Summary

Periodontitis is a disease, which affects teeth and supporting tissue with the high prevalence in adult population of developed countries. The incidence of this inflammatory disease reaches up to 30%. Periodontal tissue damage does ultimately lead to tooth loss, which is a consequence of extensive host's innate immune response to bacterial invaders and their virulence factors. At the moment, the most effective way of treating periodontitis is prophylaxis of the disease, and symptomatic treatment consisting of the mechanistic removal of the biofilm deposits from the gingival pockets. The signs of inflammation are visible not only locally in the periodontal pockets, but also systemically as elevated levels of inflammatory molecules present in the blood of periodontitis patients. These features may influence the development of atherosclerotic plaque and aggravation of other chronic inflammatory diseases, including rheumatoid arthritis, arteriosclerosis or neurodegenerative diseases. Hence, the important aim of research is to develop new treatments, which are more effective than the existing therapies. To improve therapeutic approaches the discovery of molecular aspects of exacerbated inflammation typical for periodontitis development is necessary. Endogenous control of the magnitude and duration of inflammatory signaling is executed by plethora of negative regulators, which possess a great potential to inhibit inflammatory reaction. Although the molecular basis of periodontitis is intensively studied, the role of these proteins has not yet been described in context of this disease. Therefore, the main significance and intent of the project is to explore our knowledge base of this field with major focus on role of negative signaling regulator MCPIP-1, also known as Regnase-1 in periodontitis. MCPIP-1 is intracellular protein, characterized by RNAse activity, which can effectively remove transcripts encoding proinflammatory cytokines crucial in disease progression, such as IL-6, IL-8 and IL-17. The MCPIP-1 protein was identified in majority of cells, but its exceptionally high level was documented in gingival tissue. In our proposal we would like to verify two hypotheses. The first one, assume that MCPIP-1 is modulated by periodontopathogens. Thus loss of its regulatory efficiency leads to inappropriate inflammatory responses in the gingival tissue. The second hypothesis suggests that constitutive tissue level of MCPIP-1 is a key attribute, which determines the periodontitis development. We are planning to perform the complex analysis including clinical samples analysis, experimental animal-models and *in vitro* studies to evaluate the molecular role of MCPIP-1. Our research intends to identify the novel role of MCPIP-1 in periodontitis. This will not only expand a general knowledge of its mechanisms of action in bacterial infections, but also contribute to the justification of the fact that immunological imbalance, derived from molecular signalling inaccuracy, underlies the aetiology of periodontitis.