Recent clinical reports have pointed out that basic fibroblast growth factor (bFGF) promotes scarless wound healing. The aim of present study is to elucidate this mechanism. Previous studies reported that 3-10 days of transforming growth factor-β (TGFβ) treatment were required to activate fibroblasts to express the myofibroblast phenotype — increased expression of α-smooth muscle actin (αSMA). Then we employed this technique to obtain myofibroblasts. bFGF well stimulated fibroblast-collagen gel contraction but not myofibroblast collagen gel contraction. Subsequent studies were carried out to determine if co-stimulation of fibroblasts with TGFβ and bFGF affected their ability to express the myofibroblast phenotype. In these experiments, fibroblasts in monolayer were incubated with or without 1 ng/ml of bFGF in the presence of 10ng/ml of TGFβ, harvested, and then tested for the expression level of αSMA. Levels of cellular αSMA increased after 2-4 days of TGFβ treatment alone and were consistently elevated after 5 days with unchanged levels of total actin. However it is of note that levels of cellular αSMA increased after 4-6 days of bFGF and TGFβ treatment. Other experiments were carried out to test the effects of bFGF on myosin II regulatory light chain (MLC) phosphorylation on fibroblasts and myofibroblasts. After 30 min of bFGF, levels of diphosphorylated MLC were highest in fibroblasts. In contrast, levels of diphosphorylated MLC were still elevated in myofibroblasts spontaneously, and were consistently unchanged even if stimulated with bFGF. Our results indicated that bFGF had certain roles on the expression of myofibroblast phenotype by fibroblasts and MLC phosphorylation by myofibroblasts.