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Erratum
Endogenous HMGB1 regulates GSDME-mediated pyroptosis via ROS/ERK1/2/caspase-3/GSDME signaling in neuroblastoma: Am J Cancer Res. 2023; 13(2): 436-451

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In this article, there were misusages of the **Figures 1D** and **3C** and **5A**. Hence, we would like to publish this erratum to displace the wrong figures and reflect changes. The authors have confirmed that the errors associated with this figure did not have any significant impact on either the results or the conclusions reported in this study. The authors apologize for any inconvenience or misunderstanding that these errors may have caused. The corrected **Figures 1**, **3** and **5** are shown below.

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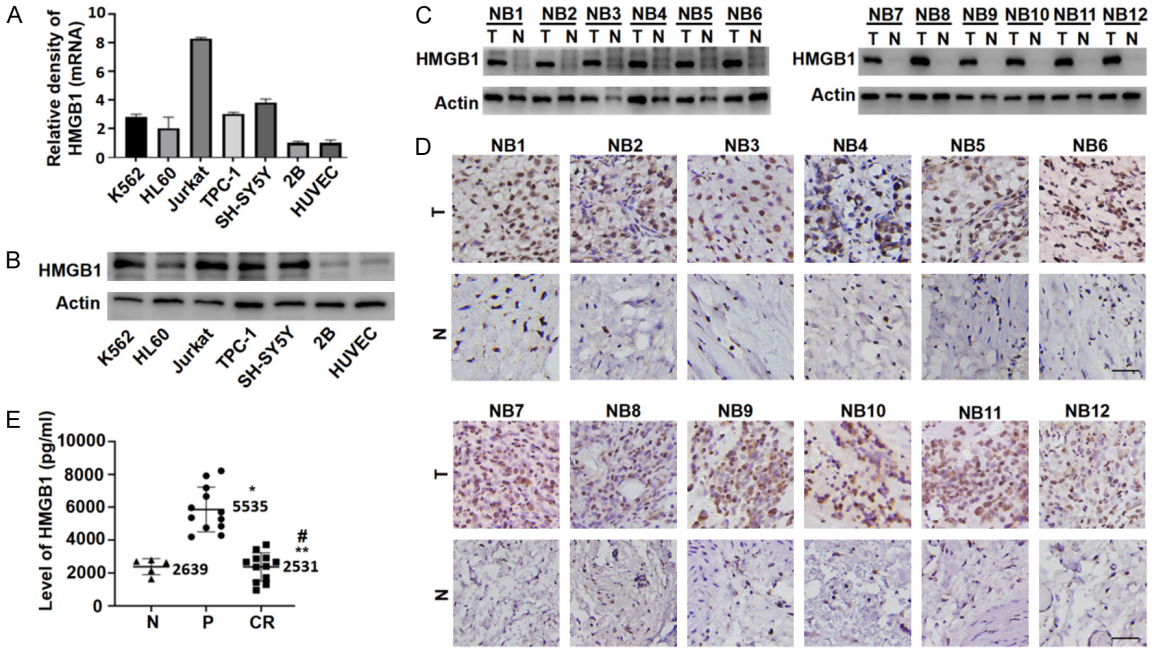

Figure 1. HMGB1 expression is up-regulated in neuroblastoma and associated with clinicopathologic features. A and B. QRT-PCR and western blot of HMGB1 and actin in various cell lines hinted at an over-expression of HMGB1 in SH-SY5Y cell lines; C and D. Western blot and IHC analysis showed that HMGB1 expression was higher in tumors than non-tumors. T, tumor; N, non-tumor; Scale bars, 100 μm; E. Expression of HMGB1 in serum of different patients and normal healthy subjects. N, normal healthy subject; P, primary; CR, complete remission; $^{*}P < 0.05$ vs. normal subject; $^{**}P > 0.05$ vs. normal subject; $^{\#}P < 0.05$ vs. primary.

