Correction to: ALDH2 protects against high fat diet-induced obesity cardiomyopathy and defective autophagy: role of CaM kinase II, histone H3K9 methyltransferase SUV39H, Sirt1, and PGC-1 α deacetylation

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The authors found a critical mistake in the assembly of Fig. 2; in Fig. 2A the right two images were erroneously

duplicated. The authors have re-analysed all the data, checked for accuracy and provided the updated Fig. 2 here. Nothing is affected with regards to data summary and conclusion.

Fig. 2 Cardiac fibrosis, hypertrophy, intracellular Ca^{2+} properties, and superoxide levels in cardiomyocytes from WT and ALDH2 mice fed low fat (LF) or high fat (HF) diet for 5 months. **a** Representative photographs of Masson trichrome staining of WT and ALDH2 mice fed LF or HF diet. **b** Quantitative analysis of interstitial fibrosis using measurements of ~100 cardiomyocytes from five mice per group. **c** Representative photographs of Lectin staining of transverse sections of left ventricular myocardium ($\times 400$) from WT and ALDH2 mice fed

LF or HF diet. **d** Quantitative analysis of cardiomyocyte cross-sectional (transverse) area using measurements of ~100 cardiomyocytes from five mice per group. **e** Electrically stimulated rise in FFI (ΔFFI). **f** Intracellular Ca^{2+} decay rate; and **g** superoxide levels using DHE fluorescence intensity. Mean $\pm$ SEM, $n = 8-11$ mice per group for Masson trichrome staining; $n = 12-16$ mice for lectin staining and $n = 60-75$ cells from three mice per group for fura-2. * $p < 0.05$ vs. WT-LF group, # $p < 0.05$ vs. WT-HF group