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Chemopreventive effects of a curcumin-like diarylheptanoid [2,6-bis(2,5-dimethoxybenzylidene) cyclohexanone] in cellular targets of rheumatoid arthritis *in vitro*

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Statement of the Problem: Synovial fibroblast has emerged as a potential cellular target in progressive joint destruction in rheumatoid arthritis development. Our preliminary findings had shown that the newly synthesized curcumin analogue [2,6-bis(2,5-dimethoxybenzylidene) cyclohexanone] or BDMC33 exhibited improved anti-inflammatory activity by inhibiting nitric oxide synthesis, PGE2 synthesis and cyclooxygenase (COX) expression in activated macrophage cells.

Aim: Aim of this study was to investigate the potency of BDMC33 on molecular and cellular basis of synovial fibroblasts (SF) *in vitro*.

Methodology & Theoretical Orientation: Synovial fibroblast cells (HIG-82) were cultured *in vitro* and induced by phorbol-12-myristate acetate (PMA) to stimulate the expression of matrix metalloproteinase (MMPs) and pro-inflammatory cytokines. The protective effects of BDMC33 were evaluated toward MMP activities, pro-inflammatory cytokine expression and nuclear factor kappa-B (NF-κB) activation by using various bioassay methods, including zymography, western blotting, reverse transcription polymerase chain reaction, immunofluorescence microscopy and electrophoretic mobility shift assay.

Results: The results showed that BDMC33 significantly inhibited the pro-gelatinase B (pro-MMP-9) and collagenase activities via suppression of MMP-1 in activated SF. In addition, BDMC33 strongly suppressed *MMP-3* gene expression as well as inhibited *COX-2* and *IL-6* pro-inflammatory gene expression. We also demonstrated that BDMC33 abolished the p65 NF-κB nuclear translocation and NF-κB DNA binding activity in PMA-stimulated SF.

Conclusion & Significance: BDMC33 represents an effective chemo-preventive agent and could be used as a promising lead compound for further development of rheumatoid arthritis therapeutic intervention.

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