

Exploring the Nutritional Modulation of Gastric Physiology and Immunity in the Context of Vitamin B12 Malabsorption

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ABSTRACT

Despite food fortification, vitamin B12 deficiency endures, now driven chiefly by malabsorption rather than inadequate intake. Pernicious anemia, the end-stage of autoimmune atrophic gastritis, emerges as the principal cause and is increasingly linked to modifiable diet. Evidence from clinical, epidemiological, and mechanistic studies shows that salt-, fat- and nitrosamine-rich diets elevate gastric pH, suppress gastric-pancreatic proteases and inflame mucosa, blocking the release of cobalamin from food. Concurrent deficits of zinc, vitamin D and other micronutrients erode antioxidant defenses and immune tolerance, hastening parietal-cell loss and intrinsic-factor failure, while ultra-processed foods drive microbiome shifts that bias Th2-mediated autoimmunity. These converging insults disrupt two critical steps—enzymatic liberation of B12 and intrinsic-factor-dependent uptake—spanning reversible gastritis to irreversible pernicious anemia. Preliminary interventions suggest that diet optimization and micronutrient repletion can restore gastric enzyme activity and lower parietal-cell antibodies, positioning targeted nutrition as a low-cost adjunct, or occasional alternative, to lifelong parenteral B12 therapy. Rigorous prospective and randomised studies are now needed.

Keywords

Vitamin B12 deficiency, Cobalamin malabsorption, Pernicious anemia, Autoimmune atrophic gastritis, Gastric intrinsic factor, Dietary patterns, Malnutrition, Micronutrient status (zinc, vitamin D), Gastric proteases, Gut microbiome, Ultra-processed foods.

List of Abbreviations

AAG: Autoimmune Atrophic Gastritis, APC: Antigen Presenting Cell, CD: Cluster of Differentiation, DC: Dendritic Cell, DNA: Deoxyribonucleic Acid, F/B: Firmicutes to Bacteroidetes Ratio, GIF: Gastric Intrinsic Factor, HCl: Hydrochloric Acid, hsCRP: High-Sensitivity C-Reactive Protein, IF: Intrinsic Factor, IgG1: Immunoglobulin G1, IL: Interleukin, mRNA: Messenger Ribonucleic Acid, PA: Pernicious Anemia, PCA: Parietal Cell Antibodies, RDA: Recommended Dietary Allowance, ROS: Reactive Oxygen Species, SOD: Superoxide Dismutase, Treg: Regulatory T Cell,

Th1/Th2: T Helper Cell Type 1 / Type 2, UHP: Ultra-High Processed (Foods), VDR: Vitamin D Receptor.

Introduction

Hypovitaminosis B12 (cobalamin) is a common ailment, with deficient values observed in 6% of U.S. adults aged 60 and above, and marginal values observed in up to 26% of the general population of the United States [1,2]. Depleted values are drastically more common in developing countries, with deficiencies observed in 40% to 80% of populations of certain developing nations. B12 deficiency has many different causes that can be separated into two main categories: dietary insufficiency and malabsorption. Dietary insufficiency is becoming increasingly rare due to efforts to fortify common food products such as flour, cereals, and nutritional yeast with adequate levels of B12. Thus, malabsorption can be attributed as the cause of most cases of insufficient B12 levels in patients. The malabsorption of B12 has various etiologies pertaining to

various organs, but by far the single most common cause of B12 malabsorption is Pernicious anemia (PA), also known as Biermer's disease, affecting an estimated 151 in 100,000 Americans yearly [3,4]. PA is an aggressive/terminal form of autoimmune atrophic gastritis (AAG), characterized by an atrophy of the fundic gastric mucosa by cell-mediated autoimmunity, the reduced secretion of gastric intrinsic factor (GIF), and the presence of anti-IF antibodies and anti-parietal cells antibodies that target the hydrogen, potassium, adenosine triphosphate pump [5]. While the underlying causes for PA and AAG are thought to be genetic in nature, there is emerging evidence to suggest that the development and prognosis of these ailments can be significantly influenced by diet and nutrition. Furthermore, there is anecdotal and empirical evidence to suggest that nutritional status and dietary patterns significantly influence the gastric environment and may either mediate or exacerbate B12 malabsorption and its underlying autoimmune pathology. This paper explores the nutritional underpinnings of B12 malabsorption, emphasizing how diet-induced changes in the gastric milieu may affect B12 bioavailability and the development of AAG.

