Cellular Physiology and Biochemistry
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**Erratum**

In the article "ATM-Mediated Phosphorylation of Cortactin Involved in Actin Polymerization Promotes Breast Cancer Cells Migration and Invasion" [Cell Physiol Biochem 2018;51:2972–2988. DOI: 10.1159/000496048] by Lang et al., the incorrect images were included in Figure 2, Figure 6 and Figure 7 due to an error in Figure preparation.

The corrected representative images are:

1. Figure 2C 21% O2/ATMi;
2. Figure 6C BT549/WT and BT549/S113A;
3. Figure 6D MDA-MB-231/ATMi+S113D and BT549/S113A;
4. Figure 7A BT549/Extract CTTN;
5. Figure 7B MDA-MB-231/Extract CTTN and Arp3.

The corrected Figure 2, Figure 6 and Figure 7 are shown below.

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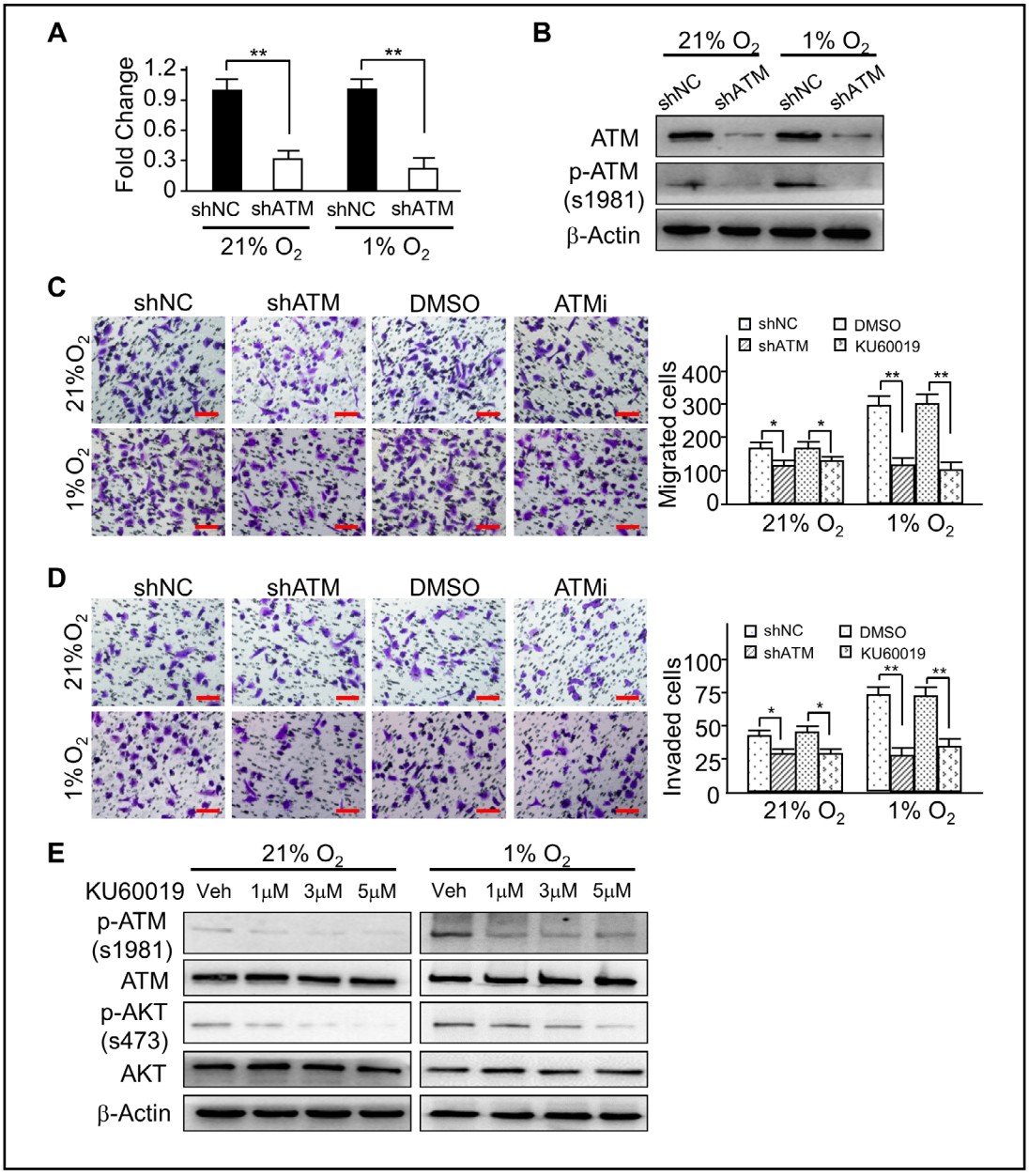


**Fig. 2.** Oxidized ATM is required for cell migration and invasion in breast cancer cells. (A, B) The knocked down efficiency of ATM by specific shRNA was measured in normoxic and hypoxic breast cancer cells BT549 using qRT-PCR (A) and western blot (B) (**, p<0.01). (C, D) BT549 cells were stably knocked down ATM or treated with Ku60019 (labeled as ATMi), the cell migration (C) and invasion (D) potentials were tested by transwell assay in normoxia and hypoxia, scale bars, 50μm (**, p<0.01). (E) BT549 cells were treated with or without an oxidized ATM inhibitor Ku60019 (1 μM to 5μM) for 6 hours in normoxia and hypoxia. The activation of oxidized ATM and AKT was measured by western blotting.

