Vitamin B production by intestinal flora through the cerebral-intestinal axis in the face of neuritis obliterans

Peng Xuan Wen Huiyi Fu Siwu

Northwest University for Nationalities，Lanzhou，Gansu，730030；

Abstract: Facial neuritis, a neurological disease, is characterized by dysfunction of the muscles responsible for facial expression. The pathogenesis of this condition involves a variety of factors, including an inflammatory response, nerve ischemia, and metabolic dysregulation.Recent studies have demonstrated that intestinal flora influence the function of both the central and peripheral nervous systems through bidirectional regulatory mechanisms of the brain-gut axis. This review will focus on the potential role of vitamin B synthesized by intestinal flora (e.g., B1, B6, B12, etc.) in facial neuritis. Vitamin B serves as an important coenzyme involved in the formation of nerve myelin and energy metabolism. Moreover, clinical studies have identified that patients with facial neuritis frequently exhibit signs of intestinal dysbiosis and diminished vitamin B levels. This observation suggests the potential for the flora-vitamin B axis to play a pivotal role in the onset and progression of the disease. This research domain offers novel insights into the prevention and treatment of facial neuritis, underscoring the necessity for further in-depth studies. These studies should investigate the specific mechanisms through which particular strains of vitamin B-producing bacteria influence facial neuritis via the brain-gut axis. Moreover, they should aim to develop suitable intervention programs.

Keywords: vitamin B; facial neuritis; intestinal flora; brain-gut axis; potential role

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1. Introduction

The intestinal microbiota, a complex micro-ecosystem within the human body, plays a pivotal role in maintaining the host's physiological functions and safeguarding its health. This intestinal flora can be likened to an "invisible metabolic organ" within the host, contributing not only to nutrient synthesis and absorption but also to the establishment of a intricate bi-directional regulatory network with the central nervous system through the brain-gut axis. Recent studies have demonstrated that vitamin B-producing microorganisms, such as Lactobacillus and Bifidobacterium, not only alleviate vitamin B deficiency in the host but also inhibit neuroinflammatory responses by regulating immunity, synthesizing neurotransmitters, and other mechanisms, thus affecting the pathogenesis of facial neuritis. Research on facial neuritis is of great significance for the following reasons: first, existing therapeutic options, such as glucocorticosteroids and antiviral drugs, have limitations in terms of efficacy and side effects; therefore, there is an urgent need to develop novel therapeutic targets based on the gut flora-brain-gut axis; second, the "metabolism-immunity-neurogenesis" and other multidimensional functions of vitamin B-synthesizing microbiota are important. Secondly, the exploration of vitamin B-synthesizing bacteria for their multifaceted functions, such as the "metabolism-immunity-neurology" axis, is imperative. This may offer novel approaches for the prevention and treatment of neuroinflammatory diseases as a potential "natural drug bank."

2. The relationship between vitamin B and facial neuritis

Facial neuritis, an inflammation of the facial nerve within the stemmed mastoid foramen, is a form of peripheral facial paralysis. The clinical manifestations of this condition include paralysis of the affected facial muscles, widening of the eye fissures, inability to close the eyelids, loss of frontal wrinkles, and ptosis of the corners of the lips [1].In patients with weakness of the facial muscles, the condition is often accompanied by pain and numbness behind the ear and in the ipsilateral cheek. The incidence of facial neuritis has been documented to range from 11.5 to 55.3 cases per 100,000 individuals.Typically, the symptoms associated with peripheral facial palsy

undergo a spontaneous resolution within a period of weeks to months. However, in certain cases, the condition can result in severe transient oral dysfunction and eyelid closure dysfunction, which may potentially lead to irreversible ocular damage. Approximately 25% of patients with facial neuritis may experience persistent mild to severe facial asymmetry, which can have a significant impact on the patient's quality of life [2].

