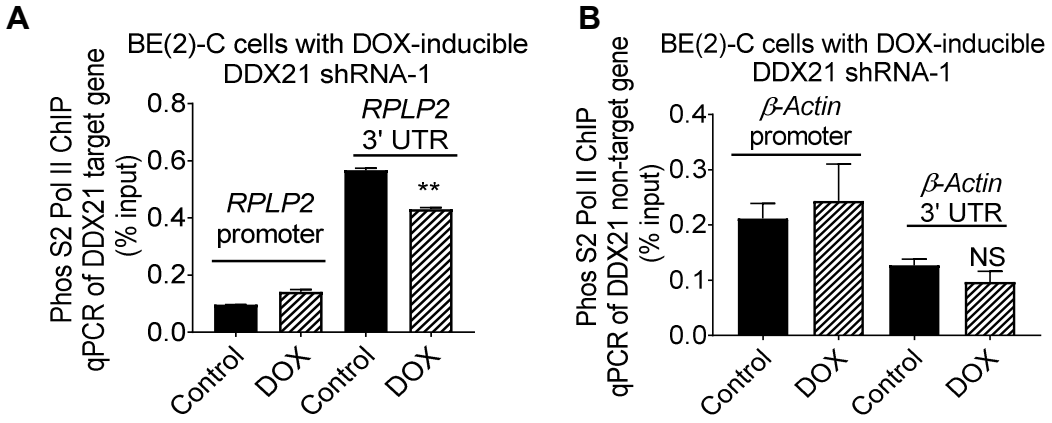
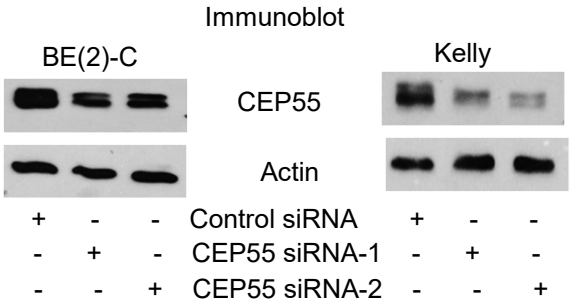
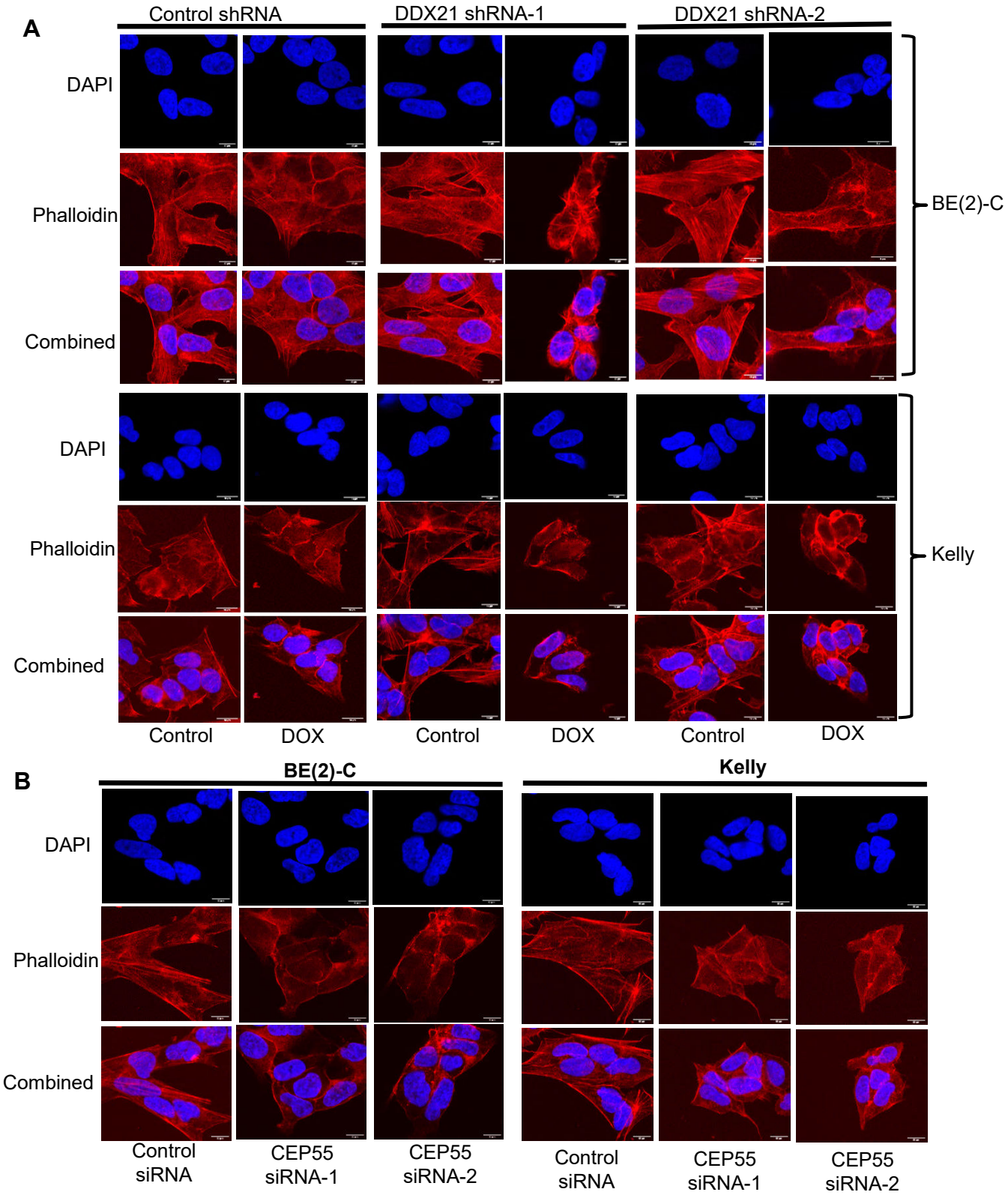


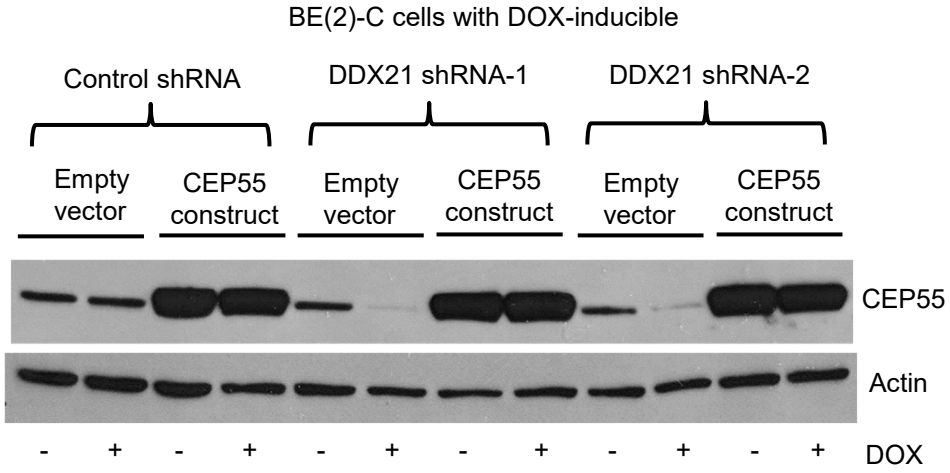
Supplementary Figure S1. DDX21 promotes RPLP2 transcriptional elongation. (A-B) BE(2)-C cells with DOX-inducible DDX21 shRNA-1 were treated with vehicle control or DOX for 48 hours, followed by ChIP assays with a control or an anti-phos S2 Pol II antibody. The immunoprecipitated DNA was examined by PCR with primers targeting the *RPLP2* or *β-actin* promoter, and the *RPLP2* or *β-actin* 3′-untranslated region (3′-UTR). Error bars represented standard error. ** indicated $P < 0.01$. NS indicated not significant.



