Cellular Physiology	Cell Physiol Biochem 2020;54:515-516	
and Biochemistry	DOI: 10.33594/000000236	© 2020 The Author(s) Published by Cell Physiol Biochem Press GmbH&Co. KG, Duesseldorf

Erratum

In the original article by Wang, et al., entitled "Peperomin E Induces Promoter Hypomethylation of Metastatic-Suppressor Genes and Attenuates Metastasis in Poorly Differentiated Gastric Cancer" [Cell Physiol Biochem 2018;50(6):2341–2364, DOI: 10.1159/000495096], there have been misplaced subfigures in Fig. 8 and 9.

The western band of Sp1 (HGC-27) was wrongly used in Fig. 8B. One of the wound healing assay pictures was wrongly used in Fig. 9B. The correct Fig. 8 and Fig. 9 and their corresponding figure legends are displayed below.

The authors confirm that all of the results and conclusions of the article remain unchanged, as well as the figure legends.

The authors sincerely apologize for this mistake.

Fig. 8. PepE inhibits AMPKα-Sp1-DNMT signaling in GC cells. (A) Sp1 siRNA concurrently decreased Sp1 and DNMTs (including DNMT1, 3a and 3b) expression in NCI-N87 cells. NCI-N87 cells were transfected with negative control SiRNA (vector) or Sp1 siRNA (1 or 2) and cultured for 2 weeks. Total cell lysates were subjected to Western blot for Sp1, DNMT1, 3a and 3b. (B) PepE inhibits Sp1 protein expression in GC cells. Reduced Sp1 expression in NCI-N87, MGC-803 and HGC-27 cells treated with indicated dosage of



PepE for 24 h. Bortezomib was used as positive control (C:control, B:Bortezomib, 100 nM). Data are presented as mean \pm SD (n=3), *P<0.05; **P<0.01. (C) PepE abolished Sp1 binding to the DNMT1, 3a and 3b promoter. EMSA was performed with nuclear extracts prepared from NCI-N87 cells untreated or treated with PepE or bortezomib. The DNMT1/Sp1, DNMT3a/Sp1 and DNMT3b/Sp1 probes are shown on the top of each panel. C indicates control; P: PepE (2 μ M), and B: bortezomib (100 nM). (D) PepE induced phosphorylation of AMPK α in NCI-N87 cells at indicated doses for 24 h. Data are presented as mean \pm SD (n=3). *P<0.05; **P<0.01 when compared with control. (E) The inhibition ability of PepE against Sp1 expression was abrogated by dorsomophin (an AMPK inhibitor). NCI-N87 cells were treated with dorsomophin (10 μ M) for 2 h before exposure of the cells to PepE (4 μ M) for an additional up to 24 h. Afterward, the expression of p-AMPK α and Sp1 were detected by Western blot. Data are presented as mean \pm SD (n=3). *P<0.01 when compared with compared with NCI-N87 cells only treated with dorsomophin.



Fig. 9. Effects of DNMT1 inhibition on suppressing the metastasis of GC cells. (A) DNMT1 siRNA decreased DNMT1 expression in NCI-N87 cells dose dependently. (B) Wound healing assay of NCI-N87 before and after DNMT1 transcription decreased by SiRNA and inhibited by 5-Aza-dC. Histograms showed the ratio of migrated cells to the denuded zone of



PepE treated cells when compared with control. (C) Migration and invasion assays of NCI-N87 before and after DNMT1 transcription decreased by SiRNA and inhibited by 5-Aza-dC. Histograms showed the ratio of migration cells and invasion cells of SiRNA and 5-Aza-dC group when compared with control. Cell numbers were counted in five randomly selected microscopic fields. All the above data is shown as mean ± SD of three independent experiments. *P<0.05; **P<0.01.