

Pharmaceutical sciences researchers to publish paper

15 November 2011

Erxi Wu, assistant professor of pharmaceutical sciences, and Shuang Zhou, doctoral student of pharmaceutical sciences in Wu's lab, co-wrote the article, "High glucose promotes pancreatic cancer cell proliferation via the induction of EGF expression and transactivation of EGFR," which will be published by *PLoS ONE*.

The paper was co-written with Qingyong Ma lab at Xi'an Jiaotong University, China. "We have established a productive collaboration with the Ma lab in finding cancer therapeutics and elucidating the mechanisms of the targeted therapy for pancreatic cancer, one of the most lethal malignancies," Wu said.

According to the authors, multiple lines of evidence suggest that a large portion of [pancreatic cancer](#) patients suffer from either hyperglycemia or diabetes, both of which are characterized by high blood glucose level. However, the underlying biological mechanism of this phenomenon is largely unknown. The authors demonstrated that the proliferative ability of two human pancreatic cancer cell lines, BxPC-3 and Panc-1, was upregulated by [high glucose](#) in a concentration-dependent manner.

Provided by North Dakota State University

Furthermore, the promoting effect of high glucose levels on epidermal growth factor (EGF) transcription and secretion, but not its receptors in these pancreatic cancer cell lines, was detected by using an epidermal growth factor-neutralizing antibody and reverse transcription polymerase chain reaction. In addition, the epidermal growth factor receptor transactivation is induced by high glucose levels in concentration- and time-dependent manners in pancreatic [cancer cells](#) in the presence of the epidermal growth factor-neutralizing antibody. These results suggest that high glucose promotes pancreatic cancer cell proliferation via the induction of epidermal growth factor expression and transactivation of [epidermal growth](#) factor receptor.

"Our findings may provide new insight on the links between high glucose level and pancreatic cancer in term of the molecular mechanism and reveal a novel therapeutic strategy for pancreatic cancer patients who simultaneously suffer from either diabetes or hyperglycemia," senior author Wu said.

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