Journal of Experimental & Clinical Cancer Research

CORRECTION

Open Access

Correction: Melatonin synergizes BRAF-targeting agent vemurafenib in melanoma treatment by inhibiting iNOS/hTERT signaling and cancer-stem cell traits

Jiaojiao Hao[1†], Wenhua Fan[2†], Yizhuo Li[2†], Ranran Tang[3†], Chunfang Tian[1], Qian Yang[1], Tianhua Zhu[1], Chaoliang Diao[1], Sheng Hu[1], Manyu Chen[1], Ping Guo[1], Qian Long[2], Changlin Zhang[2], Ge Qin[2], Wendan Yu[1], Miao Chen[2], Liren Li[2], Lijun Qin[4], Jingshu Wang[4], Xiuping Zhang[5], Yandong Ren[5], Penghui Zhou[2], Lijuan Zou[1], Kui Jiang[1*], Wei Guo[1*] and Wuguo Deng[2*]

Correction: J Exp Clin Cancer Res 38, 48 (2019)
https://doi.org/10.1186/s13046-019-1036-z

Following publication of the original article [1], authors identified errors in Figure 3c and Figure 5e. The merged image of VE group in sk-mel-28 cells (Fig. 3C) and the image of DMSO + NC group in A375 cells (Fig. 5E) has been mistakenly uploaded, respectively.

[†]Jiaojiao Hao, Wenhua Fan, Yizhuo Li and Ranran Tang contributed equally to this work.

The original article can be found online at https://doi.org/10.1186/s13046-019-1036-z.

*Correspondence:
Kui Jiang
kuijiang501@sina.com
Wei Guo
wei1015@dmu.edu.cn
Wuguo Deng
dengwg@sysucc.org.cn
[1] Institute of Cancer Stem Cells and The Second Affiliated Hospital, Dalian Medical University, Dalian, China
[2] State Key Laboratory of Oncology in South China; Collaborative Innovation Center of Cancer Medicine, Sun Yat-Sen University Cancer Centre, Guangzhou, China
[3] Nanjing Maternity and Child Health Care Hospital, Women's Hospital of Nanjing Medical University, Nanjing, China
[4] Sun Yat-Sen Memorial Hospital of Sun Yat-Sen University, Guangzhou, China
[5] Cloud Health Genomics Ltd, Shanghai, China

