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Immune Dysregulation and Nutrition in SARS-CoV-2 Infection: An Integrated Review with Oral Health Perspectives

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Abstract

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infection causes not only acute respiratory illness but also dysregulated immune responses, including autoimmunity and hypersensitivity reactions, which can worsen outcomes. SARS-CoV-2 infection, known publicly as COVID-19 disease, has presented a complex clinical landscape ranging from asymptomatic infection to SARS and multi-organ failure. Beyond direct viral damage, the pathogenesis of COVID-19 is heavily driven by immune dysregulation, which causes allergic-type reactions. Nutritional status and immunonutrition further modulate these processes and play a critical role. Vitamins C and D are essential for immune competence, with vitamin C serving as an antioxidant that supports barrier integrity and leukocyte function. In contrast, vitamin D regulates innate and adaptive responses. The intake of essential nutrients could be a strategic approach to bolster the innate and adaptive immune systems, aiming to prevent infection and mitigate disease severity. Saliva and oral health are increasingly recognized as central to COVID-19 immunopathology, both as diagnostic media and as immunological barriers. This review synthesizes recent findings on autoimmune phenomena, hypersensitivity reactions, immunonutrition, and oral health in SARS-CoV-2 infection, highlighting mechanisms and clinical implications.

Keywords: SARS-CoV-2; Autoimmunity; Hypersensitivity; Immunonutrition; Oral health

1. Introduction

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), the causative agent of Coronavirus disease 2019 (COVID-19), emerged in late 2019 and was first identified in Wuhan, China. The virus rapidly escalated into a global pandemic. SARS-CoV-2 represents a novel mutation of the SARS-CoV-1 virus responsible for earlier outbreaks [1]. COVID-19 exhibits a highly variable clinical presentation, with an incubation period averaging 3 days but extending up to 24 days. Severe cases may progress to acute respiratory distress syndrome (ARDS), characterized by alveolar fluid accumulation and severe dyspnea, frequently resulting in multi-organ failure and mortality. In the absence of specific curative therapies during the early pandemic, enhancement of host immune responses through preventive strategies and nutritional support was recognized as a critical factor in patient recovery [2].

SARS-CoV-2 infection ranges from asymptomatic cases to fatal multi-organ failure. The virus enters cells via the spike (S) protein and ACE2 receptor, initiating replication and immune activation. Early antiviral responses and a balanced adaptive immune response are critical for good outcomes [2]. While most patients recover, others suffer prolonged or delayed complications. The immune response to SARS-CoV-2 is complex and sometimes maladaptive. Besides the well-known "cytokine storm," COVID-19 may trigger autoimmune disorders and hypersensitivity reactions that damage tissue. Nutritional factors also shape immunity. Inadequate micronutrients weaken host defenses, while vitamins C and

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D may enhance immune resilience. Thus, it is essential to understand how SARS-CoV-2 induces autoimmunity, how allergic-type responses worsen disease, and how immunonutrition can mitigate these effects. This review combines recent findings on these topics to provide an updated perspective on COVID-19 immunopathology and recovery.

2. Autoimmune manifestations of COVID-19

Viral infections are well-known triggers of autoimmunity, and SARS-CoV-2 is no exception. COVID-19 can cause profound immune dysregulation, leading to loss of self-tolerance and development of autoimmune phenomena. Mechanistically, molecular mimicry (viral antigens resembling self-proteins), epitope spreading, and bystander activation of autoreactive lymphocytes have been proposed. In particular, SARS-CoV-2 infection may induce type II and IV hypersensitivity-like mechanisms, in which cross-reactive antibodies (type II) or virus-activated T cells (type IV) attack host tissues. Antibody-dependent enhancement and immune complex formation have also been implicated in promoting inflammation [2].