The etiology of facial neuritis may be related to viral infection, inflammation, local trauma, or cold, resulting in nerve inflammation that causes localized edema, demyelination, and ischemia.In the autoimmune hypothesis, facial neuritis is thought to be a single neuritis variant of Guillain-Barré syndrome. Patients with facial neuritis have been found to have decreased T-suppressor cells and increased B-lymphocytes, as well as increased serum chemokine concentrations. These findings suggest the presence of cell-mediated immune mechanisms in facial neuritis [3-6].Concurrently, the inflammatory response of the body is considered to be an independent pathogenic factor in facial neuritis. The neutrophil-to-lymphocyte (NLR) ratio serves as a marker of the inflammatory response, reflecting the inflammatory and immune status of the body.Recent studies have identified higher levels of NLR in patients with facial neuritis, and a positive correlation between NLR and House-Brackmann (H-B) classification has been observed [7].

The therapeutic approach for acute facial neuritis is primarily focused on addressing the symptoms, encompassing hormonal anti-inflammatory, neurotrophic, antiviral, dehydration, and decongestion interventions. These measures are designed to alleviate facial nerve edema, mitigate compression, regulate inflammation, nourish nerves, and enhance local blood circulation, thereby facilitating the recovery of facial nerve function [8-9].The efficacy of oral glucocorticoid therapy is substantiated by substantial evidence, including the augmentation of recovery likelihood, reduction in recovery time, alleviation of joint motor and residual facial palsy, and alleviation of clinical symptoms in the short term. However, the long-term efficacy of glucocorticoid therapy is suboptimal, and its utilization is encumbered by numerous adverse effects and contraindications [10].In contrast, valacyclovir has been shown to be more effective than acyclovir in restoring facial nerve function [11].Decompression surgery is another treatment option that has been employed.

In accordance with established clinical guidelines, the administration of B vitamins has been recommended to nourish nerves and promote myelin repair. Commonly prescribed medications include vitamin B1, B12 injections, and preparations such as methylcobalamin. Nerve growth factor (NGF) has demonstrated significant efficacy in the treatment of facial neuritis, and its mechanism of action may involve the reduction of inflammatory response by lowering the levels of inflammatory factors such as interleukin IL-17, IL-6, and IL-21 in the patient's serum. Concurrently, NGF shortens the latency period of R1, thereby promoting the recovery of damaged facial nerve function [12].Studies have demonstrated that two patients with peripheral facial paralysis who received low-intensity laser therapy (LLLT) in conjunction with vitamin B1, B6, and B12 supplementation achieved substantial improvements in facial muscle function and symmetry [13]. In addition, a substantial body of research, including both animal experiments and in vitro studies, has confirmed that B vitamins (including thiamine, pyridoxine, riboflavin, and cobalamin) have a variety of pharmacological effects, including anti-injury perception, anti-nociceptive sensitization, and anti-inflammatory effects. These vitamins have also been shown to be effective in relieving mechanical anomalies [14],which may be beneficial in the treatment of facial muscle pain.

Specifically, vitamin B1 (thiamine), a crucial cofactor in energy metabolism and antioxidant processes, plays a pivotal role in preserving the integrity of the nerve myelin sheath and ensuring normal nerve conduction function. Vitamin B6 (pyridoxine) supplementation has been shown to alleviate clinical symptoms in patients with peripheral neuropathy of various etiologies. However, vitamin B6 is not used as a standalone therapeutic agent; rather, it is utilized as part of a combination regimen, often in conjunction with other B vitamins, in the treatment of facial neuritis [15].Studies have demonstrated that vitamin B12 plays a pivotal role in the process of myelin formation by promoting DNA synthesis in oligodendrocytes [16],a mechanism that is crucial for the repair of nerve myelin in patients with facial neuritis. Additionally, vitamin B12 exerts a substantial anti-inflammatory effect, which may involve the attenuation of inflammation-mediated oxidative stress injury in patients with facial neuritis by

