CORRIGENDUM

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HSP90 inhibits apoptosis and promotes growth by regulating HIF-1α abundance in hepatocellular carcinoma

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Subsequently to the publication of the above paper, the authors contacted the Editorial Office to explain that they had found several mistakes in Figs. 1B, 2B, 6B and 7B in their paper. The PCR results shown in Fig. 1B, the flow cytometric results in Figs. 2B and 6B, and the immunohistochemistry results in Fig. 7B were inadvertently chosen incorrectly when these images were selected from the pool of raw data. However, the authors retained access to their original data, and were able to re-assemble the data in these figures as they had intended.

Consequently, the corrected versions of Figs. 1, 2, 6 and 7, containing the replacement data for Figs. 1B, 2B, 6B, and 7B, are shown below and on the next two pages. It should be emphasized that the errors that were made in assembling Figs. 1B, 2B, 6B and 7B did not have a major effect on either the results reported or the conclusions reached in this article. The authors are grateful to the Editor of *International Journal of Molecular Medicine* for allowing them the opportunity to publish this Corrigendum, and all of the authors agree to the publication of this Corrigendum. The authors sincerely apologize for their mistakes and regret any inconvenience that these errors may have caused.

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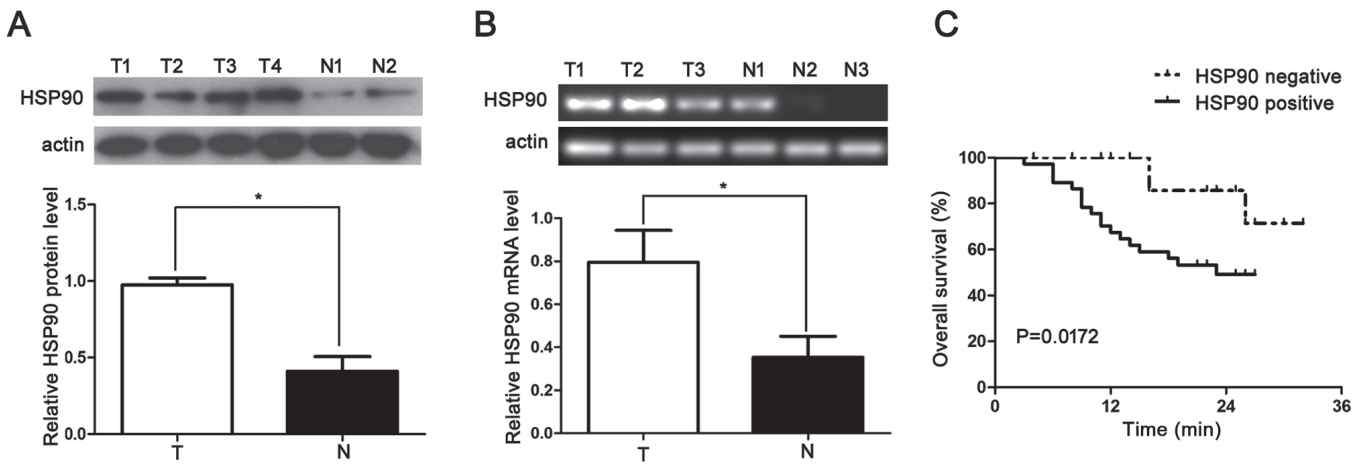

Figure 1. Expression of heat shock protein (HSP)90 and its clinical significance in hepatocellular carcinoma (HCC). (A) Western blot analysis of HSP90 expression in cancer (T) and matched tumor-adjacent tissues (N) is shown. Quantification of the data suggested that HSP90 protein expression in HCC tissues was significantly higher than that in the normal tumor-adjacent tissues. n=20; values are depicted as the means ± SEM; *P<0.05 by t-test. (B) HSP90 mRNA levels in cancer (T) and matched tumor-adjacent tissues (N) were determined by RT-qPCR. Quantification of the data showed that HSP90 mRNA in HCC tissues was significantly higher than that in the normal tumor-adjacent tissues. n=20; values are depicted as the means ± SEM; *P<0.05 by t-test. (C) Kaplan-Meier survival curves showing overall 3-year survival for HCC patients in accordance with their HSP90 protein expression. The HSP90-negative expression group (n=40), IHC score of HSP90=0; HSP90-positive expression group (n=20), IHC score of HSP90=1-3; *P<0.05 by log-rank test.

