Am J Cancer Res 2022;12(7):3486-3490
www.ajcr.us /ISSN:2156-6976/ajcr0143602

# *Erratum*
# Econazole nitrate reversed the resistance of breast cancer cells to Adriamycin through inhibiting the PI3K/AKT signaling pathway: Am J Cancer Res. 2020; 10(1): 263-274

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Received April 18, 2022; Accepted June 18, 2022; Epub July 15, 2022; Published July 30, 2022

The original version of this article contained a few mistakes, which have no influence on the final findings and conclusions, and we hereby publish these corrections. The detailed corrections were shown as followed:

In **Figure 1**, Adriamycin in panel A annotation and Econazole in panel B annotation were reversed due to error paste.

In **Figure 2**, four panels of the flow cytometry data (Econazole (3 μM) on breast cancer cells (MDA-MB-231/ADR, MDA-MB-231, MCF-7/ADR and MCF-7)) were misplaced.

In **Figure 3A**, several Western blotting (WB) panels of Econazole in MDA-MB-231 were misused. The reason is that we used similar WB data collected from other drugs at the same time.

In **Figure 4D**, the correct annotation of Econazole concentration is "3 μM", not "0 μM".

We express regrets for these unintentional mistakes caused by careless operation in the process of data acquisition and image layout.

The correct **Figures 1**-**4** are shown below.

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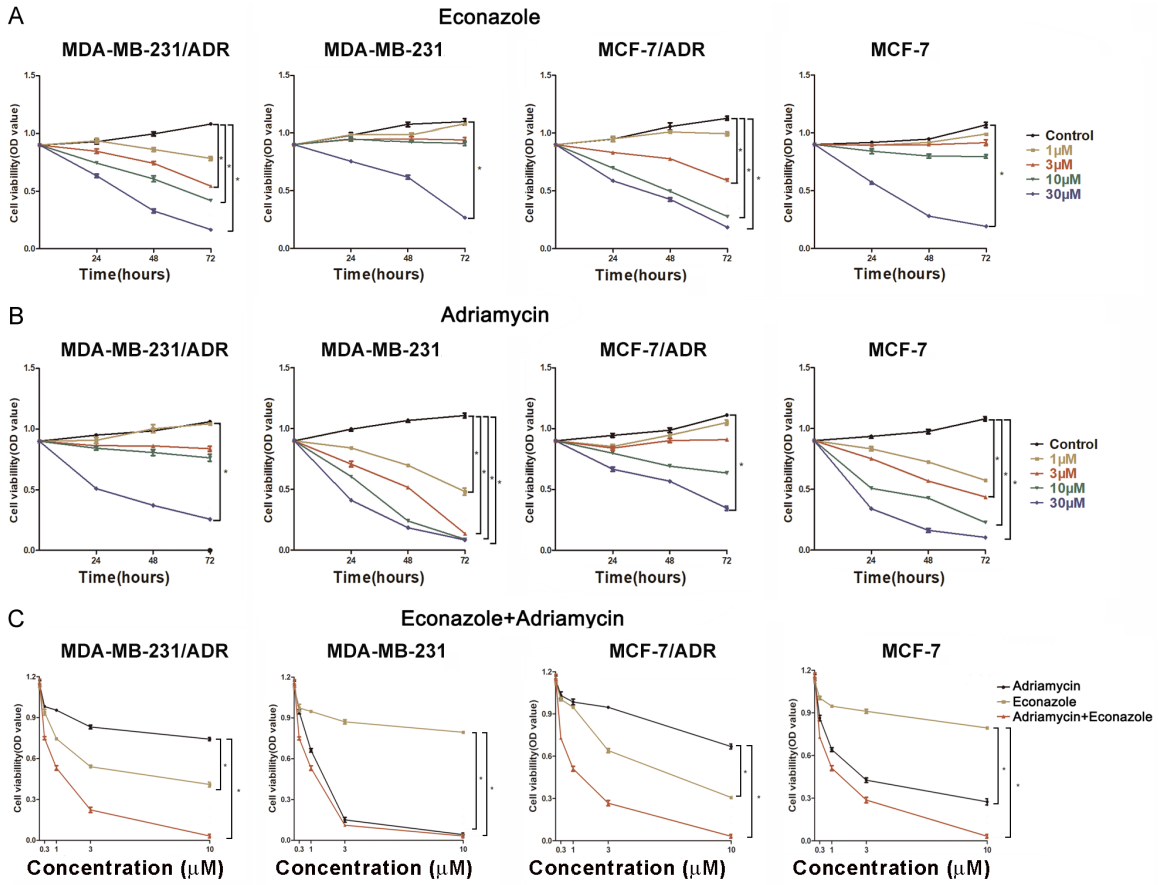


