

# New molecular therapy candidates for pancreatic cancer

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A research team from Japan investigated expression of insulin-like growth factor-I receptor (IGF-IR) in pancreatic cancer cell lines. All the cell lines examined expressed IGF-IR under culture conditions without IGF-I in the medium. They suggest that IGF-IR and phosphatidylinositol 3 kinase are good candidates for molecular therapy of pancreatic cancer.

Insulin-like growth factor-I (IGF-I) is upregulated in human pancreatic cancer tissues but is not expressed in surrounding non-cancerous tissues. Serum level of IGF-I is elevated in pancreatic cancer patients.

Histological analysis has shown that IGF-I receptor (IGF-IR) is positive in the membrane of pancreatic cancer tissues. These facts suggest that IGF-I acts as a growth factor for pancreatic cancer and inhibition of its action might be a good candidate for molecular therapy of pancreatic cancer. A possible problem is that not all pancreatic cancers produce IGF-I, which might be a reason for ineffective results of its clinical application.

A research team from [Japan](#) have proved that inhibition of IGF-IR activity results in a decrease in proliferation and motility of pancreatic cancer cell lines. Their study will be published on April 21, 2010 in the *World Journal of Gastroenterology*.

Their study indicated that IGF-IR was expressed and played a role in [proliferation](#) and motility of pancreatic cancer cell lines. Further analysis of this phenomenon could unveil a new role for a growth factor receptor and its downstream pathway in cancer. One possible mechanism would

be that the downstream pathway stimulates its upstream receptor via some unknown molecule, like a retrograde flow.

**More information:** Tomizawa M, Shinozaki F, Sugiyama T, Yamamoto S, Sueishi M, Yoshida T. Insulin-like growth factor-I receptor in proliferation and motility of pancreatic cancer. World J Gastroenterol 2010; 16(15): 1854-1858.

[www.wjgnet.com/1007-9327/full/v16/i15/1854.htm](http://www.wjgnet.com/1007-9327/full/v16/i15/1854.htm)

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