Clinically, a broad spectrum of post–COVID-19 autoimmune conditions has been reported. In the weeks to months following infection, cases of autoimmune hemolytic anemia, immune thrombocytopenic purpura, autoimmune thyroiditis (Graves' or Hashimoto's disease), and antiphospholipid syndrome have emerged [2]. Neurological syndromes such as Guillain–Barré and Miller Fisher have been documented. Some patients develop new-onset systemic lupus erythematosus or rheumatoid arthritis after COVID-19 [3]. For example, Zhou et al. found that many hospitalized COVID-19 patients had positive antinuclear or anti-SSA/Ro antibodies [2]. Importantly, recent extensive cohort studies and meta-analyses show that SARS-CoV-2 infection significantly increases the risk of multiple autoimmune diseases. In one meta-analysis, post-COVID cohorts had an elevated incidence of Behçet's Disease, Systemic Lupus Erythematosus, Rheumatoid Arthritis, Systemic Sclerosis, Psoriasis, Polymyalgia Rheumatica, Sjögren's Syndrome, Type 1 Diabetes (adults), Vasculitis, and Inflammatory Bowel Disease compared to non-infected controls. Over the post-pandemic period, a rise in such autoimmune and rheumatic conditions may be expected [3]. Autoimmunity is also implicated in "long COVID," as persistence of symptoms correlates with autoantibody production [2]. Collectively, these data underscore that SARS-CoV-2 can act as a trigger for organ-specific and systemic autoimmune disorders in susceptible individuals.

3. Hypersensitivity reactions in COVID-19

Beyond autoimmunity, hypersensitivity (allergic or immune-complex) reactions appear to play a role in COVID-19 pathogenesis, particularly in severe cases. An early study by Tan et al. found that patients with severe COVID-19 had markedly elevated IgE antibodies specific to SARS-CoV-2 spike and nucleocapsid proteins. These virus-specific IgE levels correlated with lung damage and hypoxemia. Histological analysis revealed IgE-bound, activated mast cells in the lung and intestinal tissues of COVID-19 patients. This suggests that SARS-CoV-2 can induce a type I hypersensitivity-like response: virus-IgE complexes activate mast cells (via the FcεRI receptor), causing histamine release, vascular permeability, and bronchial hyperreactivity. Such IgE-mediated mast cell activation may contribute to the "cytokine storm" and respiratory failure seen in some patients. In fact, severe cases often respond to corticosteroids, consistent with suppression of allergic inflammation. Notably, IgE levels were higher in patients with hypoxemic respiratory failure than in those with milder cases, suggesting a link between allergic inflammation and COVID-19 severity [4].

Immune complex (type III hypersensitivity) mechanisms also likely contribute. As SARS-CoV-2 triggers a robust antibody response (IgM, IgG), excess antigen–antibody complexes can form and deposit in tissues. Manzo proposed that COVID-19, particularly in its severe vascular stage, resembles a "diffuse immune complex hypersensitivity" syndrome. In this model, abundant viral antigens and antibodies generate circulating immune complexes that activate complement, leading to the generation of C3a/C5a anaphylatoxins, mast cell degranulation, and neutrophil recruitment. Deposition of these complexes in vessels causes intense inflammation, microthrombosis, and tissue injury. Indeed, SARS-CoV-2 spike protein and complement components (C5b-9) have been co-localized in the lung microvasculature of fatal cases, along with neutrophils and fibrin thrombi. This cascade – immune complexes activating complement, releasing vasoactive mediators, and recruiting neutrophils – is characteristic of type III hypersensitivity [5]. The result is severe endothelial damage and multiorgan dysfunction observed in critical COVID-19.

In summary, COVID-19 can elicit multiple forms of immune overreaction, including allergy-like responses and immune complex formation, both of which contribute to tissue damage. These mechanisms collectively drive significant inflammation and complications such as severe lung injury. This understanding indicates that therapies targeting mast cells or the complement system may offer benefit, although further research is required.

4. Immunonutrition and COVID-19

Optimal nutrition is essential for maintaining a robust immune response. Malnutrition, including deficiencies in both macronutrients and micronutrients, impairs host defenses and may exacerbate COVID-19 outcomes. A balanced diet rich in vitamins and minerals supports epithelial barrier integrity and immune cell function. Key micronutrients include vitamins A, D, C, E, B6, B12, and folate; minerals such as zinc, iron, copper, and selenium; and omega-3 fatty acids (Eicosapentaenoic Acids / EPA and Docosahexaenoic Acids / DHA). These nutrients function as cofactors or regulators at various stages of immune activity. For instance, zinc is vital for antiviral defense, vitamin A supports mucosal integrity, and vitamin E provides antioxidant protection. Omega-3 fatty acids have anti-inflammatory properties and influence cell membrane composition. Deficiencies in these nutrients can increase susceptibility to infection and worsen disease severity [6,10].

