# THE ROLE OF HEAT SHOCK PROTEINS IN PATHOGENESIS OF ATHEROSCLEROSIS

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Heat shock proteins (Hsp) or shaperons are oligomerous proteins, that help to coagulate native or denaturated proteins. They are synthesized in stress situations (therefore they are also called stress-proteins) and they are high-conservative group of proteins, that is, their structure is similar in different organisms. Cells of arterial wall product large amounts of Hsp in response to infection, fever, mechanical and oxidative stress, cytokines, heavy metals, alcohol or inhibitors of cell metabolism.

Therefore, the **aim of work** is studying of modern literature and analysis of role of heat shock proteins in pathogenesis of atherosclerosis.

**Results of work.** Found that under stress phosphorylating of small heat shock proteins Hsp27 and Hsp20 develops that influences on polymerization of actin of arterial wall. Heat shock protein 47 is the stress protein, which can be the shaperon for collagen. It is not known about its participation in pathogenesis of atherosclerosis for certain, but its localization in a fibrotic layer, regulation by height factors in parallel with a type of procollagen-1 and selective regulation by stress revealed possibility of determining stability of pre-existing plaque by Hsp47.

In recent years there were done many discoveries that allow discussing immunoinflamation mechanisms of development of atherosclerosis. Heat shock proteins Hsp60 and Hsp65 belong to the group of possible antigens for immune response. Cellular and humoral immunity to phyllogenetic conservative antigen of the Hsp60 is the triggering mechanism of development of the earliest stages of atherosclerosis. Data, which had got by scientists, show that Hsp60 contains in human atheroma and can cause damage by direct activation of macrophages. Hsp60 is the reason of inflammation cytokines formation, induction of metal-protease and low-density lipoproteins oxidation. Each of these changes affects the development of atherosclerosis. The mechanism of formation of autoimmune response to Hsp consists in an immune response for the antigens of infectious microorganism (for example, Hsp60 of Chlamydia), that cross reacts with the homologous proteins of the host organism in form of molecular mimicry.

**Conclusions.** Thus, in case of infection, organism immunity begins to be formed against Hsp60 of microorganisms. However, as a result of similarity of microbial and human Hsp60 there is a cross immunoreaction against human Hsp60, therefore autoimmune reaction that is the constituent of the infectional-autoimmune inflammation hypothesis of pathogenesis of atherosclerosis.