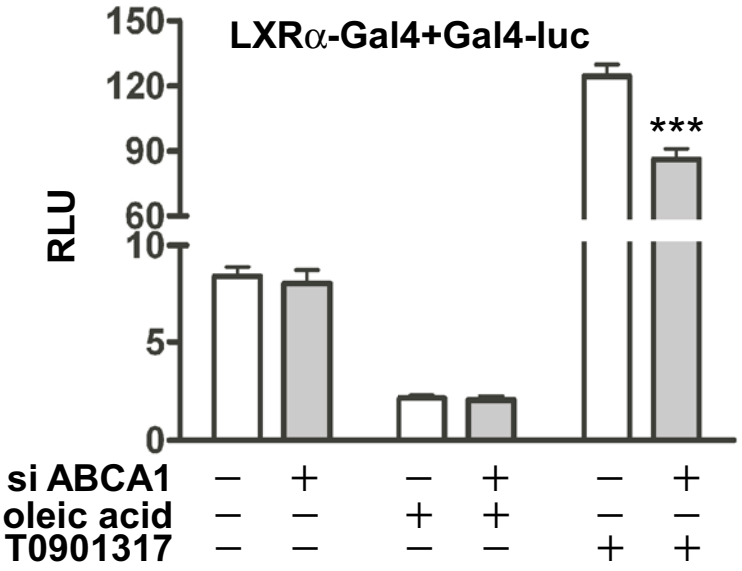
Supplemental Figure 1. ABCA1 silencing does not increase endogenous LXRα ligand binding activityin rat hepatoma cells. Control and ABCA1-silenced McA cells were transiently cotransfected with: 1) GAL4-driven luciferase reporter construct (pMH100X-TK-Luc), 2) an expression vector containing the Gal4 DNA binding domain fused to the LXR・ ligand binding domain (pCMX-Gal4-LXRα$_{LBD}$), and 3) a control vector (pRL-CMV). Cultures were stimulated with either oleate (0.8 mM) or LXR agonist (positive control, 5・ M T0901317) overnight before a dual luciferase assay was performed. Results are representative of two separate experiments and are expressed as mean ± SEM (n=4). ***, ($p<0.001$) by student's t-test.

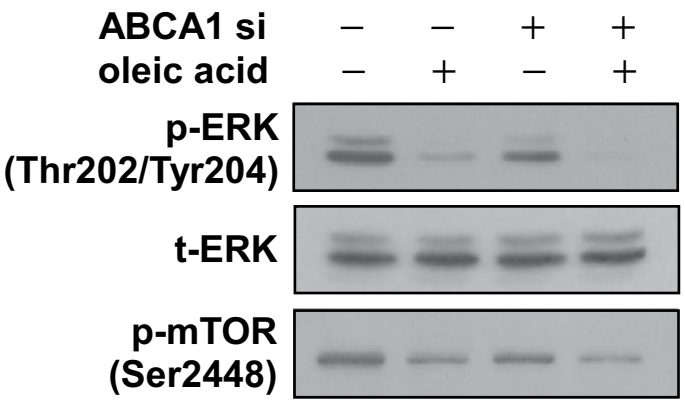
Supplemental Figure 2. ERK and mTOR activation is not responsible for elevated TG secretion in ABCA1-silenced McA cells. A. Control and ABCA1 siRNA transfected McA cells were incubated ± 0.8 mM oleate for 12 h. Cell lysates were then harvested and Western blotted for p-ERK, t-ERK and pmTOR. **B**. Control and ABCA1 siRNA transfected McA cells (25 nM for 48 h) were preincubated with 10 nM U0126 (inhibitor of ERK activation) for 1 h before addition of radiolabeling medium (5・ Ci/ml [^{3}H]-oleate + 0.8 mM oleate), followed by an additional 12 h incubation. Conditioned medium (500 µl; pooled n=3) was fractionated by FPLC and radiolabel in each fraction was determined. VLDL elution position (fractions 30-35) is denoted. **C**. Control or ABCA1 siRNA transfected McA cells were incubated with vehicle (DMSO) or 10 µM rapamycin (RAPA) for 1 h before addition of radiolabeling medium (5 µCi/ml [^{3}H]-oleate + 0.8 mM oleate). After an additional 12 h incubation, cells and medium were lipid extracted, radiolabel incorporation into TG was quantified by thin-layer chromatography, and the data (mean ± SEM, n=3) were normalized to assayed cellular protein. *, ($p<0.05$), ***, ($p<0.001$) by student's t-test.

Supplement Figure 1



Supplement Figure 2

A



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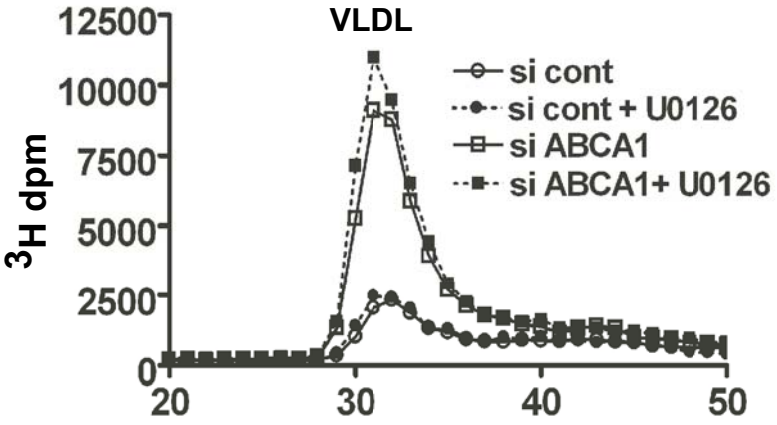

C