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CORRECTION

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Correction: AHSA1 is a promising therapeutic target for cellular proliferation and proteasome inhibitor resistance in multiple myeloma

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Correction: J Exp Clin Cancer Res 41, 11 (2022) https://doi.org/10.1186/s13046-021-02220-1

Following publication of the original article [1], an error was identified Figs. 1, 2, and 4 specifically:

- Fig. 1e - The numbers of the primary samples were mistyped, which should be consistent with Fig. 2B
- Fig. 2d - The number of the recurrent samples was mistyped, which should be 6 instead of 15
- Fig. 4f - Misplaced images and one incorrect image were used

Furthermore, Figure 1 caption has to be updated. The correct figures and the correct Figure 1 caption are presented below:

The correction do not affect the overall Conclusion of the article.

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Reference

1. Gu C, Wang Y, Zhang L, et al. AHSA1 is a promising therapeutic target for cellular proliferation and proteasome inhibitor resistance in multiple myeloma. J Exp Clin Cancer Res. 2022;41:11. https://doi.org/10.1186/s13046-021-02220-1.

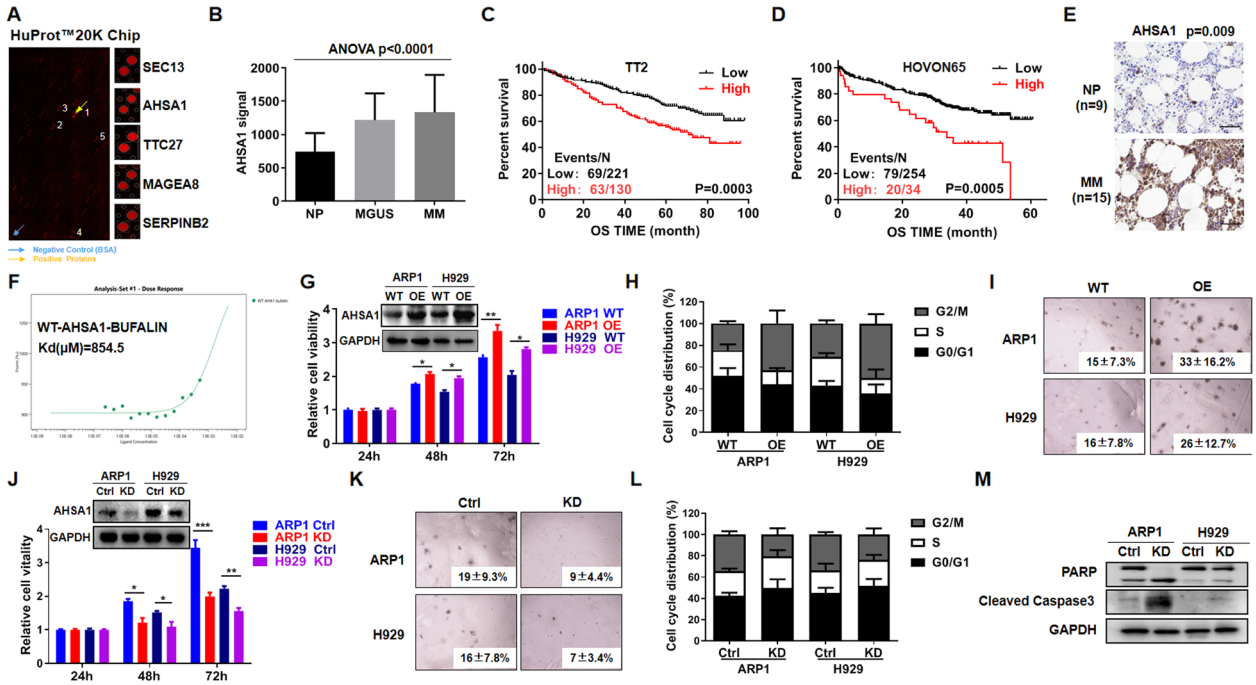

Fig. 1 Elevated AHSA1 expression confers poor survival of MM patients and promotes MM cell proliferation. (**A**) HuProt™20 K Proteome Microarray Chip indicated the top 5 protein targets binding to Bufalin. The yellow arrow indicated positive protein interacted with Bufalin, and the blue arrow represented the negative control. (**B**) Among the top 5 proteins, AHSA1 was the exclusive gene. The signal level of AHSA1 was shown on the y-axis. Patients designated as healthy donors with normal bone marrow plasma cells (NP, n = 22), monoclonal gammopathy of undetermined significance (MGUS, n = 44) or multiple myeloma (MM, n = 351) were sorted on the x-axis. (**C-D**) Increased AHSA1 mRNA expression was positively associated with poor overall survival (OS) in first diagnosis and relapsed MM patients from (C) TT2 and (D) HOVON65 patient cohort. Events/N means events of death/total patients. (**E**) Representative Immunohistochemistry staining on primary MM samples (n = 15) and normal controls (n = 9). (**F**) Microscale thermophoresis (MST) analysis for the interaction of Bufalin with human AHSA1 recombination protein. (**G**) Validation of AHSA1 overexpression in AHSA1-OE ARP1 and H929 cells relative to control cells. (**H**) Cell cycle analysis for WT and AHSA1-OE cells. (**I**) Representative images of cell colonies of WT and AHSA1-OE cells in soft agar. (**J**) Confirmation of AHSA1 protein knockdown in ARP1 and H929 cells after transfection with AHSA1 shRNA. (**K**) Representative images of cell colonies of WT and AHSA1-KD cells in soft agar. (**L**) Cell cycle analysis for WT and AHSA1-KD cells. (**M**) WB analysis of PARP and Caspase 3. The data are expressed as mean ± SD.*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

