Osteoporose. A silibinina aumenta a osteoblastogênese e protege os osteoblastos

**Osteoblastogenesis and osteoprotection enhanced by flavonolignan silibinin in osteoblasts and osteoclasts.**


**Source**

Department of Food and Nutrition, Hallym University, Chuncheon, Korea.

**Abstract**

Bone-remodeling imbalance induced by decreased osteoblastogenesis and increased bone resorption is known to cause skeletal diseases such as osteoporosis. Silibinin is the major active constituent of silymarin, the mixture of flavonolignans extracted from blessed milk thistle (Silybum marianum). Numerous studies suggest that silibinin is a powerful antioxidant and has anti-hepatotoxic properties and anti-cancer effects against carcinoma cells. This study investigated that silibinin had bone-forming and osteoprotective effects in in vitro cell systems of murine osteoblastic MC3T3-E1 cells and RAW 264.7 murine macrophages. MC3T3-E1 cells were incubated in osteogenic media in the presence of 1-20 μM silibinin up to 15 d. Silibinin accelerated cell proliferation and promoted matrix mineralization by enhancing bone nodule formation by calcium deposits. In addition, silibinin furthered the induction of osteoblastogenic biomarkers of alkaline phosphatase, collagen type 1, connective tissue growth factor and bone morphogenetic protein-2. Differentiated MC3T3-E1 cells enhanced secretion of receptor activator of NF-κB ligand (RANKL) essential for osteoclastogenesis, which was reversed by silibinin. On the other hand, RAW 264.7 cells were pre-incubated with 1-20 μM silibinin for 5 d in presence RANKL. Non-toxic silibinin markedly attenuated RANK transcription and intracellular adhesion molecule-1 expression elevated by RANKL, thereby suppressing the differentiation of macrophages to multi-nucleated osteoclasts. It was also
found that silibinin retarded tartrate-resistant acid phosphatase and cathepsin K induction and matrix metalloproteinase-9 activity elevated by RANKL through disturbing TRAF6-c-Src signaling pathways. These results demonstrate that silibinin was a potential therapeutic agent promoting bone-forming osteoblastogenesis and encumbering osteoclastic bone resorption. J. Cell. Biochem. © 2011 Wiley-Liss, Inc.

Copyright © 2011 Wiley-Liss, Inc.

PMID:

21898547