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Elusive liver factor that causes pancreatic α cell hyperplasia: A review of literature

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Abstract

Tumors and cancers of the gastrointestinal tract and pancreas are commonly derived from precursor lesions so that understanding the physiological, cellular, and molecular mechanisms underlying the pathogenesis of precursor lesions is critical for the prevention and treatment of those neoplasms. Pancreatic neuroendocrine tumors (PNETs) can also be derived from precursor lesions. Pancreatic α cell hyperplasia (ACH), a specific and overwhelming increase in the number of α cells, is a precursor lesion leading to PNET pathogenesis. One of the 3 subtypes of ACH, reactive ACH is caused by glucagon signaling disruption and invariably evolves into PNETs. In this article, the existing work on the mechanisms underlying reactive ACH pathogenesis is reviewed. It is clear that the liver secretes a humoral factor regulating α cell numbers but the identity of the liver factor remains elusive. Potential approaches to identify the liver factor are discussed.

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