BK channel is functionally expressed in neuroendocrine pancreatic tumor QGP-1 cells.

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Pancreas neuroendocrine tumors (p-NETs) are comprise of peptide-secreting tumours often with a functional syndrome. Among them, somatostatinoma is a rare disease and its pathogenic mechanism has not explained yet. Accounting for a small percentage of all pancreatic tumors, they have a good overall survival rate when diagnosed early, with surgery being curative. However, about 70% of the patients have liver metastasis and the 5-year survival rate is 30～40 %. The present pharmacotherapy is applied in the case of p-NET diagnosed to be unresectable and having a high grade of malignancy. However, there are few options of therapeutic drugs based on the pathophysiological mechanism of somatostatinoma.BK channel is expressed in many types of cancer cells and regulates the proliferation and metastasis. Each BK channel consists of a tetrameric assembly of four pore-forming α subunits (BKα) with regulatory β or γ subunits (BKβ and BKγ). Both regulatory subunits have tissue-specific distribution, contributing to tissue-dependent characteristics of electrophysiological kinetics and Ca^{2+} sensitivity of BK channel. However, the functional roles of BK channel in somatostatinoma are not clear. Whole-cell patch-clamp data showed that in neuroendocrine pancreatic tumor QGP-1 cells, BK channel currents were evoked by depolarization at pCa 6.5 and pCa 8.0 in pipette solution and suppressed by the treatment of 1mM paxilline, a specific BK channel blocker.

Expression analyses revealed BKα, BKβ2～4, and BKγ1 were expressed in QGP-1 cells at mRNA and protein levels. The after hyperpolarization in action potential (AP) was depolarized by the treatment of PAX.These results suggest that BK channel contributes somatostatin secretion and malignancy in QGP-1 cells.