The cadmium-induced death of mesangial cells results in nephrotoxicity

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摘要.

Abstract

This study summarizes our most recent findings on the mechanisms underlying the cadmium-induced death of mesangial cells, which leads to nephrotoxicity. Multiple pathways participate in cadmium-induced nephrotoxicity. In the ROS-GSK-3beta autophagy pathway, cadmium induces ROS most likely from the mitochondria, and the ROS consequently activate GSK-3beta leading to autophagic cell death. In the calcium-ERK autophagy and apoptosis pathway, cadmium stimulates calcium release from the endoplasmic reticulum, which activates ERK leading to predominantly autophagic cell death and a minor level of apoptotic cell death. In the calcium-mitochondria-caspase apoptosis pathway, cadmium-induced elevation of calcium depolarizes the mitochondrial membrane potential and then activates caspase signaling leading to apoptosis. A proposed model for cadmium-induced autophagy and apoptosis leading to nephrotoxicity is summarized in Figure 1.