




Correction to: Tat-NTS Suppresses the Proliferation, Migration and Invasion of Glioblastoma Cells by Inhibiting Annexin-A1 Nuclear Translocation

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The original version of this article unfortunately contained error in figure legends.

The article was published with incomplete figure legends for all the figures. Hence, the complete figure legends with its corresponding Figs. 1, 2, 3, 4 and 5 are presented here.

The original article has been corrected.

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The original article can be found online at <https://doi.org/10.1007/s10571-021-01134-y>.

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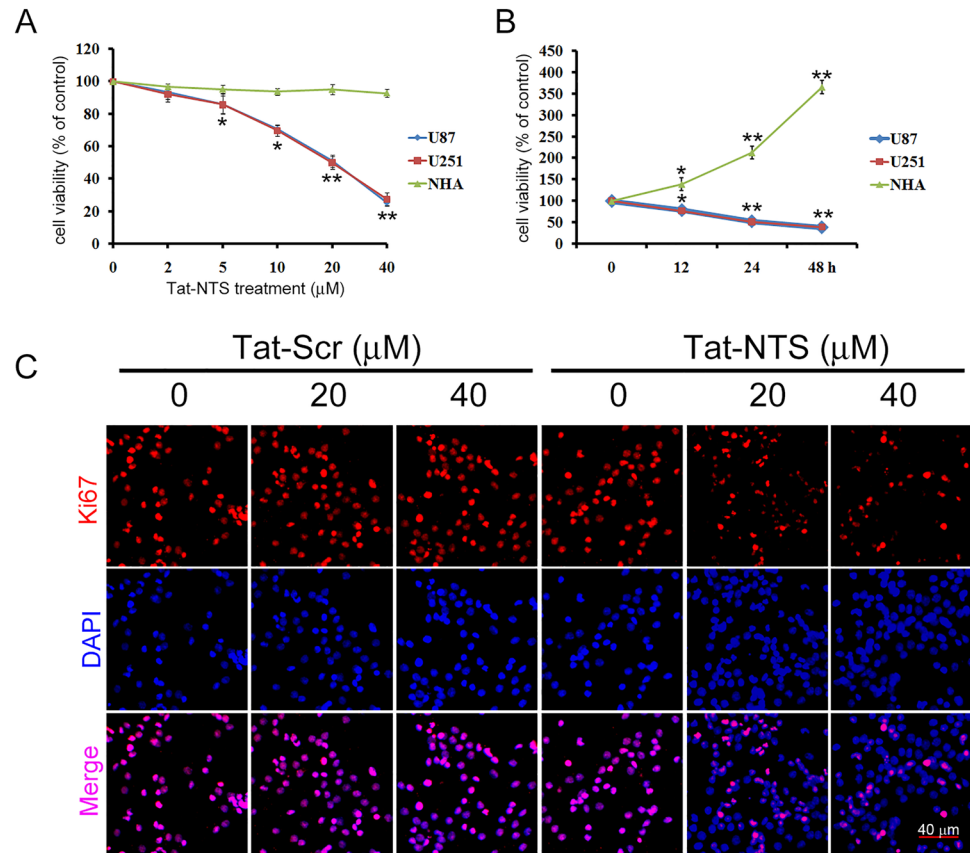
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Fig. 1 Effect of Tat-NTS on GB cell viability. Human U87 and U251 GB cells and normal human astrocytes (NHAs) were exposed to various concentrations of Tat-NTS (2, 5, 10, 20 and 40 μM) or PBS (vehicle) for 24 h. Cells were also exposed to 20 μM Tat-NTS for 12, 24 and 48 h. Cell viability was then analyzed by CCK-8 assay. Tat-NTS inhibited GB cell proliferation in a dose-dependent manner (**a**) and time-dependent manner (**b**) but had no impact on NHAs. Mean \pm SD ($n = 5$). * $P < 0.05$ and ** $P < 0.01$ vs 0 $\mu\text{mol/L}$ or 0 h. **c** Expression of the proliferation marker Ki-67 was evaluated using immunofluorescence staining for Ki67 (red) and DAPI (blue). Scale bar, 40 μm



