Supplemental Table 1. Specimens used for CD36 analysis and functional studies. Source of and clinical data for xenograft and primary human specimens used for CD36 analysis and functional studies in this manuscript, GBM = glioblastoma, TBD = to be determined.

Supplemental Figure 1. CD36 is not expressed by neural progenitor cells (NPCs) or essential for their proliferation in vivo. Representative micrographs (**A**) from the human protein atlas dataset reveal limited CD36 expression in human NPC niches as compared to a positive control (spleen). Each anatomical region and patient-specific data indicated above micrographs and each micrograph represents 1 mm from a tissue microarray core. Micrographs (**B**) and analysis of wild-type (WT) and CD36 null (CD36-/-) mice using antibodies against proliferating cell nuclear antigen (PCNA, red) or phospho-histone H3 (PH3, green) in the subventricular zone (SVZ, **C**) and subgranular zone (SGZ, **D**) reveal no major difference in proliferation in the adult mouse brain. Nuclei counterstained with dapi (blue), and scale bar represent 10 μm. In vivo quantification performed on 3 separate anatomical regions from 3 separate mice and values represent mean +/- standard deviation.

Supplemental Figure 2. CD36 is co-expressed with integrin α6 and CD133, established CSC markers. Immunofluorescence images of intracranial tumor generated from a GBM patient-derived xenograft (GBM10) indicates co-expression of CD36 (green) with integrin α6 (red, **A**) and CD133 (red, **B**). Flow cytometry analysis of patient-derived GBM xenografts (T3832, T387, T3691, GBM10) confirms co-expression of CD36 with integrin α6 (**C**) and CD133 (**D**). Xenograft staining was performed in triplicate and similar expression was observed in another patient-derived GBM xenograft (T3832). Nuclei counterstained with dapi (blue), yellow arrows indicate regions of co-localization, and scale bars represent 20 μm. Flow cytometry data represented as mean +/- standard deviation for at least 3 separate experiments.

Supplemental Figure 3. CD36 reduction results in decreased CSC maintenance signaling and self-renewal. Schematic summarizing target sequence locations of CD36 siRNA and shRNA constructs (**A**) with 5' and 3' UTR regions in gray, coding sequence in blue and location of target sequences shown in red. Immunoblotting analysis of CSCs from GBM specimens T3832 and GBM10 (**B**) following CD36 inhibition by shRNA knockdown (KD) constructs (KD1, KD3, KD4) reveals a reduction in CSC signaling pathways (Sox2, p-Akt, and p-Stat 3) as compared to non-targeting (NT) control. Actin was used as a loading control. Limiting dilution analysis (**C**) demonstrates CSCs from GBM xenografts T3832 and GBM10 transduced with CD36 shRNA KD constructs (KD1, KD3, KD4) have significantly reduced self-renewal as compared to NT control. Stem cell frequencies were also reduced by CD36 targeting and provided below limiting dilution analysis.

Supplemental Figure 4. Functional validation of 2-methylthio-1,4-naphthoquinone (MTN) by oxidized low density lipoprotein (oxLDL) uptake. Schematic (**A**) depicting the oxLDL update assay between wild-type and CD36 deficient macrophages. Quantification (**B**) of oxLDL uptake demonstrates a significant reduction in oxLDL uptake in CD36 deficient macrophages (white bar) as compared to wild-type control (black bar) macrophages. Schematic (**C**) depicting the oxLDL update assay with MTN. Quantification (**D**) of oxLDL uptake demonstrates a significant reduction in oxLDL uptake with MTN (red bard) as compared to control conditions (black bar). Uptake assays performed in at triplicate and represent mean +/- standard deviation, *** $p < 0.001$ as assessed by one-way ANOVA.