Immunopathogenesis of atherosclerosis

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Recent clinical studies indicate that the number of microbial infections (the "pathogen burden") critically determines the development and progression of atherosclerotic disease. Viruses or bacteria with a specific tropism for cells of the vascular wall may contribute to the initial vascular injury via direct cytopathic effects or via the induction of genuine autoimmune responses. Immunopathological processes such as molecular mimicry, epitope spreading, or bystander activation of self-reactive lymphocytes most likely fuel the chronic inflammatory process in the vascular wall. Recognition of atherogenesis as a pathogen-driven, immunopathological process makes this disease amenable to new treatment strategies such as vaccination or immunomodulation.

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