



The authors of "Angiopoietin-like 4 (ANGPTL4) promotes intracellular degradation of lipoprotein lipase in adipocytes" (*J. Lipid Res.* 2016. 57: 1670–1683) have advised *the Journal* that the last sentence of each of the figure legends for Figs. 5, 6, 7, 8, 9, and 10 is incorrect. The descriptions within parentheses of the R and S gel bands were inadvertently swapped. The figure legends for these figures should read as follows:

Fig. 5. ANGPTL4 lowers the amount of LPL on the adipocyte cell surface. A: Western blot of cell lysates of gWAT explants from $Angptl4^{-/-}$ and wild-type mice incubated in the absence or presence of heparin (50 IU/ml) for 3 h. Western blots were probed with antibodies against LPL and HSP90 (as loading control). B: Western blot of cell lysates of adipocytes that had been differentiated from stromal vascular fractions of WAT from $Angptl4^{-/-}$ and wild-type mice and incubated in the absence or presence of heparin (10 IU/ml) for 20 min. Western blots were probed with antibodies against LPL and HSP90 (as loading control). C: Angptl4 and Lpl mRNA levels in gWAT explants from $Angptl4^{-/-}$ and wild-type mice. D: Angptl4 and Lpl mRNA levels in adipocytes that had been differentiated from stromal vascular fractions of WAT from $Angptl4^{-/-}$ and wild-type mice. EndoH-resistant LPL (complex oligosaccharides; Golgi and cell surface LPL) is indicated with R; EndoH-sensitive LPL (high-mannose oligosaccharides, ER LPL) is indicated with S.

Fig. 6. ANGPTL4 lowers LPL secretion. A: Western blot of media of gWAT explants from $Angptl\ell^{+-}$ and wild-type mice treated in the absence or presence of heparin (50 IU/ml) for 3 h. Western blots were probed with antibodies against LPL and ADIPOQ. Coomassie blue staining was used to assess loading. B: Western blot of media of adipocytes that had been differentiated from stromal vascular fractions of WAT from $Angptl\ell^{+-}$ and wild-type mice and incubated in the absence or presence of heparin (Hep.) (10 IU/ml) for 20 min. Western blots were probed with antibodies against LPL and ADIPOQ. Coomassie blue staining was used to assess loading. C: Western blot of media of adipocytes that had been differentiated from stromal vascular fractions of WAT from $Angptl\ell^{+-}$ and wild-type mice. Cells were pretreated for adipocytes that had been differentiated from stromal vascular fractions of WAT from $Angptl\ell^{+-}$ and wild-type mice. Cells were pretreated for 20 min with heparin (10 IU/ml), washed with PBS, and incubated with heparin (10 IU/ml) for the indicated times. Western blots were probed with antibodies against LPL and ADIPOQ. EndoH-resistant LPL (complex oligosaccharides; Golgi and cell surface LPL) is indicated with R; EndoH-sensitive LPL (high-mannose oligosaccharides, ER LPL) is indicated with S.

Fig. 7. ANGPTL4-mediated loss of LPL protein occurs in a postER compartment. A: Western blot of EndoH-treated cell lysates of adipocytes that had been differentiated from stromal vascular fractions of WAT from $Angptl4^{-/-}$ and wild-type mice. Cells were treated with $5 \mu g/$ ml brefeldin A for 2 h. Western blots were probed with antibodies against LPL and HSP90 (as a loading control). B: Western blot of cell lysates of adipocytes that had been differentiated from stromal vascular fractions from WAT of Angptl4^{-/-} and wild-type mice. Cells were lysed in NP-40 lysis buffer. Western blots were probed with antibodies against LPL, HSP90 (as a loading control), and H2A (as a loading control). NP40S, NP40-soluble LPL; NP40P, NP40-precipitated LPL; s.e., short exposure; l.e., long exposure. C: Western blot of EndoHtreated cell lysates of adipocytes that had been differentiated from the stromal vascular fractions of WAT from Angptl4^{-/-} and wild-type mice. Cells were treated with 10 µM monensin (Mon.) for 3 h. Western blots were probed with antibodies against LPL and HSP90 (as a loading control). D: Western blot of EndoH-treated cell lysates from adipocytes that had been differentiated from stromal vascular fractions of WAT from Angptl4^{-/-} and wild-type mice. The cells were treated with 20 µM E64D for 24 h. Western blots were probed with antibodies against LPL and HSP90 (as a loading control). E: Western blot of EndoH-treated cell lysates of adipocytes that had been differentiated from stromal vascular fractions of WAT from $Angptl4^{-/-}$ and wild-type mice. Cells were treated with 5 mM 3MA for 10 h. Western blots were probed with antibodies against LPL and HSP90 (as a loading control). F: Western blot of EndoH-treated cell lysates of adipocytes that had been differentiated from stromal vascular fractions from WAT of $Angptl 4^{-/-}$ and wild-type mice. The cells had been treated with 10 mM leupeptin (Leup.) for 10 h. Western blots were probed with antibodies against LPL and HSP90 (as a loading control). EndoH-resistant LPL (complex oligosaccharides; Golgi and cell surface LPL) is indicated with R; EndoH-sensitive LPL (high-mannose oligosaccharides, ER LPL) is indicated with S.

