Chronic periodontitis (also known as gum disease) is a drawn-out medical condition. In the early stages, there are few symptoms including gum swelling, gingival recession, and loose teeth. According to the World Health Organization, severe periodontal disease is estimated to affect nearly 10% of the global population (WHO, 2020). The main causes and risk factors for susceptibility to periodontal diseases are namely poor oral hygiene, cigarette smoking, diabetes mellitus, genetic factors, and infections with i.e. *Porphyromonas gingivalis*, *Fusobacterium nucleatum*, and *Filifactor alocis*. Periodontal disease is also highly correlated with cardiovascular disease, preterm low birth weight, osteoporosis, and Alzheimer's disease. The fundamental reason for tissue damage and disease progression is the over-reactive host inflammatory response to periodontal pathogens. Neutrophils (also called polymorphonuclear cells – PMNs) form the body's first line of defense against invading pathogens and are the most commonly present leukocytes in periodontal pockets. Hyper-reactive PMNs are responsible for chronic inflammation and disease progression. PMNs have the shortest lifespan of all immune cells. However, during infection neutrophil survival is significantly prolonged. The overabundant presence of PMNs prolongs the process of inflammation by secreting inflammatory cytokines, which leads to damage of tissues by secreting protein enzymes and toxic oxygen. This process leads to a progression of the disease, which can be observed by loosening of the teeth in the tooth-supporting tissues and unfortunately, to fall out of the teeth.

The proposed project is willing to characterize the role of cell death of neutrophils upon infection with periodontopathogens. One of the cell death pathways is pyroptosis, an inflammatory cell death that induces the destruction of local tissues. It will be very interesting to study if the bacteria are going to inhibit it and let the cells survive producing more cytokines and antimicrobial enzymes or let them die by the pyroptosis, and make them release inflammatory agents. Therefore, this mechanism could be a reason for the periodontitis progression.

Although, the intensive research of periodontal disease, the role of cell death (apoptosis, pyroptosis, and NETosis) of neutrophils upon infection with *Fusobacterium nucleatum* and *Filifactor alocis* is yet to be described.

Two *in vitro* models will be used in the proposed research, which is human and mice neutrophil-like cell lines. This allows us to compare results obtained in the project. Also, using *in vitro* models helps limit the number of laboratory animals in the research.

The project's goal is to broaden knowledge on the necrobiology of neutrophils and molecular mechanisms of periodontosis and, which can help with other inflammatory diseases.