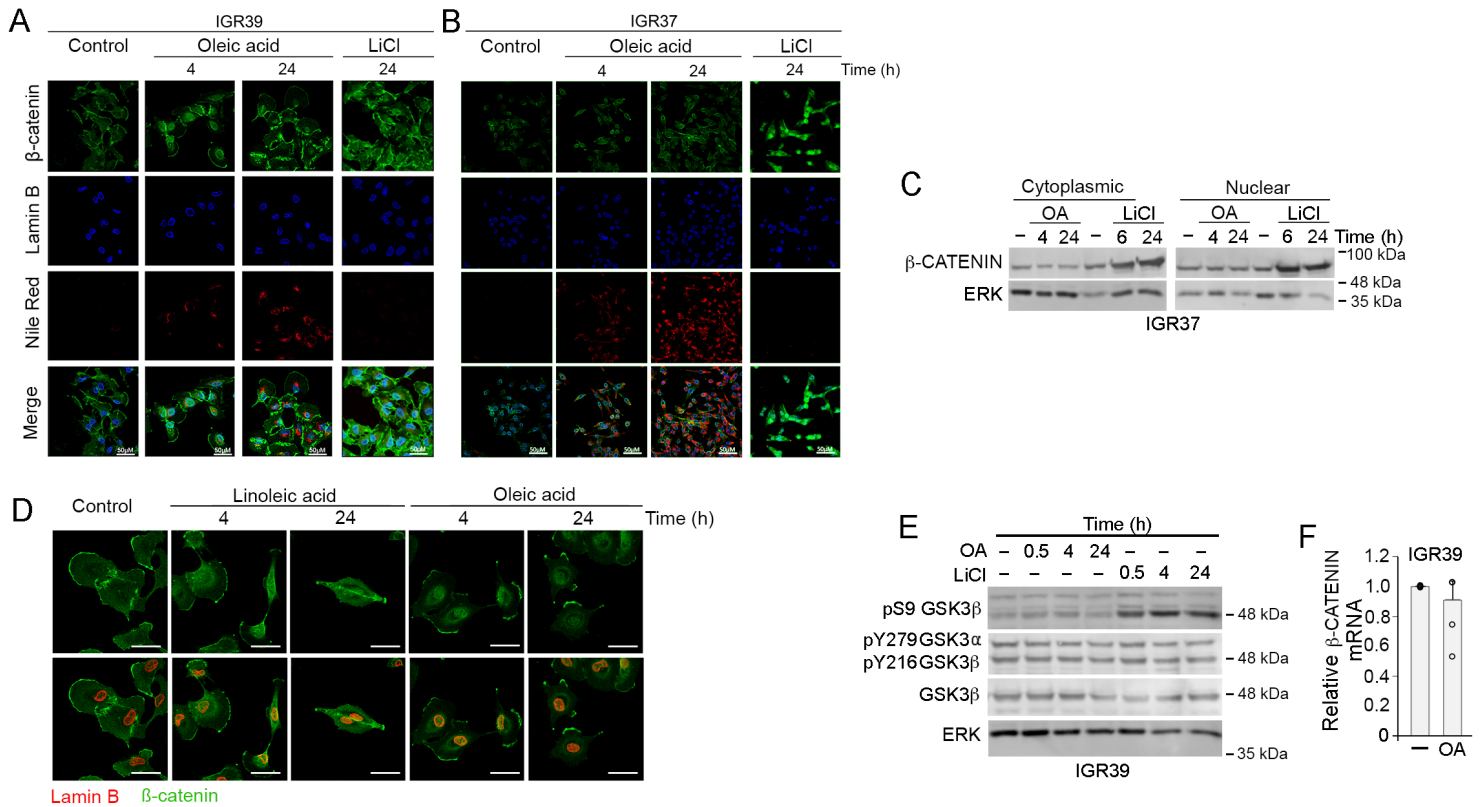
