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Erratum

The following was originally published in Volume 27, No. 2, pages 193–202, 2019 (doi: https://doi.org/10.3727/096504018X15150662230295). There was an error in the Matrigel Transwell assay in Figure 2A. A corrected version of the figure is shown here and the figure has been replaced with the corrected version in the original published article in the online site (https://www.ingentaconnect.com/contentone/cog/or/2019/00000027/00000002/art00007). All authors sincerely apologize for the error and all inconveniences caused.

Emodin Inhibits Colon Cancer Cell Invasion and Migration by Suppressing Epithelial–Mesenchymal Transition via the Wnt/β-Catenin Pathway

Juan Gu,* Chang-fu Cui,† Li Yang,‡ Ling Wang,* and Xue-hua Jiang*

*Department of Clinical Pharmacy, West China School of Pharmacy, Sichuan University, Sichuan, P.R. China
†Department of Neurology, Research Institute of China Weapons Industry, 521 Hospital, Shanxi, P.R. China
‡Microbiological Laboratory, Xinyang Vocational and Technical College, Henan, P.R. China

Colon cancer (CC) is the third most common cancer worldwide. Emodin is an anthraquinone-active substance that has the ability to affect tumor progression. Our study aims to explore the effects and the relevant mechanism of emodin on the invasion and migration of CC in vitro and in vivo. In our study, we found that emodin inhibited the invasion and migration abilities of RKO cells and decreased the expression of matrix metalloproteinase-7 (MMP-7), MMP-9, and vascular endothelial growth factor (VEGF) in a dose-dependent manner. Further research suggested that emodin inhibited EMT by increasing the mRNA level of E-cadherin and decreasing the expression of N-cadherin, Snail, and β-catenin. Emodin also significantly inhibited the activation of the Wnt/β-catenin signaling pathway by downregulating the expression of related downstream target genes, including TCF4, cyclin D1, and c-Myc. A Wnt/β-catenin signaling pathway agonist abolished the effect of emodin on EMT and cell mobility, suggesting that emodin exerted its regulating role through the Wnt/β-catenin pathway. The CC xenograft model was established to study the antitumor efficiency of emodin in vivo. The in vivo study further demonstrated that emodin (40 mg/kg) suppressed tumor growth by inhibiting EMT via the Wnt/β-catenin signaling pathway in vivo. Taken together, we suggest that emodin inhibits the invasion and migration of CC cells in vitro and in vivo by blocking EMT, which is related with the inhibition of the Wnt/β-catenin signaling pathway.

Key words: Emodin; Colon cancer (CC); Invasion; Migration; Epithelial–mesenchymal transition (EMT); Wnt/β-catenin

Address correspondence to Xue-hua Jiang, Department of Clinical Pharmacy, West China School of Pharmacy, Sichuan University, No. 3, Section 17, Renmin South Road, Wuhou District, Chengdu, Sichuan 646000, P.R. China. Tel: 028-85501370; E-mail: juanjuan20172018@163.com

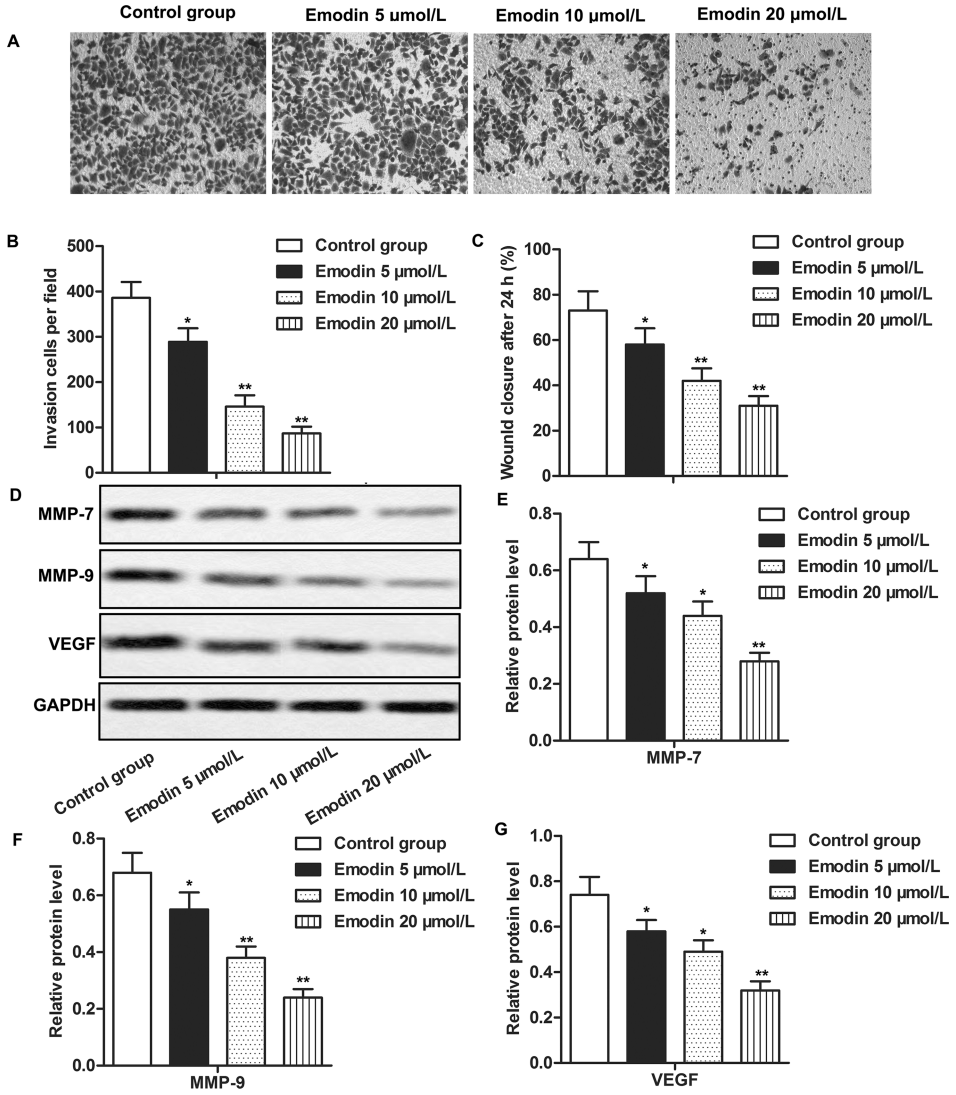

Figure 2. The inhibiting effect of emodin on invasion and migration of CC cells. RKO cells were divided into the control group and the emodin-treated groups. Cells in the control group were treated with vehicle, while cells in the emodin-treated groups were treated with different concentrations (5, 10, and 20 μmol/L) of emodin for 24 h. (A, B) The effects of emodin at different concentrations on the invasion of RKO cells were measured by Transwell invasion assay. (C) The effects of emodin at different concentrations on the migration of RKO cells as measured by wound healing assay. (D) The expression of MMP-7, MMP-9, and vascular endothelial growth factor (VEGF) was evaluated by Western blot. (E–G) Quantifications of (D). Each group was set up with triplicates, and all the experiments were repeated at least three times. GAPDH was used as an internal reference. $*p < 0.05$, $**p < 0.01$ versus the control group.