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Erratum

The following was originally published in Volume 26, Number 3, pages 411–420 (doi: https://doi.org/10.3727/096504017X14974343584989). In the original article there were some errors in the display of Figures 2C and 5C. We have revised the figures to correct these figures. Corrected versions of the figures are shown here and the figures have been replaced with the corrected versions in the original published article in the online site (https://www.ingentaconnect.com/contentone/cog/or/2018/00000026/00000003/art00008). The errors did not have an impact of the results or conclusions reported in the study. The authors apologize for any inconvenience caused.

MicroRNA-139-5p Inhibits Cell Proliferation and Invasion by Targeting RHO-Associated Coiled-Coil-Containing Protein Kinase 2 in Ovarian Cancer

Yanli Wang,* Jia Li,† Chunling Xu,† and Xiaomeng Zhang†

*Department of Gynecology, The First Hospital of Jilin University, Changchun, P.R. China
†Department of Ophthalmology, The Second Hospital of Jilin University, Changchun, P.R. China

Increasing evidence indicates that the dysregulation of microRNAs is associated with the development and progression of various cancers. MicroRNA-139-5p (miR-139-5p) has been reported to have a tumor suppressive role in many types of cancers. The role of miR-139-5p in ovarian cancer (OC) is poorly understood. The purpose of the present study was to explore the expression of miR-139-5p and its function in OC. The results showed that miR-139-5p expression was markedly downregulated in OC tissues and cell lines. In addition, underexpression of miR-139-5p was significantly associated with FIGO stage, lymph mode metastasis, and poor overall survival of OC patients. Functional analyses indicated that overexpression of miR-139-5p significantly inhibited proliferation, colony formation, migration, and invasion of OC cells. Rho-associated coiled-coil-containing protein kinase 2 (ROCK2) was identified as a direct target of miR-139-5p using luciferase reporter assays, qualitative real-time reverse transcriptase PCR (qRT-PCR), and Western blot. In addition, ROCK2 expression was upregulated and was inversely correlated with miR-139-5p levels in OC tissues. Rescue experiments showed that overexpression of ROCK2 effectively reversed the inhibitory effect of OC cells induced by miR-139-5p. Most interestingly, in vivo studies indicated that miR-139-5p markedly suppressed the growth of tumors by repressing ROCK2 expression in nude mice. Taken together, these findings demonstrated that miR-139-5p plays an important tumor suppressor role in OC by directly binding to ROCK2, providing a novel target for the molecular treatment of OC.

Key words: MicroRNAs; miR-139-5p; Ovarian cancer (OC); ROCK2; Proliferation; Invasion

Address correspondence to Xiaomeng Zhang, Department of Ophthalmology, The Second Hospital of Jilin University, 218# Ziqiang Street, Nanguan District, Changchun 130041, P.R. China. E-mail: zhangxm15106@sina.com

