PAIN. 158():S19–S28, APR 2017
DOI: 10.1097/j.pain.0000000000000779,

PMID: 27918315

The gut microbiota as a key regulator of visceral pain

Siobhain M. O' Mahony; Timothy G. Dinan; John F. Cryan

+ Author Information

Abstract

Visceral pain can range from the discomfort of indigestion to the excruciating pain of renal colic.19 Irritable bowel syndrome (IBS) is a complex and heterogeneous disorder with exaggerated visceral pain as a major distinguishing feature.44 Other visceral pain–related disorders include functional dyspepsia, functional abdominal pain syndrome, infantile colic, and interstitial cystitis. Visceral pain results from the activation of nociceptors localised in the thoracic, abdominal, and pelvic organs. Several stimuli can activate these receptors including stretch, inflammation, ischaemia, pH, bacterial products, immune mediators, and neurotransmitters.140 Increased nociceptive sensitivity leads to visceral hypersensitivity, which presents as enhanced perception of physiological signals from the viscera and/or enhanced

perception of experimental stimuli.69 Visceral pain signals travel through both spinal95 and vagal routes,162 and sensitisation of peripheral nerves can induce central sensitisation at both spinal and supraspinal levels (Fig. 1). Disorders or visceral pain significantly impact the quality of life and are associated with psychological disturbances45 as well as sleep and sexual dysfunction.55 Furthermore, treatment options for visceral pain are often unsatisfactory potentially due to the multiple factors that affect the perception and maintenance of this type of pain.