CORRIGENDUM

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S100A9 promotes the proliferation and invasion of HepG2 hepatocellular carcinoma cells via the activation of the MAPK signaling pathway

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Subsequently to the publication of the above article, an interested reader drew to the authors' attention that two pairs of data panels in Fig. 7D on p. 1008, showing the results from Transwell invasion assay experiments, contained overlapping sections such that these panels were likely to have been derived from the same original sources where they were intended to show the results from differently performed experiments.

After having consulted their original data, the authors were able to identify that two of the data panels in Fig. 7D were inadvertently selected incorrectly; specifically, the 'GST+SB203580' and 'GST-hS100A9+PD98059' panels in this figure. The revised version of Fig. 7, showing the correct data panels for the 'GST+SB203580' and 'GST-hS100A9+PD98059' panels in Fig. 7D, is shown on the next page. The authors confirm that the errors made during the assembly of Fig. 7 did not grossly affect the major conclusions presented in this paper, and are grateful to the Editor of *International Journal of Oncology* for allowing them this opportunity to publish a Corrigendum. They also apologize to the readership for any inconvenience caused.

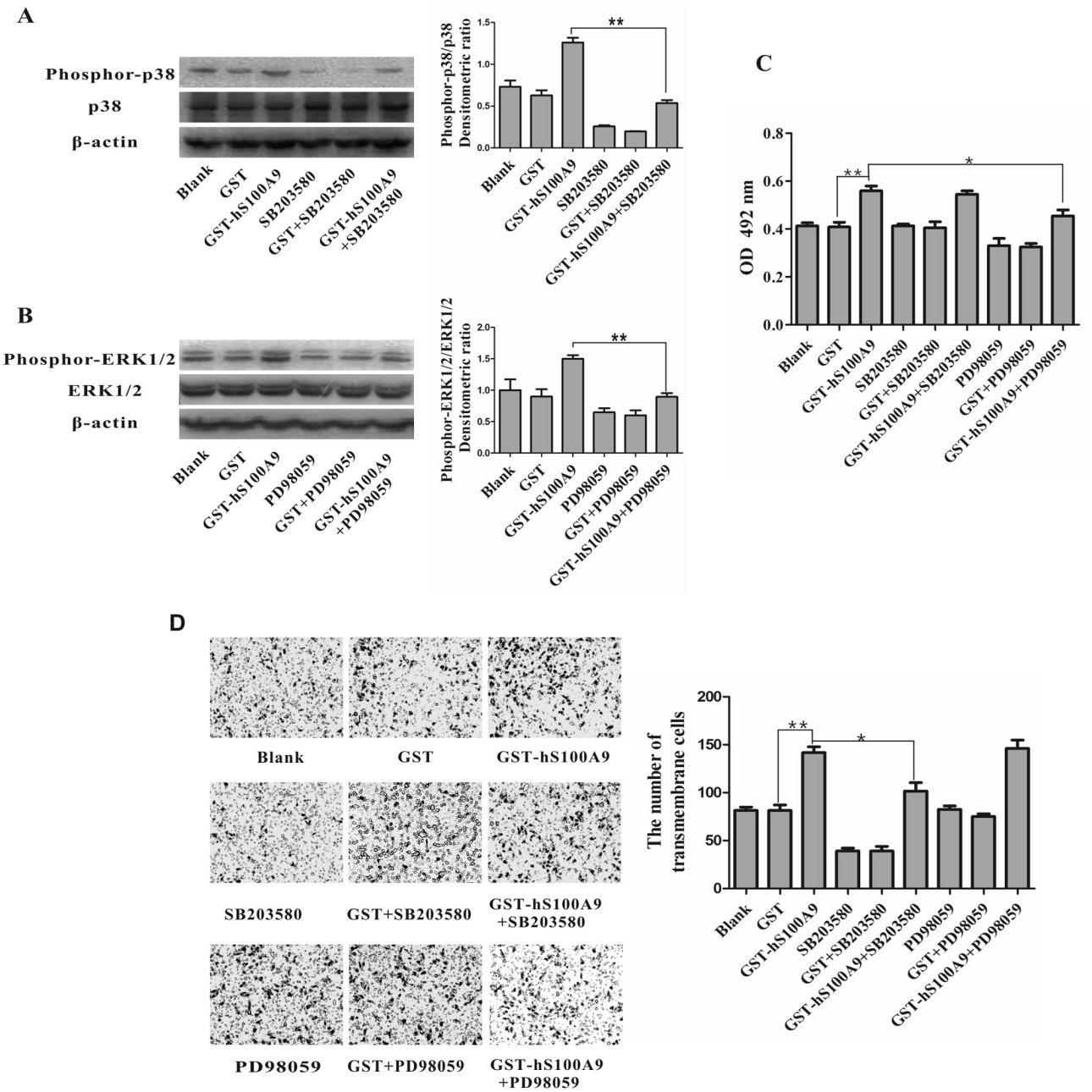

Figure 7. The promotive role of S100A9 in the proliferation and invasion of HepG2 cells. The proliferation and invasion was reversed by inhibitors of ERK1/2 (PD98059) and p38 (SB203580), respectively. (A and B) HepG2 cells were pre-treated with the inhibitors of p38 (SB203580, 10 μM) and ERK1/2 (PD98059, 20 μM) for a period of 30 min prior to treatment for 30 min with GST (20 μg/ml) or GST-hS100A9 (20 μg/ml). The cell lysates were then analyzed by western blot analysis using respective antibodies against phosphorylated p38 or ERK1/2. The densitometric ratios were compared to the control and then normalized to the β-actin control. The promotive role of S100A9 in the phosphorylation of (A) p38 and (B) ERK1/2. The phosphorylation of p38 and ERK1/2 was reversed by SB203580 and PD98059, respectively. **p<0.01, GST-hS100A9 vs. GST-hS100A9+SB203580 or GST-hS100A9+PD98059. (C) HepG2 cells were treated with GST-S100A9 (20 μg/ml) in the presence of SB203580 or PD98059 for 72 h. Cell viability was measured by MTT assay. The promotive role of S100A9 in cell proliferation was reversed by PD98059. The results represent the mean absorbance ± SEM of 3 independent experiments. **p<0.01, GST-hS100A9 vs. GST control; *p<0.01, GST-hS100A9 vs. GST-hS100A9 + PD98059. (D) HepG2 cells were treated with GST-S100A9 (20 μg/ml) in the presence of SB203580 or PD98059 for 24 h. Cell invasion was measured by transwell invasion assay. The transmembrane cells were stained with H&E and were counted under a microscope. The results weere obtained from 5 randomly selected fields for each well, and representative images of transmembrane cells are shown in the left panel; the mean number of transmembrane cells ± SEM per microscopic field of 3 independent experiments are quantified in the right panel. Magnification, ×100. **p<0.01, GST-hS100A9 vs. GST control; *p<0.05, GST-hS100A9 vs. GST-hS100A9+SB203580.