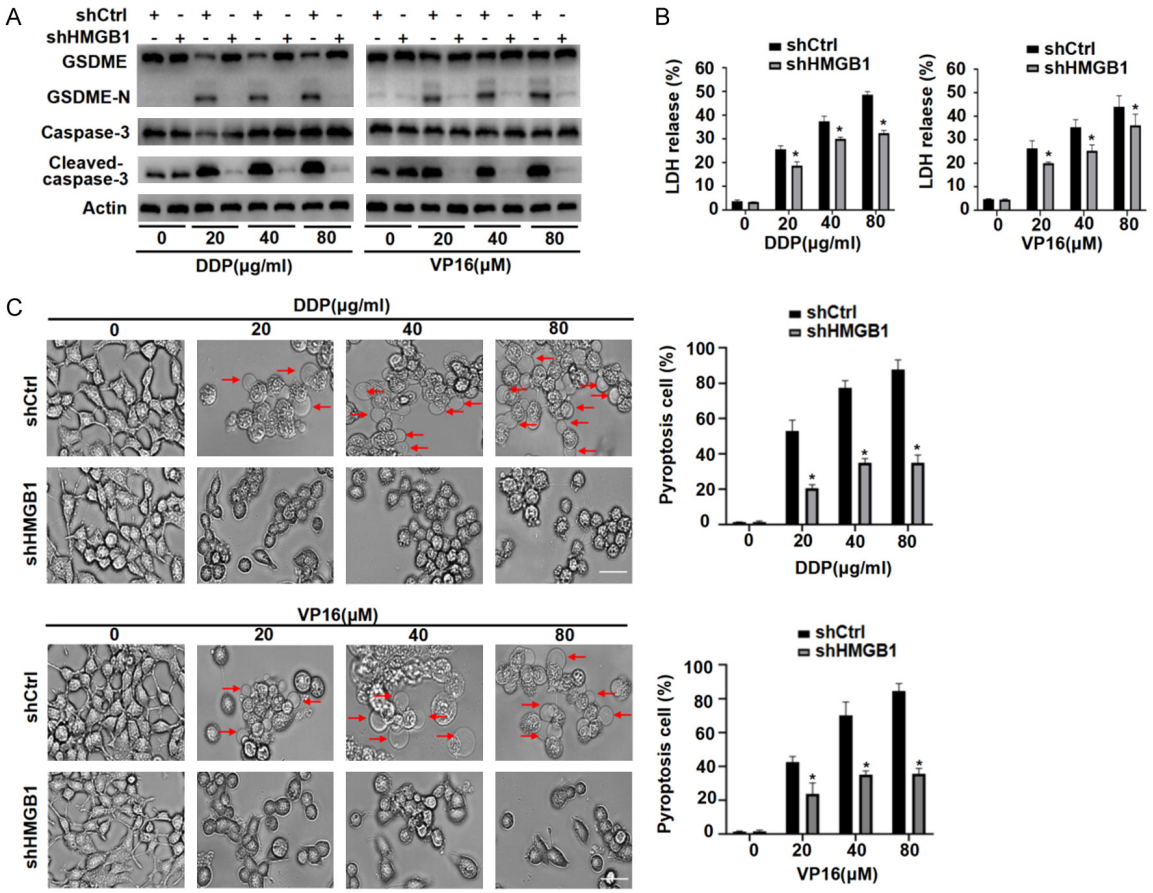

Figure 3. Depletion of HMGB1 inhibits DDP/VP16-induced pyroptosis. A and B. SH-SY5Y cells were transfected with HMGB1 shRNA and control shRNA and then treated with DDP and VP16 at the indicated doses for 24 h, respectively. GSDME-NT and cleaved caspase-3 levels were assayed by western blot. LDH-release was analyzed using LDH assay kit and expressed as mean ± SD (n = 3, **P* < 0.05 vs. shCtrl group). C. Representative light microscopy images of SH-SY5Y cells with differently treatments were detected. The red arrow indicates bubbles emerging from the plasma membrane (n = 3, **P* < 0.05 vs. shCtrl group).

