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• 計畫中文名稱	一氧化氮誘導骨母細胞凋零研究---探討 Caspases 所扮演之角色(I)		
• 計畫英文名稱	Study on Nitric Oxide-induced Osteoblast Apoptosis---The Role of Caspases (I)		
• 主管機關	行政院國家科學委員會	• 計畫編號	NSC91-2314-B038-031
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• 研究人員	陳瑞明 Chen, Ruei-Ming		
• 中文關鍵字	一氧化氮；骨母細胞；細胞凋亡；硫胱胺酸蛋白胨；亞硝鐵氰化鈉		
• 英文關鍵字	Nitric oxide (NO)；Osteoblast；Apoptosis；Caspase；sodium nitroprusside		
• 中文摘要	查無中文摘要		
• 英文摘要	Nitric oxide contributes to osteoblast metabolism. This project is designed to determine the role of different types of caspases in the nitric oxide-induced osteoblast apoptosis using primary osteoblasts from neonatal rat calvariae as the experimental model. The first year of this project determined the role of the mitochondria-dependent caspase activation pathway, including apoptotic factors and caspase-3, in the nitric oxide-induced osteoblast apoptosis. Exposure of osteoblasts to sodium nitroprusside, a nitric oxide donor, significantly increased lactate dehydrogenase release and decreased cell viability in concentration- and time-dependent manners. Sodium nitroprusside concentration- and time-dependently caused DNA fragmentation in osteoblasts. In parallel to sodium nitroprusside-induced osteoblast apoptosis, this nitric oxide donor increased the amounts of intracellular reactive oxygen species. However, ascorbic acid and N-acetyl cystein could not block sodium nitroprusside-induced reactive oxygen species in rat osteoblasts. Administration of sodium nitruprusside significantly reduced the membrane potential and NADH oxidase activity in osteoblast mitochondria. Immunoblotting analysis revealed that sodium nitruprusside decreased the levels of Bcl-2 protein in osteoblasts. The activities of caspase-3 were time-dependently increased following sodium nitroprusside treatment. Administration of sodium nitroprusside increased the levels of 17 kD activated subunits of caspase-3. The present study has shown that nitric oxide released from sodium nitroprusside could induce osteoblast insults and apoptosis, and this might be involved by modulating intracellular		

oxidative stress, mitochondrial functions, anti-apoptotic Bcl-2 protein and caspase-3.