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• 計畫英文名稱	Moelcular Signaling of Caspase-Independent Apoptosis--- The Role of Mitochondria and Calpain		
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• 英文摘要	Mitochondria are believed to be integrators and coordinators of programmed cell death in addition to their respiratory function. Using mitochondrial DNA (mtDNA)-depleted osteosarcoma cells (0 cells) as a cell model, we investigated the apoptogenic signaling pathway of cadmium (Cd) under a condition of mitochondrial dysfunction. The apoptotic percentage was determined to be around 58.0% after a 24-h exposure to 25 M Cd using flow cytometry staining with propidium iodine (PI). Pretreatment with Z-VAD-fmk, a broad-spectrum caspase inhibitor, failed to prevent apoptosis following Cd exposure. Moreover, Cd was unable to activate caspase 3 using DEVD-AFC as a substrate, indicating that Cd induced a caspase-independent apoptotic pathway in 0 cells. JC-1 staining demonstrated that mitochondrial membrane depolarization was a prelude to apoptosis. On the other hand, the intracellular calcium concentration increased 12.5-fold after a 2-h exposure to Cd. More importantly, the apoptogenic activity of Cd was almost abolished by ruthenium red, a mitochondrial calcium uniporter blocker. This led us to conclude that mtDNA-depleted cells provide an alternative pathway for Cd to conduct caspase-independent apoptosis through a mitochondria-calcium mechanism.		