Parietal cells of the fundus also secrete intrinsic factors at this step. In the duodenum, pancreatic enzymes cleave the B12 from the haptocorrin, allowing it to bind to the gastric intrinsic factor which then accompanies it to the ileum and facilitates receptor-mediated endocytosis [6,7]. It is worth noting that a minute quantity of B12, approximately 1% of consumed B12, is absorbed passively by the ileum. Thus, the majority of B12 absorption is vastly dependent on a multistep, multifactor process involving multiple proteins, enzymes, and anatomical locations. If any enzymes or conditions are altered, either directly or indirectly, it can lead to the inability to correctly absorb and metabolize B12.

Nutrition and the Gastric Milieu

The gastric milieu refers to the total gastric environment including gastric enzymes, the gastric mucosa, and the pH and composition of gastric juices. The gastric milieu and its components can be impacted by genetic and environmental determinants, and there is significant evidence to indicate that nutrition is a key factor that can alter the gastric milieu and affect the normal functioning of gastric cells, thus impeding optimal B12 absorption. It has been directly demonstrated that deficient levels of pancreatic proteases in the gastric milieu, specifically trypsin, can prevent the separation of haptocorrin from cobalamin, and as such can play at least a partial role in B12 malabsorption [9,10]. This indicates the importance of a proper gastric environment for B12 absorption. In a sample of 8 malnourished Crohn's disease patients, an abnormal gastric environment was observed. Specifically, a low amount of 4 key gastric enzymes was measured in the stomach and following a strict nutritional intervention focusing on adequate nutrients and high protein intake, the enzymes returned to near control levels. This indicates that gastric acid and pancreatic enzyme synthesis and secretion can be influenced by nutritional state [11]. Furthermore, a direct correlation has been observed in adolescents between the incidence rate of an inflamed gastric mucosa (gastritis), often partially attributed to non-homeostatic gastric conditions, and consuming diets high in sodium, fat, and processed foods [12].

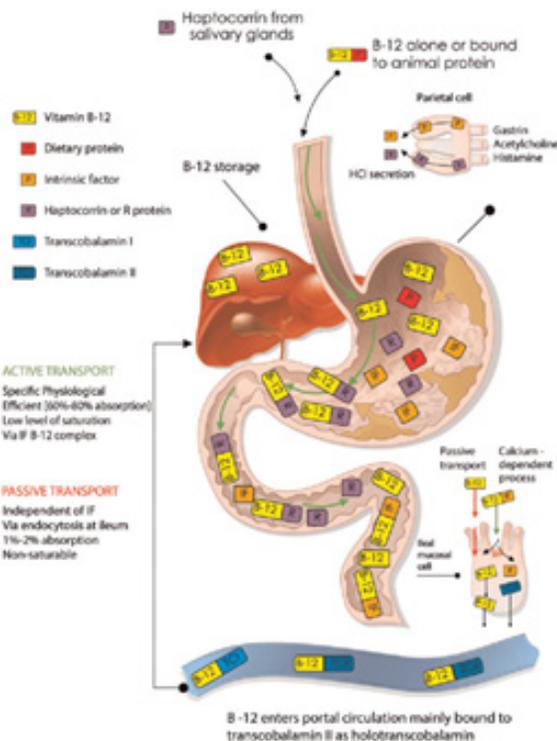


Figure 1: A diagram showing the complex process of B12 digestion and absorption [8].

Normal Cobalamin Absorption

Before exploring the maladies causing abnormal B12 absorption, it is important to have a foundational knowledge of the unaltered process of B12 absorption. Under normal conditions, B12 is absorbed in a multistep process. After the substance containing B12 is swallowed, it is first separated from the food by salivary enzymes, HCl, and gastric proteases, and then bound to haptocorrin, a salivary protein that protects it from degradation by gastric juices.

