cules following GM-CSF priming. J. Immunol. 197 : 4090–4100. doi 10.4049/jimmunol.1600051

Wang Y., Gao W., Shi X., Ding J., Liu W., He H., Wang K., Shao F. 2017. Chemotherapy drugs induce pyroptosis through caspase-3 cleavage of a gasdermin. Nature. 547 : 99–103. doi 10.1038/nature22393

Wartha F., Beiter K., Normark S., Henriques-Normark B. 2007. Neutrophil extracellular traps: Casting the NET over pathogenesis. Curr. Opin. Microbiol. 10 : 52–56. doi 10.1016/j.mib.2006.12.005

Welty D.M., Long R.D., Bailey J.R., Parnell M.J., Hoe N.P., Adams G.G., Deleo F.R., Musser J.M. 2005. Extracellular deoxyribonuclease made by group A *Streptococcus* assists pathogenesis by enhancing evasion of the innate immune response. Proc. Natl. Acad. Sci. USA. 102 : 1679–1684. doi 10.1073/pnas.0406641102

Yang D., He Y., Muñoz-Planillo R., Liu Q., Núñez G. 2015. Caspase-11 requires the pannexin-1 channel and the purinergic P2X7 pore to mediate pyroptosis and endotoxic shock. Immunity. 43 : 923–932. doi 10.1016/j.immuni.2015.10.009

Yang W.S., Stockwell B.R. 2008. Synthetic lethal screening identifies compounds activating iron-dependent, nonapoptotic cell death in oncogenic-RAS-harboring cancer cells. Chem. Biol. 15 : 234–245. doi 10.1016/j.chembiol.2008.02.010

Yang W.S., Stockwell B.R. 2016. Ferroptosis: Death by lipid peroxidation. Trends Cell Biol. 26 : 165–176. doi 10.1016/j.tcb.2015.10.014

Yano T., Kurata S. 2008. Induction of autophagy via innate bacterial recognition. Autophagy. 4 : 958–960. doi 10.4161/auto.6802

Yipp B.G., Petri B., Salina D., Jenne C.N., Scott B.N., Zbytnuik L.D., Pittman K., Asaduzzaman M., Wu K., Meijndert H.C., Malawista S.E., de Boisfleury Chevance A., Zhang K., Conly J., Kubes P. 2012. Infection induced NETosis is a dynamic process involving neutrophil multitasking *in vivo*. Nat. Med. 18 : 1386–1393. doi 10.1038/nm.2847

Yoshikawa Y., Ogawa M., Hain T., Yoshida M., Fukumatsu M., Kim M., Mimuro H., Nakagawa I., Yanagawa T., Ishii T., Kakizuka A., Sztul E., Chakraborty T., Sasakawa C. 2009. Listeria monocytogenes ActA-mediated escape from autophagic recognition. Nat. Cell Biol. 11 : 1233–1240. doi 10.1038/ncb1967

Zawrotniak M., Rapala-Kozik M. 2013. Neutrophil extracellular traps (NETs) – formation and implications. Acta Biochem. Polonica. 60 : 277–284.

Zelenay S., Reise Sousa C. 2013. Adaptive immunity after cell death. Trends Immunol. 34 : 329–335. doi 10.1016/j.it.2013.03.005

Zilka O., Shah R., Li B., Friedmann Angeli J.P., Griesser M., Conrad M., Pratt D.A. 2017. On the mechanism of cytoprotection by Ferrostatin-1 and Liproxstatin-1 and the role of lipid peroxidation in ferroptotic cell death. ACS Cent. Sci. 3 : 232–243. doi 10.1021/acscentsci.7b00028

Zong W.X., Thompson C.B. 2006. Necrotic death as a cell fate. Genes Dev. 20 : 1–15. doi 10.1101/gad.1376506

PROINFLAMMATORY MECHANISMS OF NEUTROPHIL GRANULOCYTE DEATH

S. N. Pleskova[a, b, *] and R. N. Kriukov[a]

[a] *Research and Education Center for "Physics of Solid State Nanostructures", Lobachevsky Nizhny Novgorod State University, Nizhny Novgorod, 603950 Russia*

[b] *Department of Nanotechnology and Biotechnology, Alekseev Nizhny Novgorod State Technical University, Nizhny Novgorod, 603950 Russia*

**e-mail: pleskova@mail.ru*

Neutrophil granulocytes play a central role in the innate nonspecific defense response of the human organism. They have a rapidly realizable, highly aggressive and bioactive factors (ability to produce excessive amounts of reactive oxygen and nitrogen species and a wide range of hydrolytic enzymes; cationic proteins; ability to produce cytokines, etc.). More than 10 mechanisms of cell death have been officially registered for them, including the unique formation of NET (neutrophil extracellular traps). Since neutrophil is the central part of exudative-destructive inflammation, the mechanism of its death can directly influence on the dialectic of inflammation either by helping to resolve inflammation and repair of damage, or, conversely, by increasing the effectors cascade and contributing to the development of flogogenic complications. Here it is discussed the mechanisms of neutrophil granulocytes death with proinflammatory activity.

Keywords: neutrophil granulocytes, inflammation, necrosis, necroptosis, ferroptosis, pyroptosis, neutrophil extracellular traps, autophagy