regulating the production of inflammatory factors such as interleukin-6 (IL-6)[17]. Notably, nitric oxide (NO), as a pivotal signaling molecule, plays a crucial role in pain transmission and nociceptive sensitization [18],patients with facial neuritis frequently exhibit neuropathic symptoms.Hydroxocobalamin, an active form of vitamin B12, has bee n shown to possess free radical scavenging properties and can effectively modulate the effects of NO on the va scular and nervous systems[19],this action may contribute to the alleviation of neuroinflammation and pain sympt oms in patients with facial neuritis. Vitamin B12, a crucial enzyme cofactor in the methylation process, plays a p ivotal role in the metabolic processes of nerve myelin and is a vital vitamin for sustaining the proper functionin g of the nervous system.Pro-inflammatory cytokines can be induced through the initiation of inflammatory respo nses, which can subsequently lead to oxidative stress-induced damage. Cob(II)alamin, the reduced form of vitami n B12, has been shown to possess significant antioxidant properties and exert potent reactive oxygen species (R OS) scavenging activity in the nervous system through a variety of mechanisms [20],this property is likely to contr ibute to the reduction of neurological damage and ROS levels in patients with facial neuritis.

3. Vitamin B production by intestinal flor

Bacteria of the family Bacteroidetes possess the ability to synthesize vitamin B1 autonomously, and its synth esis-related genes (e.g., thiC, thiD, dxs, and thiE) are widely found in bacteria of the families Bacteroidetes and Ruminalococcaceae[21].Studies have demonstrated that several phyla, including Fibrobacterium and Aspergillus, are potential sources of key genes involved in vitamin B1 synthesis (e.g., thiC), and that Flavobacterium spp. and Bu rkholderia spp. may play a synergistic role in vitamin B1 biosynthesis[22].In addition, Lactobacillus, Ruminalococcus, and Bifidobacterium spp. have been demonstrated to possess the capacity to synthesize vitamin B1[23].Macrogen omic analyses have revealed that the human intestinal flora is predominantly comprised of Actinobacteria, Asper gillus, and Anaplasma (256 samples, ~50%), and all possess the capability to synthesize vitamin B6 de novo. Spe cifically, the analysis included strains such as Bifidobacterium longum, Prevotella, Anaplasma fragilis, Collinsella aer ogenes, and Helicobacter pylori [24].Studies have confirmed that Bifidobacterium infantis VKPM AC-1912, Bifidobact erium adolescentis VKPM AC-1662, and Bifidobacterium longum VKPM AC-1665, as well as Lactobacillus acidophil us VKPM B- 2105, VKPM B-8238, and VKPM B-6551, Lactobacillus plantarum VKPM B-11007, Lactobacillus casei VKPM B-2873, etc. [25].Approximately 20% of the intestinal flora synthesizes vitamin B12, while the remainder req uires exogenous acquisition to maintain metabolism. Vitamin B12 is synthesized by a variety of microorganisms, i ncluding Clostridium spp., Prevotella spp., Propionibacterium spp., Anaplasma spp., Ruminalococcus spp., and Bifid obacterium spp.[26]The biosynthesis of vitamin B12 is a complex process that requires the involvement of about 30 enzymes. It begins with the condensation of uroporphyrinogen III and culminates in the assembly and linkage of the nucleotide ring to the cochineal ring [27].

4. Vitamin B acts on facial neuritis via the brain-gut axis

The brain-gut axis, a complex bidirectional regulatory pathway, plays a pivotal role in regulating central nerv ous system functions and maintaining intestinal homeostasis. The enteric nervous system (ENS) is referred to as the "second brain" due to its autonomous neural functions, with its neurons predominantly distributed across tw o functional subdivisions: the intermuscular plexus, which regulates intestinal motility, and the submucosal plexus, which regulates secretion and is located in the submucosa. This system establishes a bidirectional connection wi th the central nervous system (CNS) through sympathetic and parasympathetic nerve fibers, which collectively fac ilitate precise regulation of the digestive system [28].The intestinal flora contributes to the gut-brain axis regulation through multifaceted mechanisms. The primary mechanism involves the regulation of intestinal barrier permeabil ity, followed by modulation of sensory nerve signaling, synthesis of local neurotransmitters, activation of the muc osal immune system, and release of bioactive peptides. Conversely, the brain modulates the microbiome via the efferent nervous system, a process that depends on bacterial surface neurotransmitter-mediated regulation of gut motility, modulation of mucus secretion, and activation of the immune response [29].The gut is a major site of i nteraction, colonized by more than a thousand microorganisms (predominantly in the phylum Thick-walled Bacteri

