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Gossypin Protect against Cartilage Injury via Reduction of Pro-inflammatory Cytokines and Modulation of Cartilage Related Gene in Monosodium Iodoacetate Induced Osteoarthritic Rats

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SUMMARY. **Osteoarthritis (OA), a heterogeneous condition, leads to deformity in the joints and causes defects in the articular cartilage thereby inducing changes in the bone–joint margins. In this study, we studied the anti-osteoarthritic activity of gossypin against the monosodium iodoacetate (MIA)-induced osteoarthritis and explored the possible mechanism of action of gossypin. MIA (3 mg/50 μL) was used to induce OA in the rats. Animals were divided into seven groups and were administered with different doses of gossypin (25, 50, and 100 mg/kg) and celecoxib (60 mg/kg). Body weight and joint formation were estimated at regular intervals. At end of the study, proinflammatory cytokines, inflammatory mediators, and the expression of matrix metalloproteinases (MMPs) such as MMP-2, MMP-3, MMP-9, and MMP-13; tissue inhibitors of metalloproteinases (TIMPs); and collagen type II (COL2) were measured in the serum. Administration of gossypin significantly increased the body weight and organ weight (liver, heart, spleen, and heart) and reduced the diameter of the joint ($p < 0.001$). Gossypin significantly decreased the levels of pro-inflammatory cytokines and inflammatory mediators. Italso downregulated the expression of MMP-2, MMP-3, MMP-9, and MMP-13 and upregulatedthe expression of TIMPs and COL2. Gossypin down-regulated the mRNA expression of MMP and upregulated the expression of TIMP and COL2 byreducing the proinflammatory cytokines and inflammatory mediators.**

RESUMEN. La osteoartritis (OA), una condición heterogénea, conduce a la deformidad en las articulaciones y causa defectos en el cartílago articular, induciendo así cambios en los márgenes articulares-óseos. En este trabajo estudiamos la actividad anti-osteoartrítica de la gosipina contra la osteoartritis inducida por yodoacetato monosódico (MIA) y exploramos el posible mecanismo de acción de la gosipina. Se utilizó MIA (3 mg/50 μL) para inducir OA en las ratas. Los animales se dividieron en siete grupos y se les administraron diferentes dosis de gosipina (25, 50 y 100 mg/kg) y celecoxib (60 mg/kg). El peso corporal y la formación de articulaciones se estimaron a intervalos regulares. Al final del estudio, citocinas proinflamatorias, mediadores inflamatorios y la expresión de metaloproteinasas de matriz (MMP) como MMP-2, MMP-3, MMP-9 y MMP-13; inhibidores tisulares de metaloproteinasas (TIMP); y el colágeno tipo II (COL2) se midieron en el suero. La administración de gosipina aumentó significativamente el peso corporal y el peso de los órganos (hígado, corazón, bazo y corazón) y redujo el diámetro de la articulación ($p < 0{,}001$). La gosipina disminuyó significativamente los niveles de citocinas proinflamatorias y mediadores inflamatorios. También redujo la expresión de MMP-2, MMP-3, MMP-9 y MMP-13 y aumentó la expresión de TIMP y COL2. La gosipina regula a la baja la expresión de ARNm de MMP y regula al alza la expresión de TIMP y COL2 al reducir las citocinas proinflamatorias y los mediadores inflamatorios.

KEYWORDS: gossypin, matrix metalloproteinase, osteoarthritis, pro-inflammatory cytokines, tissue inhibitors of metalloproteinases.

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