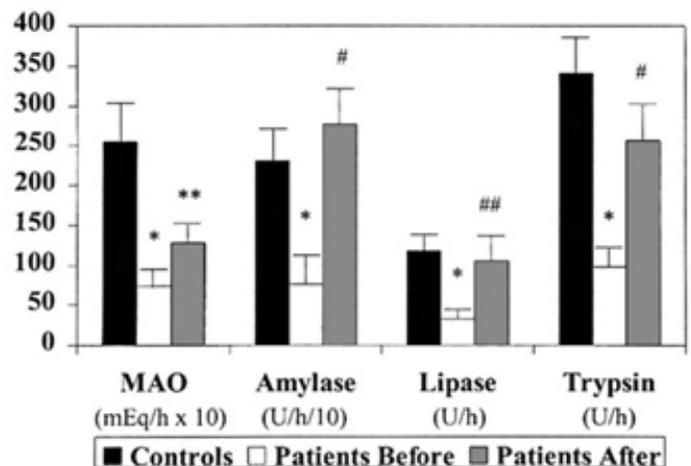


Figure 2: A diagram of gastric acid levels and pancreatic enzyme output of control subjects and malnourished Crohn's disease patients, preceding and following nutritional support [11].

Inadequate nutrition and certain lifestyle choices such as smoking have also been shown to directly influence the gastric environment, whereas adequate consumption of a balanced and nutritional diet has been shown to have a protective effect against gastric inflammation and multiple gastric ailments, including AAG. Specifically, increased levels of vegetable and fruit consumption, as well as adequate Vitamin C consumption, has been shown to have a protective effect against gastric inflammation, whereas milk consumption was linked to an increased risk of chronic gastric inflammation. Furthermore, diets consisting of high amounts of processed fats and sodium were shown to increase stomach PH and nitrate and nitrite levels, and cigarette smoking led to a twofold increased risk for developing gastric lesions [13]. A comprehensive meta-analysis examined the effects of nutrition on the gastric environment in the context of gastric tumorigenesis and found that dietary salt intake is strongly correlated with gastric inflammation and tumorigenesis, as is heavy alcohol consumption. Furthermore, substances such as red meats and spicy peppers were correlated with an increased risk of gastric cancer, whereas the consumption of fresh fruits, vegetables and certain micronutrients like folate, iron, zinc, selenium and vitamin C were observed to have protective effects in this meta-analysis [14].

Zinc Deficiency

There is further evidence to suggest that dietary micronutrient deficiencies including folic acid, B6, niacin, vitamins C or E, iron and specifically deficiencies in zinc levels, can lead to increased oxidative stress and potentially increase the risk of gastritis, metaplastic gastrointestinal cells, and tumorigenesis in digestive tract tissues [15-19]. This is because zinc in particular plays an important role in DNA replication and repair due to the prevalence of zinc finger and zinc-associated proteins in these processes. Low intracellular zinc has been shown to be correlated with up-regulation of tumor suppressor protein p53 in various cell lines, likely in response to DNA damage caused by zinc deficiency. Interestingly, low intracellular zinc has also been shown to interfere with the ability of p53 and other transcription factors to bind to a consensus DNA sequence, drastically reducing their efficacy [18].

In addition to being essential for DNA replication and repair, zinc functions as a natural antioxidant, helping to combat oxidative stress. There are multiple theories as to how zinc functions to neutralize reactive oxidative species and free radicals, but leading theories acknowledge the role of zinc in the Cu/ZnSOD complex, competing for binding sites with pro-oxidant transition metals, and regulating metallothionein metabolism [20-22]. Thus, the consumption of adequate levels of zinc is not only essential for effective DNA replication, it can also serve a protective function to help tissues combat oxidative stress. It is also worth noting that there are other dietary elements that can affect zinc absorption. Such elements include the consumption of foods high in chelators such as phytate which can bind to zinc and make it less bioavailable [19]. While much of this research examines Zinc and gastric tissues through the context of tumorigenesis, there is ample evidence to suggest that patients with gastrointestinal cancers have significantly impaired levels of intracellular B12 [23].