Incorrect Figure 3

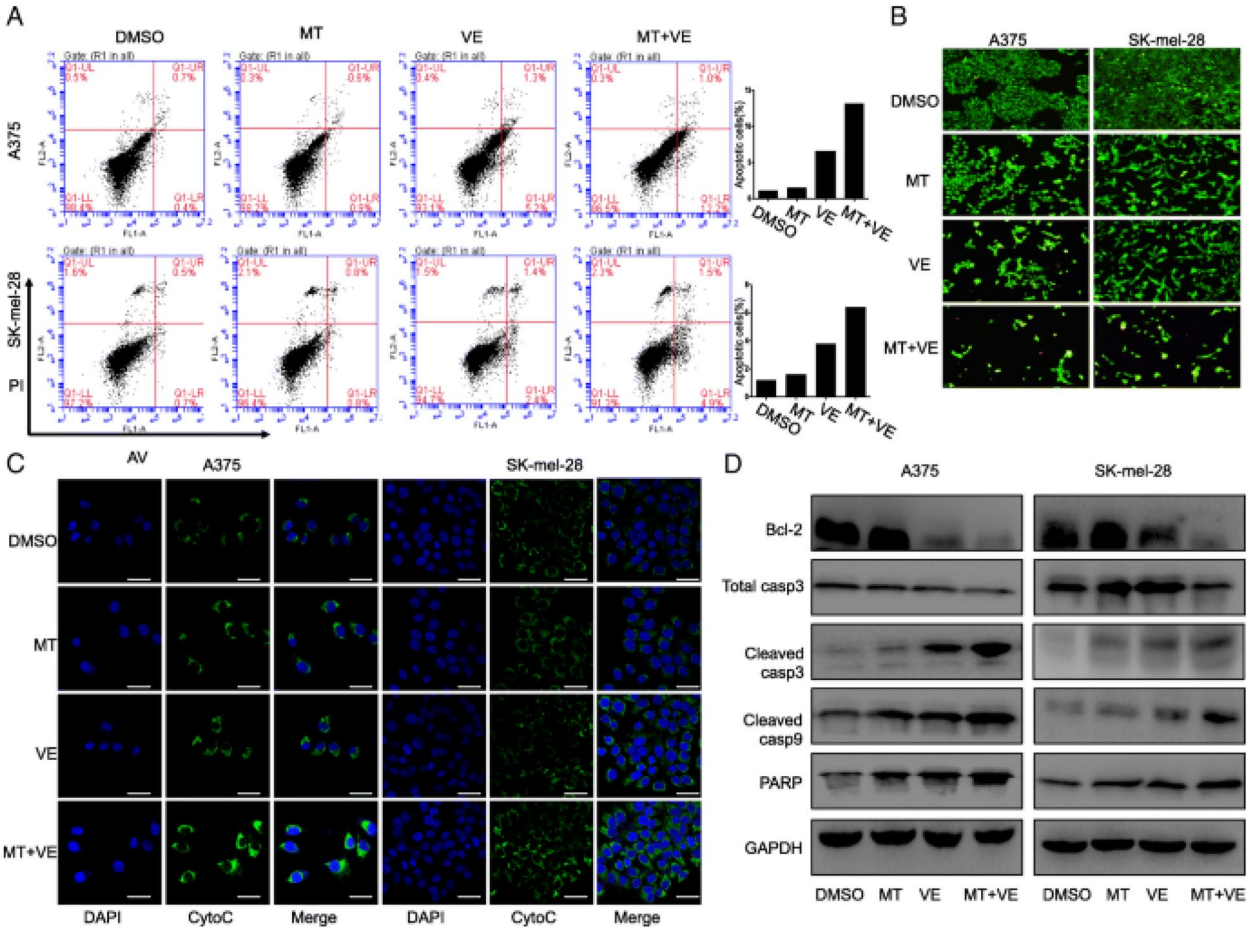

Fig. 3 Melatonin increased apoptosis induced by vemurafenib via the cytochrome c/caspase signaling pathway. Human melanoma cells were treated with vemurafenib (VE) (2.5 μM) and melatonin (MT) (1.0 mM) for 24 h. (**a**). The apoptosis was then determined by a FACS analysis. (**b**). Acridine orange/ethidium bromide fluorescence staining was performed in melanoma cells. (**c**). The release of cytochrome c (cyto-c) was monitored by immunofluorescence imaging analysis from the inter-mitochondrial space into the cytosol. (**d**). The levels of the Bcl-2, cleaved, caspase-3, 9 and PARP proteins were analyzed by Western blotting. The apoptosis are represented by relative percentages of apoptotic cells versus that in DMSO-treated cells

