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**Role of VEGF in development of chronic obstructive pulmonary disease in combination with hypertension.**

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Nowadays lot of attention is paid to issue of comorbid disease. On the basis of progressive humanity development, it is mentioned a decline of ecological situation, reduction of human motor activity, increasing of bad habits, poor nutrition and organism senilism. All these factors are lead to formation of several disease in humans at the same time. The prevalence of comorbid pathology averages 76,6%, 82% - for women, 72% - for men. One of the most common comorbid conditions is a chronic obstructive pulmonary disease (COPD) in combination with hypertension. According to the data WHO (newsletter #310, 2014) COPD and hypertension are included to the list of ten leading death causes in the world. Hypertension is found in COPD patients with varying frequency (from 6,8% to 76,3%) with average 34,3%. On this stage endothelial dysfunction can be considered as one of the most important independents risk factors for diabetes, atherosclerosis, COPD, hypertension, sepsis and growth of malignant neoplasms. One of the main endothelium functions is vascular growth regulations. One of the angiogenesis markers is vascular endothelial growth factor (VEGF). Among the angiogenic factors family, VEGF is considered to be a major one while neovascularization. Proteins which belong to the VEGF family group are glycoproteins that stimulate the formations of new blood and lymph vessels and also increase vascular permeability. One of the main factors for comobid pathology development is hypoxia and cells structure transformation under the mechanical factors influence that initiates VEGF production. By evaluation of GOLD 2014 revision COPD is characterized by a bronchial tubes affect (obstructive bronchiolitis) so as parenchymal distruction (emphysema). There are an extremely alveolar septa thinning and significantly reducing of their vascularization while progressive capillary endothelium loss and alveolar epithelium that leads to the substitution of lung tissue by the fibrous. Hypoxia promotes HIF-1a stabilization (hypoxia inducible factor-1a) which rapidly degraded at a sufficient oxygen concentration. HIF-1a and HIF-1βb is combined under hypoxia condition. Received complex is binded to a specific sensitive to the hypoxia region of the VEGF genes and increase its expression. Isolated mechanical stretching in a cell culture can contribute to VEGF realizing. Physical activity leading to the increasing of blood flow increasing shear stress and endothelial cells transformation followed by not only by increased VEGF level but also by increasing VEGFRs amount. A growth of shear stress and activation of the renin-angiotensin-aldosterone system while hypertension is also associated with increased VEGF content by mechanical influence on the vascular endothelial cells and angiotensin 2 interaction with angiotensin first type receptors which leads to the expression HIF-1a growth and increasing of VEGF synthesis. Thus VEGF and its receptors can be involved in many processes while COPD and hypertension, such as: airway walls remodeling, endothelial cell apoptosis and vascular remodeling.