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Determination of the cytotoxicity of nanosilver coated with carbosilane dendrons against B14 cells BIOOPEN 2021 – POST-CONFERENCE COMMUNICATION

MARTA KĘDZIERSKA 💿, KATARZYNA MIŁOWSKA 💿

University of Lodz, Faculty of Biology and Environmental Protection, Department of General Biophysics, Pomorska 141/143, 90-236 Lodz, Poland E-mail: marta.kedzierska1@biol.uni.lodz.pl

The aim of the study was to check the cytotoxicity of silver nanoparticles (NP) coated with carbosilane dendrons – NP Ag_{143} , NP Ag_{788} , NP Ag_{1792} – and to learn more about their mechanism of action.

The methods used were the MTT assay, assessing the viability of B14 cells, and determination of the level of reactive oxygen species (ROS) using the H_2DCFDA probe. In addition, by using a JC-1 probe, the pro-apoptotic activity of NP Ag was assessed.

All tested NP Ag showed cytotoxic properties against B14 cells. Along with an increase in NP Ag concentration, a decrease in cell viability was noticeable. The most toxic of the tested compounds was NP Ag₁₇₉₂ at a concentration of 20 μ M, where the cell viability was only 21% compared to NP Ag₁₄₃ and NP Ag₇₈₈, for which the viability was 40% and 43%, respectively. Studies with the H₂DCFDA probe have shown that NP Ag induce oxidative stress. For NP Ag₁₄₃, the highest level of ROS was observed for

the concentration of 10 μ M and it was 4.5 times higher than that of the control. For NP Ag₁₇₉₂ at a concentration of 10 μ M, the ROS content increased 4-fold. In the case of NP Ag₇₈₈, the highest level of ROS was observed after the action of the compound at a concentration of 5 μ M (a 2.7-fold increase). The experiment with JC-1 showed that all test compounds caused a significant reduction of $\Delta\Psi$ m starting from a concentration of 10 μ M. The cells incubated with NP Ag₇₈₈ at a concentration of 15 μ M for which cell viability was approx. 60% had the lowest potential value.

On the basis of the studies conducted, it can be concluded that the tested compounds are cytotoxic at higher concentrations, but the use of low concentrations of NP Ag $(1-5 \mu M)$ does not cause a significant decrease in viability. The mechanism of cytotoxicity is based, inter alia, on inducing oxidative stress and generating ROS, as well as mitochondrial dysfunction.