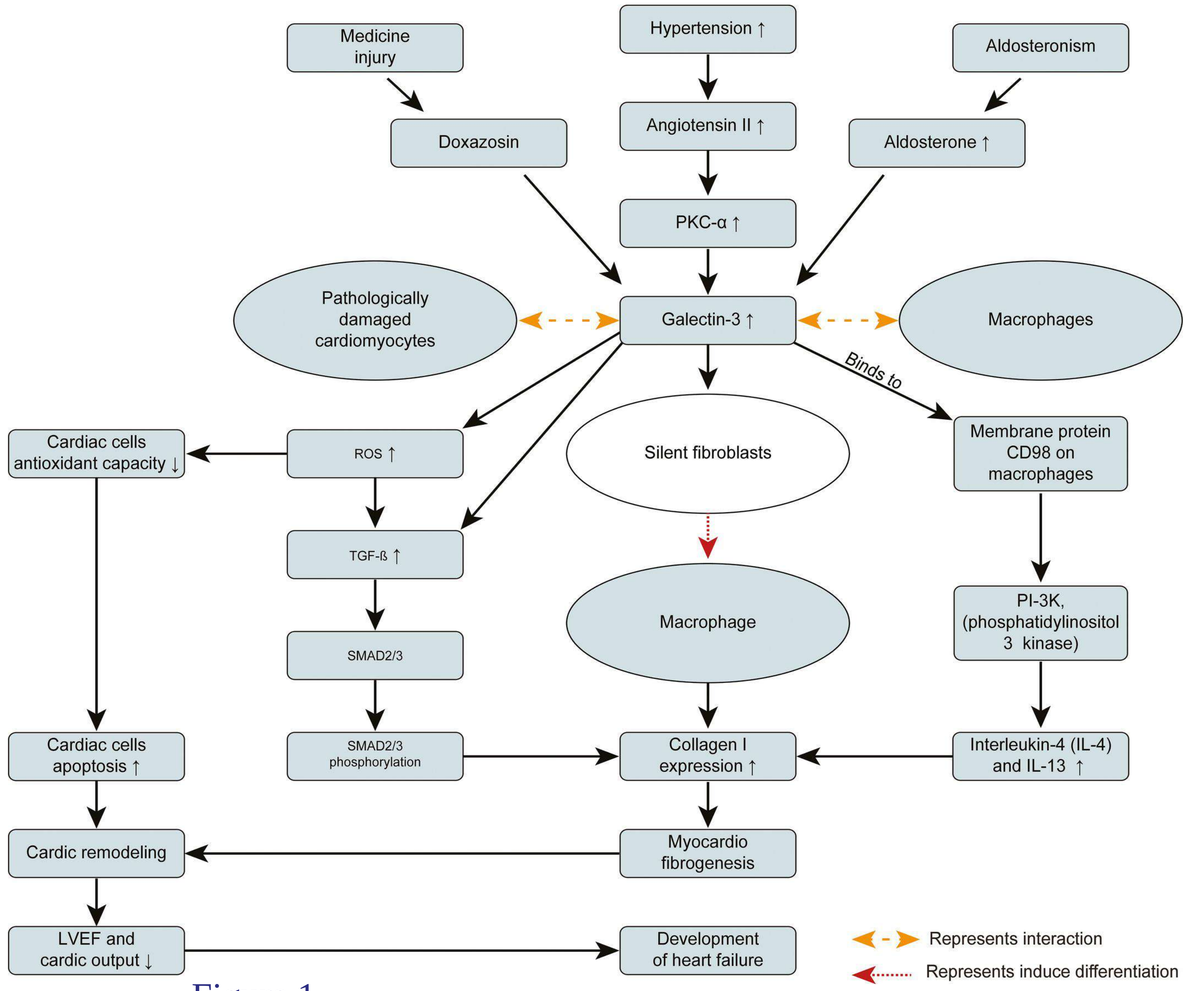

Figure 1

Conclusions: Accumulating studies demonstrate that galectin-3 is upregulated in clinical and experimental HF and plays an important role in the pathogenesis of cardiac fibrosis. Inhibition of galectin-3 activation after heart injury may provide an alternative therapeutic approach in the prevention and treatment of HF.

Keywords: cardiac remodeling, oxidative stress, fibrosis, Galectin-3.