4.1. Vitamin C

Vitamin C (ascorbic acid) is a potent water-soluble antioxidant that accumulates in leukocytes. It supports barrier function, promotes phagocytosis, and modulates cytokine production. Immunological roles of vitamin C include: enhancing neutrophil chemotaxis, phagocytosis, and microbial killing (via reactive oxygen species); supporting proliferation and function of T and B cells; scavenging free radicals during oxidative bursts to limit tissue damage; and regenerating other antioxidants (e.g., vitamin E). By stabilizing endothelial junctions, vitamin C helps maintain the integrity of the respiratory epithelium against viral invasion. It also has weak anti-histamine effects that can relieve cold/flu symptoms (e.g., sneezing, congestion) [11].

In the context of COVID-19, observational studies have found that many patients are vitamin C–deficient, especially when critically ill. Several small trials have tested vitamin C supplementation. Historical data (pre-COVID) show that regular vitamin C intake significantly reduces the incidence of pneumonia and shortens the duration of respiratory infections. For example, controlled trials reported much lower rates of pneumonia in vitamin C–supplemented groups and shorter cold duration. In Wuhan, high-dose intravenous vitamin C was used for severe COVID-19 cases, with reports of ameliorated cytokine storm and improved oxygenation [10].

However, larger randomized trials during the pandemic have yielded mixed results. A 2024 meta-analysis of 11 RCTs found that vitamin C supplementation did not significantly reduce in-hospital mortality or ICU length of stay in COVID-19 patients. The pooled risk ratio for mortality was 0.85 (95% CI 0.62–1.17; p=0.31), and ICU durations were similar between the vitamin C and control groups. Adverse events were also comparable. Thus, there is no conclusive evidence that vitamin C improves survival in established COVID-19, possibly because benefits are more preventive than therapeutic [6].

From a practical standpoint, maintaining adequate vitamin C status is advisable due to its low cost and favorable safety profile. A daily intake of 100–200 mg, obtained through diet or supplementation, is generally recommended for healthy adults. Higher doses (500–1000 mg/day) may be considered to enhance immune resistance, particularly in high-risk environments, while very high-dose intravenous vitamin C should be reserved for hospitalized patients under medical supervision [11]. In summary, vitamin C's antioxidant and immune-supportive properties make it a reasonable adjunct for the prevention of respiratory infections. However, it should not be regarded as a definitive treatment for COVID-19 [6,10].

4.2. Vitamin D

Vitamin D is a steroid hormone with broad immunomodulatory effects. The active form (calcitriol - $1,25(OH)_2D$) enhances innate immunity and tempers inflammation. Mechanisms include upregulating antimicrobial peptides (cathelicidin and β-defensin) in respiratory and gut epithelia, which can directly kill enveloped viruses and strengthen barrier defenses, and modulating immune cell responses (by binding the vitamin D receptor on macrophages, dendritic cells, and T cells). Vitamin D suppresses pro-inflammatory cytokines (e.g., IL-1, IL-6, TNF-α, IFN-γ) and promotes anti-inflammatory pathways. In vitamin D deficiency, there is a shift toward Th1 and Th17 responses, elevating IL-6 and IL-17 levels and increasing the risk of cytokine storm [9].