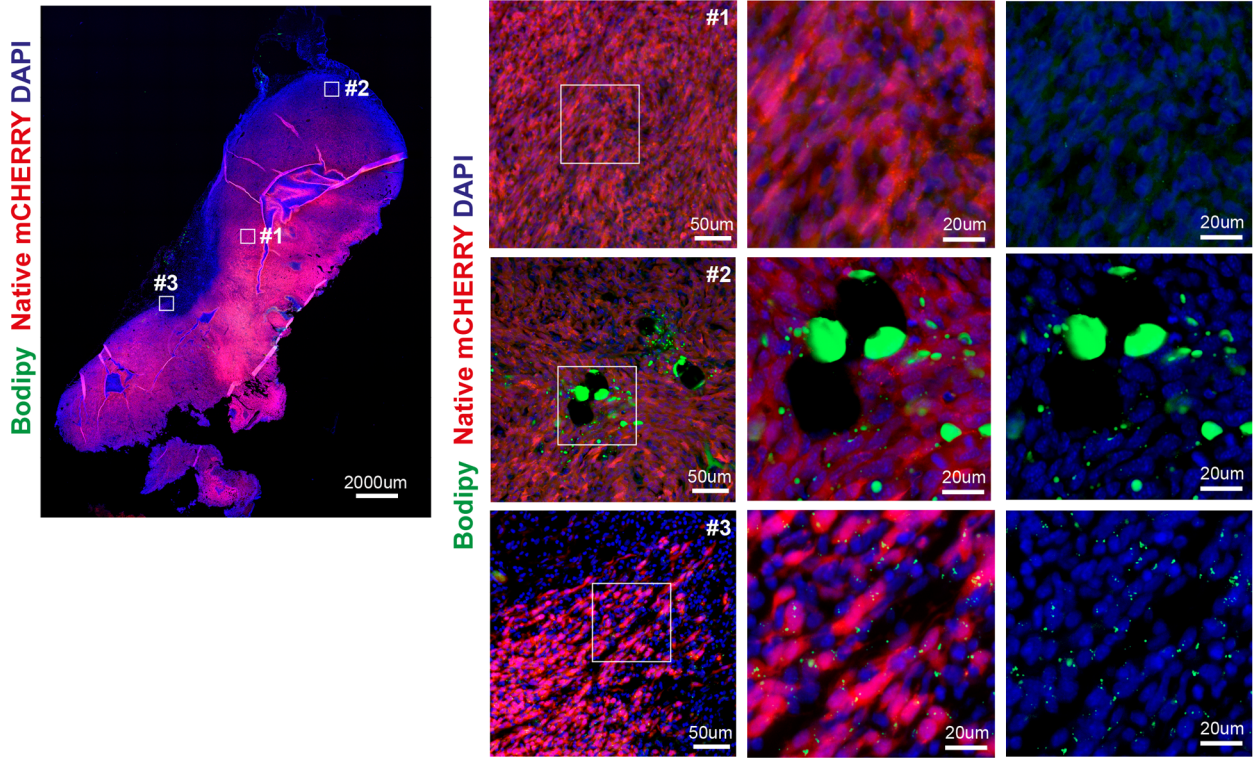