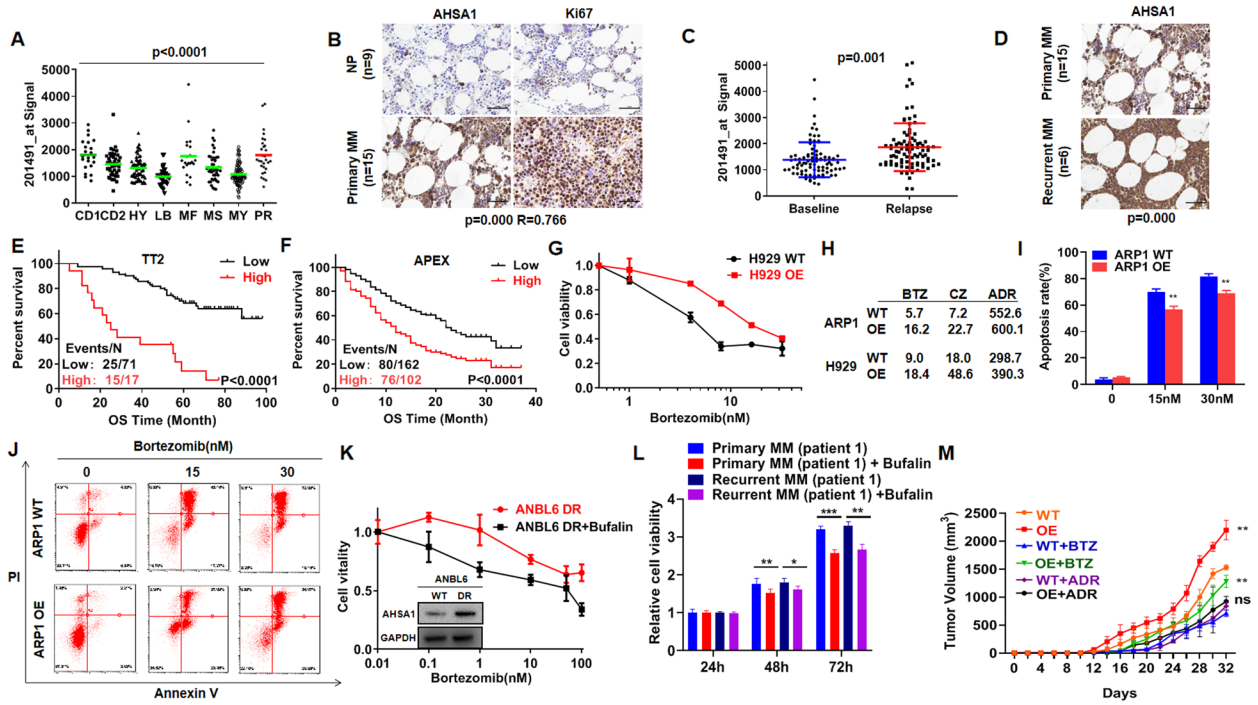

Fig. 2 AHSA1 is a high-risk MM marker and induces proteasome inhibitor resistance in vitro and in vivo. **A** Box plot representing AHSA1 expression in eight MM risk subgroups from TT2 patient cohort. **B** IHC staining of AHSA1 and Ki67 expressions in MM patient samples. **C** AHSA1 mRNA expression in paired patient MM samples collected at first diagnosis and relapse stage. **D** IHC staining of AHSA1 expression in the relapsed samples and the corresponding samples from first diagnosis. **E–F** Elevated AHSA1 expression was correlated with decreased OS in relapsed patients from the (**E**) TT2 and (**F**) APEX cohorts by long-term following up. **G** Effects of Bortezomib on cell viability of H929 cells with or without overexpression of AHSA1. **H** IC50 values of BTZ, CZ and ADR in MM cells with or without overexpression of AHSA1. **I** The rate of BTZ-induced apoptosis was shown in the histogram. **J** Effects of BTZ on cell apoptosis in ARP1 cells with or without overexpression of AHSA1. **K** Effects of Bufalin on cell viability in ANBL6 DR (Bortezomib-resistant) cells. **L** Effects of Bufalin (60 nM) on the cell viability of flow MRD-positive peripheral cells from first diagnosed