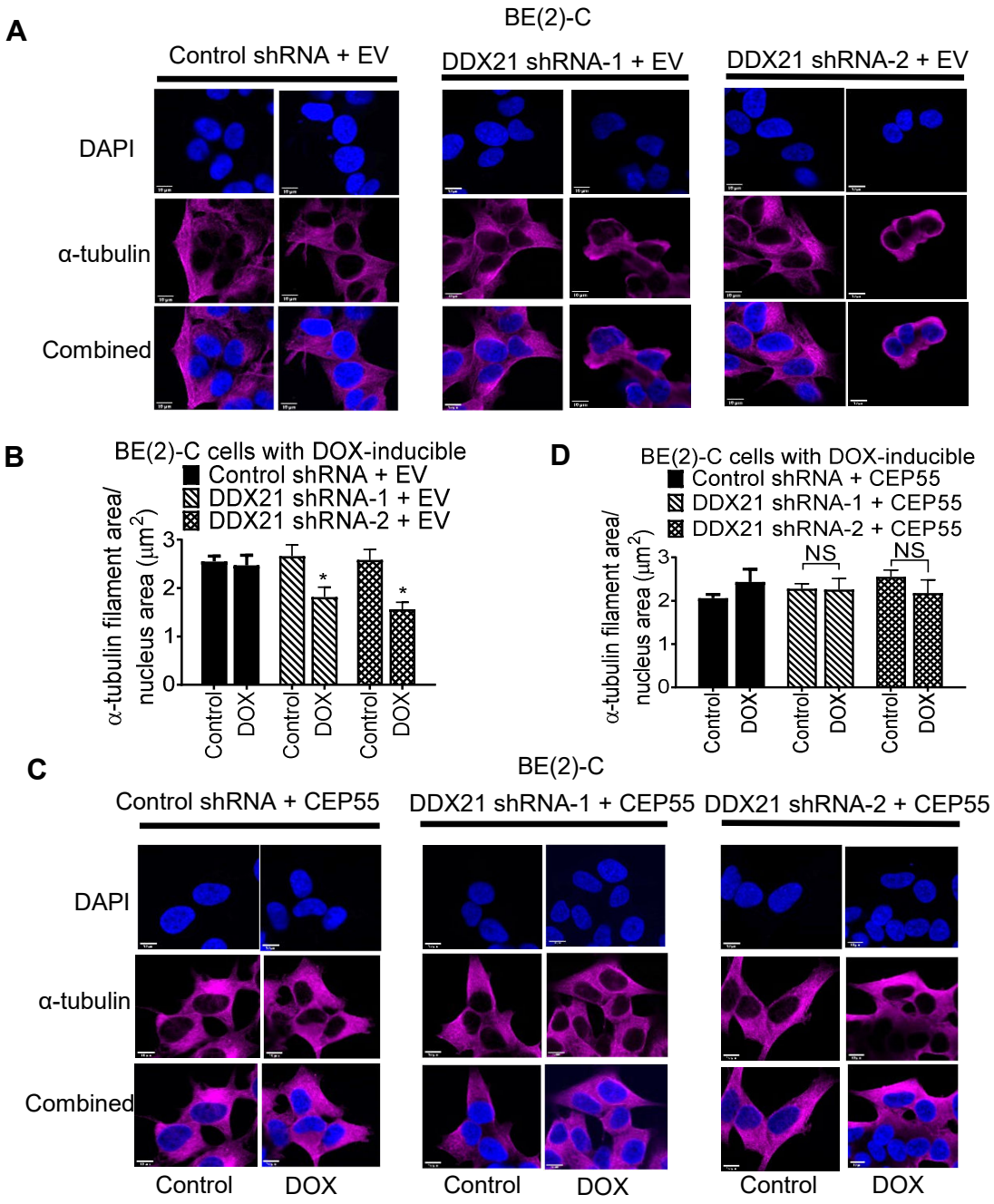
Supplementary Figure S2. CEP55 siRNAs knock down CEP55 protein expression. BE(2)-C and Kelly cells were transfected with control siRNA, CEP55 siRNA-1 or CEP55 siRNA-2 for 48 hours, followed by western blot analysis of CEP55 expression.



