

Corrigendum

The ratio of Mcl-1 and Noxa determines ABT737 resistance in squamous cell carcinoma of the skin

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Since the publication of this paper, the authors have been noted that there was an error in the description in the legend of Figure 6. The corrected legend of Figure 6 is shown below.

The authors have also noticed an error in the labelling of Figure 7b and Figure 7d. Figure 7b should be Figure 7d and Figure 7d should be Figure 7b. The corrected Figure 7 is shown in next page. The authors would like to apologize for any inconvenience caused.

Figure 6 The ratio of Mcl-1 and Noxa determines ABT737 resistance in SCC cells. Transient knockdown of Mcl-1 in MET1 and MET4 cells (a–c) or knockdown of Noxa in A5RT3 and HaCaT cells (d–f) was performed with different Mcl-1-specific (a–c) or Noxa-specific siRNA (d–f) and control siRNA as described in Materials and Methods. The successful knockdown of Mcl-1 (a) or Noxa (d) was determined by western blot analysis using the respective Noxa- and Mcl-1-specific antibodies. β -tubulin serves as control for equal loading. One representative of total of three independent experiments is shown. (b and e) For the determination of loss of MMP, the respective genetically manipulated cells were stimulated for 8 h with Enantiomer (10 μ M) or ABT737 (10 μ M), and the loss of MMP was visualized by TMRE staining and FACS analysis. One experiment of total of three independent experiments (b and e) or the quantitative summary of three independent experiments+S.E.M. is shown (a and d, above the western blot analysis). (c and f) For the analysis of ABT737-induced cell death, MET1 and MET4 cells with knockdown of Mcl-1 (a and c) or HaCaT and A5RT3 cells with knockdown of Noxa (d and f) were stimulated with diluents (DMSO), Enantiomer (10 μ M), or ABT737 (10 μ M) for 18–24 h. Surviving attached cells were quantified with crystal violet assay. Summary of three to five independent experiments is shown+S.E.M.

