**SD Figure Legends**

**SD Fig. 1** TNF-induced phosphorylation of BRMS1 is independent of NF-κB. **(A)** NSCLC H157 V/I cells were treated with TNF (20 ng/mL) and harvested at the indicated times. Protein levels of BRMS1 were evaluated by Western blot. **(B)** NSCLC H1299 cells were treated with TNF (20 ng/mL) following pretreatment with or without TBB (50 μM) and harvested at the indicated times. Endogenous BRMS1 was pulled down by immunoprecipitation, and phospho-BRMS1 was detected by a pan-phospho-Ser/Thr antibody. **(C)** NSCLCA549 and H1299 cells were treated with or without TGFβ (4 ng/mL), TNF (20 ng/mL), IL-6 (100 ng/mL), or EGF (100 ng/mL) for 2 h, and the indicated proteins were detected by immunoblot.

**SD Fig. 2** CK2α’ endogenously interacts with BRMS1. **(A)** Co-immunoprecipitations (IPs) were performed using the indicated antibodies, and BRMS1 was detected by Western blot. **(B)** Proteasome inhibitor MG132 abolishes CK2α’-mediated BRMS1 degradation. H1299 cells were transfected with HA-tagged CK2α, α’, or empty vector and were pretreated with or without MG132 (5 μM) for 2 h, followed by stimulation with TNF (20 ng/mL) for an additional 16 h. Endogenous BRMS1 was detected by Western blot.

**SD Fig. 3** 14-3-3ε interacts with BRMS1. **(A)** H157 cells were treated with TNF (20 ng/mL) or vehicle control for 1 h. Immunofluorescence assays were performed using antibody against BRMS1 (red). DAPI (blue) was used for nuclear counterstain. **(B)** 14-3-3 isoform ε, not ζ, interacts with BRMS1. Co-immunoprecipitations (IPs) were performed in H1299 cells using the indicated antibodies, and 14-3-3 expression was detected by Western blot.

**SD Fig. 4** Activation of CK2α’ enhances the invasion potential of NSCLC. **(A)** Treatment with tetracycline (TCN) induces Myc-tagged CK2α’ expression. H157stable cell lines were treated with or without TCN (1 μg/mL) for 48 h. Immunoblots show ectopic BRMS1 expression in these cells (lane 3, 4, 5, 6), and Myc-CK2α’ is induced by TCN at 48 h (lane 2, 4, 6). **(B)** S30A mutant abrogates CK2α’-induced invasion potential. H157 stable cell lines were treated with or without TCN (1 μg/mL) for 24 h, and invasion chamber assays were performed. The bar graph shows the average invaded cell numbers per cell line. \*p<0.01, compared with control; #p<0.01, compared with BRMS1 wild-type without TCN treatment.

**SD Fig. 5** Activationof CK2α’ does not affect primary tumor growth. **(A)** Mouse bioluminescent CT axial images show the positions of primary tumors in 3 representative mice from each group. **(B)** Expression of BRMS1 and CK2α’ in primary tumors in 3 representative mice from each group, examined by Western blot. **(C)** The graph represents the total cell numbers for the primary tumor in each group.

**SD Fig. 6** CK2α’ kinase activity is increased in tumors with lymph node metastasis. **(A)** Endogenous CK2α’ kinase activity in patient samples was determined in tumor and adjacent noncancerous tissues. \*p<0.01, compared with adjacent noncancerous tissues. **(B)** Patient tumors with nodal metastases exhibit higherCK2α’ kinase activity. Endogenous CK2α’ kinase activity in patient samples was determined. The bar graph shows the fold changes of tumor versus adjacent noncancerous tissue (*p*=0.025).