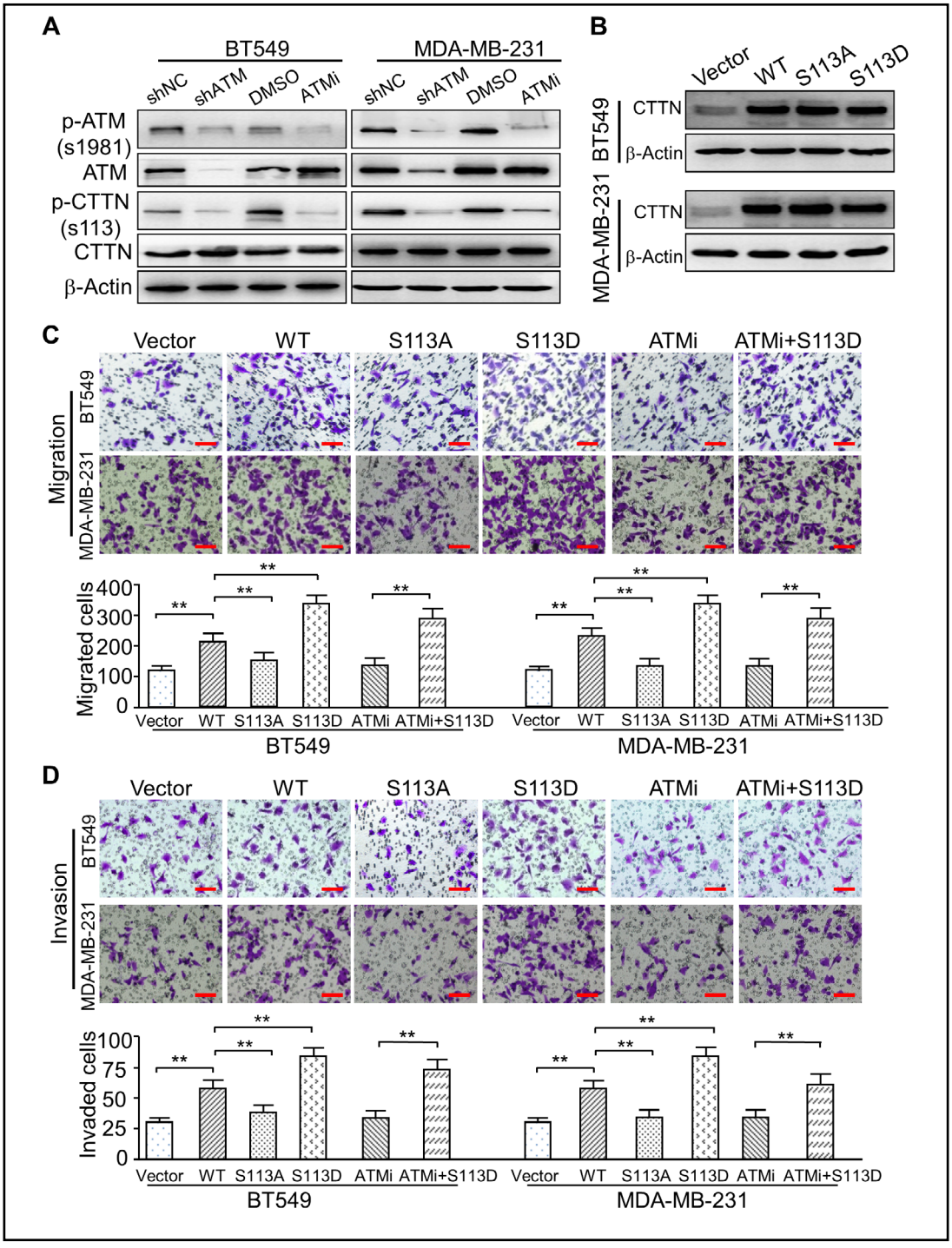


**Fig. 6.** Oxidized ATM-mediated phosphorylation of cortactin promotes breast cancer cells migration and invasion. (A) Extracts from hypoxic BT549 and MDA-MB-231 (shNC, shATM, DMSO and ATMi) cells were used to measure the protein levels of p-ATM (s1981), p-CTTN (s113), total ATM and CTTN by the indicated antibodies. (B) Western blotting to determine cortactin protein levels in hypoxic BT549 and MDA-MB-231 cells stably transfected with WT, mutant (S113A or S113D) cortactin and the control vector. β-Actin is the loading control. (C-D) Transwell assay to test cell migration (C) and invasion (D) of hypoxic BT549 and MDAMB- 231 cells stably expressing WT, mutant cortactin (S113A or S113D) and control plasmid treated with or without Ku60019 (labeled as ATMi). Scale bars, 50μm (**, p<0.01).

