Streszczenie w języku angielskim

In the face of a global increase in fungal infections, particularly those caused by the *Candida auris* strain, which is characterized by multidrug resistance and the ability to evade the host's immune response, as well as in the context of the COVID-19 pandemic, where an increase in the occurrence of fungal infections was noted, there is an urgent need to develop new, effective therapies. pGSN, due to its immunomodulating and anti-inflammatory properties, may play a key role in limiting tissue damage caused by excessive immune response, typical for severe cases of COVID-19 and *C. auris* infections.

The research objectives as part of the doctoral dissertation included assessing the impact of pGSN on the innate immune response, determining the mechanism of action at the molecular level, and analyzing the anti-inflammatory effects in relation to neutrophils and endothelial cells. Additionally, the protective action of pGSN on endothelial cells forming the blood-brain barrier was evaluated in an innovative 3D culture flow model exposed to the S1 subunit of the SARS-CoV-2 protein.

The results of the conducted research indicated for the first time that pGSN exhibits an immunomodulating action by stimulating the expression of type I class B "scavenger" receptors (SR-B) on the surface of human neutrophils, which results in the stimulation of phagocytosis. Additionally, it was observed that pGSN alleviates inflammation by suppressing the secretion of pro-inflammatory cytokines and chemokines, limiting the overproduction of neutrophil extracellular traps, and reducing the increased permeability of the blood-brain barrier caused by the S1 protein. These findings reveal a new molecular mechanism of action of pGSN and underscore its potential role as a therapeutic target in treating inflammatory states and infections.

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