Vitamin D deficiency is common worldwide and has been linked epidemiologically to higher rates of COVID-19 infection and mortality. Seasonal patterns of influenza/COVID-19 outbreaks (more in winter when vitamin D is lowest) support a potential protective role [9]. Observational studies show that COVID-19 severity is greater among patients with low 25(OH)D levels. Accordingly, multiple clinical trials have tested whether supplementation can prevent or mitigate COVID-19. The results are mixed:

Prevention and mild disease: Some trials suggest benefit. For example, a Mexican study found that 4000 IU/day of vitamin D3 for 30 days reduced SARS-CoV-2 infection rates by 77% compared to placebo. In an Indian outpatient trial, 60,000 IU daily for 7 days in vitamin-D–deficient subjects accelerated viral clearance and reduced inflammatory markers [9]. A 2024 meta-analysis of preventive trials concluded that vitamin D significantly lowered the incidence of COVID-19 and ICU admissions. In that analysis, randomized studies showed a 60% reduction in infection risk with supplementation (pooled OR ≈0.40) and similar benefits in observational studies [8].

Hospitalized and severe disease: Large RCTs in hospitalized patients have generally been negative. The UK "CORONAVIT" trial (n≈6200, population-wide test-and-treat) found that screening and supplementation of adults with deficiencies did not reduce COVID-19 incidence or severity compared with controls [8]. A Brazilian JAMA trial (Murai et al., n=240) gave a single oral bolus of a 200,000 IU dose of vitamin D3 to hospitalized patients and observed no difference in length of stay, ICU admission, need for ventilation, or mortality [9]. A French trial (COVIT-TRIAL) comparing high (400,000 IU) vs low (50,000 IU) dose found initially lower 14-day mortality with the high dose, but this advantage disappeared by 28 days. The Córdoba calcifediol study (pilot, n=76) reported a dramatic reduction in ICU admissions (2% vs 50%), but the small sample and methodological concerns limit generalizability [8].

Systematic reviews: Meta-analyses echo this mixed evidence. An umbrella meta-analysis found that preventive vitamin D (in generally healthy people) is associated with a significantly lower risk of COVID-19 infection and ICU admission [8]. In contrast, systematic reviews of treatment trials conclude that vitamin D supplementation does not significantly reduce mortality or need for ICU/mechanical ventilation in COVID-19 patients. In the Zhang et al. meta-analysis (2023), pooled RCT data showed no mortality benefit (RR≈0.94, p=0.70), whereas cohort data suggested lower mortality (RR≈0.33) but with risk of bias [7].

These findings suggest that vitamin D primarily functions as a preventive modulator in COVID-19, rather than as a therapeutic. Maintaining sufficient vitamin D levels—through daily supplementation of 1000–4000 IU or safe sun exposure—is recommended, particularly for individuals with documented deficiency [9]. Some experts advocate for a short course of high-dose vitamin D in at-risk adults or upon diagnosis, although definitive recommendations require further clinical evidence. A recent observational study among U.S. veterans reported a 33% reduction in 30-day mortality following daily vitamin D3 supplementation. In summary, vitamin D sufficiency supports both innate and adaptive immunity (see Figure 1). It may modestly reduce COVID-19 risk [8-9], but current evidence from large, high-quality trials does not support universal high-dose therapy.

