Periodontitis is a group of multifactorial, inflammatory infectious diseases, which belong to the most prevalent infections in humans. They are initiated by an ecological shift in the composition of oral bacteria, resulting in inflammation and destruction of tooth-supporting tissues. In a wider context, periodontitis is a known risk factor complications from systemic diseases. *P. gingivalis* is considered as one of the main etiological agents and key pathogen responsible for initiation and progression of chronic periodontitis. This bacterium is found in large numbers not only in oral cavity of patients with chronic periodontitis, but also in oral cavity of healthy subjects in low numbers, in increased numbers at periodontitis sites of diabetic patients, as well as in other tissues in patients suffering from cardiovascular diseases. Heme, a key virulence factor for *P. gingivalis*, is provided to the bacterial cell mostly by heme-binding protein, HmuY, and enzymes degrading proteins, gingipains. Given the high incidence of periodontitis, diabetes and cardiovascular diseases, resulting in serious health problems and economic costs, defining their mechanistic link is of high importance. Our aim is to investigate synergistic heme acquisition mechanisms of *P. gingivalis* and other bacteria, involving also host proteins with regard to their involvement not only in colonization of the healthy oral cavity, but importantly in initiation and progression of chronic periodontitis associated with systemic diseases. Our main hypothesis is that *P. gingivalis* could employ the heme acquisition mechanisms expressed by other commensal/pathogenic bacteria together with host proteins, especialy proteins modified by pathological conditions, to increase its own virulence and cause dysbiosis of oral microbiome. We suspect that also non-pathogenic, often commensal microorganisms may display the potential for heme acquisition, which contributes to production of a significant pool of available heme for *P. gingivalis*. One of such proteins may be non-canonical heme-binding protein of Streptococcus gordonii, namely glyceraldehyde-3-phosphate into glycerate-1,3-diphosphate (SgGAPDH). Moreover, glycated host hemoproteins may serve as a valuable nutrient source for periodontopathogens. We propose to examine if and how these proteins promote *P. gingivalis* growth, biofilm formation and invasion of host cells in order to cause infection. To fulfill aims of this project a variety of methods will be employed, including bacterial and host cell cultures, spectrophotometric, chromatographic, and microscopic methods. Results gained in this project will broaden current knowledge regarding novel heme acquisition, synergistic mechanism occurring between proteins produced by bacteria and human proteins. This is especially important for *P. gingivalis*, allowing this bacterium playing key pathogen role in chronic periodontitis alone or in coexistence with systemic diseases.