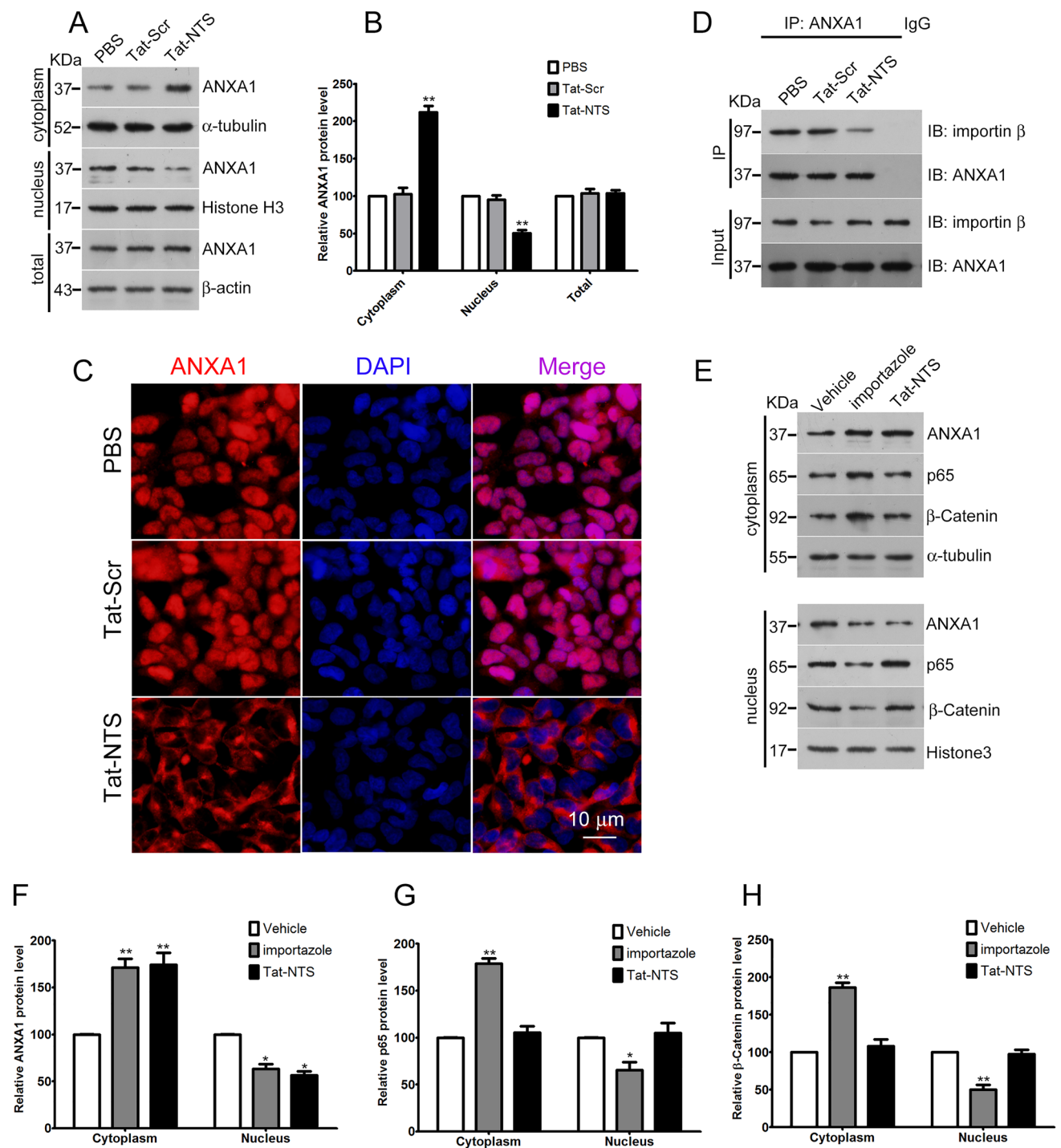