Correct Figure 3

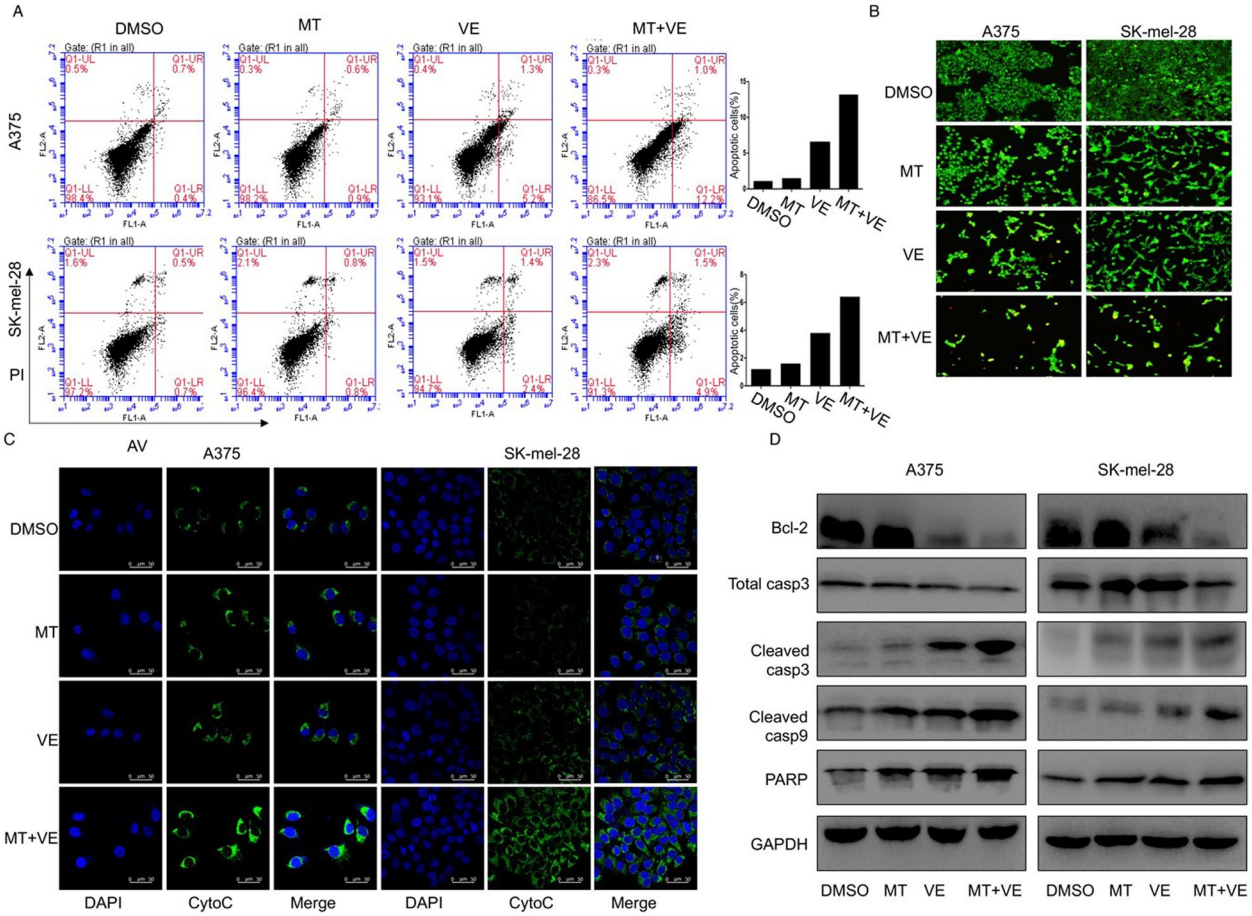

Fig. 3 Melatonin increased apoptosis induced by vemurafenib via the cytochrome c/caspase signaling pathway. Human melanoma cells were treated with vemurafenib (VE) (2.5 μM) and melatonin (MT) (1.0 mM) for 24 h. (**a**). The apoptosis was then determined by a FACS analysis. (**b**). Acridine orange/ethidium bromide fluorescence staining was performed in melanoma cells. (**c**). The release of cytochrome c (cyto-c) was monitored by immunofluorescence imaging analysis from the inter-mitochondrial space into the cytosol. (**d**). The levels of the Bcl-2, cleaved, caspase-3, 9 and PARP proteins were analyzed by Western blotting. The apoptosis are represented by relative percentages of apoptotic cells versus that in DMSO-treated cells

