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CORRECTION

Correction to: Licochalcone A induces apoptosis through endoplasmic reticulum stress via a phospholipase Cγ1-, Ca^{2+}-, and reactive oxygen species-dependent pathway in HepG2 human hepatocellular carcinoma cells

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Correction to: Apoptosis (2014) 19:682–697
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The original version of this article contained mistakes in figures. The western blot data for pro-caspase-3 and cleaved caspase-3 (Fig. 1d), β-actin (Fig. 1d), PLCγ1 (Fig. 5d), and eIF2α (Fig. 7d) are incorrect. The corrected Figs. 1d, 5d, and 7d are shown below. The corrections do not influence either the validity of the published data or the conclusion described in the article. The corrected Figs. 1d, 5d, and 7d are shown below.

The original article can be found online at https://doi.org/10.1007/s10495-013-0955-y.

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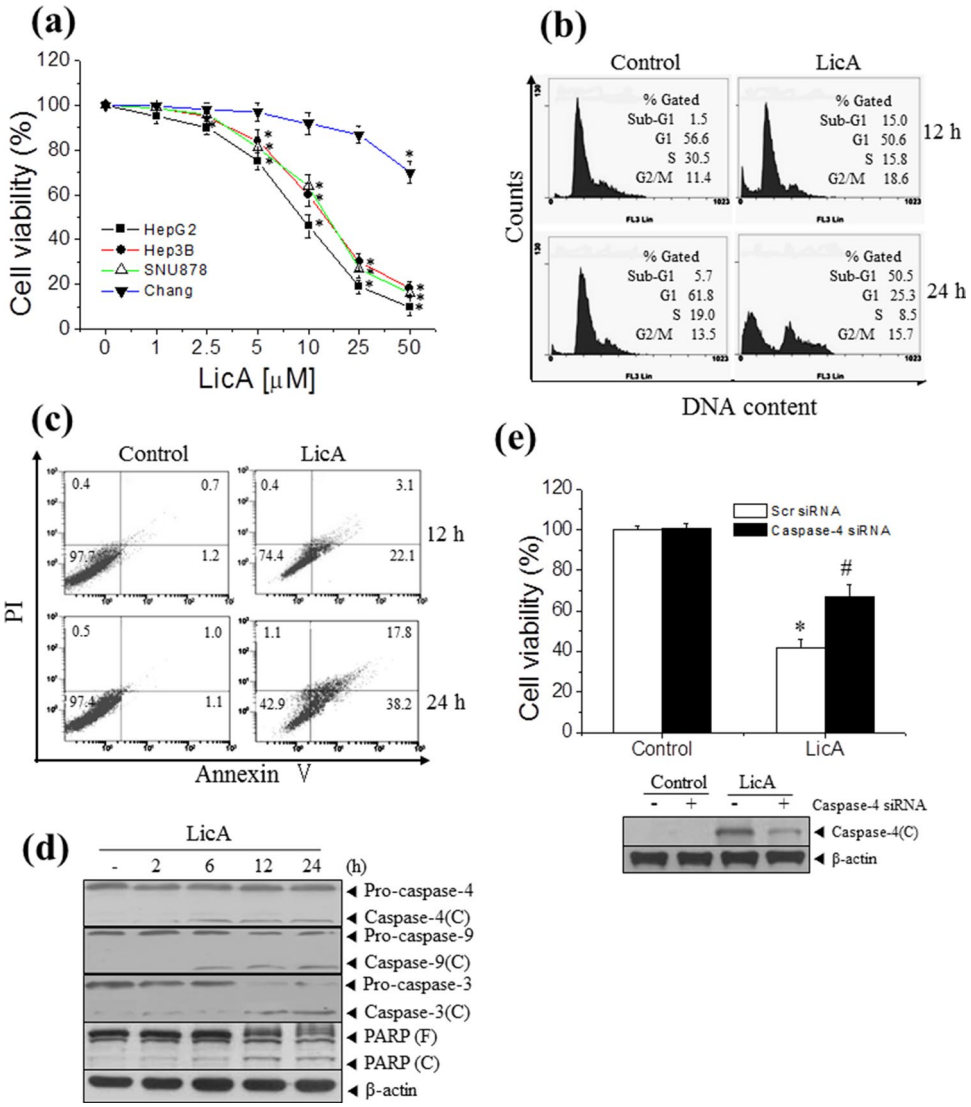

