Periodontitis (PD) is a chronic inflammatory disease of the gum with a high prevalence, reaching over 47% of the US adult population. The key etiological factors are Gram-negative, anaerobic pathogenic bacterial species, which promote dysbiosis in sub-gingival biofilm. Its formation leads to exacerbation of host innate and adaptive immune response, followed by damage of teeth-surrounding tissue and alveolar bone loss. *Porphyromonas gingivalis* is considered to be the most important pathogen in PD development. These bacteria express several virulence factors, including LPS, fimbriae and cysteine proteases, known as gingipains, which ensure infection success. The increased number of clinical data show the high incidence of viral infection in PD patients, mainly of the oral cavity and lungs, especially viruses from the *Herpesviridae* family, such as HSV-1, EBV-1 and HCMV, but also HSV-2 and VZV. Herpesviruses were shown to participate in the progression and severity of PD. On the other hand little is known about the benefits for viruses, facilitation of their replication and progression of viral infection, resulting from the microenvironment formed by periodontal pathogens in inflamed gingiva. Recent observation revealed the higher susceptibility of PD patients for viral infection, including HIV-1, influenza, HBV/HCV, and even SARS-Cov-2, however, the molecular mechanism of the above phenomenon was barely studied. It was documented that PD promotes the primary viral infection facilitating the virus entry to the host cells or favors reactivation of the silent form of the virus. Our unpublished data revealed that *P. gingivalis* efficiently impairs the antiviral immune pathway promoting the virus replication. **Therefore, we aimed to find the molecular explanation of such "united superinfection" and link *P. gingivalis* activity with host immune response to the virus**. We would like to examine the hypothesis that a highly invasive and unique equipped *P. gingivalis* facilitates the formation of the gate of infection for viruses, influencing the primary virus infection. Moreover, we will determine the role of *P. gingivalis* in the reactivation and propagation of the latent form of the virus. In vitro studies are going to be expanded using probes collected from the gingiva of periodontitis patients. We plan to identify viruses associated with this disease, determining their tissue localization together with *P. gingivalis*-determinants.

Taken together, the evaluation of the role of periodontal pathogens and periodontitis on the susceptibility of patients to viral infection is a novel approach. It's of high importance due to the high comorbidity of periodontal diseases with viral infections. We hope that our studies will allow us to explain the molecular mechanism of this phenomenon. **We postulated that PD might be considered as the risk factor of viral infection thus** the prevention and treatment of a periodontal disease may occur to be beneficial.