Platelets have during the recent decades become accepted as immune cells, and not only as key-players in the haemostasis. They express surface receptors e.g. toll-like receptors, capable of recognizing and binding pathogens and mediate inflammatory responses and engagement of more immune cells. Platelets can release a plethora of pro- and anti-inflammatory mediators upon bacterial activation and thereby recruit more immune cells to the site of infection and initiate an inflammatory process. Periodontitis is a chronic inflammation associated with the degeneration of tissues and bones supporting the teeth. Porphyromonas gingivalis is one of the etiological agents in periodontitis and known to have several mechanisms to modulate and evade the host immune response. Periodontitis is suggested to be associated with cardiovascular atherosclerotic disease. The present thesis elucidates the role of platelets in sensing bacterial infection, the provoked platelet immune response upon stimulation with P. gingivalis and the role of platelets as a possible linker between periodontitis and atherosclerosis.