**INFLAMMATION FOLLOWING MYOCARDIAL INFARCTION PLAYS AN IMPORTANT ROLE IN COLLAGEN SYNTHESIS AND CARDIAC REMODELLING**

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Current therapies substantially ameliorate the prognostic after MI, but do not restore the organ structure and function. Therefore, the main goal of the modern cardiology is to design strategies to optimize cardiac repairing and remodelling. The increased collagen content was associated with decreased ventricular function, therefore inhibition of myofibroblast was considered an appropriate therapeutical goal. Recently was shown that increased collagen content even supports the contractility and preserves heart function. Our hypothesis is that leukocyte infiltration would be able to influence the structure of the final scar and implicitly the function of the left ventricle. Cultured myofibroblasts produce mainly collagen type IV and I, but also significant amounts of collagen VI, XII, XIV, XVIII and III. Coincubation with mononuclear cells in hypoxic conditions reestablish the levels of collagen I and to a certain extent collagen IV and reduces the levels of collagen VI. Coincubation with neutrophils decreases significantly the level of collagen XII. Samples from control heart showed increased levels of collagen XIII, XVII, IX and X, and moderate levels from the other subtypes. The final scar presented reduced levels of collagen XIII, XVII, IX, X, but increased levels of collagen III, VIII, XIV. Beside the similarities in collagen III and XIV, the differences between the in vitro and in vivo experiments demonstrate the existence of multiple unknown factors influencing collagen synthesis. Collagen synthesis is a complex process in which the inflammatory reaction can play a decisive role. Manipulating leukocyte recruitment can provide a tool for controlling scar composition.