Fig. 1 Effects of LicA on apoptosis and caspase cleavage in HepG2 cells. **a** HepG2, Hep3B, SNU878 HCC cells, and Chang liver cells were treated with vehicle (DMSO) or various concentrations (1–50 μM) of LicA for 24 h. Cell viability was determined by the MTT assay and the percent viabilities are plotted as the mean ± standard deviation of at least three experiments. $^{*}P < 0.01$ compared with vehicle-treated control cells. **b, c** Cells were treated with vehicle (DMSO, Control) or 10 μM LicA for 12 h or 24 h. LicA-treated cells were evaluated by flow cytometry after staining with PI (**b**) or annexin V-FITC and PI (**c**). $n = 3$ for each experimental group. **d** HepG2 cells were treated with 10 μM LicA for the indicated times (2–24 h). The cells were lysed, and total cell extracts were resolved by SDS-PAGE. The protein levels were detected by western blot analysis using antibodies against the pro- and cleaved(C) form of caspase-4, caspase-9, and caspase-3, and against the full length PARP(F), cleaved PARP(C) product, and β-actin. **e** HepG2 cells were transfected with control scrambled (Scr) siRNA and caspase-4 siRNA for 24 h and then treated with LicA for 24 h. Cell viability was determined by MTT assay and the percent viabilities are plotted as the mean ± standard deviation of at least three experiments. $^{*}P < 0.01$ compared with vehicle- or Scr siRNA-treated control cells. $^{\#}P < 0.01$ compared with Scr siRNA-transfected and LicA-treated cells

