

**МІНІСТЕРСТВО ОСВІТИ І НАУКИ УКРАЇНИ  
СУМСЬКИЙ ДЕРЖАВНИЙ УНІВЕРСИТЕТ  
ФАКУЛЬТЕТ ІНОЗЕМНОЇ ФІЛОЛОГІЇ  
ТА СОЦІАЛЬНИХ КОМУНІКАЦІЙ**



# **СОЦІАЛЬНО-ГУМАНІТАРНІ АСПЕКТИ РОЗВИТКУ СУЧАСНОГО СУСПІЛЬСТВА**

**МАТЕРІАЛИ V ВСЕУКРАЇНСЬКОЇ НАУКОВОЇ КОНФЕРЕНЦІЇ СТУДЕНТІВ,  
АСПРАНТІВ, ВИКЛАДАЧІВ ТА СПІВРОБІТНИКІВ**

**(Суми, 20-21 квітня 2017 року)**

**Суми  
2017**

## THE ROLE OF OSTEOPONTIN IN THE DEVELOPMENT OF THE HEART VALVE CALCIFICATION

I. A. Morozova, I. M. Zakorko, R. A. Moskalenko

**Relevance:** Calcific aortic valve stenosis (CAVS) is an important clinical problem affecting 2.8% of adults over 75 years of age in the developed countries. Studies suggest that the progression of CAVS is actively regulated with valve endothelial injury leading to inflammation, fibrosis and calcification. Multiple biological pathways are responsible for aortic valve degeneration, with studies suggesting that the progression of CAVS is triggered by injury to the valve's endothelial cell lining and is actively regulated. In addition to endothelial cell loss, injury leads to infiltration of circulating inflammatory cells, extracellular matrix deposition and concomitant activation of proinflammatory cytokines.

Identifying markers that play a role in the pathogenesis of CAVS is therefore crucial in understanding the progression of this disease and may reveal potential therapeutic targets for its treatment. Indeed, similar to vascular smooth cells, valvular interstitial myofibroblast-like cells showed expression osteopontin, osteocalcin, and Runx2, which suggests an active mineralization process prior to the development of end-stage calcification (macrocalcification). Osteopontin (OPN) was originally linked to bone mineralization, but has since been shown to be a multifunctional proinflammatory cytokine with important roles in promoting inflammation and tissue remodelling including fibrosis and angiogenesis.

**Objective:** To analyze English articles in foreign scientific journals regarding the role of osteopontin in the development of calcification.

**Materials and Methods:** 17 English-language articles were analyzed, they have been placed in the public domain on the website Pub Med. All articles were published in the professional journals, which are the members of the scientific register SCOPUS.

**Research results:** OPN is an acidic phosphoprotein normally found in mineralized tissues such as bones and teeth, and it is involved in regulation of mineralization by acting as an inhibitor of apatite crystal growth, as well as promoting osteoclast function. Although OPN is not found in normal arteries, but some scientists reported, that OPN is abundant at sites of calcification in human atherosclerotic plaques and in calcified aortic valves. Studies demonstrated that a subset of valve macrophages actively synthesize osteopontin protein in aortic valvular tissue. Other studies similarly have demonstrated that macrophages may synthesize

osteopontin mRNA and contain osteopontin protein in human atherosclerotic plaques; therefore, detection of osteopontin in macrophages has precedent. It also has been shown by immunohistochemistry. Osteopontin binds readily to hydroxyapatite and may mediate adherence of osteoblasts and osteoclasts to bone matrix through an arginine-glycine-aspartate integrin-binding sequence and amplify the calcification. In contrast, other literature sources state that osteopontin regulates negatively mineral deposit formation, and it is necessary to myofibroblast differentiation and activity, which are produced in the response to a profibrotic cytokine, transforming growth factor-1. OPN elaborated by stromal or inflammatory cells at sites of ectopic mineralization, binds to bioapatites and initially physically inhibits crystal growth. Binding of OPN to bioapatite simultaneously provides a recognition site and/or concentration gradient for macrophages and giant cells thereby leading to localized accumulation.

**Conclusion:** there is no definitive data about the role of osteopontin in the development of calcific aortic valve stenosis, that requires further studying.

## THE NOBEL PRIZE IN PHYSICS 2016: REVIEW

Lisovenko M., *group PhEm.-61*

Mulina N. I., *Ph.D., EL Advisor*

For the past 120 years, the Nobel Prize has honored scientists for discoveries that the committee feels most benefit mankind. And theoretical discoveries of topological phase transitions and topological phases of matter lead David J. Thouless (the University of Washington, Seattle), Duncan M. Haldane (Princeton University) and J. Michael Kosterlitz (Brown University) to getting The Nobel Prize in Physics 2016. Their work started in the USA in the early 1970s by identifying a completely new type of phase transition in 2D systems with topological defects. This became a powerful breakthrough in the theoretical understanding of matter mysteries, which created new perspectives on the development of innovative materials. [1]

The main idea of this work comprises two main terms – phase transition and topology. Phase transition it is a phenomenon when a substance suddenly changes its material properties. The simplest example is when water turns into ice with dropping temperature. Phase transition does