Astilbin ameliorates intestinal inflammation and suppresses colorectal cancer cell proliferation by regulating NLRP3 inflammasome and nuclear factor-kappa B signaling pathway

Lu Han[#], Xiao Zhong Deng[1#], Ya Li[2], Jinqing Hui[3]

Abstract:

OBJECTIVE: The inflammation-responsive NLRP3 inflammasome and nuclear factor-kappa B (NF-κB) dependent signaling pathways are critically connected with inflammatory conditions and disorders such as colorectal cancer (CRC). Where phytochemicals may be added as preventive natural supplements to control CRC. In this study, we investigated whether astilbin (AST) interacted with anti-inflammatory NLRP3 and NF-κB-dependent molecular events in CRC.

BACKGROUND: The network of growth signaling redox-sensitive transcription factor NF-κB interacting with the NLRP3 inflammasome in CRC progression needed targeted research. AST is a flavonol reported for anti-inflammatory, immune-suppressive, and antioxidant properties, which are sought to assess CRC growth inhibition through NF-κB and NLRP3.

METHODS: AST was applied to HCT116 and HT-29 cells of human origin to examine cell survival, apoptosis, progression, and DNA fragmentation to determine the analysis of gene and protein expression and molecular mechanisms.

RESULTS: AST inhibits the proliferation of HCT116 and HT29, and both cell lines depend on IC50 values of 128 and 144 events. AST caused induction of apoptosis in CRC cells via intrinsic mechanism involving caspase-9 and caspase-3 activation and caused arrest of the G2/M phase cell cycle. AST (100 μm) inhibited colony formation abilities to 54% and wound healing to 62% in both cell lines. In the azoxymethane/dextran sodium sulfate (AOM/DSS)-induced colitis-associated colon cancer mice, the total tumor count was reduced from 14 to 4.3 by the AST 20 mg/kg group, significantly reducing the large tumor count. AST (20 mg/kg) suppressed the colonic inflammation as shown by decreased expression of NF-κB (1.8-fold) and NLRP3 (1.5-fold) against the control (1.0-fold). AST restored colon length and histopathological changes caused by AOM/DSS. AST inhibited the production of COX-2, INOS, and pro-inflammatory cytokines and chemokines, especially interleukin-6 (IL-6), IL-1β, and IL-10, by approximately 50% at 20 mg/kg. AST suppressed intestinal tissue ASC and IL-1β NLRP3 and NF-kB by approximately 1.3 times compared to control.

CONCLUSIONS: AST inhibited colorectal carcinoma growth by blocking the expression of NLRP3 and NF-κB and inducing an apoptotic cascade and suppressing iNOS-COX2 and IL-1β as regulators of inflammation and growth signal. This study advocates the application of phytopharmaceutical supplements for the management of colorectal carcinoma.

Keywords:

Astilbin, colorectal cancer, inflammasome, NLRP3, nuclear factor-kappa B, phytopharmacology

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