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Corrigendum to: Thapsigargin triggers cardiac contractile dysfunction via NADPH oxidase-mediated mitochondrial dysfunction: Role of Akt dephosphorylation:

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The authors regret to report errors in Fig. 3 and Fig. 5 as reported. During the assembly of representative gel bands in Fig. 3, non-continuous gel bands were used without proper acknowledgement of the non-continuous nature. In Fig. 5, representative DHE images were wrongly copied and pasted in 3 panels. All Western blotting and DHE staining data were re-analysed for accuracy and the summary graphs presented were minimally changed in values which did not affect the original conclusion for these figures. The authors would like to apologise for any inconvenience caused.

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Fig. 3. Effect of ER stress on ER protein markers, protein/mitochondrial damage, level of pro-apoptotic protein and cell survival

ER stress was induced with *in vivo* thapsigargin (3 mg/kg, i.p. for 48 hrs) prior to assessment of ER stress, protein carbonyl formation, cell survival, mitochondrial aconitase activity and the pro-apoptotic protein BAD in WT and MyAkt mouse hearts. A: *Gadd153* expression; B: GRP78 expression; C: phosphor-eIF2α (peIF2α) expression; D: Protein carbonyl levels; E: Cell survival using MTT assay; F: Aconitase activity. G: BAD expression; H: pBAD expression; and I: pBAD-to-BAD ratio. Insets: Representative gel blots depicting *Gadd153*, GRP78, peIF2α, BAD and pBAD using specific antibodies (β-actin was used as the loading control); Mean ± SEM, n = 5–7 hearts per group, * p < 0.05 vs. WT group, # p < 0.05 vs. WT-Thapsigargin (TG) group.

Fig. 5. Effect of ER stress on superoxide (O_2^-) production and NADPH oxidase
 ER stress was induced with *in vivo* thapsigargin challenge (3 mg/kg, i.p., 48 hrs) before O_2^- production and p47^{phox} subunit of NADPH oxidase were evaluated by DHE fluorescence and western blot, respectively, in myocardium from adult WT and MyAkt mice. A–D: DHE fluorescence images depicting O_2^- fluorescence intensity from WT (A), WT-TG (B), MyAkt (C) and MyAkt-TG (D) groups; E: Quantified data displaying O_2^- fluorescent intensity from 9–11 fields; and F: p47^{phox} expression; Inset: Representative gel blots depicting p47^{phox} and β -actin (loading control) using specific antibodies; Mean $\pm$ SEM, n = 5–7 mice per group, * p < 0.05 vs. WT group; # p < 0.05 vs. WT-Thapsigargin (TG) group.