**TNF-MEDIATED AND FASL-MEDIATED APOPTOSIS AT DIFFERENT STAGES OF CHRONIC CARRAGEENAN-INDUCED GASTROENTEROCOLITIS**

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**Introduction.** The food additive carrageenan (E407) has been widely used as thickener in food industry. However, carrageenan has been known to be a very potent stimulator of inflammatory processes since 1960s. Despite this fact, mechanisms of carrageenan-induced inflammation are still poorly understood.

**Aim.** The investigation was designed to study TNF-mediated and FasL-mediated apoptosis in rats with chronic carrageenan-induced gastroenterocolitis.

**Materials and methods.** Thirty female Wistar rats were used in the experiment. They were subdivided into 3groups (the 1st group – intake of carragenan *per os* within 4 weeks; the 2nd group – intake of carragenan *per os* within 2 weeks; the 3rd group – control group consisting of healthy rats). The intake of carrageenan led to the development of chronic gastroenterocolitis (this fact was proven morphologically and biochemically). Blood levels of TNF-α and Fasligand,as well as caspase-3 activity, were measured by ELISA.

**Conclusion.** It was found that carragenan-induced gastroenterocolitis led to elevation of caspase-3, TNF-α, and Fas-ligand. An increase in both caspase-3 activity and TNF-α level was more pronounced in animals from the first group. On the contrary, FasL levels were higher in animals from the second group compared to the first one. The correlation analysis shows that FasL-mediated apoptosis prevails at the earlier stage of the disease, whereas TNF-mediated apoptosis is more active at the later stage.