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Folate deficiency triggered apoptosis of synoviocytes: Role of overproduction of reactive oxygen species generated via NADPH oxidase/mitochondrial complex II and calcium perturbation

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Despite a plethora of literature has documented that osteoarthritis (OA) is veritably associated with oxidative stress-mediated chondrocyte death and matrix degradation, yet the possible involvement of synoviocyte abnormality as causative factor of OA has not been thoroughly investigated. For this reason, we conduct the current studies to insight into how synoviocytes could respond to an episode of folate-deprived (FD) condition. First, when HIG-82 synoviocytes were cultivated under FD condition, a time-dependent growth impediment was observed and the demise of these cells was demonstrated to be apoptotic in nature mediated through FD-evoked overproduction of reactive oxygen species (ROS) and drastically released of cytosolic calcium (Ca2+) concentrations. Next, we uncovered that FD-evoked ROS overproduction could only be strongly suppressed by either mitochondrial complex II inhibitors (TTFA and carboxin) or NADPH oxidase (NOX) inhibitors (AEBSF and apocynin), but not by mitochondrial complex I inhibitor (rotenone) and mitochondrial complex III inhibitor (antimycin A). Interestingly, this selective inhibition of FD-evoked ROS by mitochondrial complex II and NOX inhibitors was found to correlate excellently with the suppression of cytosolic Ca2+ release and reduced the magnitude of the apoptotic TUNEL-positive cells. Taken together, we presentthe first evidence here that FD-triggered ROS overproduction in synoviocytes is originated from mitochondrial complex II and NOX. Both elevated ROS in tandem with cytosolic Ca2+ overload serve as final arbitrators for apoptotic lethality of synoviocytes cultivated under FD condition. Thus, folate supplementation may be beneficial to patients with OA.

Biography

Hung-Chih Hsu completed his MD degree from Taipei Medical University and PhD from Chang Gung University, Taiwan. He finished his residency at the Department of Physical Medicine and Rehabilitation (PM&R) at Chang Gung Memorial Hospital and fellowship in Washington Medical Center in Seattle, USA. In addition to PM&R, he is also a Specialty Doctor of Geriatric Medicineand Medical Diagnostic Ultrasound. He served as the Director of PM&R and Center of Developmental Pediatricsat Chia-Yi Chang Gung Memorial Hospital. He is now the Supervisory Board of Taiwan Academy of Physical Medicine and Rehabilitation, Taiwan Myopain Society, Taiwan Sports Medicine Association. He is specialized in myofascial pain treatment, electro diagnostic medicine and regeneration medicine. He has published more than 50 papers in reputed journals and three books in his specialty.

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