Correction

Correction: Cdc6 contributes to cisplatin-resistance by activation of ATR-Chk1 pathway in bladder cancer cells

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This article has been corrected: Due to errors during Figure preparation, the wrong image was selected for placement in Figure 3D (UMUC3 cells, Si-NC panel). This image contained a partial duplicate of an image in Figure 3C (UMUC3 cells, Si-Cdc6 column). The corrected Figure 3 is shown below. In addition, the legend of Figure 3 contains a spelling error - the word 'invision' has been changed to 'invasion'. The authors declare that these corrections do not change the results or conclusions of this paper.

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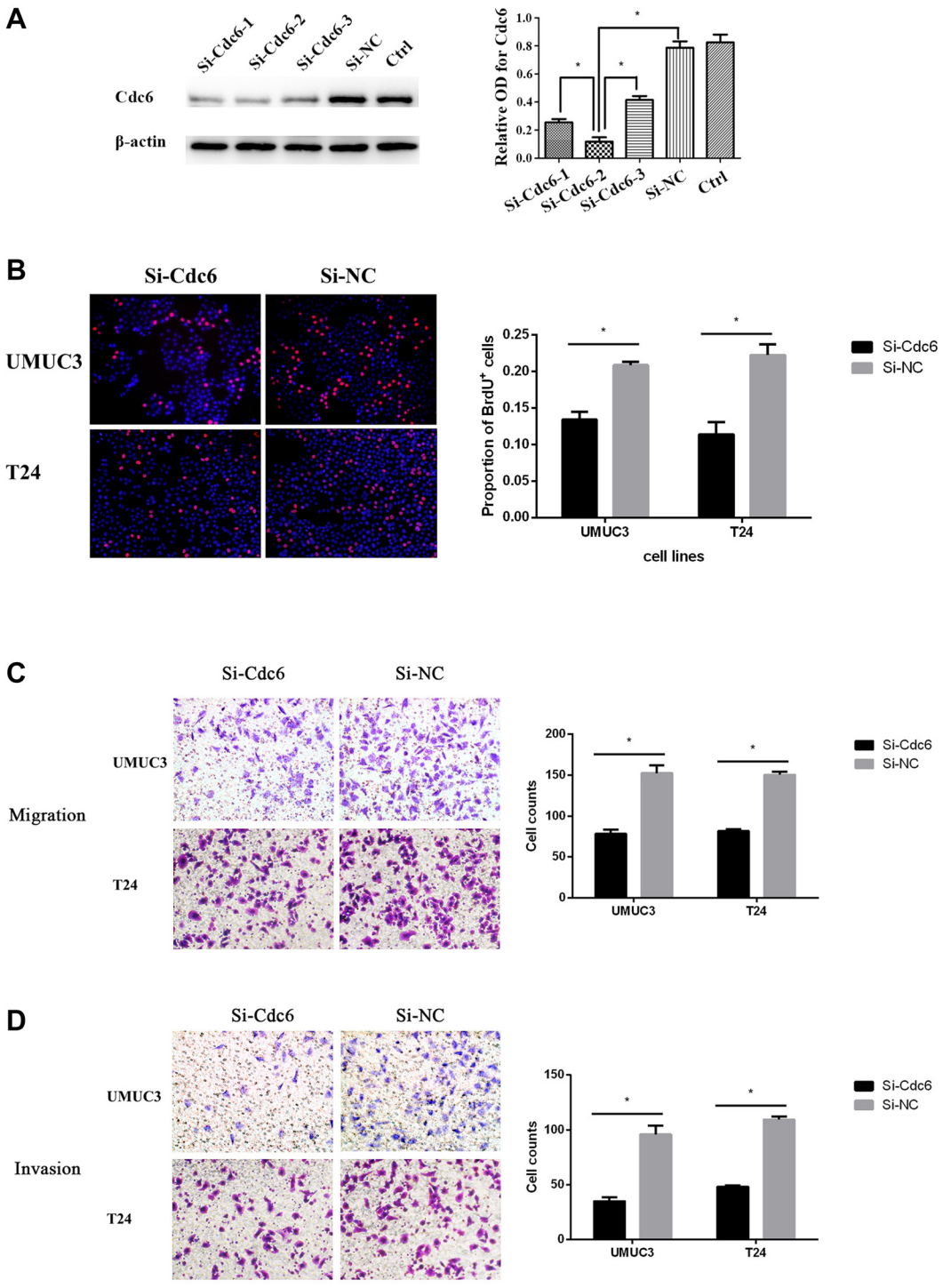

Figure 3: Cdc6 depletion reduced DNA replication, migration and invision in bladder cancer cells. (**A**) UMUC3 Cells were transfected with Cdc6 siRNA-1, 2, 3 or negative control siRNA (Si-NC) for 48 h. Cdc6 protein level was analyzed by Western blot. Beta-actin was used as the loading control. Data are expressed as optical density (OD) fold difference related to beta-actin from 3 duplicate experiments, $^{*}P < 0.05$. (**B**) UMUC3 or T24 Cells were transfected with Cdc6 siRNA-2 or Si-NC for 48 h, BrdU incorporation assays were performed to evaluate DNA synthesis after transfection for 48 h; Transwell migration assay (**C**) and transwell invasion assay (**D**) UMUC3 or T24 Cells were transfected with Cdc6 siRNA-2 or Si-NC for 24 h, cells were plated on the upper chambers. After 24 h, cells of migration and invasion were counted. Data are shown as mean ± SD of three independent experiments (right panel), $^{*}P < 0.05$.