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Erratum

The authors of the original article by Wu, et al., entitled "Osterix Decreases the Chemosensitivity of Breast Cancer Cells by Upregulating GALNT14" [Cell Physiol Biochem 2017;44(3):998-1010, DOI: 10.1159/000485400], would like to correct two accidentally and incorrectly placed pictures in two figures, which happened during manuscript preparation: For Fig. 2e in the lower panel, and for the micrograph of GALNT14 siRNA-2 of 231-OE6 cells in Fig. 4d.

The authors confirm that all of the results and conclusions of the article remain unchanged, as well as the figure legends.

The authors sincerely apologize for this mistake.

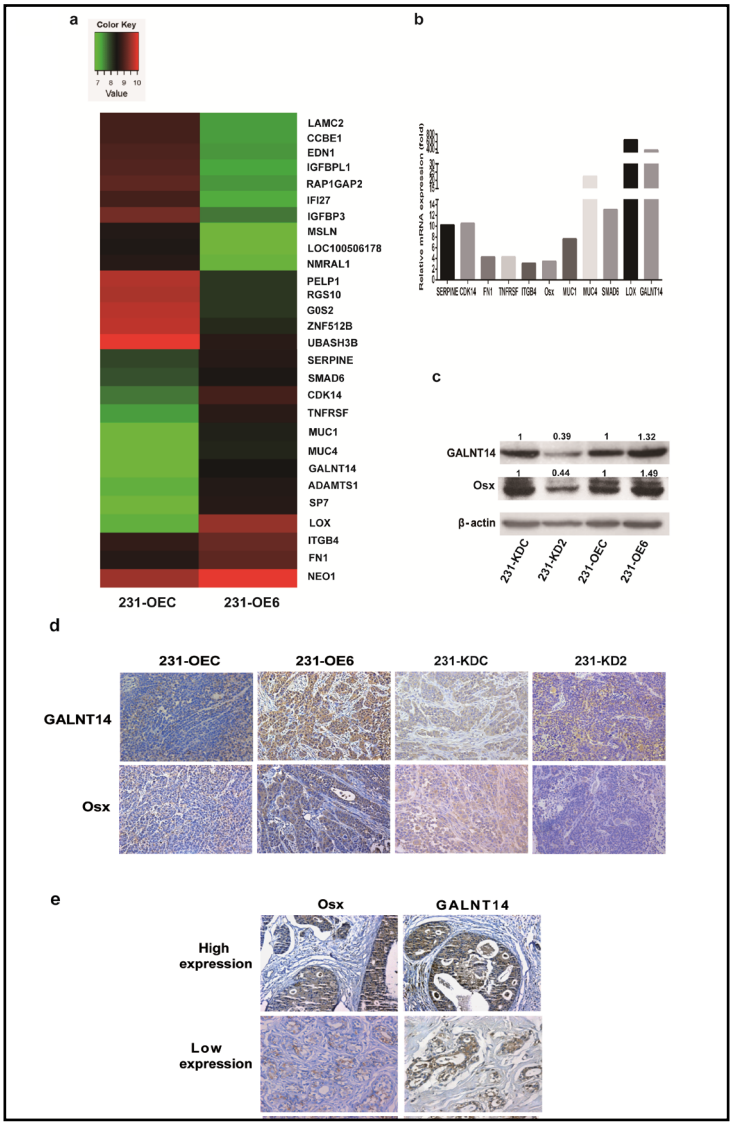

Fig. 2. GALNT14 is a target gene of Osx. (a) Heatmap representing the expression levels of differential expressed genes between 231-OE6 and in 231-OEC cells. The red and green colours indicate the up- and down-regulated genes, respectively. (b) Partially upregulated genes in 231-OE6 were demonstrated by qRT-PCR. β-Actin was used as an internal control. (c) Cell lysates from 231-KD2, 231-KDC, 231-OE6, and 231-OEC cells were immunoblotted with anti-GALNT14 and anti-Osx antibodies. β-Actin served as a loading control. The 64-kDa blot of GALNT14 and 45-kDa blot of Osx were cropped from the same gel and were run under the same experimental conditions. The fold change of KD or OE vs. control was calculated after quantification and was presented above each blot. (d) Expression of Osx and GALNT14 in breast carcinoma sections from a nude mouse model established by the injection of 231-OE6, 231-KD2 and their corresponding control cells into the mammary fat pad of female nude mice, as detected by the IHC assay. (e) Expression of Osx and GALNT14 in human breast cancer tissues, as detected by the IHC assay.

