

5. SUMMARY

In summary, in the present study it is shown that cultured cardiomyocytes and aortic vascular smooth muscle cells are sources of bradykinin B1 and B2 receptors. In cultured cardiomyocytes, stimulation of cytokine IL1 β significantly upregulated both B1R and B2R mRNA expression. This effect was much more stronger on B1R (a factor of 25 fold) than on B2R (a factor of 3 fold). In aortic vascular smooth muscle cells, expression of both B1 and B2 receptor mRNA could be detected, however, IL1 β treatment did not influence their expression in the cells. In cardiac fibroblasts, expression of both B1 and B2 receptor mRNA was not detectable, and the addition of IL1 β could not induce their expression. *In vivo* studies showed that, after MI induction, administration of IL1 β converting enzyme inhibitor downregulated the bradykinin B1 receptor expression in the heart, but this treatment caused no significant changes in B2R expression. These data suggest the known elevated release of cytokine IL1 β early after myocardial infarction may, at least in part, upregulate bradykinin B1 and B2 receptor expression through a CMC-specific pathway.