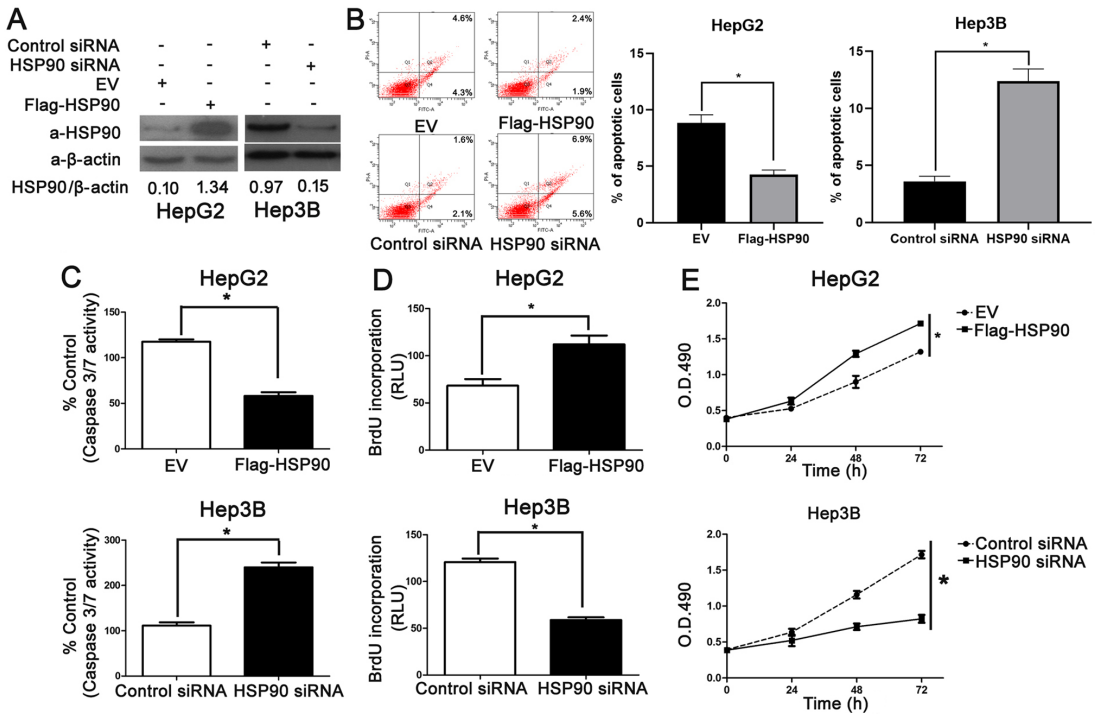

Figure 2. Heat shock protein (HSP90) regulates the proliferation and apoptosis of hepatocellular carcinoma (HCC) cells. (A) Hep3B and HepG2 cells were transfected with HSP90 siRNA and Flag-HSP90, respectively, and then subjected to western blot analysis for HSP90. The data are representative of multiple repeats with similar results. (B) Flow cytometric quantification of the apoptotic cell population. HSP90 knockdown increased the percentage of apoptotic Hep3B cells compared with the control cells, and HSP90-overexpressing HepG2 cells were composed of a smaller subset of apoptotic cells. *P<0.05 by t-test; n=3 repeats with similar results. (C) The activity of caspases-3 and -7 was downregulated following HSP90 overexpression in HepG2 cells and upregulated after HSP90 knockdown in Hep3B cells. *P<0.05 by t-test; n=3 repeats with similar results. (D) Cell proliferation was measured by BrdU assay which was inhibited by HSP90 knockdown in Hep3B cells and promoted by HSP90 overexpression in HepG2 cells. *P<0.05 by t-test; n=3 repeats with similar results. (E) Using MTT assays, the viability of HepG2 cells was enhanced after HSP90 overexpression, and HSP90 knockdown reduced the viability of Hep3B cells. *P<0.05 by two-way ANOVA; n=3 repeats with similar results. Values are depicted as the means ± SEM.