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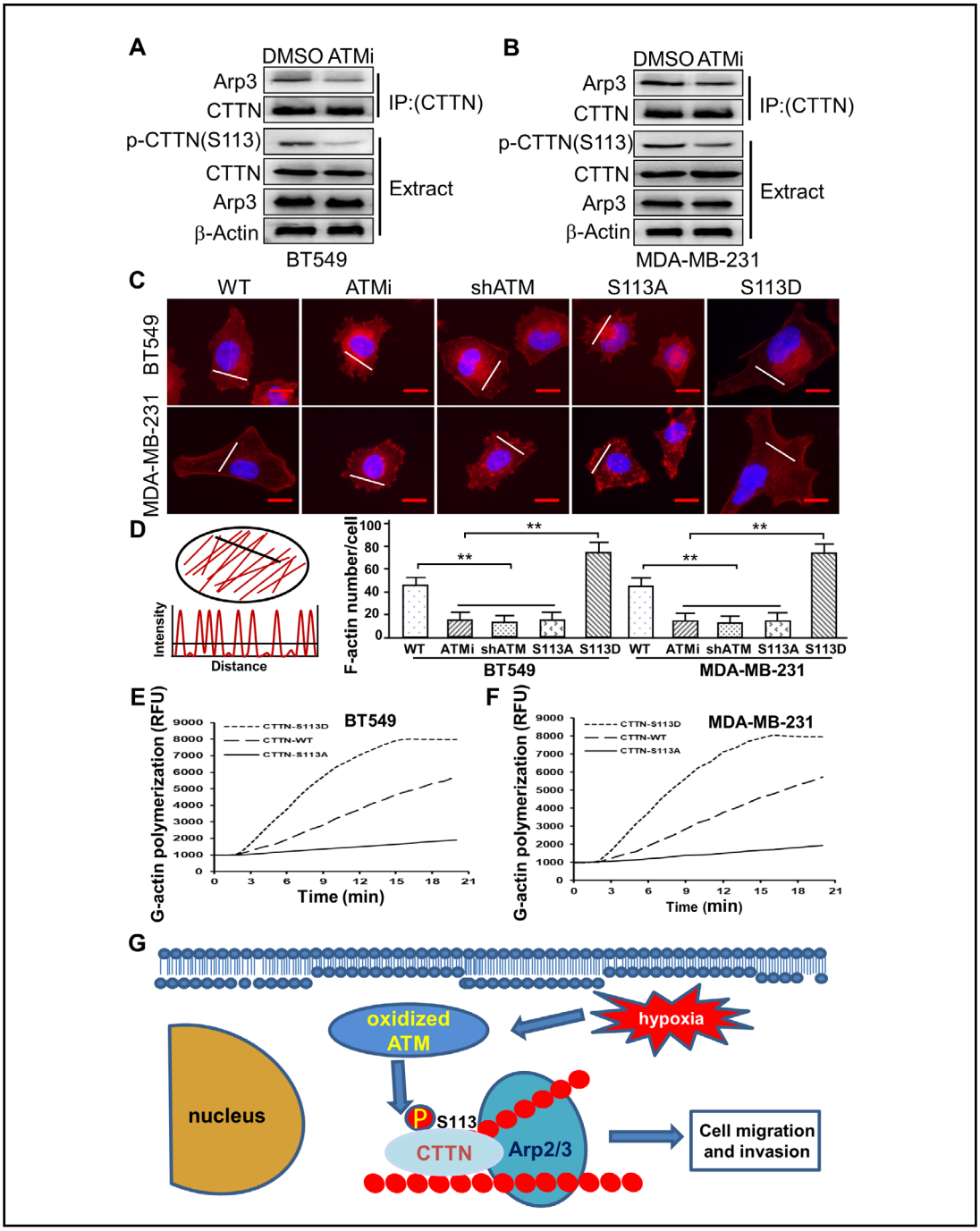


**Fig. 7.** The phosphorylated cortactin binding with Arp2/3 promotes actin polymerization. (A, B) Cortactin was immunoprecipitated from extracts of BT549 (A) and MDA-MB-231 cells (B) treated with or without Ku60019 under hypoxia, followed by immunoblotting with indicated antibodies. The protein binding between cortactin and Arp2/3 is shown. (C, D) The polymerized actins (F-actin) were displayed by immunofluorescence staining using rhodamineconjugated phalloidin in hypoxic BT549 and MDA-MB-231 cells stably expressing WT, mutant (S113A or S113D) cortactin, shATM, or control cells treated with Ku60019 (labeled as ATMi). Scale bars, 10μm. (E, F) Equal amounts of protein extracts from BT549 cells (E) and MDA-MB-231 cells (F) transfected with cortactin WT, S113A or S113D construct were incubated with pyrene actin monomers, and the kinetics of actin polymerization was measured as described in Materials and Methods. (G) A proposed model to show oxidized ATM in promoting breast cancer cells migration and invasion via phosphorylated-cortactin/Arp2/3-mediated actin polymerization.