CORRIGENDUM

DOI: 10.3892/ijo.2022.5301

Receptor for activated C kinase 1 (RACK1) promotes the progression of OSCC via the AKT/mTOR pathway

XUEFENG ZHANG, NA LIU, DANHUA MA, LING LIU, LU JIANG, YU ZHOU, XIN ZENG, JING LI and QIANMING CHEN

Int J Oncol 49: 539-548, 2016; DOI: 10.3892/ijo.2016.3562

Subsequently to the publication of the above article, an interested reader drew to the authors' attention that Fig. 3 (showing how RACK1 silencing alters the protein expression levels of tumor malignant progress markers in OSCC *in vivo*) contained an overlapping data panel, such that the data were derived from the same original source where they were intending to depict the results from experiments performed under different experimental conditions and Fig. 4 (showing how RACK1 expression is positively correlated with p-AKT in OSCC tissues and cells) contained a clearly duplicated pair of data panels.

The authors were able to re-examine their original data, and have identified the data that were intended to have been shown for these figure parts. The corrected versions of Figs. 3 and 4 are shown on the next two pages, featuring the correct data for the E-cadherin experiments in Fig. 3B and the correct enlargement panel for the RACK1 / Moderate dysplasia experiment in Fig. 4A. The authors confirm that these inadvertent errors did not have any major impact on the conclusions reported in their paper, are grateful to the Editor of *International Journal of Oncology* for allowing them this opportunity to publish a Corrigendum, and apologize to the readership for any inconvenience caused.

