

# Experimental insulin-like growth factor receptor inhibitor reduced pancreatic cancer growth

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Researchers at Amgen are testing a fully human monoclonal antibody that inhibits the activity of insulin-like growth factors (IGF-1 and IGF-2) and appears to reduce pancreatic cancer cells in early testing, according to a report in *Molecular Cancer Therapeutics*, a journal of the American Association for Cancer Research.

Pancreatic cancer is one of the deadliest cancers, and less than 4 percent of the 200,000 patients diagnosed annually live more than five years. The only available clinical treatment is gemcitabine, but this has yet to show a survival benefit.

Scientists are testing a variety of experimental therapies to bring pancreatic cancer under control. At Amgen, Pedro J. Beltran, Ph.D., a principal scientist in oncology research, is experimenting with AMG 479, a fully human anti-IGF-1 monoclonal antibody.

"We know that insulin-like growth factors play a role in cancer development, particularly in mediating cell survival. This is the first drug that specifically targets the receptor for these growth factors without cross-reacting with the closely related insulin receptor," said Beltran.

In the in vitro study, AMG 479 bound to IGF-1R and blocked both IGF-1 and IGF-2 binding factors 1 and 2. It also completely inhibited ligand-induced activation in some growth factors, which led to a decreased cellular viability. When Beltram and colleagues measured the effect of AMG 479 on pancreatic cancer cells in vivo, the inhibition rate was approximately 80 percent inhibition of [tumor growth](#) and receptor expression was observed.

"These data clearly show that AMG 479 is a clinical candidate for pancreatic [cancer therapy](#), either alone or in combination with gemcitabine,"

he said.

Beltran said researchers are currently testing AMG 479 in nine separate phase II studies of various cancer types; he expects the effect will be seen beyond [pancreatic cancer](#).

Source: American Association for Cancer Research ([news](#) : [web](#))

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