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Effects of silver nanoparticles on adult neural stem cell differentiation and beta-catenin signaling.

Silver nanoparticles (AgNP) are distinguished by their unique antimicrobial actions. Since AgNPs inhibit the growth of bacteria, they are incorporated into common consumer products such as clothing, electronics and medicine. In concentrations as high as 50-100µg/mL, AgNP cause cytotoxicity in neural cells; yet in low concentrations of 30µg/mL and below, cytotoxicity decreased and cell viability remained. Most people will not be exposed to toxic doses of AgNP, but rather a sub-lethal exposure which may cause subtle effects. AgNP can disrupt the blood brain barrier, cross cell membranes, and accumulate in the brain, resulting in chronic low-level exposure. My project used cultured adult neural stem cells (NCS) to test the effects of AgNP exposure at 1µg/mL, well below cytotoxic levels. NCS play a key role in learning, memory and repair and provide a cell culture model for brain cells. We previously found that low level AgNP exposure disrupted the cytoskeleton structure in cultured NSC, resulting in inhibition of neurite dynamics and the formation of f-actin puncta. This research aims to investigate β-catenin signaling pathways as a potential target of AgNP effects on NSC cytoskeleton function. We found that β-catenin and f-actin puncta co-localize after AgNP treatment. Also, the subcellular localization of β-catenin in differentiating NCS is altered after exposure to AgNP. This suggests an interaction between AgNP and β-catenin, resulting in the formation of cytoskeletal disruptions during NCS differentiation. Consequently, as β-catenin signaling contributes to healthy neural physiology and development, disruption by low-level AgNP could affect proper growth and function.