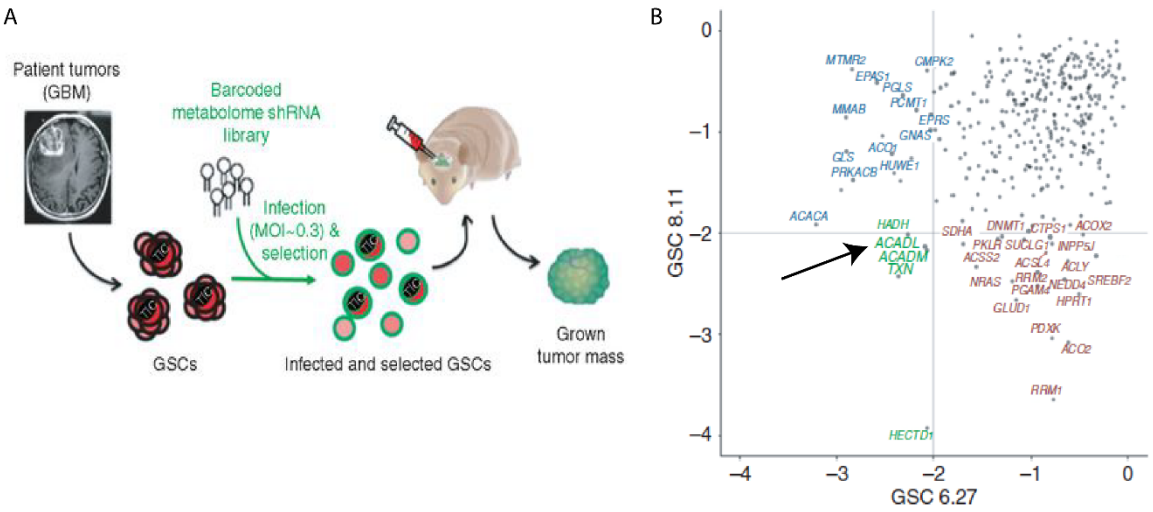

Figure S1. ACADL gene was idenitified from a metabolome shRNA screen as a key dependency for tumor growth in Glioblastoma. A. Schematics of experimental design for intracranial metabolome shRNA screens in patient-derived GSCs. The lentiviral library was transduced at a low MOI (less than one integrant/cell). B. Gene–rank analysis highlighting the significance of each gene on tumor growth for two patient-derived glioma sphere-forming cells (GSC 6.27 and GSC 8.11). The genes highlighted in green show significant impact in both GSCs when these genes are silenced using shRNA. The ACADL gene is indicated by the arrow.
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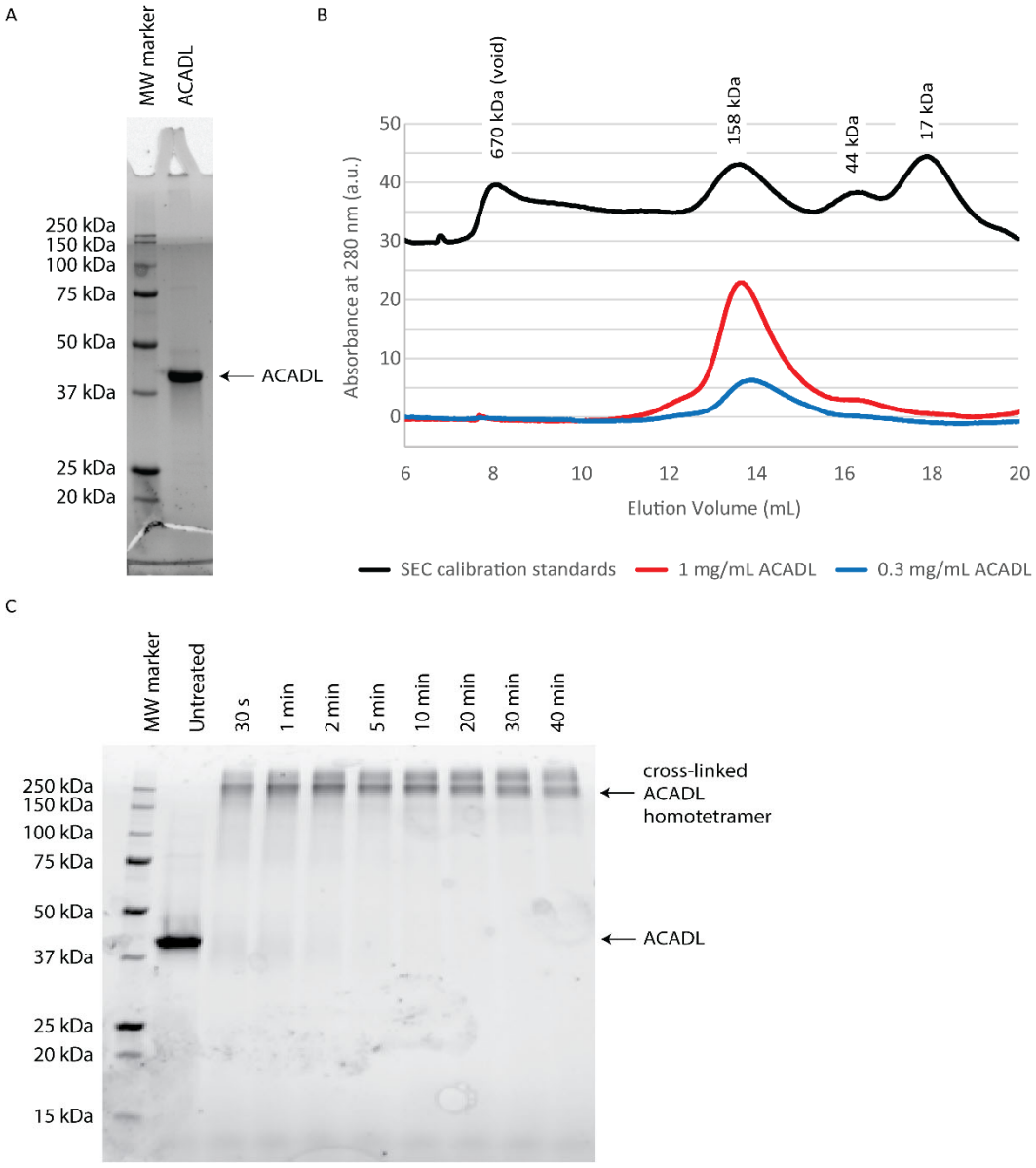

Figure S2. A. SDS PAGE gel analysis for purified Human ACADL holoenzyme (residues 31-430). B. Size exclusion chromatography chromatogram for 100 µL of ACADL protein loaded at 1 mg/mL (-) and 0.3 mg/mL (-) concentrations. The elution profile of a protein mixture containing Thyroglobulin (670 kDa), γ-globulin (158 kDa), ovalbumin (44 kDa), myoglobulin (17 kDa) and Vitamin B12 (1.35 kDa) is shown above for comparison. The ACADL protein elutes as a homotetramer (MW_{calc} = 190.6 kDa). C. Time-course experiment for a glutaraldehyde cross-linking reaction using 1 mg/mL ACADL and 0.2% (v/v) glutaraldehyde. 1 min reaction time appears optimal for this cross-linking reaction.