Escuela de Osteopatía de Madrid Internacional

HEPATOLOGÍA
BIOQUÍMICA POR ALIMENTACIÓN

HÍGADO Y NERVIO VAGO

AL ENFRENTARNOS AL ESTUDIO Y TRATAMIENTO DEL SNV:

Y si irritamos el vago a nivel hepático: ¿qué ocurre?

La excitación de las aferentes vagales que inervan el hígado, estómago distal y duodeno proximal y que se proyectan a través de la rama hepatica del vago abdominal→ desencadenan durante la inflamación en la víscera:

1.- respuestas de enfermedad (inmovilidad, reducción de la interacción social)

2.- descenso en la ingesta, aversion a nueva comida, anorexia,

3.-fiebre

4.- aumento del sueño,

5.-cambios endocrinológicos, malestar

6.-comportamiento hiperalgésico).

Auton Neurosci. 2000 Dec 20;85(1-3):49-59.

Vagal immune-to-brain communication: a visceral chemosensory pathway.

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Author information

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Abstract

The immune system operates as a diffuse sensory system, detecting the presence of specific chemical constituents associated with dangerous micro-organisms, and then signalling the brain. In this way, immunosensation constitutes a chemosensory system. Several submodalities of this sensory system function as pathways conveying immune-related information, and can be classified as either primarily brain barrier associated or neural. The vagus nerve provides the major neural pathway identified to date. The initial chemosensory transduction events occur in immune cells, which respond to specific chemical components expressed by dangerous micro-organisms. These immune chemosensory cells release mediators, such as cytokines, to activate neural elements, including primary afferent neurons of the vagal sensory ganglia. Primary afferent activation initiates local reflexes (e.g. cardiovascular and gastrointestinal) that support host defense. In addition, at least three parallel pathways of ascending immune-related information activate specific components of the illness response. In this way, immunosensory systems represent highly organized and coherent pathways for activating host defense against infection.

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Estas células quimiosensoriales inmunes liberan mediadores, tales como citocinas, para activar elementos neurales, incluyendo neuronas aferentes primarias de los ganglios sensoriales vagales.

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La activación aferente primaria inicia reflejos locales (p. ej., cardiovasculares y gastrointestinales) que apoyan la defensa del huésped.
Además, al menos tres vías paralelas de información relativa a la inmunidad ascendente activan componentes específicos de la respuesta a la enfermedad.
De esta manera, los sistemas inmunosensoriales representan vías altamente organizadas y coherentes para activar la defensa del huésped contra la infección

El sistema inmune funciona como un sistema sensorial difuso, detectando la presencia de componentes químicos específicos asociados con microorganismos peligrosos, y luego señalizando al cerebro.

De esta manera, la inmunosensibilidad constituye un sistema quimiosensorial.
Varias submodalidades de este sistema sensorial funcionan como vías de transmisión de información relacionada con el sistema inmune, y pueden clasificarse como principalmente barrera cerebral asociada o neural.

El nervio vago proporciona la principal vía neural identificada hasta la fecha. Los eventos iniciales de transducción quimiosensorial ocurren en las células inmunes, que responden a componentes químicos específicos expresados por microorganismos peligrosos.

Y si irritamos el vago a nivel hepático : ¿ qué ocurre? Y… qué aporta la osteopatía → reduce TNFalfa.

Se cree que las aferentes vagales que inervan el hígado se activan medinte citoquinas proinflamatorias [IL-1], IL-6, factor de necrosis tumoral alfa[TNF] liberados por las células de Kuppfer hepáticas (que son macrófagos activados), células dendríticas y leucocitos que señalizan estos fenómenos al cerebro y resultan en respuestas de enfermedad.

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J Auton Nerv Syst. 1996 Dec 14;61(3):287-91.

The afferent discharges from sensors for interleukin 1 beta in the hepatoportal system in the anesthetized rat.

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Abstract

The effect of intraportal administrations of interleukin 1 beta (IL-1 beta) on the afferent activity of the hepatic branch of the vagus nerve was observed in urethane anesthetized rats. An intraportal injection of IL-1 beta in doses of 10 pg and 100 pg per animal (300-400 g body wt.) resulted in dose-dependent increase in the afferent activity, which lasted about 70-100 min. Further, intraportal injection of IL-1 beta (100 pg) induced reflex activation of efferent activity of the splenic (sympathetic) nerve and vagal thymic nerve. This reflex activation was not observed in hepatic vagotomized rat and after an administration of the same dose of IL-1 beta into the systemic vein in normal rat. The results suggest the existence of sensors for IL-1 beta which send information on IL-1 beta in the portal venous blood to the central nervous system through the hepatic branch of the vagus nerve and play some role in reflex regulation of immune function.

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Vagus-brain communication in atherosclerosis-related inflammation: a neuroimmunomodulation perspective of CAD.

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Abstract

The current understanding of the pathophysiology of atherosclerosis leading to coronary artery disease (CAD) emphasizes the role of inflammatory mediators. Given the bidirectional communication between the immune and central nervous systems, an important question is whether the brain can be "informed" about and modulate CAD-related inflammation. A candidate communicator and modulator is the vagus nerve. Until now, the vagus nerve has received attention in cardiology mainly due to its role in the parasympathetic cardiovascular response. However, the vagus nerve can also "inform" the brain about peripheral inflammation since its paraganglia have receptors for interleukin-1. Furthermore, its efferent branch has a local anti-inflammatory effect. These effects have not been considered in research on the vagus nerve in CAD or in vagus nerve stimulation trials in CAD. In addition, various behavioural interventions, including relaxation, may influence CAD prognosis by affecting vagal activity. Based on this converging evidence, we propose a neuroimmunomodulation approach to atherogenesis. In this model, the vagus nerve "informs" the brain about CAD-related cytokines; in turn, activation of the vagus (via vagus nerve stimulation, vagomimetic drugs or relaxation) induces an anti-inflammatory response that can slow down the chronic process of atherogenesis.

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