a and Bacteroidetes). These microorganisms form a dynamic homeostatic network with the host, and this homeo static system safeguards the health of the gut through a three-fold mechanism: maintenance of the structural int egrity of the tissues, inhibition of pathogen colonization, and precise modulation of the immune response of the host. The gut-brain axis constitutes a complex bidirectional communication network, comprising the central nerv ous system (CNS), the enteric nervous system (ENS), the autonomic nervous system (ANS), and the hypothalamic -pituitary-adrenal axis (HPA). Consequently, extant studies have accentuated the pivotal function of the microbiota in facilitating information exchange between the gut and the brain, thereby giving rise to the novel concept of the "microbiota-gut-brain axis (MGB)" [30]. The gut microbiota communicates with the brain through three parall el and interacting pathways [31]: neural, immune, and endocrine pathways.The mechanisms by which the gut micr obiota regulates neurotransmission consist of three primary pathways: (1) the ability of certain gut microbiota to encode genes for enzymes necessary for the synthesis of neurotransmitters, which in turn catalyze the synthesis of substrates for the corresponding neurotransmitters or precursors; and (2) the ability of gut microbiota to pro duce neuroactive compounds, e.g., Lactobacillus synthesizes acetylcholine and γ-aminobutyric acid, and Bifidobacte rium produces γ-aminobutyric acid. These neurotransmitters enter the blood circulation through the intestinal mu cosa, which in turn affects the function of the central nervous system or directly activates the vagus nerve to tr ansmit messages to the brain. (3) The gut microbiota also regulates the synthesis and release of neurotransmitte rs from enteroendocrine cells through their metabolites. While the precise molecular mechanisms and transmissio n pathways through which vitamin B substances synthesized by the intestinal microbiota act on patients with fac ial neuritis via the brain-gut axis remain to be fully elucidated by existing studies, it is reasonable to infer that i ntestinal vitamin B may exert a potentially positive effect in the treatment of facial neuritis based on the bidirec tional regulatory function of the brain-gut axis theory.

5. Discussion

Facial neuritis, a neurological disease, is characterized by dysfunction of the muscles responsible for facial ex pression. Its pathogenesis is multifactorial, involving inflammatory response, nerve ischemia, and metabolic dysreg ulation.Recent studies have demonstrated that intestinal flora influence the function of both the central and peri pheral nervous systems through bidirectional regulatory mechanisms of the brain-gut axis. This review will focus on the potential role of vitamin B synthesized by intestinal flora (e.g., B1, B6, B12, etc.) in facial neuritis. Vitami n B serves as an important coenzyme involved in the formation of nerve myelin and energy metabolism. Moreo ver, clinical studies have identified that patients with facial neuritis frequently exhibit signs of intestinal dysbiosis and diminished vitamin B levels. This observation suggests the potential for the flora-vitamin B axis to play a piv otal role in the onset and progression of the disease. This research domain offers novel insights into the preven tion and treatment of facial neuritis, underscoring the necessity for further in-depth studies. These studies shoul d investigate the specific mechanisms through which particular strains of vitamin B-producing bacteria affect facia l neuritis through the brain-gut axis. Moreover, they should aim to develop suitable intervention programs.

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Project Number: X202410742340
Corresponding author: FU Siwu
Author's Name: Peng Xuan
Gender: Female
Ethnicity: Han
Native Place: Changde, Hunan
Date of Birth: August 9, 2003
Education: Bachelor's Degree
Title: None
Research Direction: Facial Nerve Inflammation
Postal Code: 730030
Work Unit: Northwest Minzu University
Project Title: Research on the Correlation between Facial Nerve Inflammation and B Vitamins