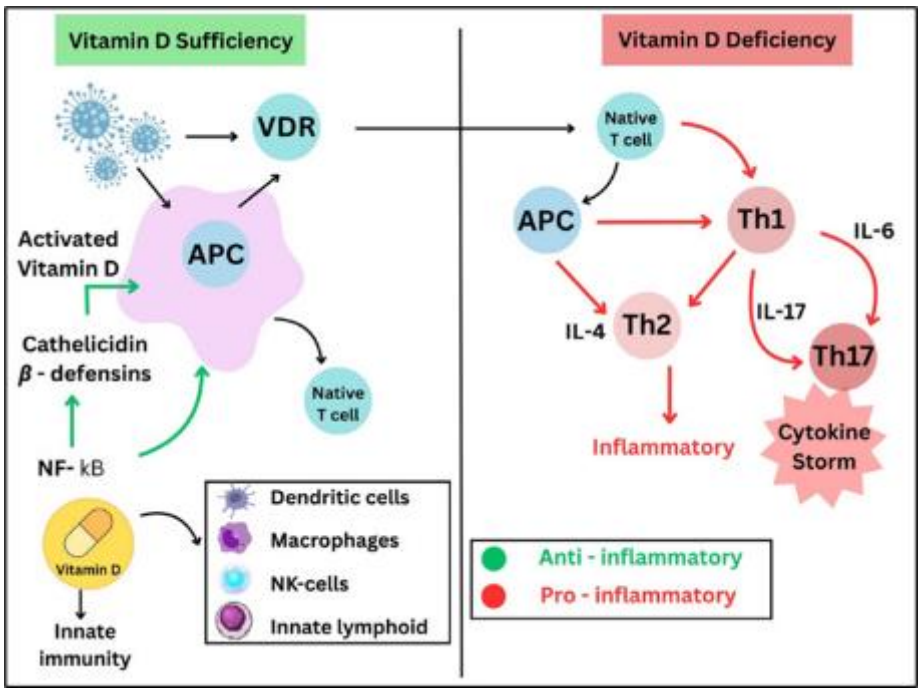

Figure 1 *Vitamin D modulates immune responses in SARS-CoV-2 infection.* Adequate vitamin D (left) activates the vitamin D receptor (VDR), promotes antimicrobial peptide production (cathelicidins, β-defensins), and inhibits NF-κB, resulting in a balanced anti-inflammatory response. Vitamin D deficiency (right) shifts immunity toward Th1/Th17 polarization, leading to excessive IL-6 and IL-17 production and a cytokine storm [9]

5. Saliva, oral health, and COVID-19

Saliva is increasingly recognized as a valuable biofluid and constitutes an essential first-line immune barrier in the oral cavity. It contains epithelial cells, neutrophils, dendritic cells, lymphocytes, and a rich mix of adaptive and innate immune factors – notably secretory IgA (sIgA) antibodies, antimicrobial peptides (e.g. defensins, histatins), and enzymes such as lysozyme and lactoferrin – that can neutralize pathogens at mucosal surfaces. These salivary components act in concert to prevent viral adhesion and replication. SARS-CoV-2 RNA is consistently detectable in saliva, with heterogeneous viral shedding patterns that can serve as biomarkers for disease progression and transmission risk. sIgA specific to SARS-CoV-2 can bind viral particles and block entry into epithelial cells, while peptides like lactoferrin and lysozyme have broad-spectrum antiviral activity [12-14].

The oral cavity is a critical site for SARS-CoV-2 entry, as ACE2 receptors are highly expressed in salivary glands and oral mucosa. The oral microbiome and mucosal immune network further shape this defense. The oral mucosa – populated by commensal bacteria and covered by saliva – regulates immune homeostasis and provides a barrier against pathogens. A balanced, diverse microbiome contributes to host resistance, whereas dysbiosis may predispose to disease [14]. Studies show that in mild-to-moderate COVID-19, the salivary microbiota remains stable, mainly demonstrating resilience. However, severe disease can transiently disrupt microbiome diversity, implying that dysbiosis and uncontrolled inflammation are linked in severe infection. Lower oral microbial diversity and altered salivary cytokine profiles were associated with greater COVID-19 severity, and models combining salivary microbiome and cytokines could predict patient outcomes [15-16]. Such evidence suggests a feed-forward loop in which a healthy oral ecosystem promotes robust mucosal immunity, while loss of microbial balance may weaken mucosal defenses and exacerbate systemic inflammation.