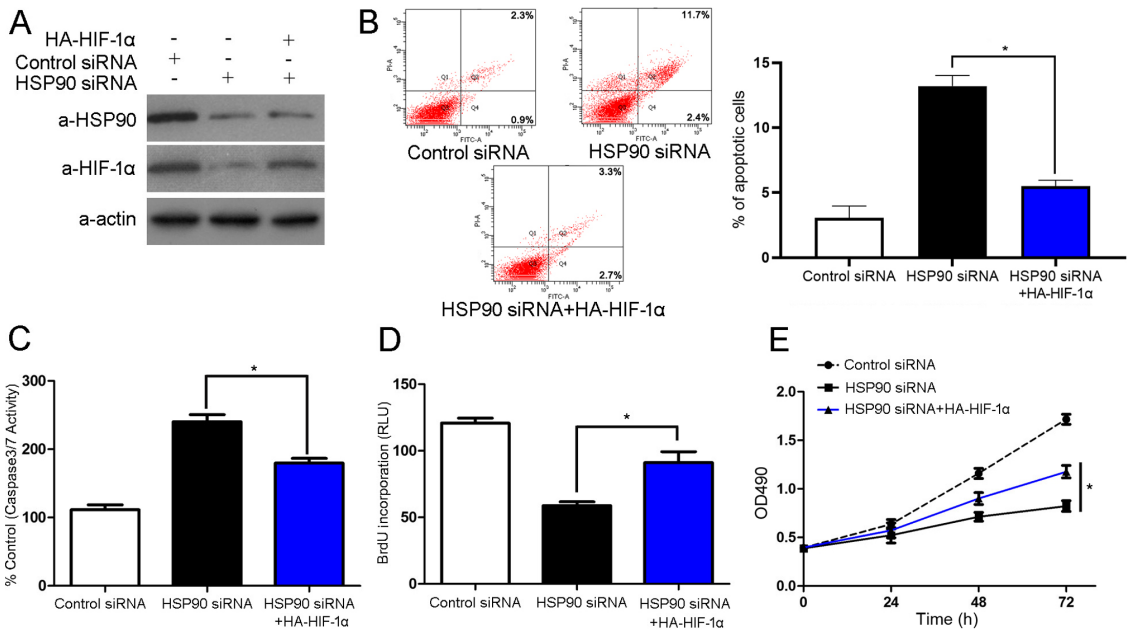

Figure 6. Heat shock protein (HSP)90 siRNA-induced suppression of Hep3B cell growth is partially reversed by hypoxia-inducible factor (HIF)-1α. (A) HSP90 siRNA-transfected Hep3B cells successfully downregulated HSP90 protein expression. HSP90 knockdown in Hep3B cells reduced the levels of HIF-1α. HSP90-knockdown cells which were then transfected with HA-HIF-1α partially rescued the HIF-1α expression. The data are representative of multiple repeats with similar results. (B) Flow cytometry was used to measured apoptotic cells. Restoring HIF-1α expression decreased the number of apoptotic cells in HSP90 siRNA-transfected Hep3B cells. *P<0.05 by one-way ANOVA; n=3 repeats with similar results. (C) In HSP90-knockdown Hep3B cells, the activity of the pro-apoptotic caspases-3 and -7 was decreased after HA-HIF-1α transfection. *P<0.05 by one-way ANOVA; n=3 repeats with similar results. (D) The results of BrdU assay suggested that HIF-1α promoted proliferation in HSP90-knockdown Hep3B cells. *P<0.05 by one-way ANOVA; n=3 repeats with similar results. (E) Performing MTT assays showed that HIF-1α enhanced the viability of HSP90-knockdown Hep3B cells. *P<0.05 by two-way ANOVA; n=3 repeats with similar results. Values are depicted as the means ± SEM.