**Figure 1.** The cytotoxic effects of Econazole and Adriamycin in ADR and sensitive breast cancer cell lines. A. Econazole (1, 3, 10, and 30 μM) efficiently decreased the viability of ADR breast cancer cells (MDA-MB-231/ADR and MCF-7/ADR) at 24, 48, and 72 hours, as measured by MTT assays. Interestingly, MDA-MB-231 and MCF-7 cells were not sensitive to low concentrations (<10 μM) of Econazole. B. Adriamycin (1, 3, 10, and 30 μM) efficiently decreased the viability of sensitive breast cancer cells (MDA-MB-231and MCF-7) at 24, 48, and 72 hours, as measured by MTT assays. MDA-MB-231/ADR and MCF-7/ADR cells showed resistance to low concentrations (<10 μM) of Adriamycin. C. The combination of Econazole (0.3-10 μM) and Adriamycin (0.3-10 μM) synergistically decreased the viability of ADR breast cancer cells, as measured by MTT assays at 72 hours. Statistical analysis was performed by one-way ANOVA (Analysis Of Variance), and the difference was considered significant if $P<0.05$, which is marked with an asterisk (*) in the figures.

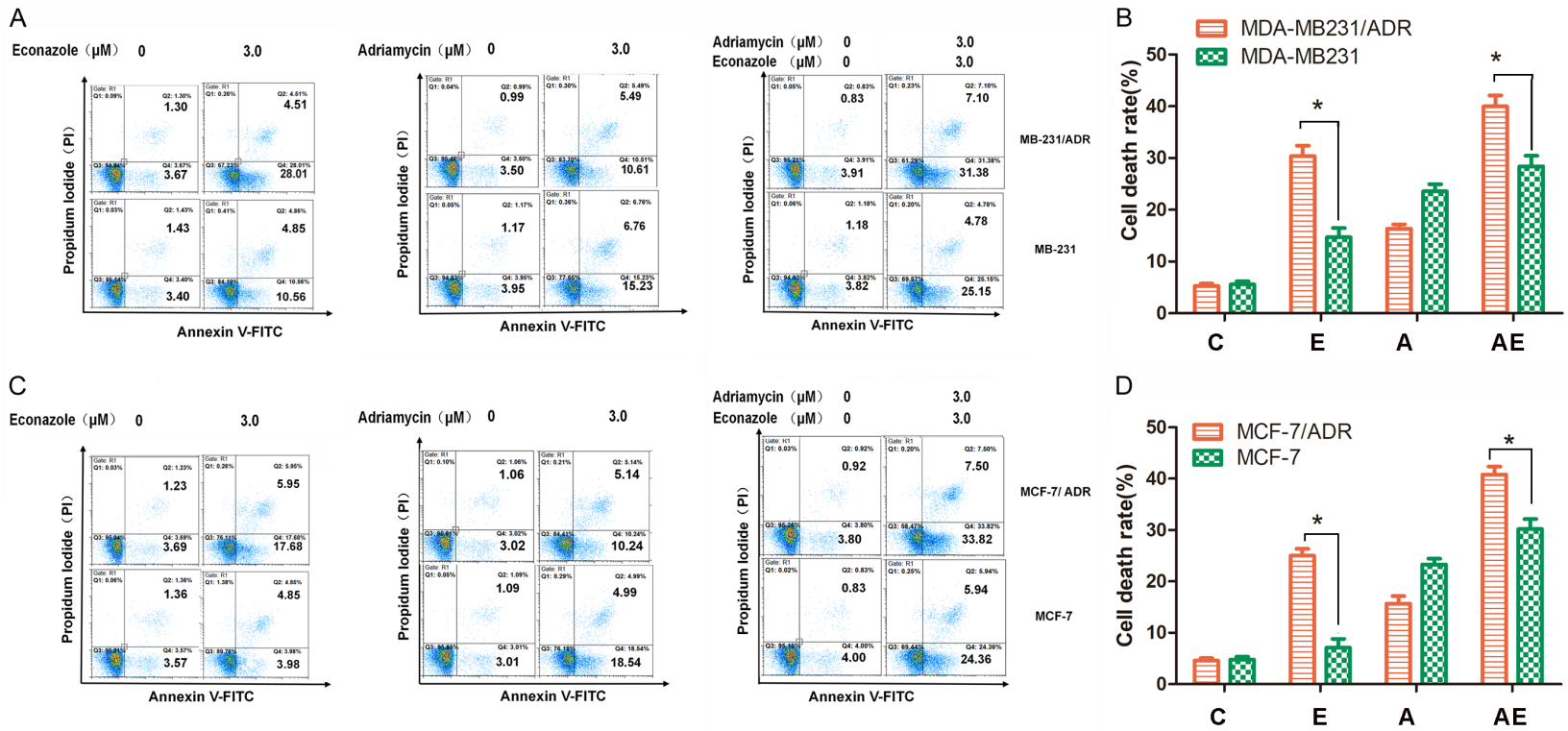


**Figure 2.** Econazole and Adriamycin induced apoptosis in ADR and sensitive breast cancer cell lines. A. Econazole (3 μM), Adriamycin (3 μM), and the combination of Econazole (3 μM) with Adriamycin (3 μM) treatment (24 hours) induced apoptosis in MDA-MB-231/ADR and MDA-MB-231 breast cancer cells. Apoptosis was measured by Annexin V/PI staining and flow cytometry (P<0.05). B. The quantitative results of A. C. Econazole (3 μM), Adriamycin (3 μM), and the combination of Econazole (3 μM) with Adriamycin (3 μM) treatment (24 hours) induced apoptosis in MCF-7/ADR and MCF-7 breast cancer cells. Apoptosis was measured by Annexin V/PI staining and flow cytometry (P<0.05). D. The quantitative results of C.

