OBJECTIVE: To study the role of IIIb isoform of human fibroblast growth factor receptor 1 (FGFR1-IIIb) in proliferation of pancreatic ductal cells and its effects on mitogen-activated protein kinase (MAPK).

METHODS: Human pancreatic ductal cells of the line TAKA-1 were cultured. The plasmid of human full-length FGFR1-IIIb isoform, pSVK4/FGFR1-IIIb, was stable transfected into the cultured TAKA-1 pancreatic ductal cells facilitated by lipofectamine. Un-transfected TAKA-1 cells and TAKA-1 ductal cells transfected with blank plasmid were used as controls. The expression, distribution and character of protein of FGFR1-IIIb in the TAKA-1 cells were estimated by Western blotting, Northern blotting, immunofluorescence assay, and glycosylation assay. The function and mechanism of FGFR1-IIIb in the transfected pancreatic ductal cells stimulated by FGF were examined by MTT assay and MAPK assay. Tunicamycin, an inhibitor of N-terminal glycoprotein synthesis, was added into the culture fluid of the FGFR1-IIIb transfected TAKA-1 cells to observe the changes of the FGFR1 bands.

RESULTS: FGFR1-IIIb, a glycosylated receptor at various levels at 120 kDa and between 130 - 150 kDa, was localized at moderate levels at the cell membrane and cytoplasm and at higher level in the perinuclear region of the cytoplasm of the pSVK4/FGFR1-IIIb-transfected cells. FGF-1, -2, and -4 significantly increased the growth of FGFR1-IIIb-transfected TAKA-1 cells, and at the same time induced the p44/p42 MAPK phosphorylation.

CONCLUSION: Human FGFR1-IIIb receptor is a functional receptor in pancreatic ductal cells. FGF-1, -2, and -4 can increase the growth of FGFR1-IIIb-transfected pancreatic ductal cells, and the mechanism is that they can induce the p44/p42 MAPK phosphorylation.

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