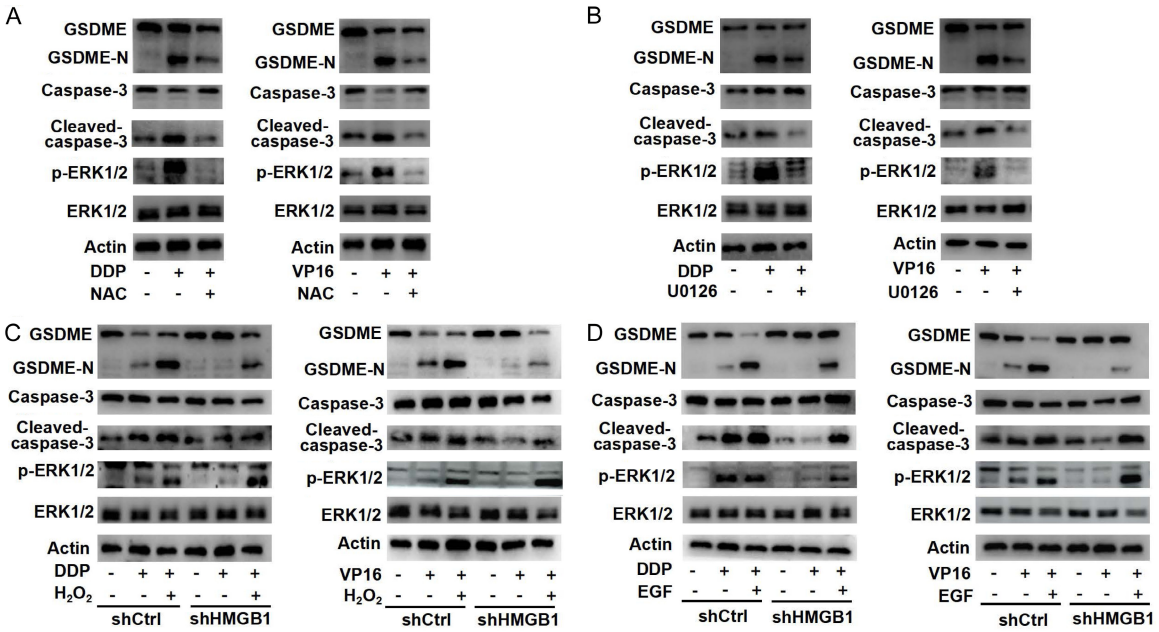

Figure 5. Endogenous HMGB1 regulates DDP/VP16-induced pyroptosis in a ROS-ERK1/2 pathway. A. SH-SY5Y cells were pretreated with or without NAC (20 μM) for 12 h, and then treated with DDP (40 μg/ml) and VP16 (40 μM) for 24 h, respectively. GSDME-NT, cleaved caspase-3 and p-ERK1/2 levels were assayed by western blot. B. SH-SY5Y cells were pretreated with or without U0126 (10 μM) for 12 h, and then treated with DDP (40 μg/ml) and VP16 (40 μM) for 24 h, respectively. GSDME-NT, cleaved caspase-3 and p-ERK1/2 levels were assayed by western blot. C. SH-SY5Y cells were transfected with HMGB1 shRNA and control shRNA, with or without H_2O_2 (50 μM) for 48 h, and then treated with DDP (40 μg/ml) and VP16 (40 μM) for 24 h, respectively. GSDME-NT, cleaved caspase-3 and p-ERK1/2 levels were assayed by western blot. D. SH-SY5Y cells were transfected with HMGB1 shRNA and control shRNA, or pretreated with or without EGF (50 ng/ml) for 48 h, and then treated with DDP (40 μg/ml) and VP16 (40 μM) for 24 h, respectively. GSDME-NT, cleaved caspase-3 and p-ERK1/2 levels were assayed by western blot.