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AgNPs mechanism, AgNP-CAZ synergism, and anisotropic AgNPs activity against Burkholderia pseudomallei

Burkholderia pseudomallei is the causative agent of melioidosis, an endemic infectious disease primarily found in northern Australia and Southeast Asia. *B. pseudomallei* (BP) is intrinsically resistant to most common antibiotics. Our research interest is to find alternative in treatment of melioidosis. We observed the activity of AgNPs against several strains of BP and mechanism behind. Firstly, we studied the mechanism of AgNPs. AgNPs exhibited two-phase mechanism: cell death induction and ROS induction. The first phase was a rapid killing step within 5 min causing direct damage of the cytoplasmic membrane of the bacterial cells. The second phase, the ROS induction, occurred 1-4 h after AgNPs treatment where the Ag+ interacted with biomolecules resulting in ROS formation. This is the direct kinetic evidence of AgNPs killing mechanism by which cell death is separable from ROS induction. Secondly, we determined FIC index of AgNPs-CAZ combined activity, giving FIC index = 0.5 and 0.258 for *B. pseudomallei* H777 (wild type) and *B. pseudomallei* 316c (ceftazidime-resistance), respectively, indicating the synergistic effect. Combined AgNPs-CAZ induced increasing uptake of AgNPs in cell when compare with AgNPs or CAZ alone. Thirdly, we examined the anti-BP activity of the anisotropic AgNPs (asymmetrical shape). MIC of anisotropic AgNPs is lower than normal AgNPs, giving 8 µg/mL for *B. pseudomallei* H777. AgNPs and AgNPs-CAZ may be considered as a potential candidate to develop a novel alternative agent for melioidosis with fast action.

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