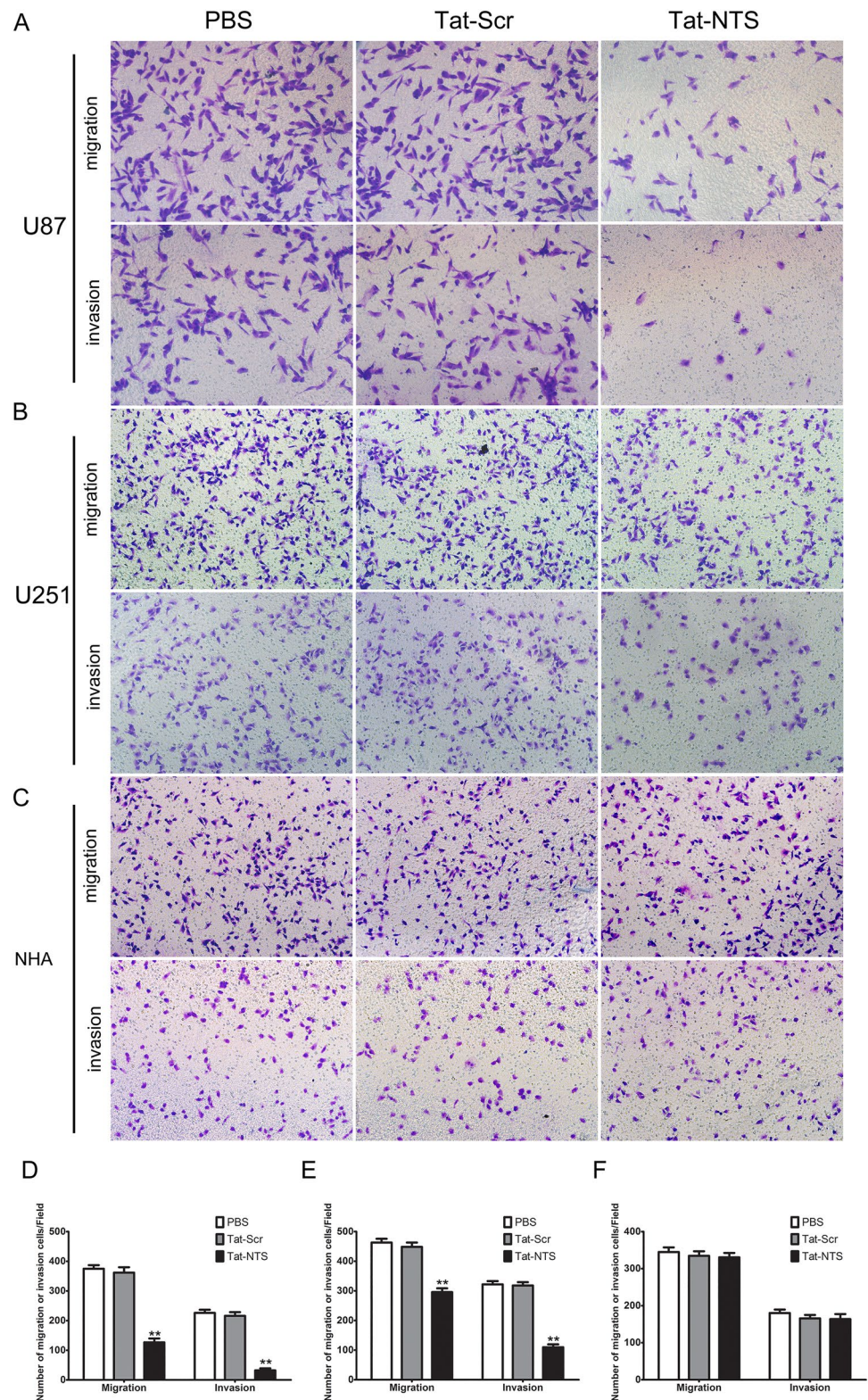