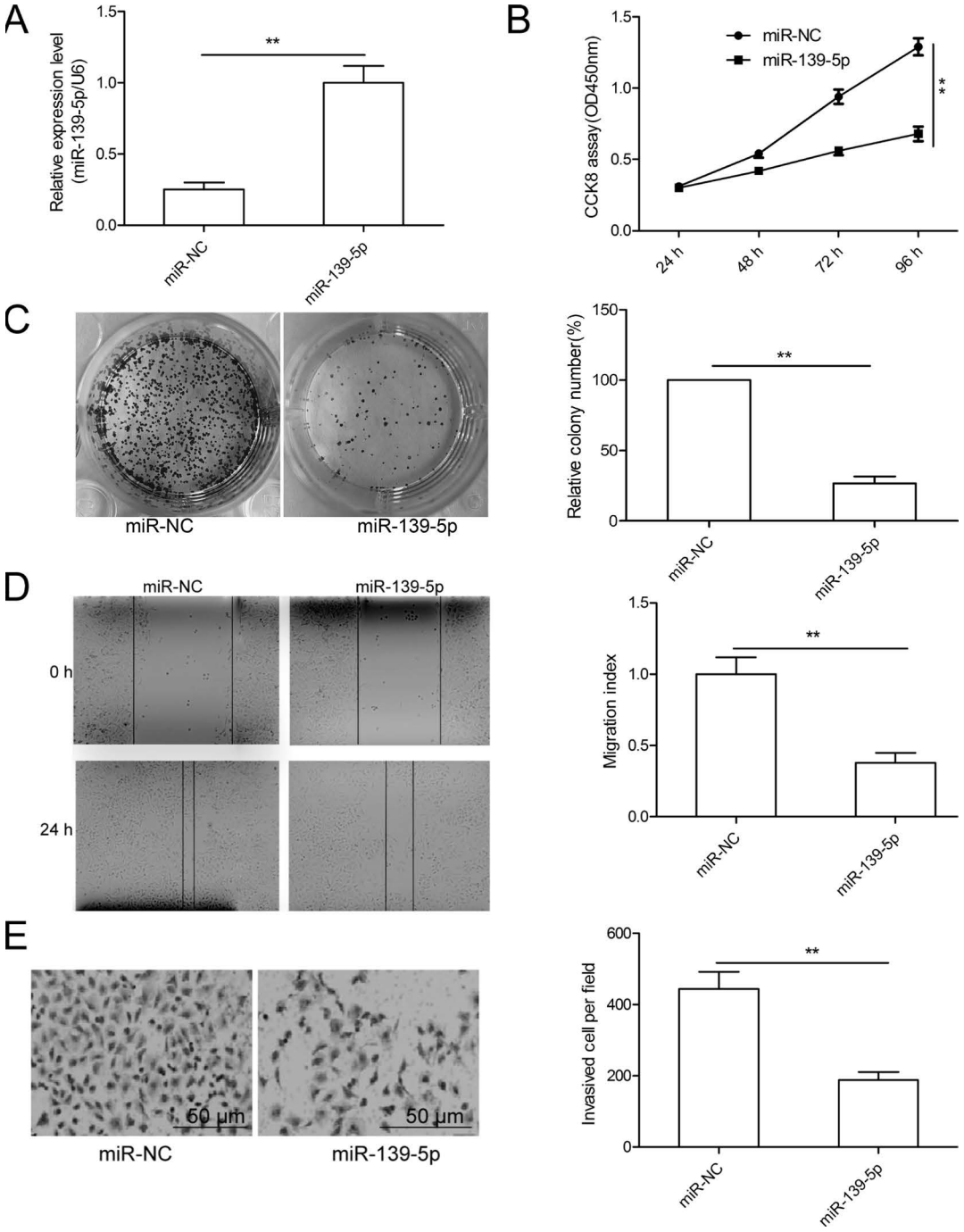

Figure 2. miR-139-5p inhibits OC cell proliferation, migration, and invasion in vitro. (A) The expression level of miR-139-5p was measured in SKOV3 cells transfected with miR-139-5p mimic or its negative control (miR-NC) by qRT-PCR. (B–E) Cell proliferation, colony formation, migration, and invasion were determined in SKOV3 cells transfected with miR-139-5p mimic or miR-NC. $^{**}p < 0.01$.