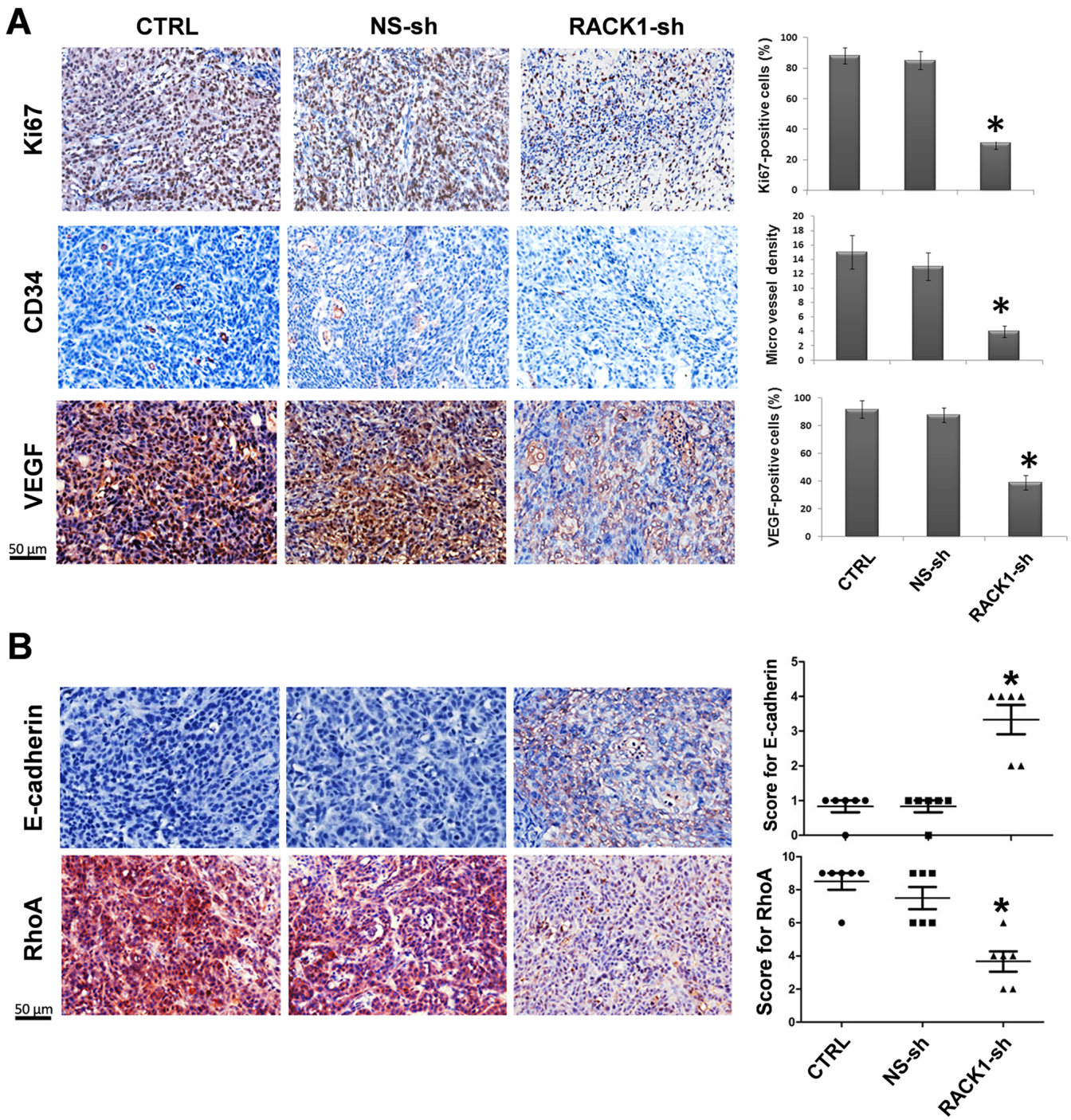

Figure 3. RACK1 silencing changes protein expression levels of tumor malignant progress markers in OSCC *in vivo*. (A) Percentages of Ki67-positive nuclei in RACK1-sh group were significantly lower than those in the two control groups (mean ± SD, *P<0.05). Angiogenesis in tumors was detected by CD34 and induced VEGF. The average number of microvessels and VEGF expression were significantly lower in RACK1-sh group tissues compared with those in the two control groups (mean ± SD, *P<0.05). (B) RACK1 silence could decrease the expression of RhoA and increase the expression of E-cadherin of OSCC *in vivo* (mean ± SD, *P<0.05)..

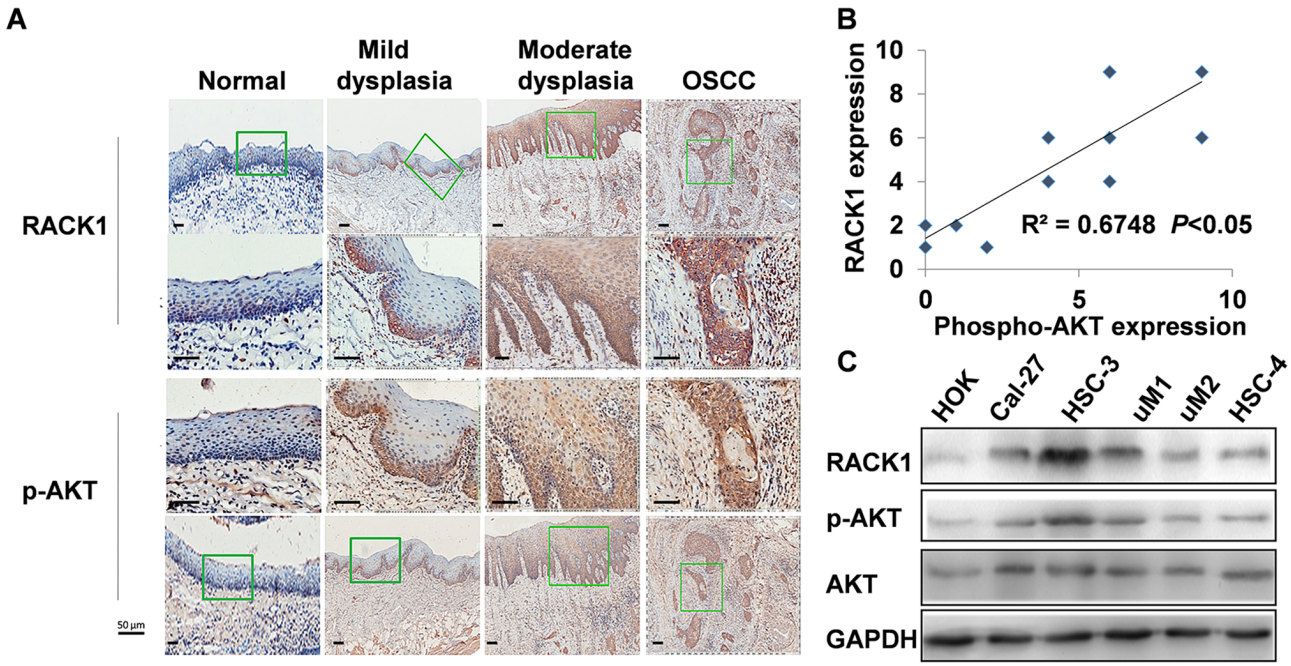

Figure 4. RACK1 expression was positively correlated with p-AKT in OSCC tissues and cells. (A) Protein level of RACK1 and p-AKT have a similar tendency in different stages of oral carcinogenesis tissue. (B) RACK1 expression was positively correlated with p-AKT in OSCC tissues. (C) RACK1 expression was positively correlated with p-AKT in OSCC cells.