Fig. 2 Tat-NTS inhibits ANXA1 translocation to the nucleus in U87 cells. U87 cells were treated with PBS, Tat-Scr or Tat-NTS (20 μM) for 24 h. Nuclear and cytosolic protein extracts were then subjected to Western blot analysis with specific antibodies. **a** Representative images of Western blot analysis for ANXA1 levels in the cytoplasmic and nuclear extracts. α-tubulin and histone H3 were used as cytoplasmic and nuclear internal controls, respectively, while β-actin was used as a loading control of total proteins. **b** The relative level of ANXA1 protein was quantified by densitometric analysis. Data are represented as the mean ± SD from three independent experiments. ***P* < 0.01 vs the Tat-Scr control. **c** Representative immunofluorescence staining results of ANXA1 (red) and nuclei (blue) showing the

subcellular distribution of endogenous ANXA1 in U87 cells. Scale bar = 10 μm. **d** Representative Co-IP results showing the interaction of ANXA1 with importin β in GB cells treated with Tat-NTS or Tat-Scr peptide. **e** Representative images following Western blot analysis for ANXA1, p65 NF-κB, and β-catenin in cytoplasmic and nuclear extracts of U87 cells treated with vehicle, importazole or Tat-NTS peptide. α-Tubulin and histone H3 served as cytoplasmic and nuclear loading controls, respectively. **f–h** Quantification of the relative protein levels of ANXA1, p65 NF-κB and β-catenin. The intensity of the bands was quantified by densitometric analysis. **P* < 0.05 and ***P* < 0.01 vs the Vehicle group; all data are presented as the mean ± SD from three independent experiments

Fig. 3 Effect of Tat-NTS on the migration and invasion capacity of GB cells in vitro. **a–c** U87 GB cells, U251 GB cells and NHAs were treated with PBS, Tat-Scr (20 μ M) or Tat-NTS (20 μ M) for 24 h, and cell migration was then determined with a Transwell system. Migrated and invaded cells on the bottom surface of the membrane were fixed and stained with crystal violet. Original magnification: $\times 200$. **d–f** Quantification of migrating and invading cells corresponding to **a–c**. The migrating and invading cells were counted under a microscope in five random fields. Data are presented as the mean \pm SD from three independent experiments. ****** $P < 0.01$ vs the Tat-Scr group



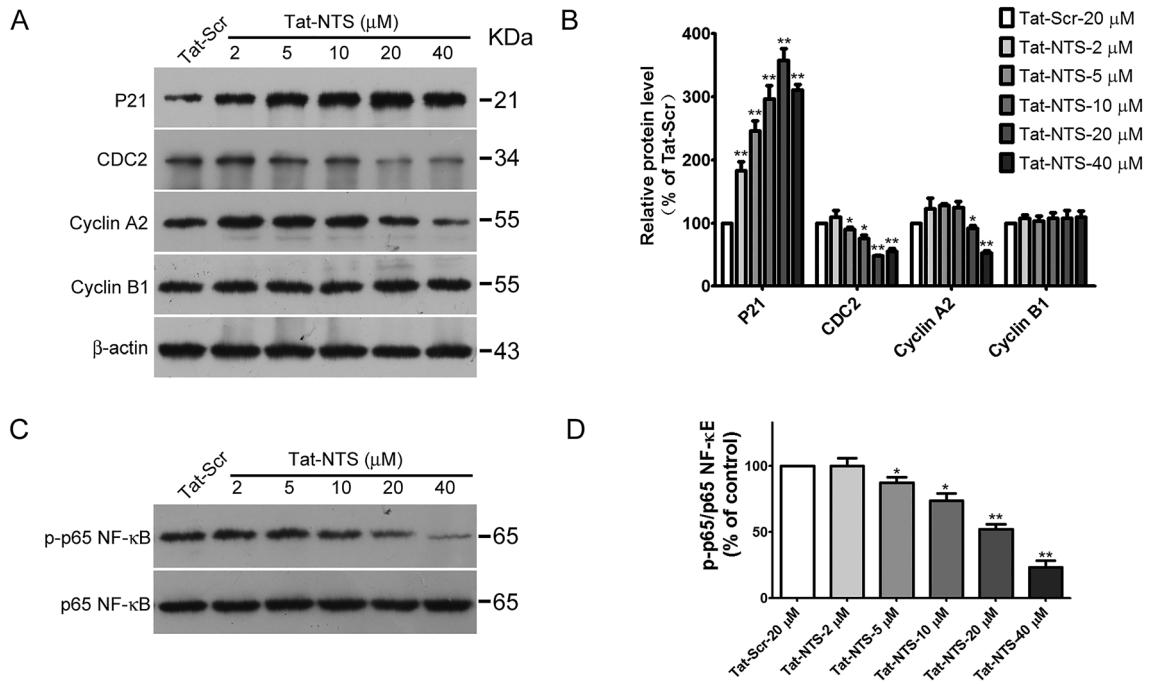


Fig. 4 Tat-NTS induces G2/M cell cycle arrest and inhibits activation of the NF-κB signaling pathway in GB cells. U87 cells were treated with the indicated concentrations of Tat-NTS (2, 5, 10, 20 and 40 μM) for 24 h, and the expression levels of P21, CDC2, Cyclin A2, Cyclin B1, and phosphorylated p65NF-κB in total-cell lysates were

determined by Western blot analysis. **a** and **c** Representative protein blots evaluated by Western blot analysis. **b** and **d** Densities of the protein bands were normalized to those of β-actin and p65 NF-κB. Data are shown as the mean ± SD from three independent experiments. **P* < 0.05 and ***P* < 0.01 vs the Tat-Scr group