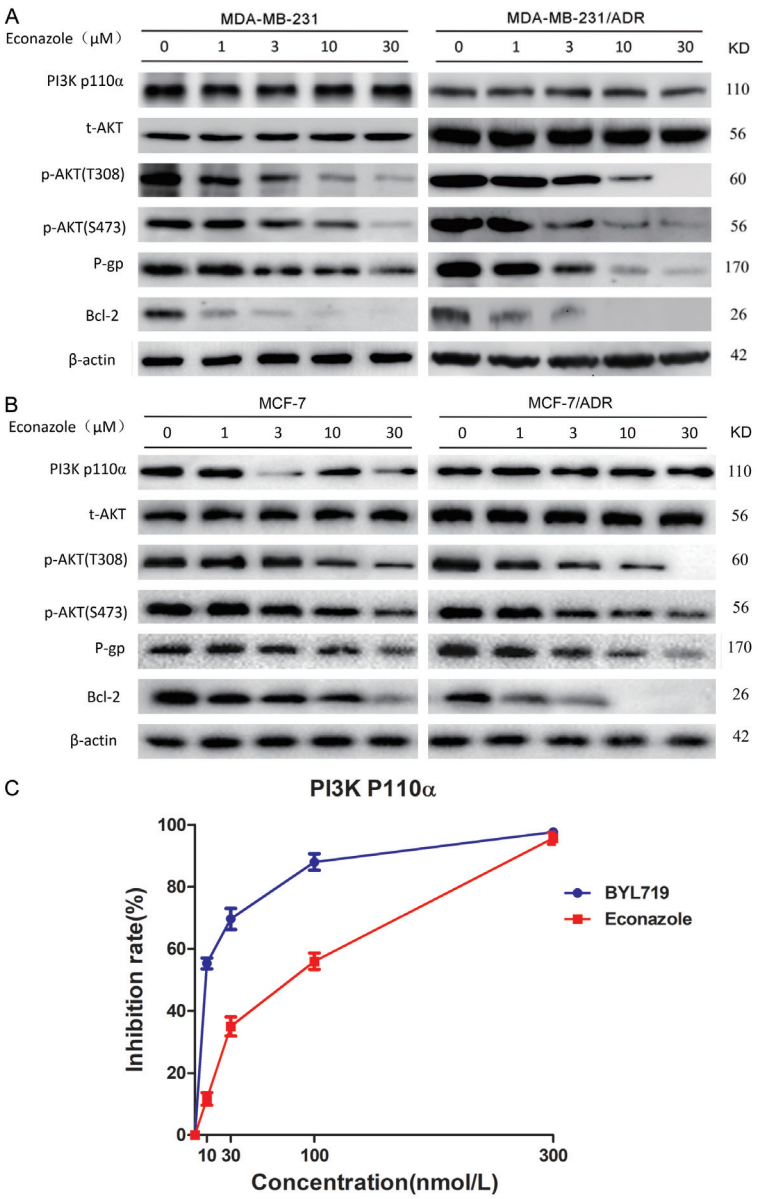


**Figure 3.** Econazole inhibited the PI3K/AKT pathway in ADR and sensitive breast cancer cell lines. A. The expression levels of p-AKT (S473 and T308), P-gp, and Bcl-2 were decreased by Econazole in MDA-MB-231/ADR and MDA-MB-231 cells in a dose-dependent manner. The cells were treated with increasing concentrations of Econazole (0, 1, 3, 10, and 30 μM) for 24 hours. Econazole had no effect on PI3K p110α or t-AKT. β-actin was used as a loading control. B. The expression levels of p-AKT (S473 and T308), P-gp, and Bcl-2 were decreased by Econazole in MCF-7/ADR and MCF-7 cells in a dose-dependent manner. The cells were treated with increasing concentrations of Econazole (0, 1, 3, 10, and 30 μM) for 24 hours. Econazole had no effect on PI3K p110α or t-AKT. β-actin was used as a loading control. C. Econazole and BYL719 significantly inhibited PI3Kα kinase activity in a dose dependent manner ($IC_{50}$: 79.29±6.97 nM and 8.68±2.13 nM).