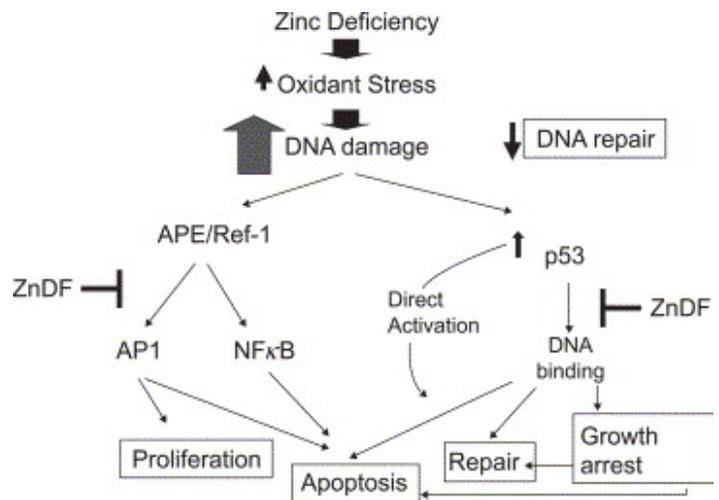


Figure 3: Summary of potential systemic effects of zinc deficiency on DNA [19].

B12 Malabsorption Comorbidities

There are various conditions that often coexist with B12 malabsorption pathologies such as AAG. The most common of which are other autoimmune diseases like Hashimoto's disease and type 1 diabetes mellitus, and chronic inflammatory diseases such as Crohn's disease [24]. In some cases, the coexisting conditions can directly cause autoimmune atrophic gastritis, as has been observed in thyroiditis patients. A longitudinal study observed abnormally high levels of parietal cell antibodies (PCA) and intrinsic factor antibodies (IFA) a sample of thyroiditis patients, and followed up after 5 years, observing that approximately a quarter of all patients initially positive for PCA/IFA had developed symptoms in line with autoimmune atrophic gastritis [25]. While this is an instance of a direct causal relationship regarding B12 malabsorption, in other cases there is evidence to suggest that comorbidities can indirectly affect B12 absorption due to the nutritional underpinnings of those diseases. For instance, patients with Celiac disease often present with B12 deficiencies because of the abnormalities in enteric brush border proteins and digestive enzymes that result in celiac patients whose diets contain gluten [26-29]. There is a noticeable lack of PCA and IFA antibodies in these patients, indicating that while B12 is being malabsorbed, it is not the result of autoimmune gastritis, but rather the result of a less understood pathology resulting from gastric endothelial irritation [28]. Furthermore, in most patients, B12 deficiency reversed following adherence to a gluten free diet, although parental B12 supplements were required to treat symptomatic patients [29,30]. This evidence indicates the nutritional basis of comorbidities can influence B12 absorption. Another example of this is in obese patients. There is a well-documented inverse correlation between obesity and serum B12 levels [31,32]. In these studies, it was clear that obese participants were consuming low-quality diets, but it remains unclear if low B12 levels are a result of dietary inadequacy of B12 or malabsorption, however there is an indication it could be the latter although this needs further investigation.

Nutritional Influence on Autoimmune Gastritis Development and Treatment

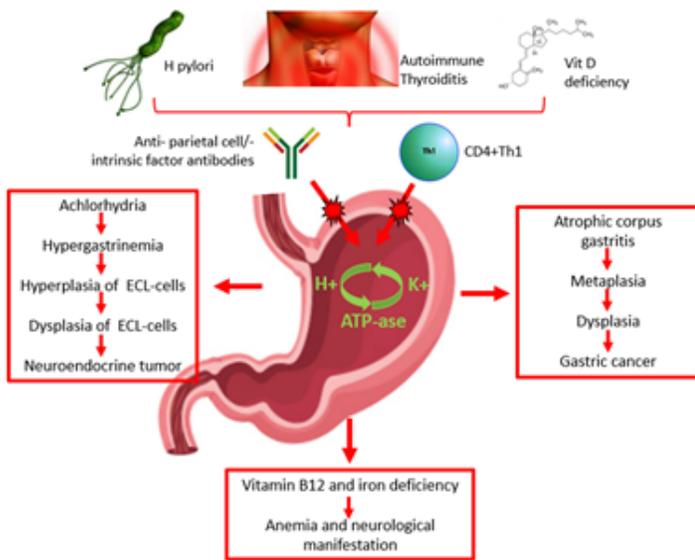


Figure 4: Etiopathogenesis, symptoms, and prognosis of Autoimmune Gastritis [33].