Incorrect Figure 5

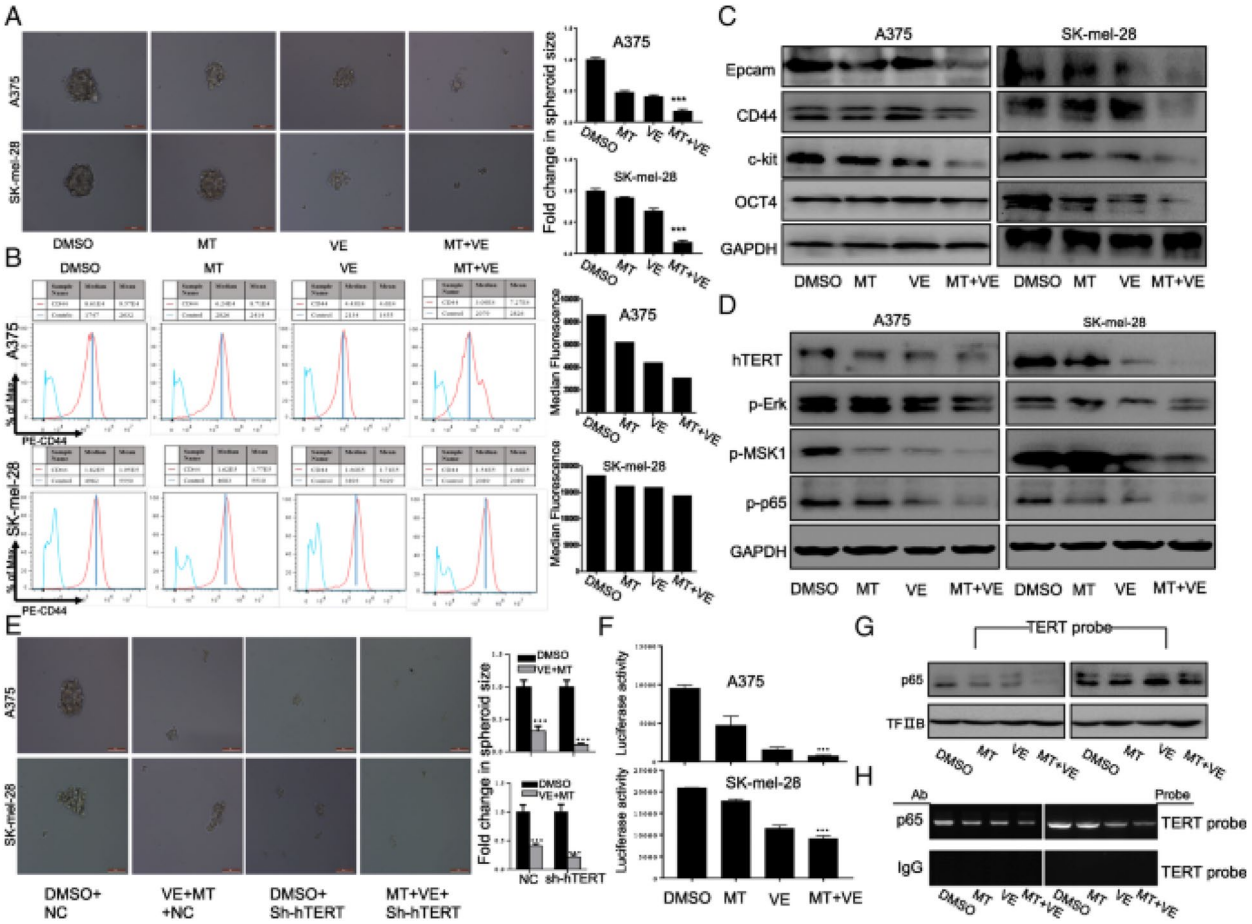

Fig. 5 Combination of vemurafenib and melatonin inhibited cancer stem cell traits by down-regulating hTERT in melanoma cells. Human melanoma cells were exposed to vemurafenib (VE) (2.5 μM) with or without melatonin (MT) (1.0 mM) for 48 h. (**a**). The representative images of tumor sphere formation of melanoma cells with indicated treatment. (**b**). CD44 expression on the surface of melanoma cells was analyzed by FACS. (**c**). The expression of CSC-related markers Epcam, CD44, c-kit and Oct4 were determined by western blot in A375 and SK-mel-28 cells with the indicated treatment. (**d**). The expression of hTERT-p-MSK1-p65 pathway were determined by western blot in A375 and SK-mel-28 cells with the indicated treatment. (**e**). The representative images of tumor sphere formation of melanoma cells treated with DMSO or vemurafenib (2.5 μM) combined with MT (1.0 mM) for 24 h after pretreatment with the hTERT targeting shRNA. (**f**). Melanoma cells were co-treated with the plasmids of hTERT promoter driven-luciferase and vemurafenib (VE) (2.5 μM) with or without melatonin (MT) for 48 h followed by a dual-luciferase assay. The relative luciferase intensity per mg protein was calculated in the treated cells. (**g**). The streptavidin–biotin pulldown assay was performed to analyze the binding of P65 protein to hTERT promoter in melanoma cells with the indicated treatment. (**h**). Binding of p65 to the hTERT promoter in chromatin structure by ChIP assay. IgG, a negative control for ChIP in melanoma cells with the indicated treatment. The data are presented as the mean ± SD of three separate experiments. * $P < 0.05$, significant differences between treatment groups and DMSO control groups