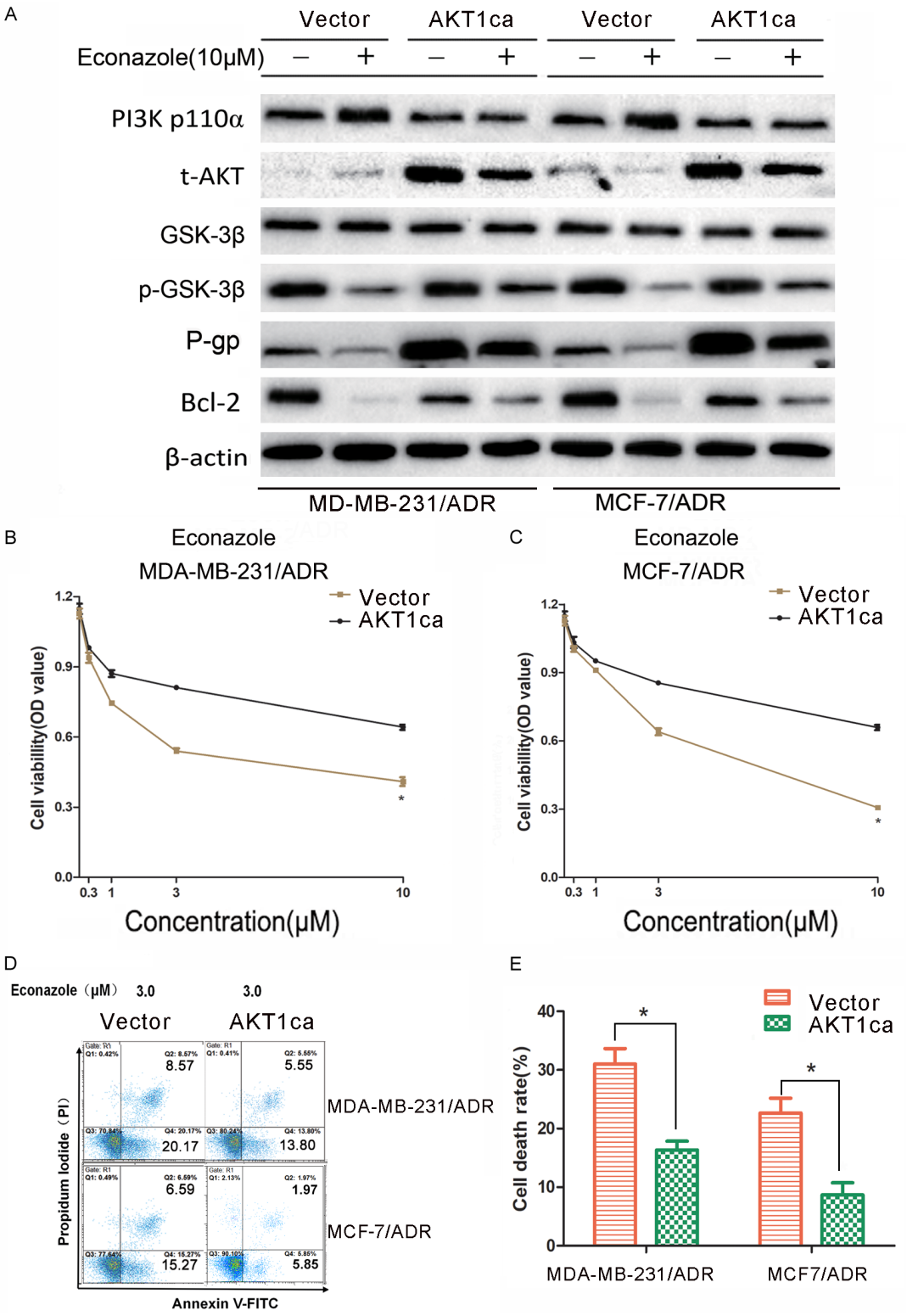


**Figure 4.** The overexpression of AKT1ca gene decreased Econazole-induced apoptosis in MDR breast cancer cell lines. A. AKT1ca overexpression decreased Econazole-induced decreases of P-gp and Bcl-2 protein levels in two ADR breast cancer cell lines (MDA-MB-231/ADR and MCF-7/ADR), as measured by Western blotting. P-GSK3β (S9) was used as a positive control because it is phosphorylated by AKT. β-actin was used as a loading control. B. AKT1ca overexpression significantly decreased the Econazole-induced loss of cell viability in MDA-MB-231/ADR, as measured by MTT assays at 72 hours ($P<0.05$). C. AKT1ca overexpression significantly decreased the Econazole-induced loss of cell viability in MCF-7/ADR, as measured by MTT assays at 72 hours ($P<0.05$). D. AKT1ca overexpression significantly decreased Econazole-induced (3 μM) apoptosis in both MDA-MB-231/ADR and MCF-7/ADR cells at 24 hours. Apoptosis was measured by Annexin V/PI staining and flow cytometry. E. The quantitative results of D ($P<0.05$).