Supplemental Fig. S1. Fatty acid uptake by IGR29 and IGR37 cells. (A) Oil red O staining of IGR37 and IGR39 melanoma cells after 4 h exposure to oleic acid (OA). Scale bar = 50 μm . (B) Schematic showing pre-loading of 3T3-L1 adipocytes with BODIPY (green) before removal of free BODIPY by washing and co-culture with melanoma cells in a chamber separated by a 3 μm pore size membrane that permits passage of small molecules but not cells or cell contact. (C) BODIPY-FL uptake from pre-loaded 3T3-L1 cells by IGR37 or IGR39 cells co-cultured for 24 h using mono-cultured melanoma cells as a negative control. Both melanoma cell lines are positive, indicating their ability to promote release and uptake from the BODIPY-labelled 3T3-L1 cells.

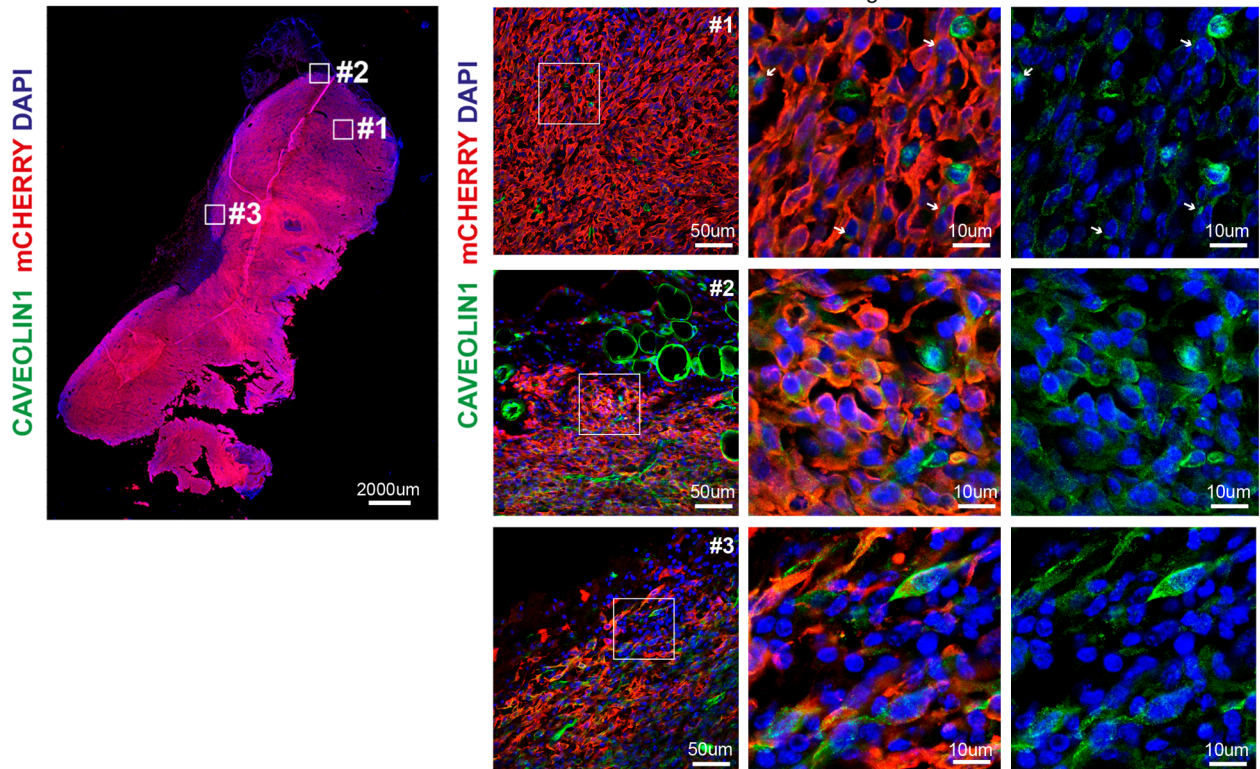


Supplemental Fig. S2. Effect of Oleic acid on β -catenin in IGR37 and IGR39 cells. (A,B) Immunofluorescence showing subcellular localization of β -catenin (green) following treatment with 100 μ M oleic acid or 20 mM LiCl. Anti-Lamin B (red) was used to highlight the nuclear periphery. Scale bars = 50 μ m. (C) Western blot of fractionated IGR37 cells following treatment with 100 μ M oleic acid or 20 mM LiCl. (D) Immunofluorescence using anti-Lamin B (red) and anti- β -catenin green after treatment of cells with 100 μ M oleic acid or linoleic acid as indicated. Scale bars = 50 μ m. (E) Western blot over time of IGR39 cells treated with 100 μ M oleic acid or 20 mM LiCl. (F) qRT PCR showing expression of β -catenin mRNA relative to GAPDH in IGR39 cells treated with 100 μ M oleic acid for 24 h. Error bars =SD. N=3.