Fig. 8. Physiological regulation of ANGPTL4 expression affects levels of EndoH-resistant LPL in vivo. A: Western blot of gWAT lysates of $Angptl4^{-/-}$ and wild-type mice, as analyzed by SDS-PAGE and native PAGE. Western blots were probed with antibodies against LPL and HSP90. Coomassie blue staining was used to assess loading. B: Western blot of EndoH-treated and PNGase-treated gWAT lysates of $Angptl4^{-/-}$ and wild-type mice. Western blots were probed with antibodies against LPL and HSP90 (as a loading control). C: Western blot of EndoH-treated gWAT lysates prepared from ad libitum-fed, overnight-fasted, or refed $Angptl4^{-/-}$ and wild-type mice. The tissue samples were taken from an experiment described earlier (13). Western blots were probed with an antibody against LPL; Coomassie blue staining was used to assess loading. D: Western blot of gWAT lysates prepared from ad libitum fed, overnight-fasted, or refed wild-type mice. The tissue samples were taken from an experiment described earlier (13). Western blots were probed with an antibody against ANGPTL4; Coomassie blue staining was used to assess loading. E: Western blot of EndoH-treated gWAT lysates from $Angptl4^{-/-}$ and wild-type mice that were either not perfused or perfused with PBS containing heparin (50 IU/ml). Western blots were probed with antibodies against LPL and HSP90 (as a loading control). F: Western blot of EndoH-treated gWAT lysates prepared from fed and overnight-fasted $Angptl4^{-/-}$ and wild-type mice that had been given an intravenous injection of heparin (100 IU/kg). Western blots were probed with antibodies against LPL and HSP90 (as a loading control). F: Mestern blot of EndoH-treated gWAT lysates from fed and overnight-fasted $Angptl4^{-/-}$ and wild-type mice that had been given an intravenous injection of heparin (100 IU/kg). Western blots were probed with antibodies against LPL and HSP90 (as a loading control). EndoH-treated gWAT lysates **charides; Golgi and cell surface LPL**) is indicated with R; EndoH-sensitive LPL (Fig. 9. Levels of EndoH-resistant LPL are inversely related to *Angptl4* expression. A: Western blot on 0.75 μ l of plasma from *Angptl4^{-/-}* and wild-type mice. Western blots were probed with an antibody against LPL; Coomassie blue staining was used to assess loading. B: Western blot of EndoH-treated WAT lysates prepared from *Angptl4^{-/-}*, *Angptl4^{+/-}*, and wild-type mice. Western blots were probed with an antibody against LPL; Coomassie blue staining was used to assess loading. C: Western blot of EndoH-treated WAT lysates prepared from *Angptl4^{-/-}*, wild-type, and *Angptl4^{-/-}* g mice. Western blots were probed with an antibody against LPL; Coomassie blue staining was used to assess loading. D: Western blot of EndoH-treated lysates of 3T3-F442a adipocytes that had been treated with rosiglitazone (10 μ M) or DMSO control for 6 h. Western blots were probed with antibodies against LPL, ANGPTL4, and HSP90 (as a loading control). E: Western blots were probed with antibodies against LPL, ANGPTL4, and HSP90 (as a loading control) for 6 h. Western blots were probed with rosiglitazone (10 μ M) or DMSO control for 6 h. Western blots were probed with antibodies against LPL, and HSP90 (as a loading control). E: Western blots were probed with antibodies against LPL, and HSP90 (as a loading control). E: Western blots were probed with antibodies against LPL, and HSP90 (as a loading control). E: Western blots were probed with antibodies against LPL, and HSP90 (as a loading control). EndoH-resistant LPL (complex oligosaccharides; Golgi and cell surface LPL) is indicated with R; EndoH-sensitive LPL (high-mannose oligosaccharides, ER LPL) is indicated with S.

Fig. 10. ANGPTL4 regulates levels of EndoH-resistant LPL in adipose tissue, but not heart. A: Western blot of EndoH-treated BAT lysates from $Angptl \ell^{/-}$ and wild-type mice exposed to cold (4°C) or thermoneutral temperature (28°C) for 10 days. The tissue samples were taken from an experiment described earlier (14). Western blots were probed with antibodies against LPL and HSP90 (as a loading control). B: Western blot on EndoH-treated heart lysates from $Angptl \ell^{/-}$ and wild-type mice fed ad libitum or fasted overnight. Western blots were probed with antibodies against LPL and HSP90 (as a loading control). EndoH-resistant LPL (complex oligosaccharides; Golgi and cell surface LPL) is indicated with R; EndoH-sensitive LPL (high-mannose oligosaccharides, ER LPL) is indicated with S.