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ERRATUM

Erratum to: DAPK plays an important role in panobinostat-induced autophagy and commits cells to apoptosis under autophagy deficient conditions

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**Erratum to: Apoptosis (2012) 17:1300–1315
DOI: 10.1007/s10495-012-0757-7**

The original version of the article unfortunately contained a mistake. In Fig. 4 the correct image for LBH 24 h was included. In Fig. 5 the mix-up between the 24 and 48 h control images was corrected. In Fig. 6b the correct image for GAPDH was included. This mistake had no influence on the scientific conclusion neither of the figure nor of the paper. The correct versions of Figs. 4, 5, and 6 are given below.

There was a mislabeling between the house keeping genes (HKG) ß-actin and GAPDH:

Figure 1: HKG for pDAPK is GAPDH

Suppl. 7B: HKG is GAPDH

The online version of the original article can be found under doi:10.1007/s10495-012-0757-7.

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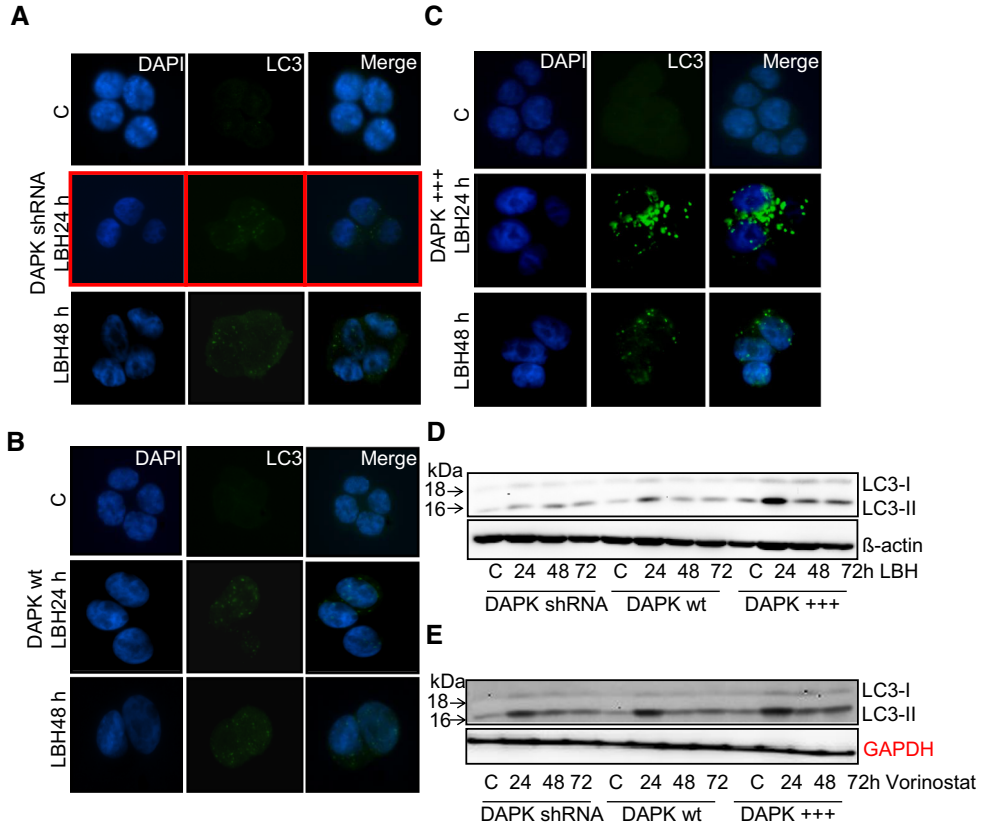

