Results: FGFR1 and GAPDH.

Materials and Methods: Fracture model: Closed middiaphyseal fractures and analyzed the spatial and temporal gene expression for bFGF and FGFR1. In the present study, we employed a standardized rat model of fracture healing and assessed the gene expression of FGF ligands and receptors have not yet been fully established. Based on the results, we discuss a possible role for FGF-FGFR1 signaling in fracture repair.

Discussion: The present results demonstrate that gene expression for FGFR1 is rapidly upregulated after fracture, and maintained at relatively high levels even in the later stages of healing. In spite of such widespread expression of FGFR1, little expression for bFGF mRNA was detected by our ISH and RPA analyses. Previous studies showed that a considerable amount of bFGF peptide is stored in bone matrix (5). Thus we suggest that, during fracture healing, FGFR1 is produced locally but its ligand (bFGF) is mainly released from the bone matrix. From the expression patterns of FGFR1, we suggest that FGF-FGFR1 signaling may preferentially contribute to cell proliferation during initial callus formation, and to bone remodeling in the later stages. In this study, we also revealed that FGFR1 mRNA was expressed not only in osteoblastic cells but also in mature osteoclasts in fracture calluses, suggesting that FGFs directly act on osteoclasts in vivo. When bFGF will be used for fracture treatment, it should be noted that role of FGF-FGFR1 signaling may not necessarily be consistent throughout healing, but may vary depending on the healing stage.


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