Yanqiu Liu, Medicinal & Aromatic Plants, Volume 7
DOI: 10.4172/2167-0412-C1-017

Canonical and non-canonical Wnt signaling is involved in the stimulation of BMSC proliferation and osteogenic differentiation by components from Er-Zhi-Wan

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Deficient in osteoblastogenesis from Bone Marrow Mesenchymal Stem Cells (BMSC) may cause a decrease in bone mass, resulting in osteoporosis. Regulation of BMSC survival and differentiation becomes essential for developing targeting drugs. Here, we evaluated the potential influence on osteoblastogenesis by combing wedelolactone with oleonuzhenide, two chemical components from Er-Zhi-Wan, a traditional Chinese formula which has been proved to be effective in treating osteoporosis. Wedelolactone at 2 μg/ml and oleonuezhenide at 10 μg/ml enhanced osteoblast differentiation and bone mineralization level. The enhanced effect was more potent when BMSC was treated with wedelolactone plus oleonuezhenide. Similarly, the expression of osteoblastogenesis-related marker genes including osteorix, osteocalcin and runx2 increased. At the molecular level, oleonuezhenide did not affect GSK-3β phosphorylaton induced by wedelolactone, but elevated Casein kinase 2-alpha (CK2α) expression, resulting in β-catenin and runx2 nuclear translocation. Addition of Disaster Medical Assistance Team (DMAT), a CK2α inhibitor, blocked oleonuezhenide-induced Alkaline Phosphatase (ALP) activity and correspondingly suppressed β-catenin nuclear accumulation induced by oleonuezhenide or combined with wedelolactone. Additionally, high dose (10 μg/ml) of wedelolactone-induced cytotoxicity in BMSC was relieved by addition of 10 μg/ml oleonuezhenide and these BMSC protected by oleonuezhenide maintained osteblastic activity. Oleonuezhenide increased Wnt 5a and CK2α expression. Wedelolactone-reduced Extracellular Signal-Regulated Kinase (ERK) phosphorylation was reversed by oleonuezhenide. However, addition of DMAT decreased ERK phosphorylation induced by oleonuezhenide. Together, these data demonstrated that oleonuezhenide enhanced wedelolactone's action on osteoblast differentiation and activity through Wnt/CK2α/β-catenin pathway and prevented wedelolactone-induced cytotoxity through Wnt5a/ CK2α/ERK pathway.

Biography

Yanqiu Liu has completed her PhD from Shenyang Pharmaceutical University and Postdoctoral studies from Dalian Institute of Chemical Physics, Chinese Academy of Sciences. She is the Associate Director of laboratory, Institute of Integrative Medicine, Dalian Medical University. She has published more than 30 papers in reputed journals and has been serving as an Editorial Board Member of repute.

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