Fig. 4 DAPK-dependent autophagy induction after LBH589 treatment. **a–c** The three cell types were seeded on glass coverslips and treated with LBH589 for various time points as indicated. Endogenous LC3 aggregation was detected using immunofluorescence with anti-LC3 antibody. The appearance of punctuated signals of LC3 is a hallmark of autophagy. The picture shows one representative experiment out of two independent experiments. **d** The indicated cell types were treated with or without LBH589 for various time points. Protein levels of LC3 were detected by Western Blotting. The data are representative of two independent experiments. **e** HCT116 cells having different DAPK status were stimulated with 2 μM Vorinostat for 24, 48 and 72 h. After the treatment, whole-cell protein lysates were prepared and Western Blotting analysis was performed against LC3

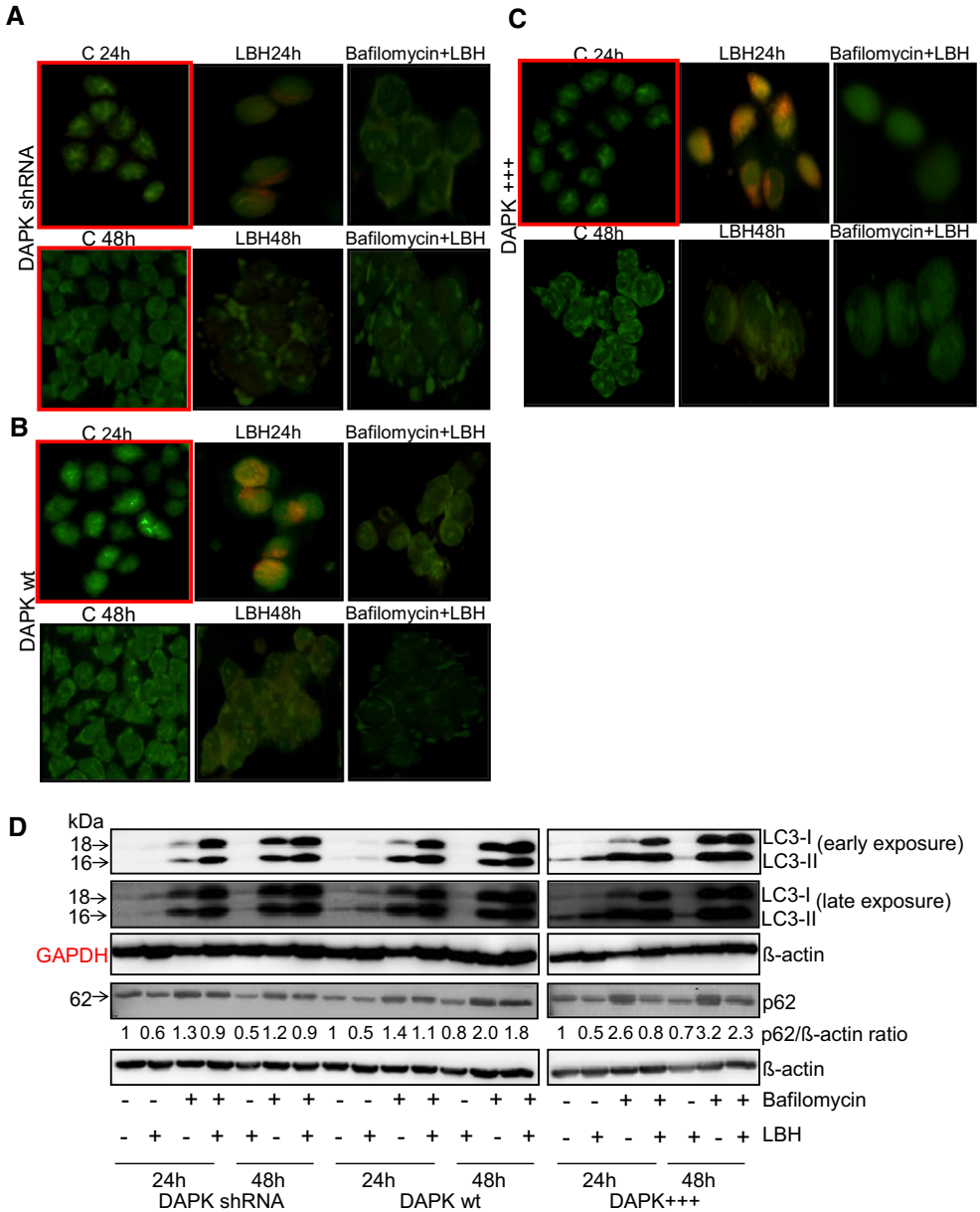

Fig. 5 LBH589 induces formation of acidic autophagic vacuoles and p62 protein degradation in a DAPK-dependent manner. **a–c** The three cell types were treated with