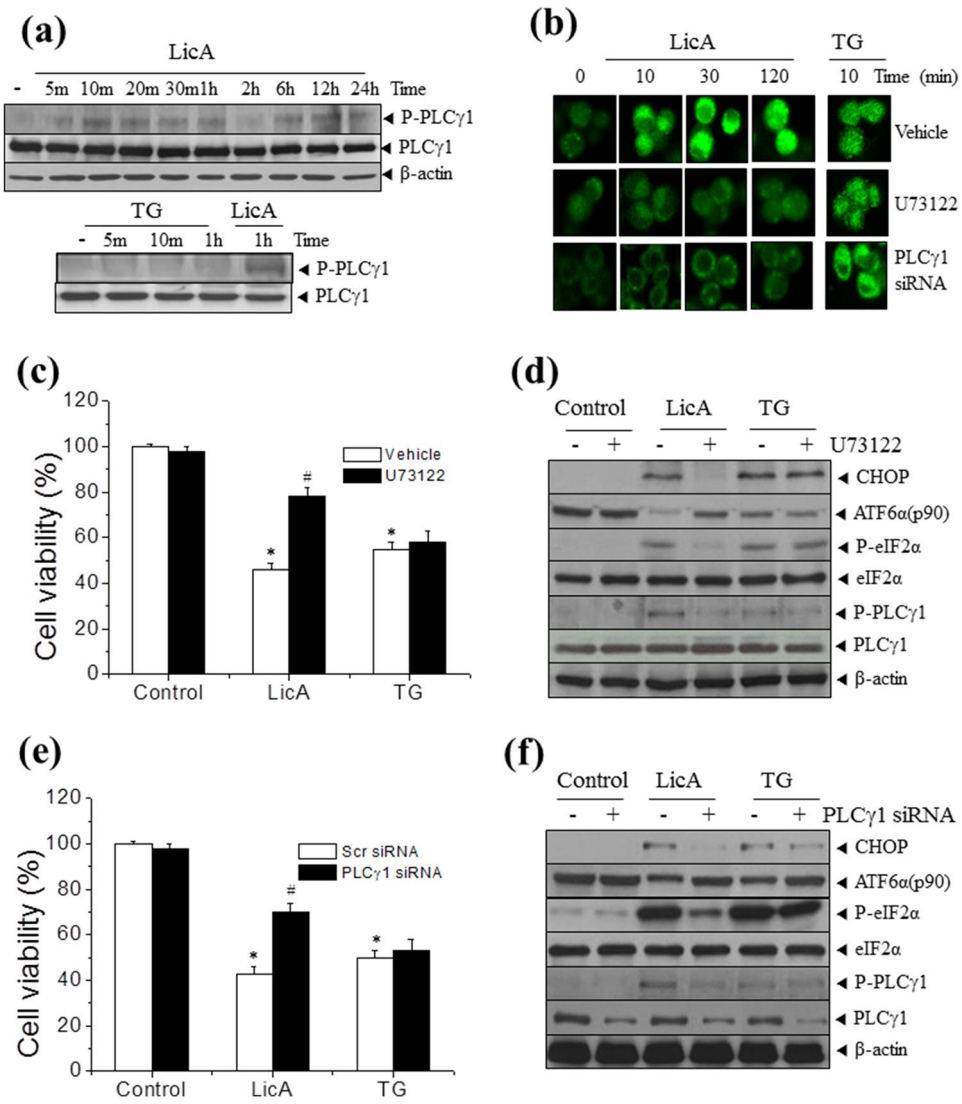

Fig. 5 Involvement of PLCγ1 in LicA-induced cell death and ER stress in HepG2 cells. **a** HepG2 cells were treated with 10 μM LicA or 5 μM TG for the indicated times. Cell lysates were resolved by SDS-PAGE and analyzed by western blot using antibodies against P-PLCγ1, PLCγ1, and β-actin. **b** HepG2 cells were pretreated with 10 μM U73122 or transfected with PLCγ1siRNA, then treated with LicA or TG for the indicated times. The cells were then treated with 5 μM Fura-2AM, and fluorescence was detected by confocal microscopy. **c** HepG2 cells were pretreated with U73122 for 1 h. then treated with LicA or TG for 24 h. Cell viability was determined by the MTT assay and the percent viabilities are plotted as the mean ± standard deviation of at least three experiments. $^{*}P < 0.01$, compared with vehicle-treated control cells. $^{\#}P < 0.01$ compared with LicA- or TG-treated cells without U73122. **d** HepG2 cells were pretreated with U73122 for 1 h, then treated with LicA or TG for 1 h (for P-eIF2α and P-PLCγ1) or 24 h (for CHOP and ATF6α). The cell lysates were subjected to western blot analyses using antibodies against CHOP, ATF6α(p90), P-eIF2α, eIF2α, P-PLCγ1, PLCγ1, and β-actin. **e** HepG2 cells were transfected with scrambled or PLCγ1 siRNA for 24 h, then treated with LicA or TG for 24 h. Cell viability was determined by the MTT assay and the percent viabilities are plotted as the mean ± standard deviation of at least three experiments. $^{*}P < 0.01$, compared with vehicle-treated control cells. $^{\#}P < 0.01$ compared with LicA- or TG-treated cells without PLCγ1 siRNA transfection. **f** HepG2 cells were transfected with scrambled or PLCγ1 siRNA for 24 h, then treated with LicA or TG for 1 h (for P-eIF2α and P-PLCγ1) or 24 h (for CHOP and ATF6α). The cell lysates were subjected to western blot analyses using antibodies against CHOP, ATF6α(p90), P-eIF2α, eIF2α, P-PLCγ1, PLCγ1, and β-actin. $n = 3$ for each experimental group (**a**–**f**)