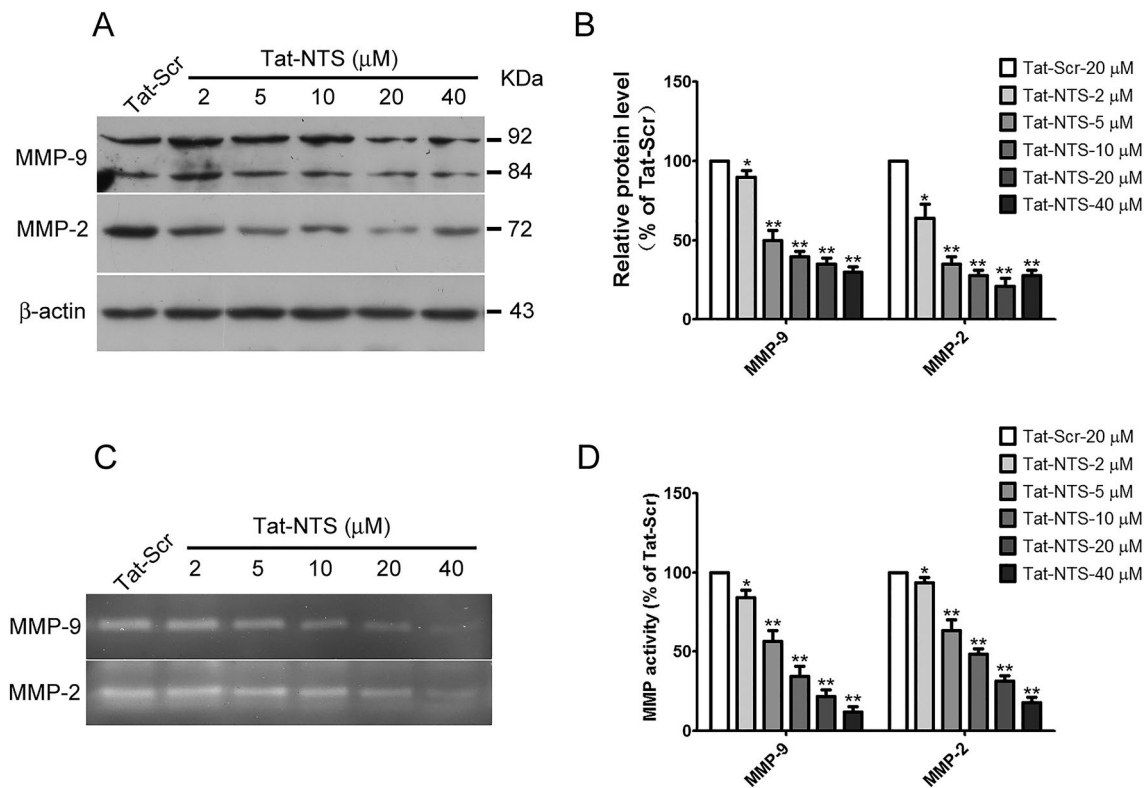


Fig. 5 Tat-NTS inhibits the expression and activity of MMP-2 and MMP-9. U87 cells were treated with Scr control (20 μM) or different concentrations of Tat-NTS (2, 5, 10, 20 and 40 μM) for 24 h. Both cell lysates and conditioned media were collected. Cell lysates were analyzed by Western blotting, and conditioned media were analyzed by gelatin zymography assay. **a** Representative images fol-

lowing Western blot analysis for MMP-2 and MMP-9 expression in U87 cells. **b** Quantification of relative protein levels of MMP-2 and MMP-9 in U87 cells. **c** Representative images of gelatin zymography of MMP-2 and MMP-9. **d** Quantification of the relative bands of MMP-2 and MMP-9 in U87 cells ($n=3$). * $P<0.05$ and ** $P<0.01$ vs the Tat-Scr group