In the past decade, the increased prevalence of cardiovascular disease and mortality among patients with periodontitis has received increased attention. *Porphyromonas gingivalis* is the major pathogen causing periodontal disease and has been localized in atherosclerotic plaques. Inflammation and host inflammatory responses have been attributed as the key factor for the pathogenesis of atherosclerosis. Smooth muscle cells are the main components of vascular wall. During the atherosclerotic process, activated vascular smooth muscle cells migrate and proliferate in the intima and contribute to the formation of atherosclerotic plaques. Although, *Porphyromonas gingivalis* has been considered to play a significant role in the development of atherosclerosis, how this bacterium affects the innate and adaptive immunity and its role in maintaining a chronic inflammatory condition is far from clear understanding. The aim of this thesis was to clarify the mechanisms of *Porphyromonas gingivalis* interaction with the vessel wall by exposing human aorta smooth muscle cells to live *Porphyromonas gingivalis* in vitro.