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Recently, interest has increased in the possibility that infection may cause atherosclerosis. A considerable body of seroepidemiological evidence supports a role for certain bacteria, notably Chlamydia pneumoniae, & certain viruses notably cytomegalovirus in the etiology of atherosclerosis. Although proof that bacteria or viruses can cause atherosclerosis remains elusive, it is quite plausible that infection may potentiate risk factors, such as hypercholesterolemia. Cells within atheroma itself may be a site of infection e.g. Macrophages existing within established atherosclerotic lesion might become infected with C. Pneumoniae, which could spur their activation & accelerate the inflammatory pathways that are currently believed to operate within atherosclerotic intima. Specific microbial products such as lipopolysaccharides, heat shock proteins or other virulence factors might act locally at the level of arterial wall to potentiate atherosclerosis in infected lesion.

Extra vascular infection might also influence the development of atheromatous lesion & provoke their
complications. Acute phase response to an infection in a nonvascular site might affect the incidence of thrombosis complications of atherosclerosis, by increasing fibrinogen or plasminogen activator inhibitor-1 (PAI-1) or altering the balance between coagulation and fibrinolysis.

Acute infection might also produce hemodynamic alteration that could trigger coronary events e.g. Tachycardia & metabolic demand of fever could augment O₂ demand of heart, precipitating ischemia in an otherwise compensated individual. These various scenarios illustrate, how infectious processes, either local in atheroma or extra vascular might aggravate atherogenesis particularly in preexisting lesions.