

Correction

Correction: The role of the Niemann-Pick disease, type C1 protein in adipocyte insulin action



The *PLOS ONE* Staff

In Figure 2C, the labels are missing. Please see the correct, complete Figure 2 here.

Citation: The *PLOS ONE* Staff (2014) Correction: The role of the Niemann-Pick disease, type C1 protein in adipocyte insulin action. *PLoS ONE* 9(7): e103797. doi:10.1371/journal.pone.0103797

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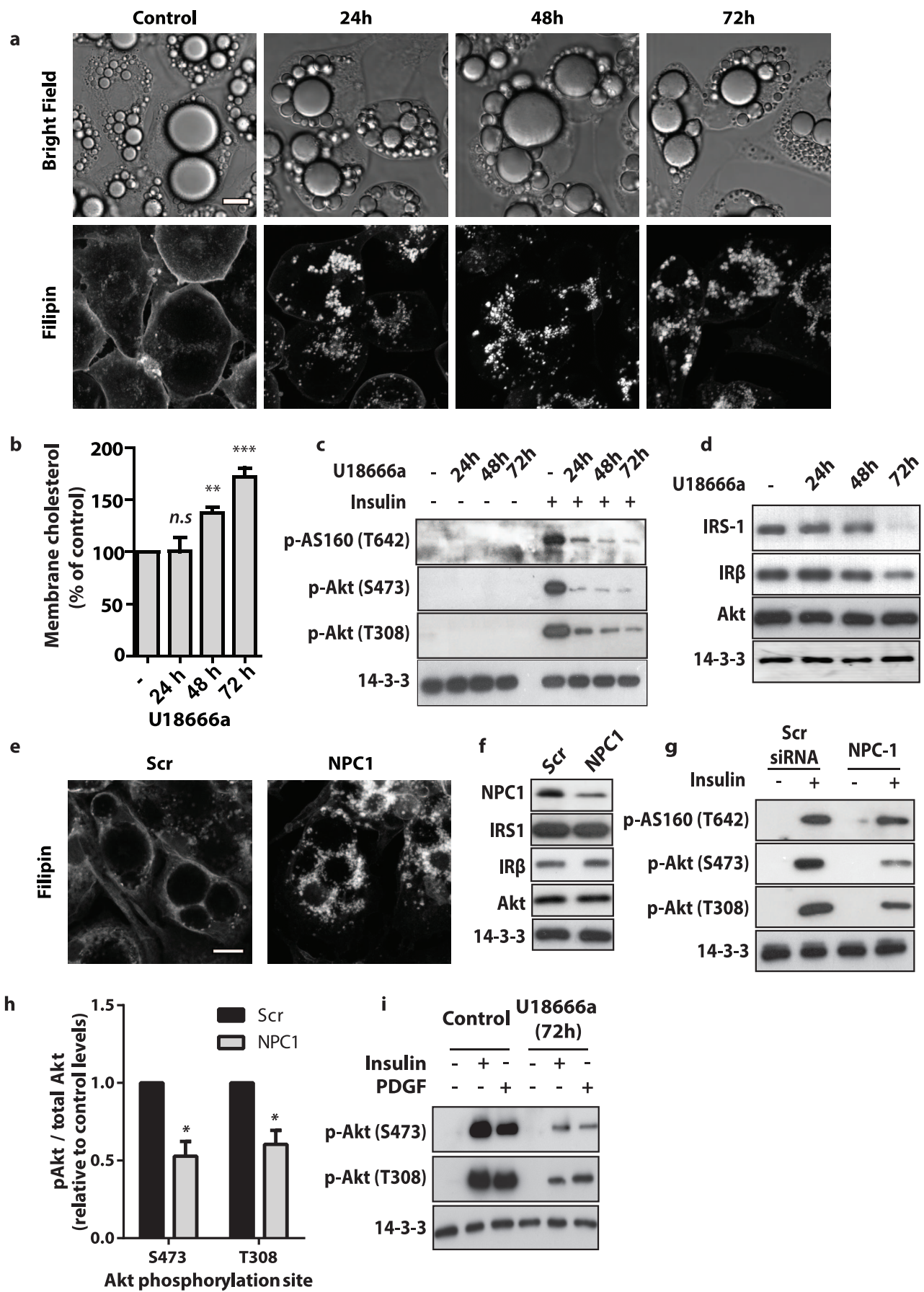


Figure 2. Pharmacological and genetic inhibition of NPC1 reduced insulin signalling in 3T3-L1 adipocytes. NPC1 was inhibited in 3T3-L1 adipocytes through treatment with U18666a or through siRNA-mediated knockdown of NPC1. (a) Subcellular localisation of cholesterol in 3T3-L1 adipocytes after treatment with U18666a for indicated times was visualised by Filipin staining (scale bar = 10 μ m), bottom panel. Corresponding bright field images are provided (top panel). Images are representative of $n \geq 300$ cells viewed per condition over 3 experiments. (b) Total membrane cholesterol levels within the total membrane fraction. Data are mean $\pm$ S.E.M, $n = 3$ independent experiments, one-way ANOVA. (c) Insulin signalling was assessed by immunoblotting total cell lysates of unstimulated and insulin-stimulated (100 nM, 20 min) 3T3-L1 adipocytes for phospho-T308 Akt, phospho-S473 Akt, phospho-T642 AS160 and 14-3-3. (d) Total protein expression of key proteins in the insulin pathway determined by immunoblotting for IRS-1, IR β and Akt. (e) Subcellular localisation of cholesterol after treatment with scrambled or NPC1 siRNA was visualised by Filipin staining (scale bar = 10 μ m). Images are representative of $n \geq 300$ cells viewed per condition over 3 experiments. (f) The extent of siRNA mediated knockdown of NPC1 and the effect of knockdown on expression of proximal insulin signalling components was measured by probing for NPC1, IRS-1, IR β and Akt. (g) The effect of NPC1 knock-down on insulin signalling was assessed by immunoblotting with phospho-T308 Akt, phospho-S473 Akt, phospho-T642 AS160 and 14-3-3 antibodies. (h) The levels of insulin-stimulated pS473 and pT308 of Akt in cells NPC1 knockdown cells was quantified relative to control cells. Data are mean $\pm$ S.E.M, $n = 3$ independent experiments, two-sample t-test. (i) 3T3-L1 adipocytes overexpressing PDGF receptor were treated with U18666a for 72 hr prior to assessment of insulin and PDGF-mediated (20 ng/mL, 20 min) signalling. Signalling was assessed by immunoblotting with phospho-T308 Akt, phospho-S473 Akt and 14-3-3 antibodies. All data are representative of at least $n = 3$ independent experiments, significance calculated compared to control cells, n.s = non-significant, *, = $p < 0.05$ ** = $p < 0.01$ *** = $p < 0.001$, statistical tests as indicated.

doi:10.1371/journal.pone.0095598.g002

Reference

1. Fletcher R, Gribben C, Ma X, Burchfield JG, Thomas KC, et al. (2014) The Role of the Niemann-Pick Disease, Type C1 Protein in Adipocyte Insulin Action. PLoS ONE 9(4): e95598. doi:10.1371/journal.pone.0095598