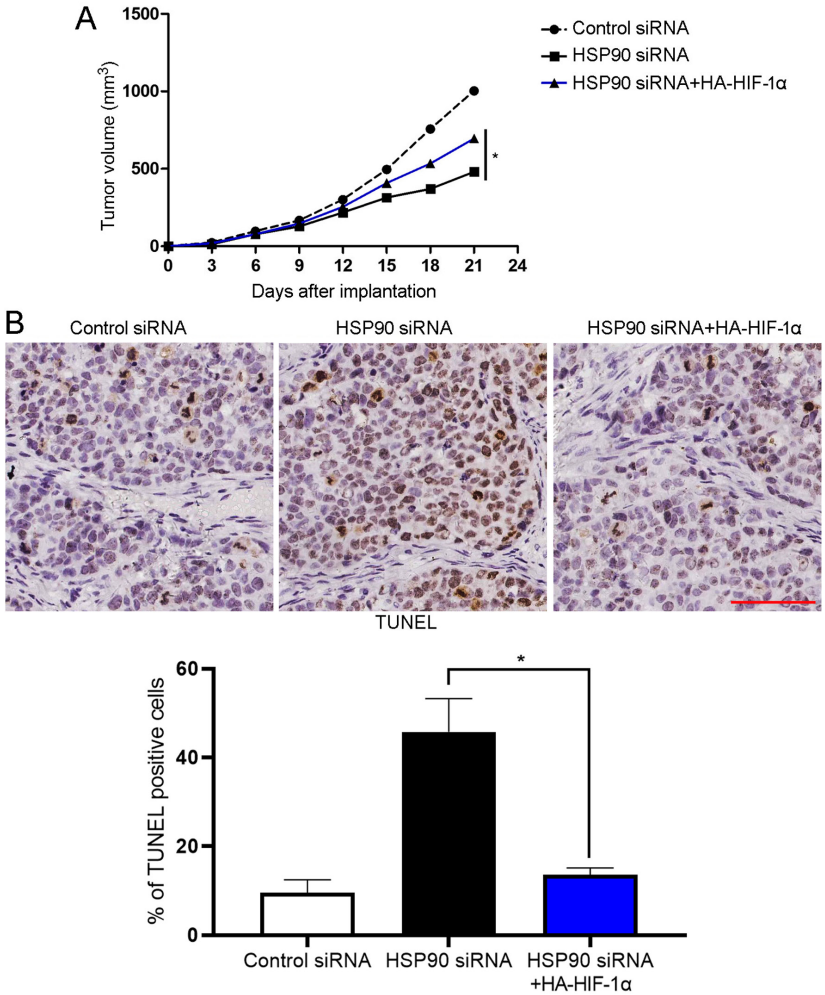

Figure 7. Hypoxia-inducible factor (HIF)-1α partially abolishes heat shock protein (HSP)90 siRNA-induced suppression of tumor growth. (A) Control Hep3B cells (control siRNA, n=6), HSP90-knockdown Hep3B cells (HSP90 siRNA, n=6) and co-expressing Hep3B cells (HSP90 + HA-HIF-1α, n=6), respectively, were implanted into nude mice by subcutaneous injection. A caliper was used to measure the tumor nodules at different times after implantation. HSP90-knockdown Hep3B cells exhibited a greater tumor-inhibiting effect than control cells; however, compared with the HSP90 siRNA group, restoring HIF-1α expression promoted tumor growth. *P<0.05 by two-way ANOVA. (B) Tumor nodules were subjected to TUNEL assays and quantitative analysis. TUNEL assays showed that HSP90 knockdown markedly increased the percentage of apoptotic cells. Scale bar, 70 µm; n=6; values are depicted as the means ± SEM; *P<0.05 and by one-way ANOVA.