



CORRECTION

Author Correction: AZD9291 inactivates the PRC2 complex to mediate tumor growth inhibition

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Correction to: *Acta Pharmacologica Sinica*, <https://doi.org/10.1038/s41401-019-0248-2>, published online 06 June 2019

After publication, the authors realized that the western blotting bands of EZH2 (Fig. 2a), and β -actin (Fig. 2a, Fig. 3a) in MDA-MB-453

cells were misplaced. And in Fig. 5c, the representative images of miR-NC group cells were found to be misplaced by accident. The correct figures are reproduced and presented. The conclusions are not affected by this correction. We sincerely apologize for our mistakes and any inconvenience this might have caused.

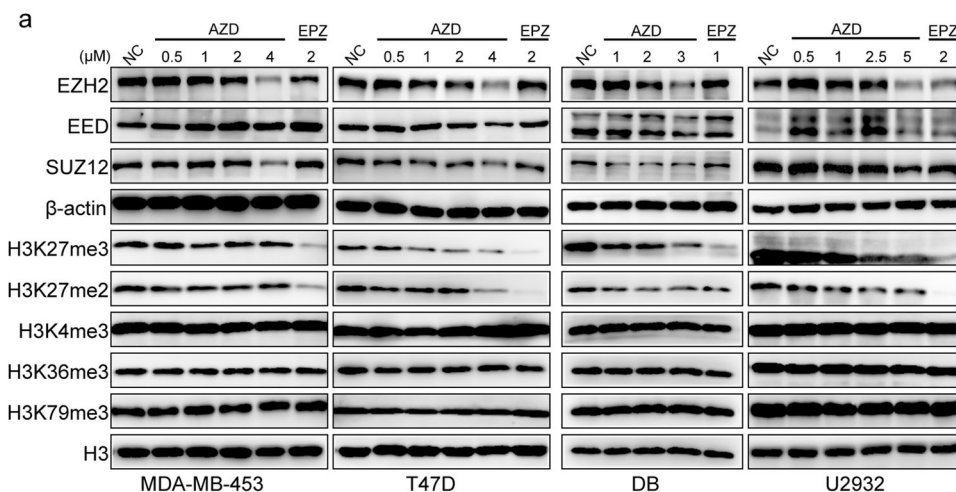


Fig. 2 AZD9291 selectively inhibits H3K27 methylation and exhibits anti-tumor activity. **a** EZH2, EED, SUZ12, H3K27me3, and other histone lysine methylation or acetylation marks levels in MDA-MB-453 and T47D breast cancer cells and DLBCL cell line U2932 (EZH2 WT type) and DB (EZH2 mutant type) were shown by following treatment with the indicated concentrations of AZD or EPZ for 72 h. The β -actin and total histone H3 was shown as a loading control. AZD AZD9291, EPZ EPZ-6438.

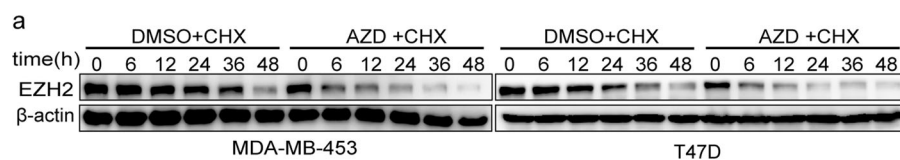


Fig. 3 AZD9291 decreases EZH2 expression and inhibits genes expression which EZH2 activated. **a** Cells were treated with Cycloheximide (100 μ M) combining with vehicle (DMSO) or AZD (5 μ M) for the indicated times, and harvested for detection of protein level by Western blotting. AZD AZD9291, CHX cycloheximide.

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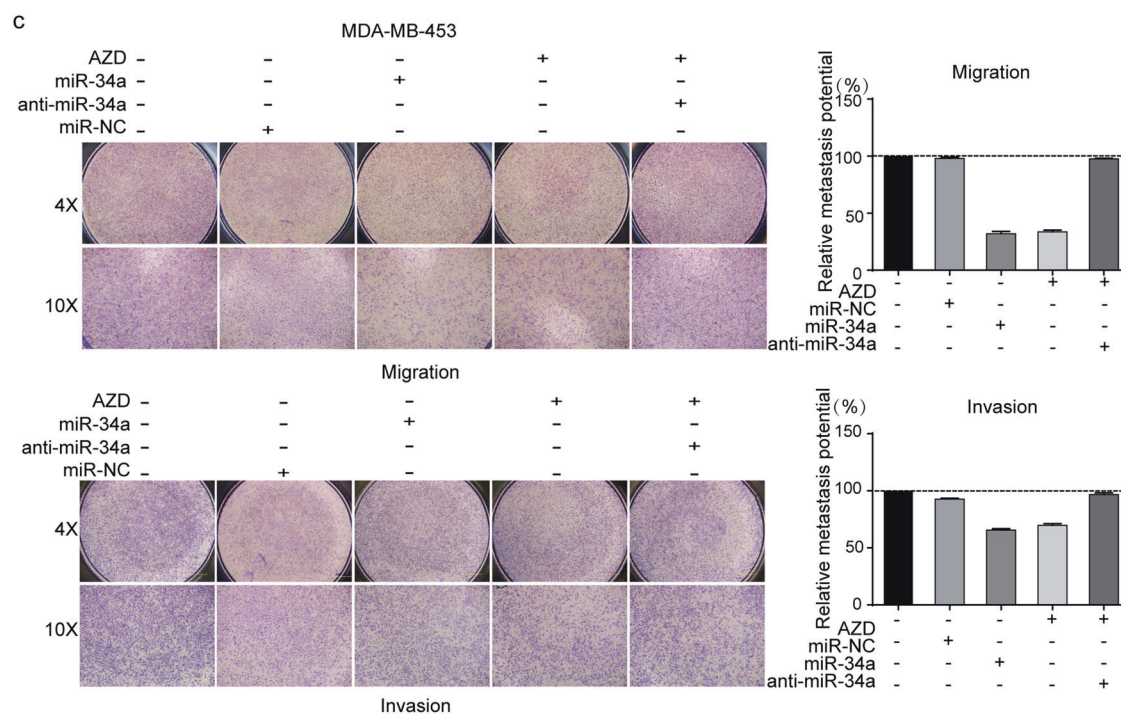


Fig. 5 The influences of AZD9291 and miR-34a on cancer cell metastasis. c AZD 2.5 μ M or miRNAs 100 nM treated MDA-MB-453 cells for 48 h were subjected to transwell experiment and matrigel invasion experiment. Scale bar, 2 mm. AZD AZD9291.