Reply

Growth hormone and interstitial fibrosis

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Beranek suggests that growth hormone rather prevents than reduces interstitial fibrosis in non-infarcted areas following experimental myocardial infarction in rats. We appreciate his thorough reading of our paper and his important contribution on the possible mechanisms of growth hormone on myocardial structure.

Indeed, we agree that Fig. 3B (showing the non-infarcted area of the left ventricle in an untreated rat), visualizes reactive as well as reparative fibrosis [1]. His conclusions fit well in the current concept of the complex remodeling process following myocardial infarction.

In our paper, we had to address a great number of different parameters, that had been obtained by appropriate methods. Extracellular matrix proteins were measured by quantitative image analysis to assess interstitial fibrosis of the myocardium, which has significant impact on left ventricular performance. However, we had not measured cardiomyocyte number nor the amount of adaptive versus reparative interstitial fibrosis. For the purpose of our study hypothesis, interstitial fibrosis of either origin was measured and found to be significantly reduced by growth hormone as compared to vehicle along with enhanced cardiomyocyte hypertrophy [1]. It would be of particular interest, to focus on adaptive fibrosis in non-infarcted areas. However, this would request a complex different methodological approach compared to the one we used in this study. Meanwhile, we started to investigate apoptosis. 4 weeks after the initial insult, apoptosis was not detected in non-infarcted areas (treated and untreated) using TUNEL staining (unpublished results).

However, before a clear conclusion on the potential role of apoptosis is possible, its time-course has to be assessed. Furthermore, the role of neurohormonal systems like the renin-angiotensin-aldosterone system and the sympathetic nervous system, has to be coinvestigated. They are considerably involved in the progression of heart failure and recent studies [2–4] could demonstrate important – partially dual – effects on apoptosis (angiotensin II).

Progress results from hard data. Because we had not assessed any of the possibly involved mechanisms, that were mentioned by Beranek, we restricted our discussion on interstitial fibrosis. However, we share his stimulating speculations about potential mechanisms of growth hormone, that must be addressed by further studies.

References


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