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Titel des Beitrags: Neural plasticity in pancreatitis and pancreatic cancer.

Abstract: Pancreatic nerves undergo prominent alterations during the evolution and progression of human chronic pancreatitis and pancreatic cancer. Intrapancreatic nerves increase in size (neural hypertrophy) and number (increased neural density). The proportion of autonomic and sensory fibres (neural remodelling) is switched, and are infiltrated by perineural inflammatory cells (pancreatic neuritis) or invaded by pancreatic cancer cells (neural invasion). These neuropathic alterations also correlate with neuropathic pain. Instead of being mere histopathological manifestations of disease progression, pancreatic neural plasticity synergizes with the enhanced excitability of sensory neurons, with Schwann cell recruitment toward cancer and with central nervous system alterations. These alterations maintain a bidirectional interaction between nerves and non-neural pancreatic cells, as demonstrated by tissue and neural damage inducing neuropathic pain, and activated neurons releasing mediators that modulate inflammation and cancer growth. Owing to the prognostic effects of pain and neural invasion in pancreatic cancer, dissecting the mechanism of pancreatic neuropasticity holds major translational relevance. However, current in vivo models of pancreatic cancer and chronic pancreatitis contain many discrepancies from human disease that overshadow their translational value. The present Review discusses novel possibilities for mechanistically uncovering the role
of the nervous system in pancreatic disease progression.

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