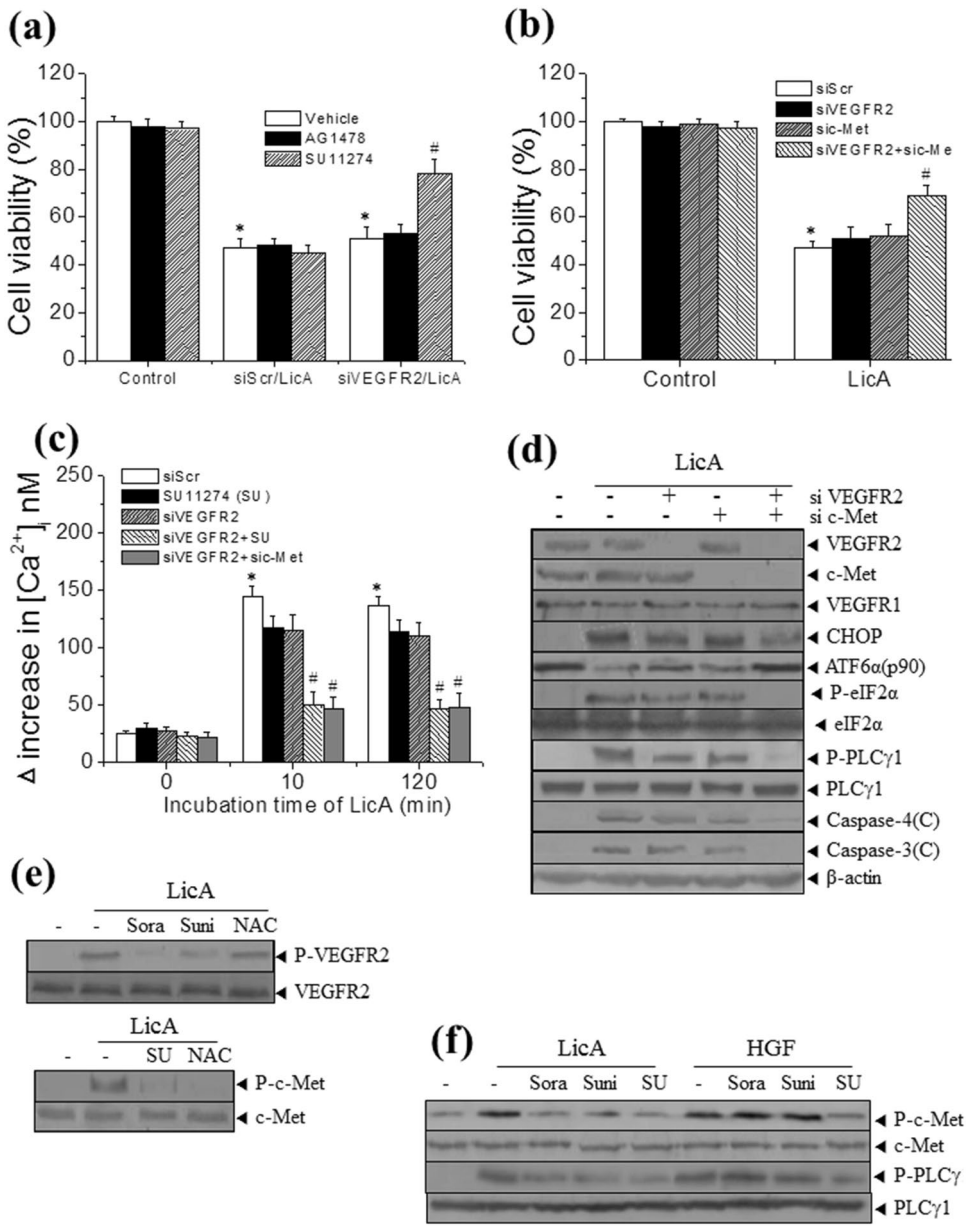

Fig. 7 Involvement of both VEGFR2 and c-Met in LicA-induced cell death, Ca^{2+}, and ER stress in HepG2 cells. **a** After transfection with control scrambled (Scr) and VEGFR2 siRNAs for 24 h, HepG2 cells were preincubated with specific inhibitors of EGFR (10 μM AG1478) or c-Met (10 μM SU11274) for 1 h, then treated with LicA for 24 h. Cell viability was determined by the MTT assay and the percent viabilities are plotted as the mean ± standard deviation of at least three experiments. $^{*}P < 0.01$ compared with vehicle-treated control cells. $^{\#}P < 0.01$ compared with LicA-treated and VEGFR2 siRNA-transfected cells without SU11274. **b** HepG2 cells were transfected with scrambled control (siScr), VEGFR2 siRNA (siVEGFR2), c-Met siRNA (sic-Met), or a mixture of VEGFR2 and c-Met siRNAs for 24 h, then treated with LicA for 24 h. Cell viability was determined by the MTT assay and the percent viabilities are plotted as the mean ± standard deviation of at least three experiments. $^{*}P < 0.01$ compared with vehicle-treated control cells. $^{\#}P < 0.01$ compared with LicA-treated cells without the mixture of VEGFR2 and c-Met siRNAs. **c** After transfection with control, VEGFR2 siRNA, c-Met siRNA, or the mixture of VEGFR2 and c-Met siRNAs for 24 h, HepG2 cells were loaded with Fura-2AM for 30 min and incubated in the presence or absence of SU11274 for 1 h, then treated with LicA for 10 or 120 min. Fluorescence was monitored at 37 °C using a fluorescence plate reader. **d** After transfection with control, VEGFR2 siRNA, c-Met siRNA, or the mixture of VEGFR2 and c-Met siRNAs for 24 h, HepG2 cells were treated with LicA for 1 h or 24 h and the cell lysates were analyzed by western blot using antibodies against CHOP, VEGFR2, c-Met, VEGFR1, ATF6α(p90), P-eIF2α, eIF2α, P-PLCγ1, PLCγ1, caspase-4 (C), caspase-3 (C), and β-actin. **e** HepG2 cells were pretreated with 0.1 μM sorafenib (Sora), 1 μM sunitinib (Suni), 5 mM NAC, or 10 μM SU11274 (SU) for 1 h, then treated with LicA for 1 h. The cell lysates were analyzed by western blot using antibodies against P-VEGFR2 (Tyr1175), P-c-Met (Tyr1234/Tyr1235), VEGFR2, or c-Met. **f** HepG2 cells were preincubated with Sora, Suni, or SU for 1 h, then treated with LicA or 20 ng/ml HGF for 1 h. The cell lysates were analyzed by western blot using antibodies against P-c-Met, c-Met, P-PLCγ1, or PLCγ1