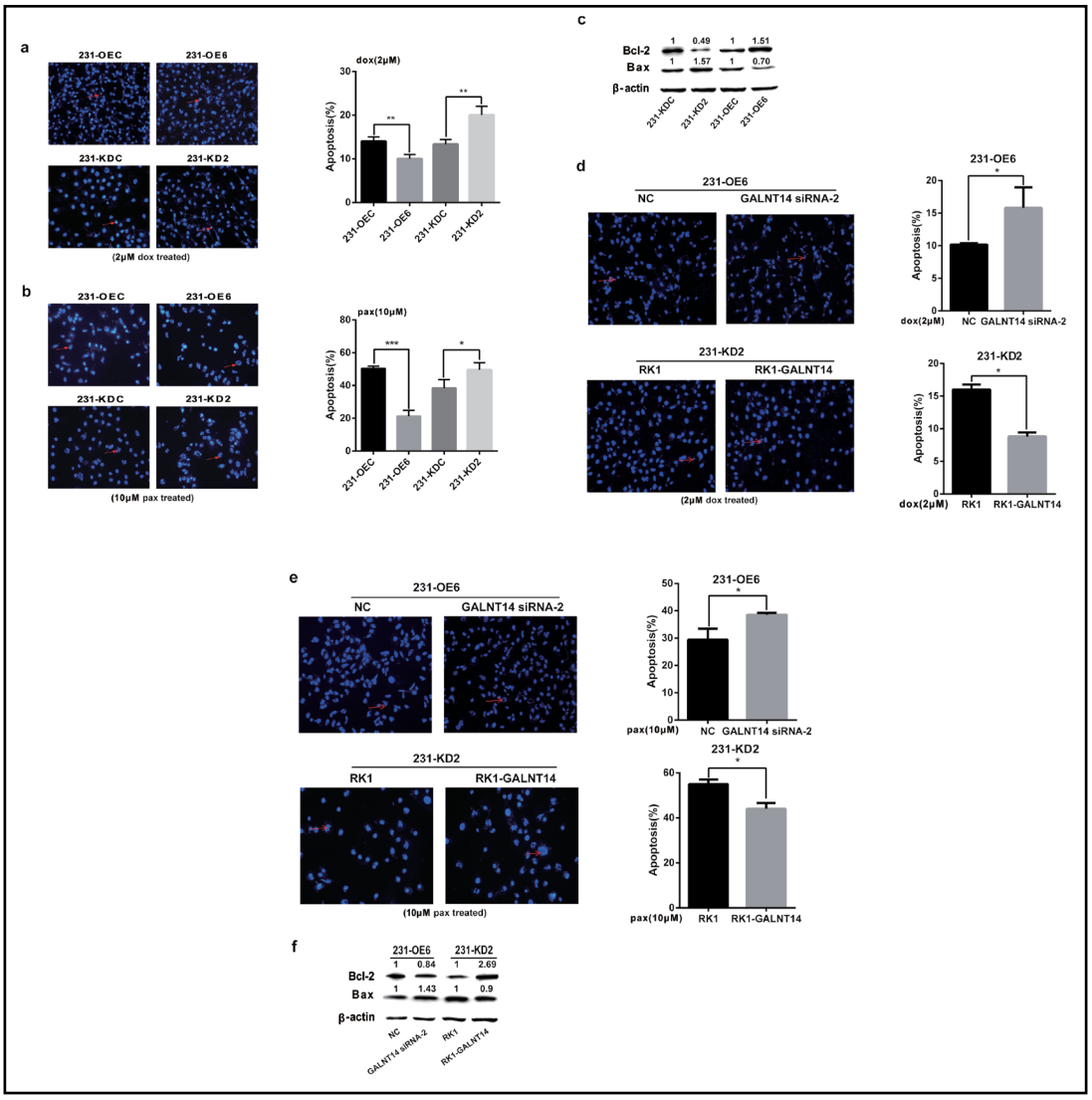

Fig. 4. Osx enhances the anti-apoptosis of breast cancer cells. (a and b) 231-OE6 and 231-KD2 cells, as well as their corresponding control cells, were treated with 2 μM dox or 10 μM pax for 36 h. Apoptotic cells were assayed with Hoechst staining and highlighted by a red arrow (left). Quantification of apoptotic cells is shown in the right panels. (c) Bcl-2 and Bax protein were detected by western blot analysis in 231-OE6 and 231-KD2 cells, as well as in their corresponding control cells. β-Actin served as an internal control. The 26-kDa blot of Bcl-2, 21-kDa blot of Bax and 42-kDa blot of β-actin were cropped from the same gel and were run under the same experimental conditions. The fold change of KD or OE vs. control was calculated after quantification and was presented above each blot. (d and e) GALNT14 siRNA-2 and NC were transiently transfected into 231-OE6 cells. The RK1-GALNT14 expression plasmid and RK1 were transiently transfected into 231-KD2 cells. Next, the transfected cells were treated with 2 μM dox or 10 μM pax for 36 h. Apoptotic cells were assayed by Hoechst staining and highlighted by a red arrow (left). Quantification of apoptotic cells are shown in right panels. The results are the means ± SD of three independent experiments. * $p<0.05$, ** $p<0.01$. (f) Bcl-2 and Bax protein were detected by western blot analysis in 231-OE6 cells transfected with GALNT14 siRNA-2 or NC, as well as in 231-KD2 cells transfected with the RK1-GALNT14 expression plasmid or RK1 vector. β-Actin served as an internal control. The 26-kDa blot of Bcl-2, 21-kDa blot of Bax and 42-kDa blot of β-actin were cropped from the same gel and run under the same experimental conditions. The fold change of KD or OE vs. control was calculated after quantification and are presented above each blot.