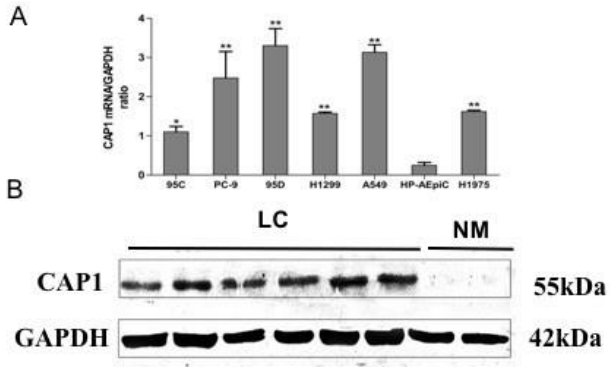

Figure S1. CAP1 expression of lung cancer cell lines and normal lung cell line was analyzed in PCR (A) and (B) Western blotting with the antibody against CAP1. GAPDH served as the loading control. (**P < 0.01; *P< 0.05 vs. normal lung cell line.).

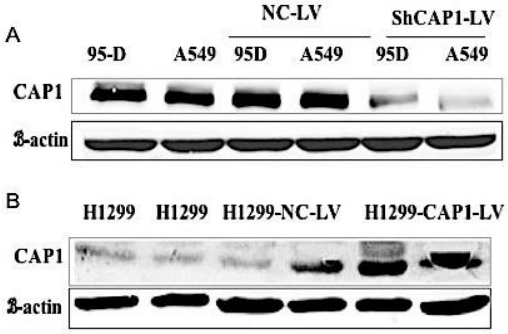

Figure S2. (A) Efficient knockdown of CAP1 in the A549 and 95-D cancer cells was confirmed by Western blotting with the antibody against CAP1. GAPDH served as the loading control. (B) Expression of CAP1 was increased by overexpression of CAP1 in H1299 cancer cell.

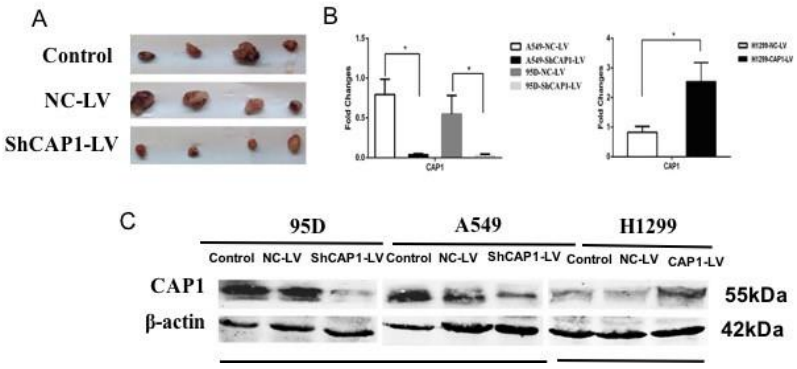

Figure S3. (A) The tumor volume was significantly lower in CAP1 gene knockout tumor tissue than that in empty virus control group and blank control group. CAP1 was increased or decreased by overexpression or inhibition of CAP1 in tumor samples by WB (C) and PCR (B).

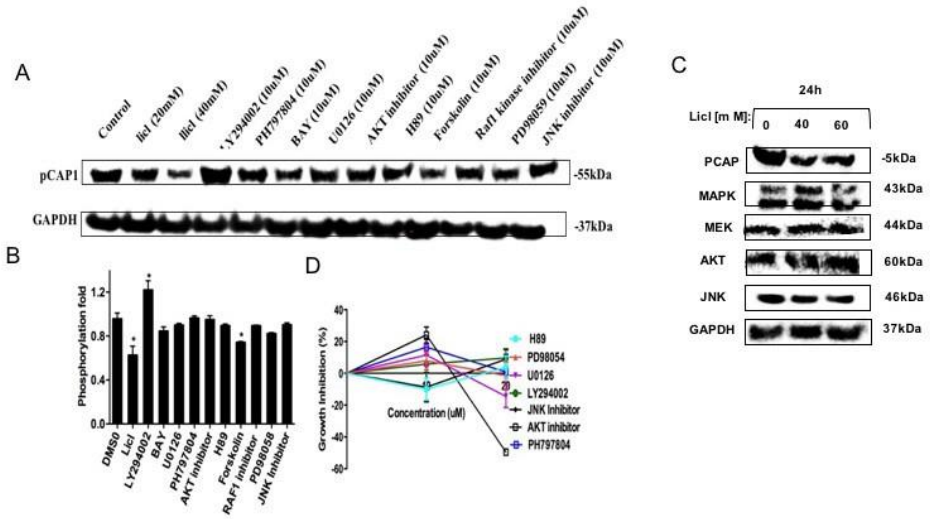

Figure S4. (A, C) Treatment of lung cells with GSK3 inhibitors LiCl reduced CAP1 phosphorylation at S307/S309. Cells were treated with the inhibitors for 24 hrs, and cell lysates were prepared for western blotting with both CAP1 and phospho-CAP1 antibodies. (B) Signals from three independent experiments were measured using densitometry and analyzed using Student's *t*-test and plotted in the graph; error bars represent±s.e.m; **P*<0.05. (D) MTT assays showed the re-expression of CDD mutant in the CAP1 knockdown A549 cells. All of the MTT experiments were repeated three times, with the error bar representing S.D.