Cellular Physiology	Cell Physiol Biochem 2020;54:794-796	
and Biochemistry	DOI: 10.33594/000000258	© 2020 The Author(s) Published by Cell Physiol Biochem Press GmbH&Co. KG, Duesseldorf www.cellphysiolbiochem.com

In the original article by Li, et al., entitled "Synergistic Antitumor Effect of BKM120 with Prima-1<sup>Met</sup> Via Inhibiting PI3K/AKT/mTOR and CPSF4/hTERT Signaling and Reactivating Mutant P53" [Cell Physiol Biochem 2018;45(5):1772-1786, DOI: 10.1159/000487786], during data transmission the results of 29T cells were confused and incorrect images were used for Figures 1d, 2b, 3b and 4a. The correct Fig. 1 to Fig. 4 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. 1.** Effect of the combination treatment of NVP-BKM120 and Prima-1Met on cell proliferation in thyroid cancer cells. (a) Chemical structure of NVP-BKM120 and Prima-1Met. (b) Thyroid cancer cells (THJ-16T, THJ-21T, THJ-29T, FTC-133, BCPAP) and normal thyroid cells (Nthy-ori-3-1) were treated with the indicated doses of Prima-1Met alone or in combination with BKM120 (1  $\mu$ M) for 48 hours, and cell viability was determined by MTT assay. (c) The IC50 values of Prima-1Met were determined for cell viability inhibition in cells treated with Prima-1Met alone or in combination with BKM120 (1  $\mu$ M). The data were presented as mean ± SD of three independent experiments. \*P<0.05 and \*\*P<0.01, significant differences compared to the control groups. (d) Colony formation was shown for two thyroid cancer cell lines (THJ-29T, FTC-133) treated with BKM120 (1  $\mu$ M), Prima-1Met (30  $\mu$ M) or the two in combination for 48 hours. (e) THJ-29T and FTC-133 cells were treated with BKM120 (1  $\mu$ M), Prima-1Met (30  $\mu$ M) or the two in combination for 48 hours. (e) THJ-29T and FTC-133 cells were treated with BKM120 (1  $\mu$ M), Prima-1Met (30  $\mu$ M) or the two in combination for 48 hours. The protein levels of p110α, p110β, total and phosphorylated Akt, p-PDK1, p-c-Raf, p-PTEN, and mutant p53 were analyzed by Western blot. β-actin served as the loading control. All the experiments were repeated 3 times, and the data were shown as mean ± SD. P-values were calculated by student's t-test.



**Fig. 2.** Effect of the combination treatment of NVP-BKM120 and Prima-1Met on migration and invasion of thyroid cancer cells. (a) Cell migration was analyzed by wound healing assay. THJ-29T and FTC-133 cells were grown to 70-80% confluency. The cell monolayers were wounded with a sterile pipette tip, and washed with medium to remove detached cells from the plates. Then the cells were left either untreated or treated with BKM120 (1  $\mu$ M), Prima-1Met (30  $\mu$ M), or the two in combination. After 48 hours, the wound gap was observed and photographed. \*P<0.05, significant difference between the BKM120 + Prima-1Met treated group and the BKM120 or Prima-1Met treated groups. (b) FTC-133 and THJ-29T cancer cells were subjected to Matrigel invasion assay and photographed (magnification 20×, scale bar 100  $\mu$ m). (c) Left: Migration capability of cells in (a) was calculated; Right: invasion capability of cells in (b) was calculation. The data were presented as mean ± SD of three independent experiments. \*\*\*P<0.005, significant difference between the treatment and control groups. (d) Protein levels of Vimentin,  $\beta$ -catein, E-Cadherin and N-Cadherin were detected after treatment with BKM120 (1  $\mu$ M), Prima-1Met (30  $\mu$ M) or the two in combination. \*P<0.05, significant difference between treatment group and DMSO control group. \*\*P<0.05, significant difference between combination treatment group and single-agent treatment group. All the experiments were repeated 3 times, and the data were shown as mean ± SD.





**Fig. 3.** Effect of the combination treatment of NVP-BKM120 and Prima-1Met on apoptosis of thyroid cancer cells. (a) THJ-29T and FTC-133 cells were treated with BKM120 (1  $\mu$ M) and Prima-1Met (30  $\mu$ M). 48 hours after treatment, apoptosis was determined by FACS analysis and presented as percentages of apoptotic cells. (b) Acridine orange/ethidium bromide fluorescence staining was performed in THJ-29T and FTC-133 cells. (c) The levels of cleaved PARP, Bax, Bcl-2 and p21 proteins were analyzed by Western blot. \*P<0.05, significant difference between the BKM120/Prima-1Met-treated group and the BKM120 or Prima-1Met-treated group. (d) The release of cytochrome-c from the inter-mitochondrial space into the cytosol was analyzed by immunofluorescence analysis.



**Fig. 4.** Effect of the combination treatment of NVP-BKM120 and Prima-1Met on stem-like traits of thyroid cancer cells and the expression of thyroid-specific differentiation markers. (a) THJ-29T and THJ-21T cells were grown in 3D, treated with drugs as indicated for 14 days, and quantitated for structural integrity. (b) Sphere formation efficiency of the cells in (a) was calculated. (c) Protein level of CD44, CD133, OCT4, ABCG2 and Nanog were determined in THJ-29T and THJ-21T cells treated with BKM120, Prima-1Met or the two in combination. (d) mRNA level of NIS and Tg were determined in THJ-29T and THJ-21T cells treated with BKM120, Prima-1Met or the two in combination. (e) The expression level of NIS in THJ-29T and THJ-21T cells treated with BKM120, Prima-1Met or the two in combination were analyzed by Western blot.