Supplementary Figure S1. Effect of L1CAM Suppression on Cell Growth, Cell Adhesion and Downstream Targets of Integrin Signaling Pathway. (A-B) Cell Proliferation was measured by MTS assay for H460 and H125 cells over a period of 4-5 days and no significant differences were observed between knockdown and control lines. Cell number (MTS assay absorbance at 490 nm) is plotted against time of incubation. Results shown represent mean ± SEM of 3 biological replicates. (C) Western blot analysis was used to show the effect of L1CAM suppression on proteins associated with integrin signalling pathway: total and activated FAK, Src, Akt, and Erk1/2. H125-shL1CAM and control cells were either serum-starved for 24 hours or stimulated with 10% serum for 24 hours prior to harvesting. The levels of β-actin were used as internal protein loading control. (D) The effect of L1CAM suppression on cell adhesion to fibronectin and collagen substrates was assessed and no significant differences were observed.
between knockdown and control lines. Results shown represent mean ± SEM of 3 biological replicates.

**Supplementary Figure S2.** L1CAM mRNA and Protein Expression Levels in Xenograft Tumors Formed by H460- and H125-shL1CAM Cells. (A) qRT-PCR was used to confirm mRNA L1CAM levels in tumors formed in mice bearing shL1CAM or control (*** p<0.0001). (B) We randomly selected several xenograft tumors in two different shRNA clones (H460-shL1CAM2.4 and shL1CAM5.4) and re-cultured these tumors on plastic. Cells derived from these tumors were subjected to puromycin selection for 2 weeks to isolate human tumor cells from mouse cells. Western blot analysis was used to assess the L1CAM protein levels. The levels of β-actin were used as internal protein loading controls. 1 of 3 experiments with comparable results is shown. (C) Ratio of tumor weight to body weight was calculated for each animal per group at endpoint (* p<0.01, ** p<0.001, *** p<0.0001, ns: not significant).