AAG primarily sees immune cells target the catalytic α and the glycoprotein β subunits of the H^+/K^+ ATPase pump in gastric parietal cells, which leads to their gradual destruction. This immune-mediated attack is primarily orchestrated by gastric dendritic cells that activate $CD4^+$ T lymphocytes in perigastric lymph nodes. Once activated, these T-cells recognize the proton pump ATPase as an autoantigen, perpetuating the immune response and leading to parietal cell apoptosis and subsequent gastric atrophy [34,35]. There is burgeoning evidence to suggest that nutrition plays a crucial role in the development of autoimmune gastritis, as well as in general immune and inflammatory responses. Dietary components can influence immune tolerance, gut microbiota composition, and systemic inflammatory responses, all of which can potentially modulate autoimmune reactivity against parietal cells. One dietary component that could potentially impact immune responses is ultra-high processed foods (UHP). As defined by the NOVA classification guidelines, consumption of UHPs can lead to a demonstrable increase in cytokine secretion in human subjects. One study found that with every 100 g increase in ultra-processed food intake, a corresponding 4.0% increase in serum levels of the inflammatory cytokine, high-sensitivity C-reactive protein (hsCRP) was observed [36]. While this protein is normally used as a biomarker to assess cardiovascular risk, there is evidence to suggest that it is strongly correlated with and can serve as a predictive, although nonspecific, marker for gastritis [37]. Another way in which nutrition can impact autoimmune development is by modulating the gastric microbiome, specifically by altering the Firmicutes to Bacteroidetes (F/B) ratio in the intestinal microbiome. Changes to this ratio and the composition of the microbiome can cause a disproportionate bias towards the Th2 response in $CD4^+$ T cells, and it is the Th2 associated immunoglobulin IgG1 that is the primary autoantibody isotype to the H^+/K^+ ATPase pump

[34,38–40]. Thus, the composition of the microbiome can affect the mechanisms underlying AAG. In regard to the role nutrition plays, a direct link has been established between the quality and composition diet and the composition of the intestinal microbiome across multiple studies. One study demonstrated that switching from a low-fat, plant polysaccharide-rich diet to a high-fat, high-sugar diet can significantly alter the configuration of the microbiome in as little as one day [41]. Another study compared the microbiomes of African children, who eat diets low in fat and high in fiber, starch, and various plant polysaccharides, with those of European children who eat diets lacking fiber and high in sugar, starch and fat. This study found that the African children had a significantly lower and thus less inflammatory F/B ratio [42]. This indicates a link between nutrition and microbiome health, which in turn indicates a correlation between nutrition and proliferation of autoantibodies.

Perhaps the most well understood nutritional factor influencing autoimmunity is serum Vitamin D levels. Vitamin D plays an important role in immune regulation as many tissues, specifically those involved directly in immune regulation, express the Vitamin D Receptor (VDR) and 1-alpha-hydroxylase, an enzyme that converts inactive Vitamin D (calcidiol) to its active form of calcitriol [43,44]. It is posited that Vitamin D contributes to the autocrine and paracrine regulation of both innate and adaptive immunities by means of this receptor. Numerous studies confirm that calcitriol enhances the innate immune response while simultaneously exercising an inhibitory influence on the adaptive immune system by regulating the interaction between lymphocytes and antigen presenting cells (APCs), as well as by inhibiting their effector mechanisms [45–47]. Perhaps most pertinent to AAG specifically, the activation of VDRs in dendritic cells (DCs)—a vital component in the genesis of AAG—leads to a limited proliferation of DCs, decreased expression of CD40, CD80 and CD86, reduced production of proinflammatory IL-12 but increased production of anti-inflammatory IL-10, and decreased prevalence of the Th1 response in T cells [48–50]. The presence of vitamin D also influences the induction of Tregs instead of T-effectors, which directly suppresses the development of autoimmune disorders pathologies [50]. These responses elicited by Vitamin D are analogous to those prompted by commonly prescribed immunosuppressants and function to create conditions precluding the genesis of AAG. Thus, clearly Vitamin D plays an important role in preventing various autoimmune disorders, and there is evidence to suggest that Vitamin D supplementation in humans can be used to reduce the severity of symptoms of various autoimmune disorders [51,52].

