Periodontitis affects a large proportion of human population. The disease is very frequent and the symptoms intensify with age. Initial symptoms, such as bleeding of gums are often ignored, which allows the disease to progress and gradually damage the tissues supporting the teeth. Progression of periodontitis can be observed as gradual lowering of the gum line and exposure of the neck of the tooth, associated with degradation of the tooth supporting tissues. This leads to increased mobility and eventually loss of the teeth. It usually takes years for the disease to develop fully, therefore loss of the tooth may be falsely associated with the aging. Currently it is known that microorganisms colonizing oral cavity, especially those on the surface of tooth below line of gums, are responsible for periodontitis. The difference should be noted between natural, oral-health associated microbiota, and those responsible for pathological state, when microorganisms trigger uncontrolled, sustained immune response of the host. Now it is known that the switch from good to pathogenic microbiota is mainly driven by only few bacterial species found among hundreds of species colonizing the oral cavity. One of those bad ones is *Porphyromonas gingivalis*, which produces numerous factors that can stimulate and alter the response of the host to the presence of bacteria in gingival pockets. Action of those factors leads to changes in the composition of microflora manifested by proliferation of pathogenic species on the tooth surface. This leads to the development of chronic inflammatory state, which directly causes the damage of the tooth supporting tissues. It is also worth noticing that periodontitis is associated with increased risk of developing rheumatoid arthritis, another common inflammatory disease. It is speculated that the connection between periodontitis and rheumatoid arthritis results from the production of citrullinated proteins in the tissues affected by periodontitis. The only known bacterial species that can citrullinate proteins is *P. gingivalis*, which possesses unique enzyme PPAD responsible for this modification. Citrullination of some factors produced by human host leads to their inactivation. This can result in lowering antibacterial mechanisms of the host, as well as slowing down the process of tissue regeneration. PPAD is therefore a factor involved in development and progression of periodontitis. Recently it was discovered that two forms of PPAD exist in *P. gingivalis* strains isolated from periodontitis patients, and the newly described variant is twice as active as the one known before.

Because PPAD is involved in development and progression of periodontitis, it is crucial to understand how increased production of citrullinated proteins by *P. gingivalis* can contribute to this disease. Research planned in this project is aimed firstly at characterizing the new, more active form of PPAD by measuring its ability to modify various substrates, including host proteins involved in the immune response. The structure of this new enzyme will also be investigated to explain how point mutations contribute to changes in efficiency of the catalyzed reaction. Next step will be to analyze if citrullinated proteins of *P. gingivalis* can trigger pathological processes linked to initiation and progression of periodontitis. The critical step will be the design of a fast method for detection of PPAD variants in the microflora of periodontitis patients. To this end the PCR technique will be used, which will allow for detection of genetic variants of PPAD in samples from patients. The designed method will be used for screening a large group of patients and healthy volunteers to look for correlation between occurrence of *P. gingivalis* strains expressing different variants of PPAD and the severity of the disease. Finding such correlation may lead to development of a new diagnostics approach to recognize potentials for development of more severe periodontitis and its association with rheumatoid arthritis.