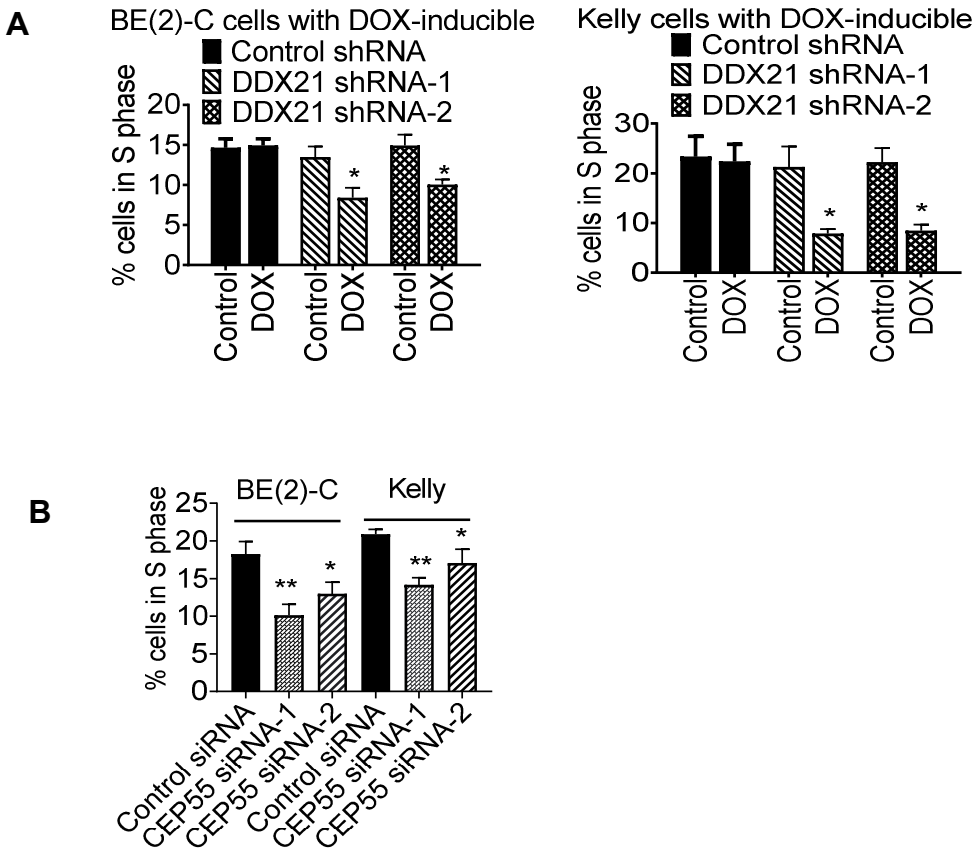
Supplementary Figure S3. DDX21 and CEP55 play a role in neuroblastoma cell cytoskeletal stability. (A) Doxycycline (DOX)-inducible control shRNA, DDX21 shRNA-1 or DDX21 shRNA-2 BE(2)-C and Kelly cells were treated with vehicle control or DOX for 72 hours, followed by immunostaining of phalloidin. DNA was counterstained with DAPI. (B) BE(2)-C and Kelly cells were transfected with control siRNA, CEP55 siRNA-1 or CEP55 siRNA-2 for 72 hours, followed by immunostaining of phalloidin. DNA was counterstained with DAPI. Scale bar: 10 μm.



Supplementary Figure S4. Transfection of CEP55 over-expression construct results in CEP55 over-expression. DOX-inducible control shRNA, DDX21 shRNA-1 or DDX21 shRNA-2 BE(2)-C cells were transfected with an empty vector or CEP55 over-expression construct, then treated with vehicle control or DOX for 48 hours, followed by western blot analysis of CEP55 expression.



Supplementary Figure S5. Over-expression of CEP55 rescues the phenotypes induced by DDX21 knockdown. (A-B) DOX-inducible control shRNA, DDX21 shRNA-1 or DDX21 shRNA-2 BE(2)-C cells were transfected with an empty vector (EV) construct, then treated with vehicle control or DOX for 72 hours, followed by immunostaining with an anti-α-tubulin antibody and DNA was counterstained with DAPI (A). The areas covered by α-tubulin filaments, relative to nucleus, were quantified (B). (C-D) DOX-inducible control shRNA, DDX21 shRNA-1 or DDX21 shRNA-2 BE(2)-C cells were transfected with a CEP55 over-expression construct, then treated with vehicle control or DOX for 72 hours, followed by immunostaining an anti-α-tubulin antibody and DNA was counterstained with DAPI (C). The areas covered by α-tubulin filaments, relative to nucleus, were quantified (D). Error bars represented standard error. * indicated $P < 0.05$. Scale bar: 10 μm. NS indicated not significant.



Supplementary Figure S6. DDX21 and CEP55 promote neuroblastoma cell proliferation. (A) DOX-inducible control shRNA, DDX21 shRNA-1 or DDX21 shRNA-2 BE(2)-C and Kelly cells were treated with vehicle control or DOX for 72 h, followed by staining with propidium iodide and cell cycle analysis by flow cytometry. (B) BE(2)-C and Kelly cells were transfected with control siRNA, CEP55 siRNA-1 or CEP55 siRNA-2 for 72 hours, followed by staining with propidium iodide and cell cycle analysis by flow cytometry. The percentage of cells in S phase was shown. Error bars represented standard error. * indicated $P < 0.05$ and ** $P < 0.01$.