Non-Genetic factors of Gastric Intrinsic Factor Secretion

Reduced secretion of gastric intrinsic factor in the fundus is a key clinical manifestation of PA. This can result from the autoimmune degradation of parietal cells but can also result from ectopic and abnormal expression of the GIF. Abnormal expression refers to producing variable levels of GIF or incorrect forms, and ectopic expression refers to expression by the incorrect cell types in the wrong anatomical location. There is evidence to suggest that

the abnormal expression of GIF is not solely genetic and can be influenced by environmental factors such as inflammation. A landmark study observed a twofold increase in ectopic IF expression in a group of rats when a sample of their gastric mucosa was exposed *in vitro* to interleukin 1- β and observed a 9.8% increase in ectopic expression under the same experimental conditions *in vivo* [53]. There also exists a gradient in ectopic GIF expression between inflamed and non-inflamed areas, indicating that a diffusible messenger molecule signals for the ectopic expression of GIF. This molecule has been posited to be a cytokine, secreted by inflammatory cells. This provides strong evidence that GIF expression can be modulated in response to inflammation, a theory that has been anecdotally observed in human pathological studies as well [54]. While the clinical significance of ectopic intrinsic factor expression remains unconfirmed, it is worth noting that in the human study, ectopic expression of GIF appeared to be substantially higher in the 5 patients exhibiting severe food-cobalamin malabsorption than in the other 4 patients that were studied.

Conclusions and Future Directions

Cobalamin malabsorption has emerged as a dominant driver of global B-12 deficiency now that overt dietary scarcity is increasingly offset by food fortification. This review synthesizes evidence that the gastric milieu—and therefore the efficiency of B-12 uptake—is profoundly shaped by nutritional factors. Diets high in sodium, saturated fat, and nitrosating preservatives elevate gastric pH, suppress key proteases, and accelerate mucosal inflammation, while micronutrient deficits (especially zinc and vitamin D) impair DNA-repair pathways, antioxidant defenses, and immune tolerance. These changes converge on two mechanistic bottlenecks: (i) disrupted cleavage of cobalamin from haptocorrin and (ii) autoimmune loss or ectopic dysregulation of GIF-secreting parietal cells. The result is a spectrum of malabsorptive states that ranges from reversible atrophic gastritis to irreversible pernicious anemia.

Collectively, the data argue that B12 malabsorption cannot be understood—or managed—solely through a genetic or autoimmune lens. Nutritional status modulates gastric enzyme output, shapes the gut microbiome's Th1/Th2 balance, and regulates dendritic-cell function via vitamin D signaling, thereby influencing both the initiation and progression of autoimmune attack. Equally important, emerging clinical observations show that correcting macronutrient quality, restoring trace-element sufficiency, and reducing pro-inflammatory dietary triggers can normalize gastric protease activity and lower parietal-cell antibody titers in subclinical disease. These findings position targeted nutrition as a plausible adjunct—or even alternative—to lifelong parenteral B12 therapy in selected patients. Yet the field rests largely on associative or small interventional studies. To translate these insights into practice, three priorities stand out:

1. Developing prospective cohort studies that quantify dietary patterns, gastric biomarkers, and incident malabsorption over time to establish temporality and dose–response relationships,
2. Executing randomised nutrition-intervention trials in early

autoimmune gastritis that test whether micronutrient repletion, anti-inflammatory diets, or microbiome-directed foods can halt or reverse histological atrophy and restore intrinsic-factor secretion.

3. Exploring mechanistic work using organoids and gnotobiotic models to dissect how specific nutrients, microbial metabolites, and cytokine milieu interact to trigger parietal-cell apoptosis or ectopic intrinsic-factor expression.

Addressing these gaps will clarify causality, refine dietary prescriptions, and identify biomarkers that predict which patients are most likely to benefit. Given the low cost, safety, and scalability of nutritional interventions—especially in resource-limited settings where B12 injections are impractical—this research agenda offers a high-yield path toward mitigating the neurological and hematological burden of cobalamin deficiency worldwide.

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