LOW-GRADE SYSTEMIC INFLAMMATION AS A GENERAL FRAMEWORK OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND COMORBID CONDITIONS

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Abstract

Epidemiological studies have shown frequent association of COPD with diseases of the cardiovascular system, obesity, metabolic syndrome, type 2 diabetes and many other diseases, characterized by dominating low grade inflammation (LGI) and insulin resistance (IR). This review highlights current understanding of LGI and IR, their role in the etiology and pathogenesis of COPD, and management of this condition, based on molecular mechanisms of LGI. In particular, it is emphasized that the LGI is the key mechanism that binds together all the risk factors for COPD and explains the frequency of comorbid conditions. Authors underline the differences between acute local inflammation, such as pneumonia, and systemic low-intensity inflammation. Latest data on etiology of LGI (the role of stress, including psychosocial, smoking, western type diet, physical inactivity and chronic infection) are presented. The data on the role of nuclear transcription factor kappa B (NFkB) and the peroxisome proliferator activating receptors (PPAR) in the molecular mechanisms of LGI and IR are also presented. Literature data indicate the presence of the bidirectional relationship between COPD and LGI, when pulmonary infection and chronic local inflammation may be the source of LGI and LGI, initially, may contribute to the occurrence and progression of COPD. Prevention and treatment strategies of COPD should consider the role, known factors and molecular mechanisms of LGI. Prevention and treatment of COPD using natural factors and pharmacological blockers of proinflammatory NFkB and activators of antiinflammatory PPAR, particularly, metformin and pioglitazone, seem to be promising approaches.

Key words: chronic obstructive pulmonary disease, systemic inflammation, insulin resistance, metformin, pioglitazone.

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