and relapsed MM patients. **M** Time course of tumor growth in ARP1 AHSA1 WT/OE xenografts taken from NOD-SCID mice treated with vehicle, BTZ, or ADR. The data are expressed as mean ± SD.*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

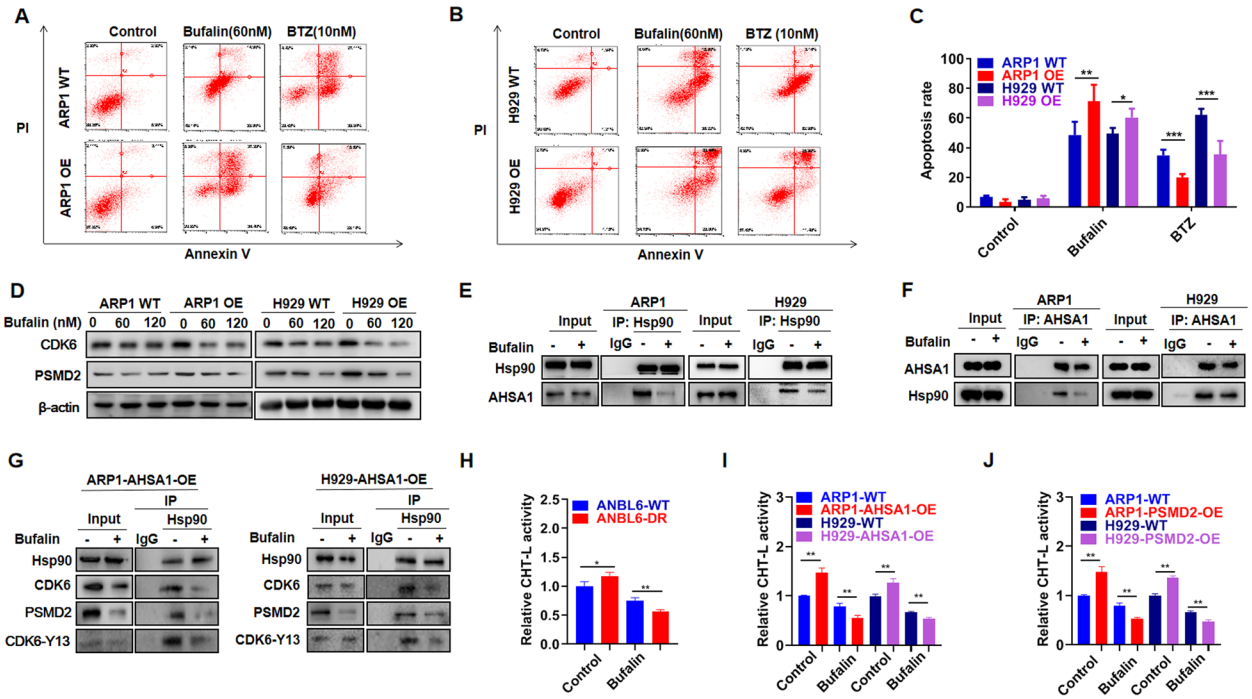

Fig. 4 Bufalin decreases cellular proliferation and PI resistance induced by AHSA1/HSP90 in MM cells. **A-B** Effects of Bufalin (60 nM) and BTZ (10 nM) incubation for 48 h on cell apoptosis of ARP1 (**A**) and H929 (**B**) WT and AHSA1-OE cells. **C** The rate of drug-induced apoptosis was shown in the histogram. **D** Effects of Bufalin on the expression of CDK6 and PSMD2 in ARP1 and H929 WT and AHSA1-OE cells. **E–F** Co-IP assay revealed that Bufalin interfered the interaction between HSP90 and AHSA1 in ARP1 and H929 cells. **G** Co-IP assay confirmed the interaction between AHSA1, HSP90, CDK6, PSMD2 and the activated form of CDK6, phosphorylation of Y13 site at CDK6. **H-J** Proteasome activity assay verified that Bufalin inhibited proteasome activity in (**H**) ANBL6 WT/DR cells, **I** ARP1 and H929 AHSA1 WT/OE cells and **J** PSMD2 WT/OE cells. The data are expressed as mean ± SD.*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$