# LETTER TO THE EDITOR



# Would periodontitis be a facilitating factor for COVID-19 progression?

Dear Editor.

The pandemic caused by the new coronavirus (SARS-CoV-2) has led to an exponential increase in scientific publications about the symptoms, virulence potential, risk of infection, and particularly methods for the treatment and prevention of the disease called COVID-19 (PAHO, 2020).

We are seeing different hypotheses that have been raised to explain the interaction between the virus and periodontitis, including the approaches taken by Martín Carreras-Presas et al., (2020) and Patel and Woolley (2020). However, because this viral agent is so new, there are no clinical or population studies with greater power of scientific evidence to prove this association.

When inflamed, periodontal tissues undergo several changes in their shape and structure and should, therefore, be investigated in the active form of the disease. Pfützner et al., (2020) believe that epithelial mucosal ulceration caused by periodontitis leads to the loss of protection against viral agents, which can render the individual with periodontitis more vulnerable to SARS-CoV-2 infection.

Some studies suggest that molecular factors present in the healthy oral mucosa may facilitate the interaction between the virus and host cells. Angiotensin-converting enzyme 2 (ACE-2) present in fibroblasts of the periodontal ligament and in the gums has an affinity for the SARS-CoV-2 virus, which easily penetrates the target cells. Therefore, all cells that express ACE-2 are susceptible to infection (Madapusi Balaji et al., 2020).

Wu and Yang (2020) demonstrated increased production of these cytokines in the blood of patients with moderate and severe COVID-19. Thus, the increase in serum interleukins may be linked to worsening of the disease (Sahni & Gupta, 2020). Within the context of preventing possible respiratory complications in patients with COVID-19, the absence of other inflammatory factors such as periodontitis would be extremely important. In the case of patients with active periodontitis, periodontal treatment proved to be efficient in reducing serum levels of IL-17 (Cheng et al., 2014). Hence, it is possible that periodontal treatment of patients with COVID-19 may also decrease these blood interleukins, attenuating the pulmonary inflammatory response.

Another physiological mechanism of periodontitis that might be associated with COVID is related to neutrophil extracellular traps (NETs). NETs represent an alternative form of cell death that causes

tissue damage directly or indirectly through immune mechanisms (Gupta & Sahni, 2020) Periodontitis triggers the production of NETs by activating interferon-alpha. Cell death via NETs can also be stimulated by viruses such as SARS-CoV-2. Exacerbation of NET production is observed in more advanced stages of COVID-19, which may be linked to the development of a hyper-inflammatory state called "cytokine storm" (Mozzini & Girelli, 2020).

The available studies are still not sufficient to answer how periodontitis interacts with SARS-CoV-2, although there is theoretical evidence of how these two pathologies are likely to be associated. Clinical, experimental and longitudinal approaches are particularly useful to provide reliable scientific evidence in the current pandemic scenario.

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# **CONFLICT OF INTEREST**

The authors declare that they have no conflict of interests.

# **AUTHOR CONTRIBUTIONS**

Andressa Chang Rodrigues Fernandes da Silva: Conceptualization; Formal analysis; Writing-original draft; Writing-review & editing. Luanderson Lopes Pereira: Conceptualization; Writing-original draft. Felipe Barreto Lemos: Conceptualization; Writing-original draft. Fernanda Conceição Machado: Conceptualization; Writing-original draft. Andreia Leal Figueiredo: Conceptualization; Writing-original draft; Formal analysis.

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Andressa Chang Rodrigues Fernandes da Silva

Luanderson Lopes Pereira

Felipe Barreto Lemos 🗓

Fernanda Conceição Machado

Andreia Leal Figueiredo 🛡

School of Dentistry, Federal University of Bahia, Salvador, Brazil

# Correspondence

Andressa Chang Rodrigues Fernandes da Silva, School of Dentistry, Federal University of Bahia, 62, Araújo Pinho Avenue, Canela, 40110-150 Salvador, Bahia, Brazil. Email: dessa.chang@hotmail.com

### ORCID

Andressa Chang Rodrigues Fernandes da Silva Phttps://orcid.

org/0000-0003-1641-8922

Luanderson Lopes Pereira https://orcid.

org/0000-0003-4461-9465

Felipe Barreto Lemos https://orcid.org/0000-0003-4731-2450

Andreia Leal Figueiredo https://orcid.

org/0000-0002-5338-6811

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