LBH589 for 24 or 48 h in the presence or absence of autophagic inhibitor bafilomycin A1. The formation of acidic vesicular organelles seen by acridine orange staining was observed using inverted fluorescence microscope. **d** Cells were prestimulated with bafilomycin A1 for 1 h and then stimulated with LBH589 for 24 and 48 h. Western Blotting was performed against LC3 and p62 protein. The band intensities were quantified by densitometry analysis. In each cell line the control was adjusted to one after normalization to β-actin. Bafilomycin A1 increased the accumulation of autophagosomes and p62 protein. All data are representative of two independent experiments

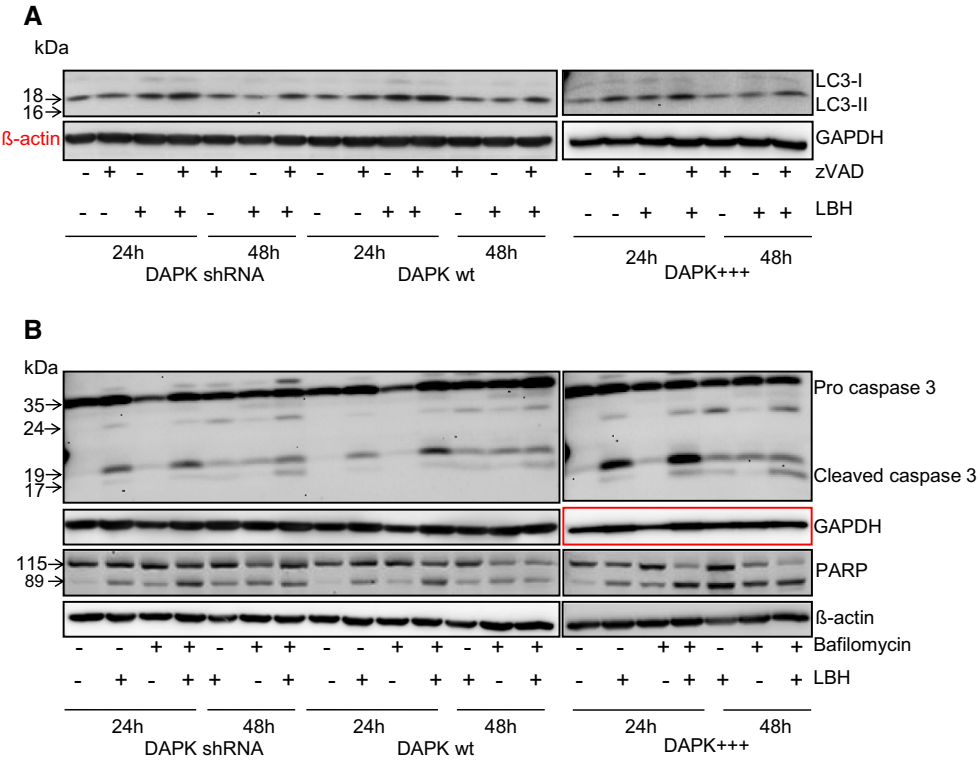

Fig. 6 Crosstalk between autophagy and apoptosis. The three cell types were treated with LBH589 in the presence or absence of either zVAD or bafilomycin A1 for various time points as indicated. **a** The cross inhibition and/or activation were analysed by Western Blotting against LC3, **b** Caspase 3 and PARP. The data are representative of two independent experiments