CYREX® Laboratories

CLINICAL SPECIFICATIONS

ENTERIC NERVE + VASOACTIVE INTESTINAL POLYPEPTIDE

Function:

Enteric neurons (ENs) represent a vast neural network that is organized into two major ganglia, the myenteric and submucosal plexuses. This neural network is distributed throughout the entire alimentary tract and extends out to the biliary tract and pancreas.[1] ENs are involved in the sensory information sent between the enteric nervous system (ENS) and the central nervous system (CNS). Vasoactive Intestinal Peptide (VIP) is a widely distributed neuropeptide in both the central and peripheral nervous system. It acts as a neuromodulator in many organs/tissues including heart, lung, thyroid gland, kidney, immune system, urinary tract, and genital organs. VIP has also been shown to inhibit LPS-induced production of inflammatory cytokines.

Associated With:

Inflammatory enteric neuropathy[1,2]
Ganglionitis[1,2]
Multiple sclerosis with constipation[3,4]
Gut dysmotility[2]
Asthma[5]

Known Cross-Reactions: $A\beta_{42}$ peptide[6]

Clinical Significance:

ENs are the major source of neuropeptides called tachykinins, which secrete proinflammatory cytokines into the mucosa during inflammatory episodes, exacerbating the intestinal inflammation.[7] Intestinal inflammation leads to gastrointestinal complications with motility. Patients with neurological disorders including multiple sclerosis,[3,4] Alzheimer's or Parkinson's diseases often manifest symptoms suggestive of disturbed bowel transit, including either diarrhea or constipation.[2] These associated conditions are likely due to underlying ENS dysfunction. Irritable bowel syndrome (IBS) is a major gut functional disorder that affects 10-20% of the population worldwide. The loss of many neurons and autoimmune reactivity against ENs may be important factors in underlying symptoms such as abnormal defecation that is associated with abnormal pain. Anti-ENs anti bodies are detected in up to 84% of patients with gut motility disorders. This suggests that autoimmune damage and loss of neurons in the ENS contributes to the disease pathology.[8] While ENs can contribute to inflammation and gut motility disorders, VIP acts as an inflammation inhibitor.[8] In the gut, VIP controls the inflammatory effects of intestinal lipopolysaccharides (LPS) and other bacterial toxins which are potent triggers of inflammation. The depletion of VIP by specific antibodies may interfere with VIP regulation of T cells and inflammatory cells and result in further production of autoreactive immunological responses.[9] If VIP actions are dampened, inflammation and autoimmunity may ensue. Vojdani and Vojdani demonstrated a significant antibody cross-reactivity between $A\beta_{42}$ and both ENs antigens and VIP;[6] based on this, it is likely that antibodies made against $A\beta_{42}$ may contribute to gut irritation and motility disorder, as well as inhibit the anti-inflammatory action of VIP. Therefore, if IBS and gut motility disorders are not properly treated, the end result may be AD and other neurodegenerative disorders.

References:

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