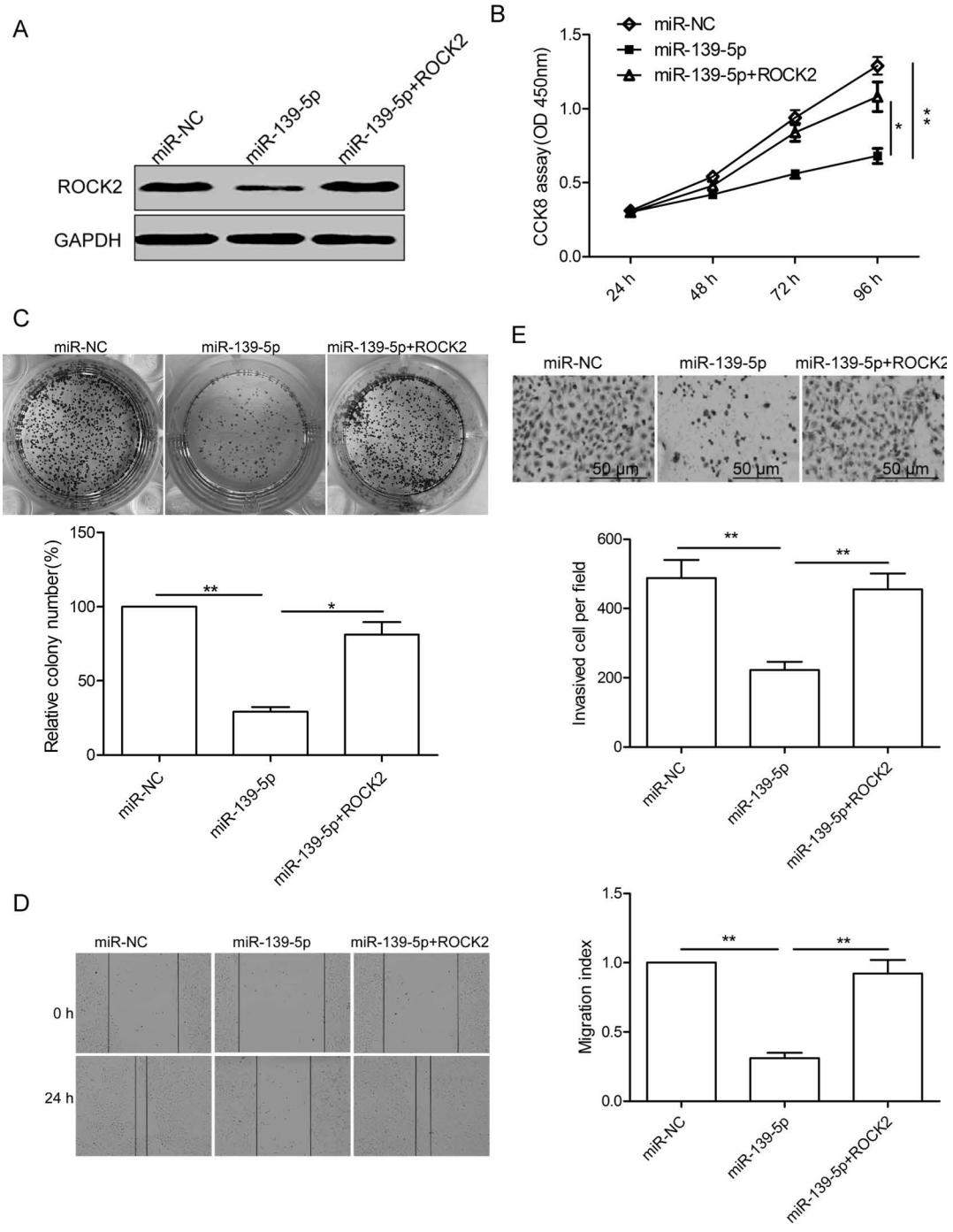

Figure 5. Overexpression of ROCK2 ablates the inhibitory effects of miR-139-5p in OC cells. (A) Western blot analysis of ROCK2 levels in SKOV3 cells after transfection with miR-139-5p mimic or miR-NC, along with (or without) ROCK2 cDNA vector lacking the 3 -UTR region. GAPDH was used as an internal control. (B–E) Cell proliferation, colony formation, migration, and invasion were determined in SKOV3 cells after transfection with miR-139-5p mimic or miR-NC, along with (or without) ROCK2 cDNA vector lacking the 3 -UTR region. *$p < 0.05$, **$p < 0.01$.