Correct Figure 5

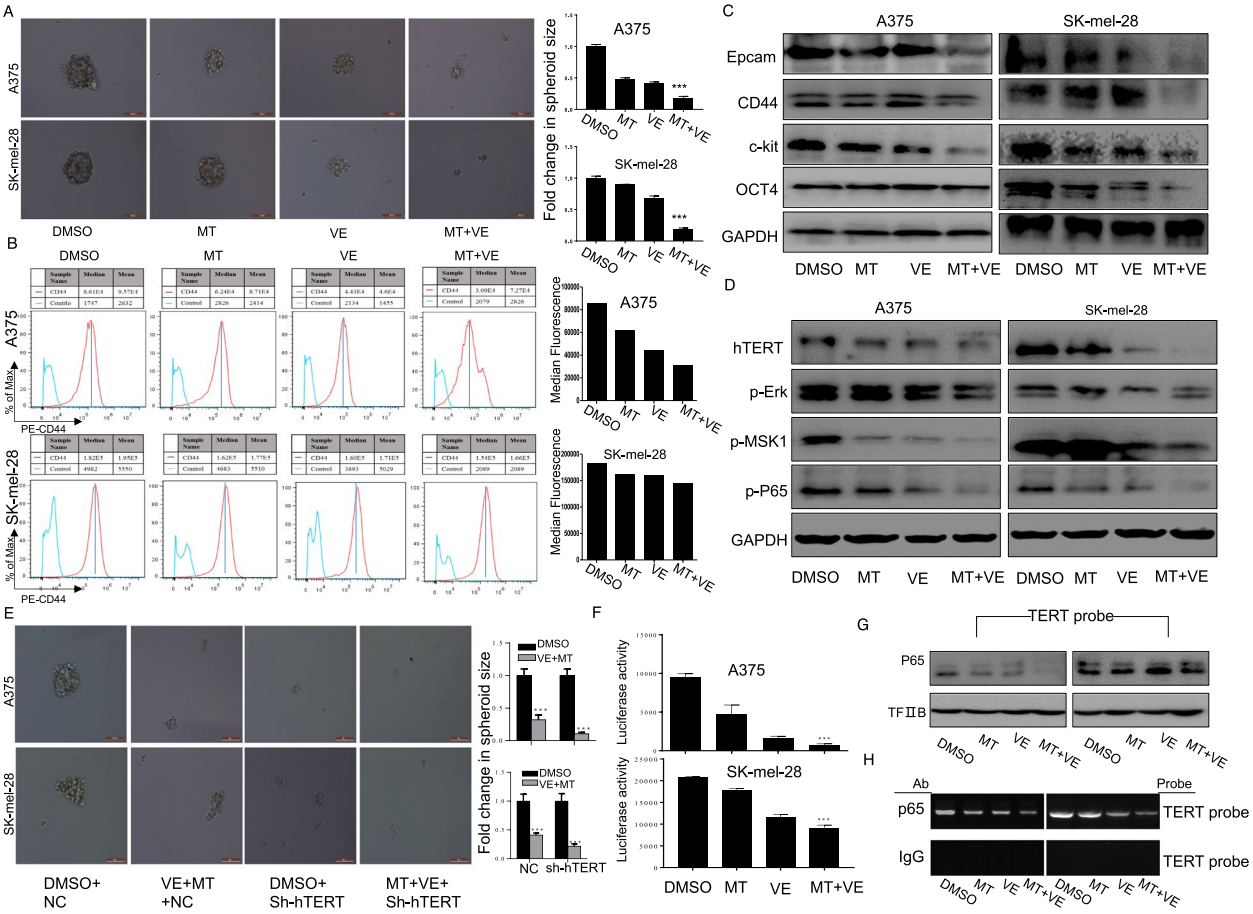

Fig. 5 Combination of vemurafenib and melatonin inhibited cancer stem cell traits by down-regulating hTERT in melanoma cells. Human melanoma cells were exposed to vemurafenib (VE) (2.5 μM) with or without melatonin (MT) (1.0 mM) for 48 h. (**a**). The representative images of tumor sphere formation of melanoma cells with indicated treatment. (**b**). CD44 expression on the surface of melanoma cells was analyzed by FACS. (**c**). The expression of CSC-related markers Epcam, CD44, c-kit and Oct4 were determined by western blot in A375 and SK-mel-28 cells with the indicated treatment. (**d**). The expression of hTERT-p-MSK1-p65 pathway were determined by western blot in A375 and SK-mel-28 cells with the indicated treatment. (**e**). The representative images of tumor sphere formation of melanoma cells treated with DMSO or vemurafenib (2.5 μM) combined with MT (1.0 mM) for 24 h after pretreatment with the hTERT targeting shRNA. (**f**). Melanoma cells were co-treated with the plasmids of hTERT promoter driven-luciferase and vemurafenib (VE) (2.5 μM) with or without melatonin (MT) for 48 h followed by a dual-luciferase assay. The relative luciferase intensity per mg protein was calculated in the treated cells. (**g**). The streptavidin–biotin pulldown assay was performed to analyze the binding of P65 protein to hTERT promoter in melanoma cells with the indicated treatment. (**h**). Binding of p65 to the hTERT promoter in chromatin structure by ChIP assay. IgG, a negative control for ChIP in melanoma cells with the indicated treatment. The data are presented as the mean ± SD of three separate experiments. * $P < 0.05$, significant differences between treatment groups and DMSO control groups

Published online: 26 June 2024

Reference

1. Hao J, Fan W, Li Y, et al. Melatonin synergizes BRAF-targeting agent vemurafenib in melanoma treatment by inhibiting iNOS/hTERT signaling and cancer-stem cell traits. J Exp Clin Cancer Res. 2019;38:48. https://doi.org/10.1186/s13046-019-1036-z.