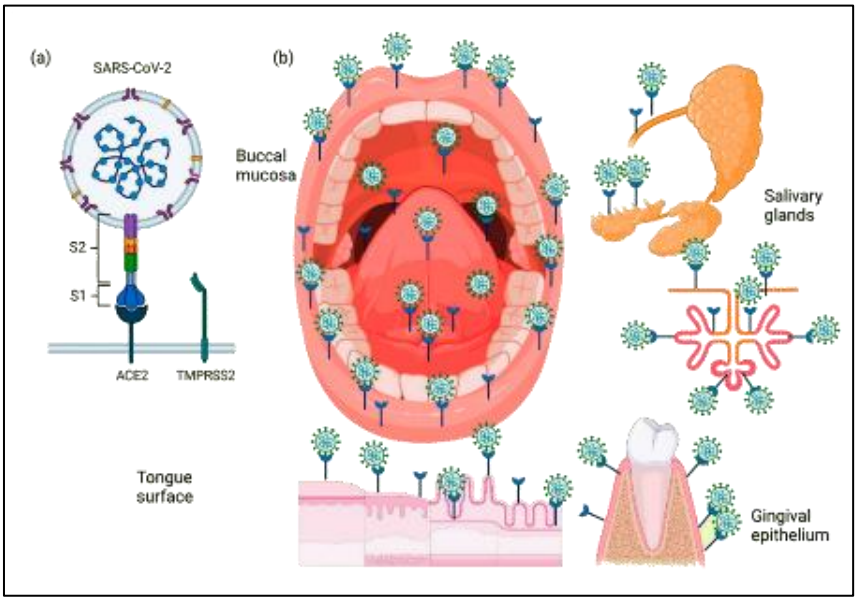

Figure 2 *Schematic representation of the oral cavity as a frontline defense against SARS-CoV-2*. (a) The spike glycoproteins (S1 and S2 subunits) of SARS-CoV-2 bind to ACE2 and TMPRSS2 receptors on host epithelial cells, enabling viral entry, replication, and rapid activation of the innate immune response. This process involves immune cell infiltration and increased production of proinflammatory cytokines. (b) The oral cavity, including the buccal mucosa, tongue, gingival epithelium, and salivary glands, shows viral particles binding to ACE2-expressing surfaces, highlighting the mouth as a key entry point for infection [16]

Oral health also influences COVID-19 risk and severity. Poor oral hygiene and periodontal disease (PD) – chronic inflammatory conditions of the gums – have been associated with worse outcomes in COVID-19 patients. Epidemiological studies and meta-analyses report significantly higher odds of severe COVID-19 symptoms, intensive care admission, and even death in individuals with PD. Mechanistically, periodontitis may exacerbate COVID-19 by acting as a reservoir of systemic inflammation: inflamed periodontal tissues release cytokines and harbor pathogens that can enter the circulation, fueling the "cytokine storm" of severe COVID-19. Periodontal inflammation has also been linked to increased expression of the ACE2 receptor in the oral cavity, potentially enhancing viral entry and dissemination. Viral replication in oral tissues contributes to transmission and local tissue damage. Clinically, COVID-19 patients often present with xerostomia, mucosal ulcerations, candidiasis, and taste disturbances. Persistent oral

lesions and altered salivary flow have also been reported in long COVID, suggesting ongoing immune imbalance and potential autoimmunity [12,16].

Nutritional strategies directly influence oral immunity. Adequate vitamin C enhances salivary antioxidant capacity, reducing oxidative stress in oral tissues and stabilizing epithelial junctions against viral invasion. Vitamin D promotes the production of antimicrobial peptides in oral epithelia, thereby strengthening salivary defenses against viral persistence. Additionally, diets rich in probiotics and omega-3 fatty acids may restore oral microbial balance, mitigating inflammatory cascades linked to severe COVID-19 outcomes. [13]

6. Conclusion

The immunopathology of SARS-CoV-2 infection extends beyond direct viral cytotoxicity, encompassing autoimmune and hypersensitivity reactions, nutritional modulation, and oral health. Clinicians should recognize that SARS-CoV-2 infection may precipitate autoimmune disorders long after the acute phase, and that allergic inflammation or immune-complex disease can contribute to severe respiratory failure. Saliva and oral health represent critical but often overlooked dimensions of COVID-19, serving both as diagnostic media and as immunological barriers. Salivary immune factors (sIgA, enzymes, peptides) constitute a frontline defense that can neutralize viruses at mucosal portals. A healthy oral microbiome and good periodontal status support this immunity, whereas dysbiosis or periodontitis can heighten infection risk and worsen outcomes. Maintaining optimal nutrition—including sufficient vitamin C and vitamin D—supports systemic and oral immunity. A comprehensive approach that addresses viral pathology, immune dysregulation, nutritional status, and oral health is likely to yield the best outcomes in the management of SARS-CoV-2 infection.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors declare no financial interests, personal relationships, or competing affiliations that may have influenced the outcomes or interpretation of this study.

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