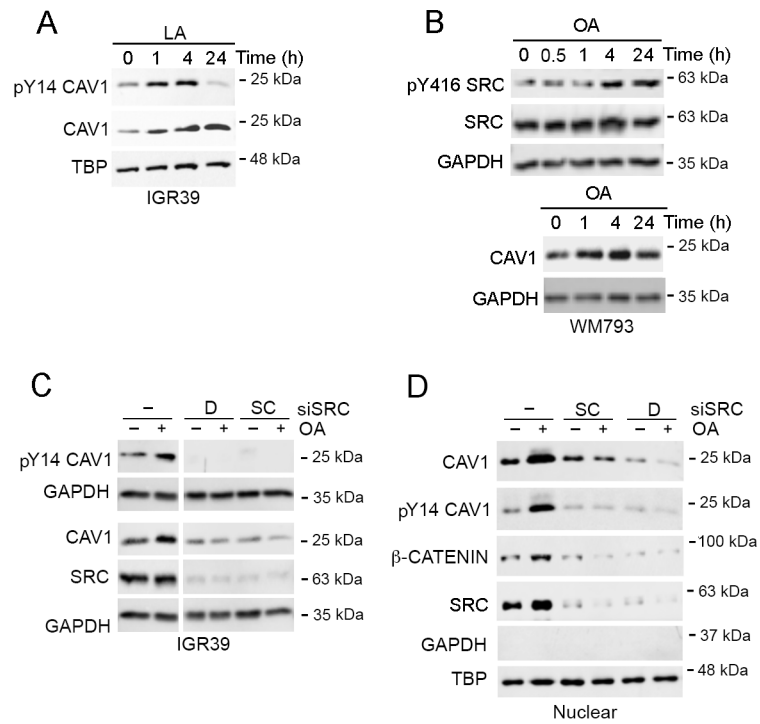
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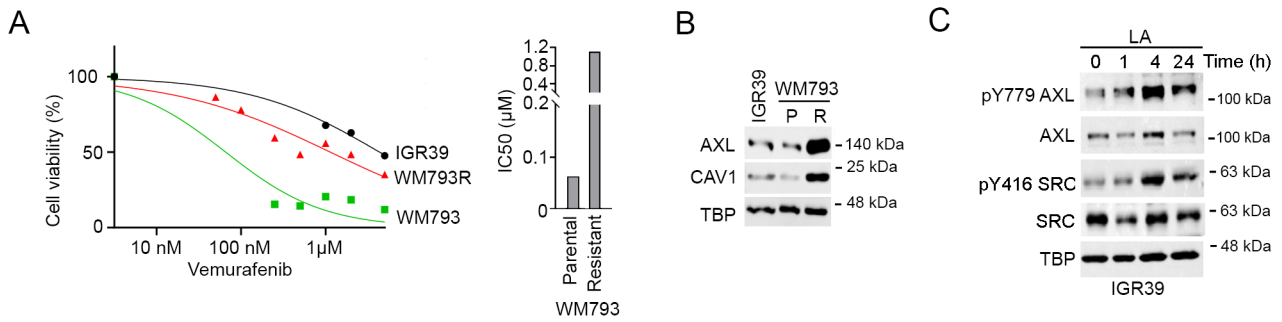
B



Supplemental Fig. S3. Elevated CAV1 expression in mouse adipocyte-proximal melanoma cells. (A, B) Left panels: Sections through a murine mCHERRY-labeled YUMM1.7 allograft melanoma tumor. Areas of interest indicated by insets corresponding to: 1, the tumor core; 2, melanoma cells in proximity to adipocytes; and 3 the invasive front. Right panels: Fluorescence of indicated regions 1-3 showing mCHERRY-positive melanoma cells (red), DAPI (blue) and either BODIPY (green) (A), or immunofluorescence using anti-Cav1 (Green)) at indicated magnifications. Scale bars for whole tumors shown in left panels = 2 mm. For regions of interest panels scale bars = 50 μ m; for middle and right panels in (A) scale bars = 20 μ m; for middle and right panels in (B) scale bars = 10 μ m.



Supplemental Fig. S4. SRC depletion blocks nuclear accumulation of CAV1 and β -catenin. (A-D) Western blots from IGR39 cells or WM793 cells as indicated, treated with either 100 mM linoleic acid (A) or 100 μ M oleic acid (B-D) and where indicated depleted of SRC using two different siRNAs (SC), or a Dharmacon pool. Note that the CAV1 and GAPDH control tracks for panel (C) are the same as those from Fig. 5F and were run on the same gel as part of the same experiment.



Supplemental Fig. S5. Induced BRAFi cells express high levels of CAV1 and AXL. (A) Dose response curve of IGR39 and WM793 melanoma cells, and WM793R cells that were selected for BRAFi resistance by culture in increasing concentrations of Vemurafenib (left), and half maximal inhibitory concentration (IC50) of the parental and resistant MITF^{Low} WM793 melanoma cells for Vemurafenib (right). (B) Western blot of whole cell extracts for indicated proteins from IGR39 cells as reference for comparison with WM793 parental and resistant WM793R cells. (C) Western blot of whole cell extracts from IGR39 cells exposed to linoleic acid (LA) for the indicated times to detect activation by phosphorylation of AXL and SRC as indicated. TBP served as loading control.