CORRIGENDUM

DOI: 10.3892/ijo.2020.5113

Increased infiltration of macrophages to radioresistant lung cancer cells contributes to the development of the additional resistance of tumor cells to the cytotoxic effects of NK cells

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Int J Oncol 53: 317-328, 2018; DOI: 10.3892/ijo.2018.4394

Subsequently to the publication of the above paper, the authors have realized that the western blots featured in Fig. 5B were inadvertently copied across from Fig. 4B owing to an error made during the figure compilation process. The corrected version of Fig. 5 is featured on the next page, showing the correct data for the western blot analysis of the programmed death receptor ligand 1 level in radioresistant lung cancer cells under the specified experimental conditions.

Note that these changes do not affect the interpretation of the data or the conclusions reported in this paper, and all the authors agree to this correction. The authors apologize to the Editor and to the readership of the Journal for any inconvenience caused.

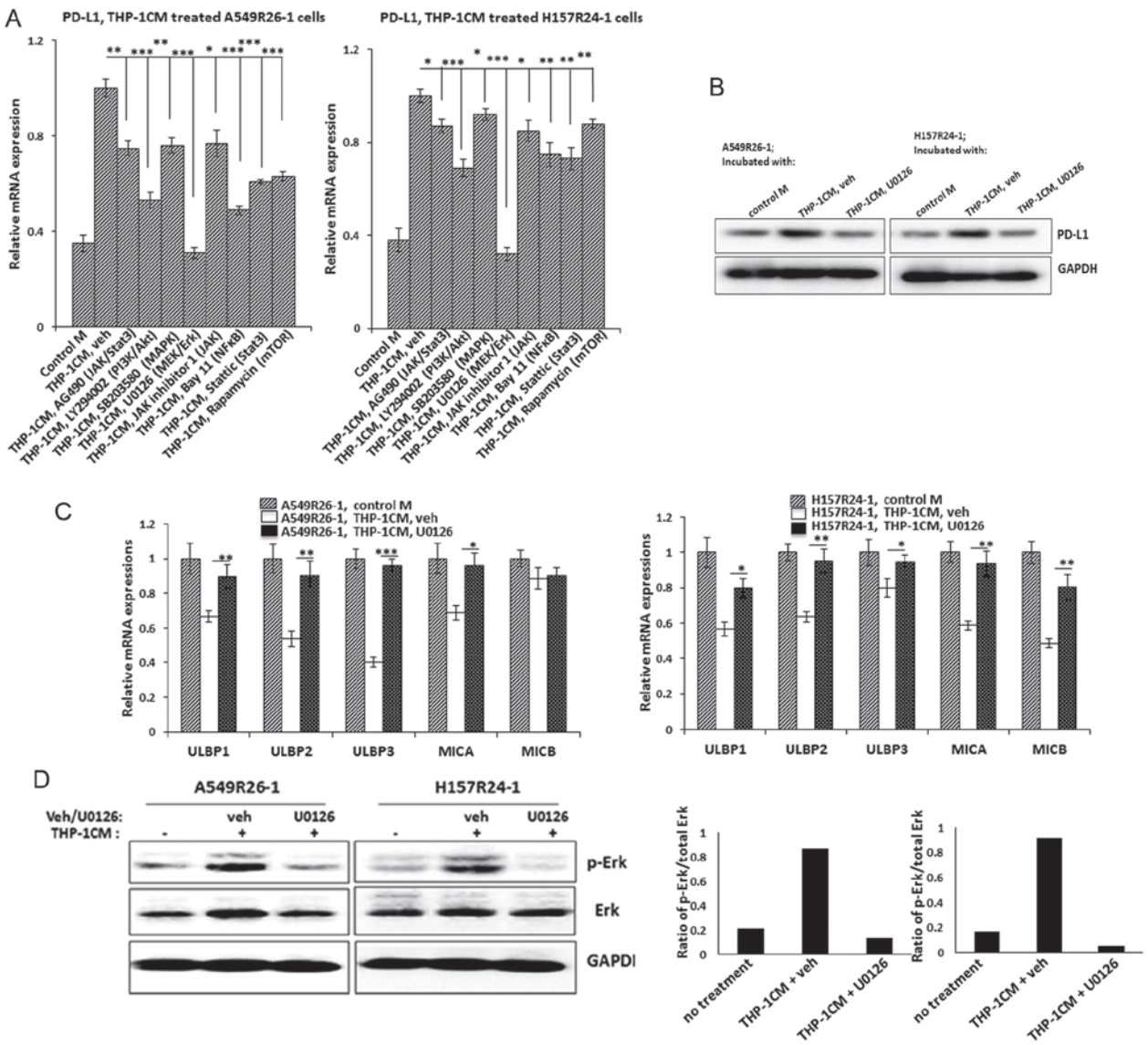

Figure 5. Searching for the IL-6 downstream signaling that is responsible for triggering the THP-1 CM effect. (A) RT-qPCR analyses of PD-L1 in radioresistant lung cancer cells treated with CM of PMA/IL-4-treated THP-1 cells after the addition of inhibitors of individual candidate signaling pathways. (B) Western blot analysis of the PD-L1 level in radioresistant lung cancer cells treated with conditioned medium (CM) of PMA/IL-4-treated THP-1 cells, with the addition of the MEK/Erk inhibitor, U0126. (C) RT-qPCR analysis of NKG2D ligand levels in radioresistant lung cancer cells treated with CM of PMA/IL-4-treated THP-1 cells after the addition of the MEK/Erk inhibitor, U0126. Left panel shows data of A549R26-1 cells and right panel shows data of H157R24-1 cells. (D) Western blot analyses of p-Erk and total Erk levels in radioresistant lung cancer cells treated with CM of PMA/IL-4-treated THP-1 cells, with the addition of U0126. Quantification of p-Erk to total Erk and GAPDH is shown in the lower panels. *P<0.05, **P<0.01 and ***P<0.001.