Am J Cancer Res 2016;6(4):876-877
www.ajcr.us /ISSN:2156-6976/ajcr0027320

Erratum
Upregulation of H19 indicates a poor prognosis in gallbladder carcinoma and promotes epithelial-mesenchymal transition: Am J Cancer Res. 2016; 6(1): 15-26

Shou-Hua Wang*, Xiao-Cai Wu*, Ming-Di Zhang, Ming-Zhe Weng, Di Zhou, Zhi-Wei Quan

*Department of General Surgery, Xinhua Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai 200000, China. *Equal contributors.*

Received March 4, 2016; Accepted March 7, 2016; Epub March 15, 2016; Published April 1, 2016

Abstract: The imprinted oncofetal long non-coding RNA H19 has been reported to be involved in many kinds of human cancers. However, whether lncRNA H19 implicate in oncogenesis and cancer progression in gallbladder cancer remain largely unknown. In the present study, compared with adjacent normal tissues, the level of H19 was significantly upregulated in gallbladder cancer tissues and was positively associated with lymphatic metastasis and tumor size. The overall survival is shorter in those who had higher H19 expression among GBC patients. *In vitro*, both TGF-β1 and IL-6 treatment induced upregulation of H19, downregulated the protein level of E-cadherin while increased Vimentin, indicating an epithelial-mesenchymal transition (EMT) phenotype in GBC. The overexpression of H19 in GBC cells enhanced tumor invasion and promoted EMT by upregulated transcription factor Twist1. On the contrary, Loss of function studies indicated that H19 interference in GBC suppressed tumor cell invasion and promoted mesenchymal-epithelial transition (MET) via suppressing Twist expression. *In vivo*, the volume of the tumors in H19-inteference group was significantly decreased compared to those in the control group of nude mice. Both western-blot and immunohistochemistry confirmed that a MET phenotype existed in the H19 interference group when compared to control group. These results defined H19 as a novel prognostic factor for GBC, and indicated that it might play important regulatory roles in the EMT process.

Keywords: Gallbladder cancer, H19, epithelial-mesenchymal transition, tumor invasion

In this article, a minor concern regarding **Figure 5** have been noticed by Shouhua Wang *et al*, which is no impact on the final finding and conclusion. In **Figure 5A**, NOZ cells was used for loss of function analyses for its high expression of H19 and in **Figure 5B**, GBC-SD cells was used for gain of function assays for its relative low expression of H19. They made an error for posted the exactly same photos in Vimentin expression, both in **Figure 5A** and **5B**. In addition, the pictures of DAPI in twist1 expression of **Figure 5B** were reversed. Therefore, in order to clarify the EMT phenotype of H19 in GBC, a modified composite figure has now been created. The amended figure is shown below. The authors express regrets for their error.

Address correspondence to: Dr. Zhi-Wei Quan, Department of General Surgery, Xinhua Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai 200000, China. Tel: 86-20-25078999; Fax: 86-20-25078999; E-mail: Quanzhiwei3@163.com

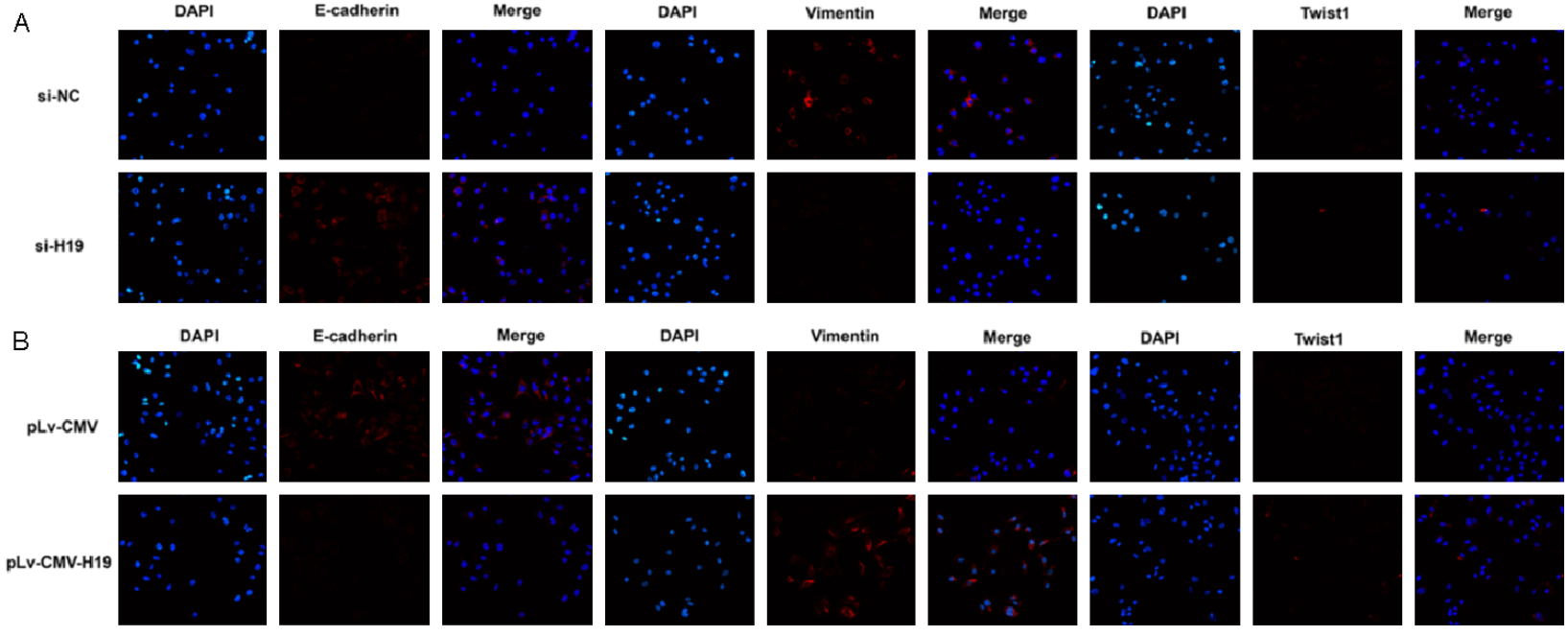

Figure 5. The expression of Twist1 and E-cadherin and Vimentin were detected by immunofluorescence (A) The expression of EMT key transcription factors Twist1 and marker genes E-cadherin and Vimentin were evaluated after silencing H19 expression in Noz cells (B) The expression of EMT key transcription factors Twist1 and marker genes E-cadherin and Vimentin were evaluated after overexpression of H19 in GBC-SD cells.