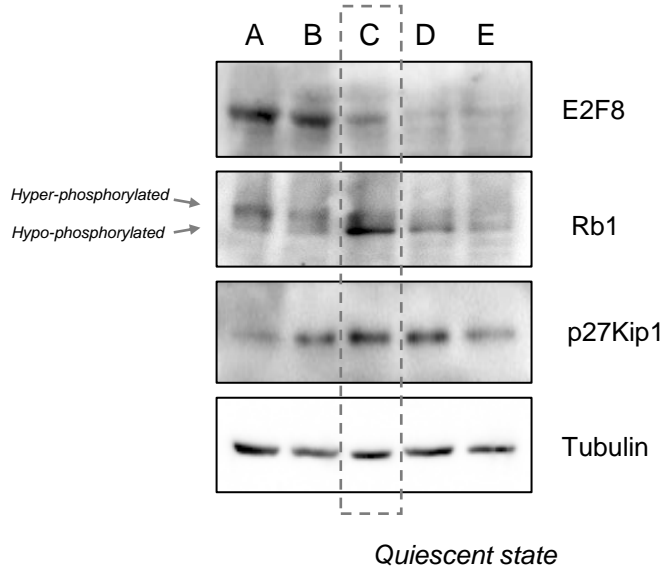
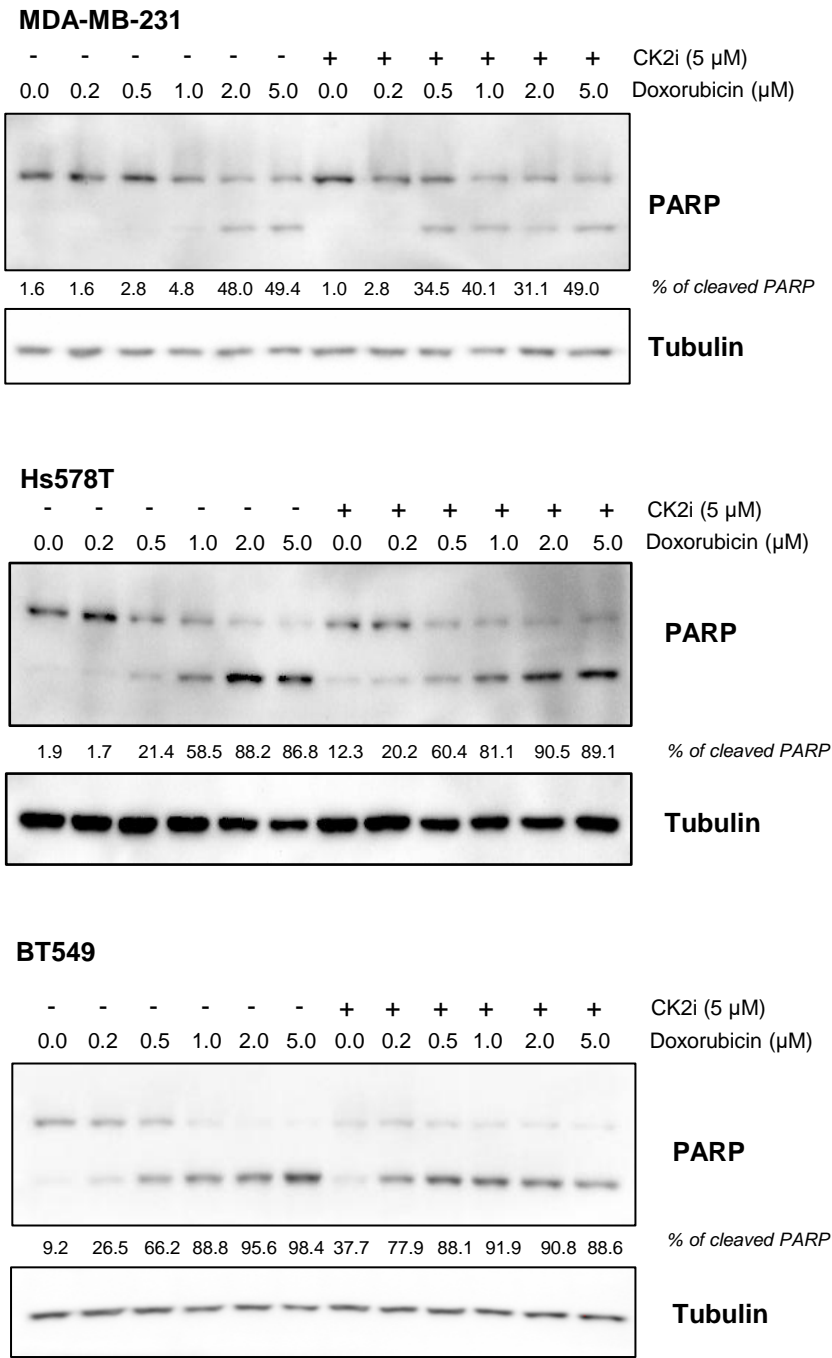


Supplementary Figure 1:



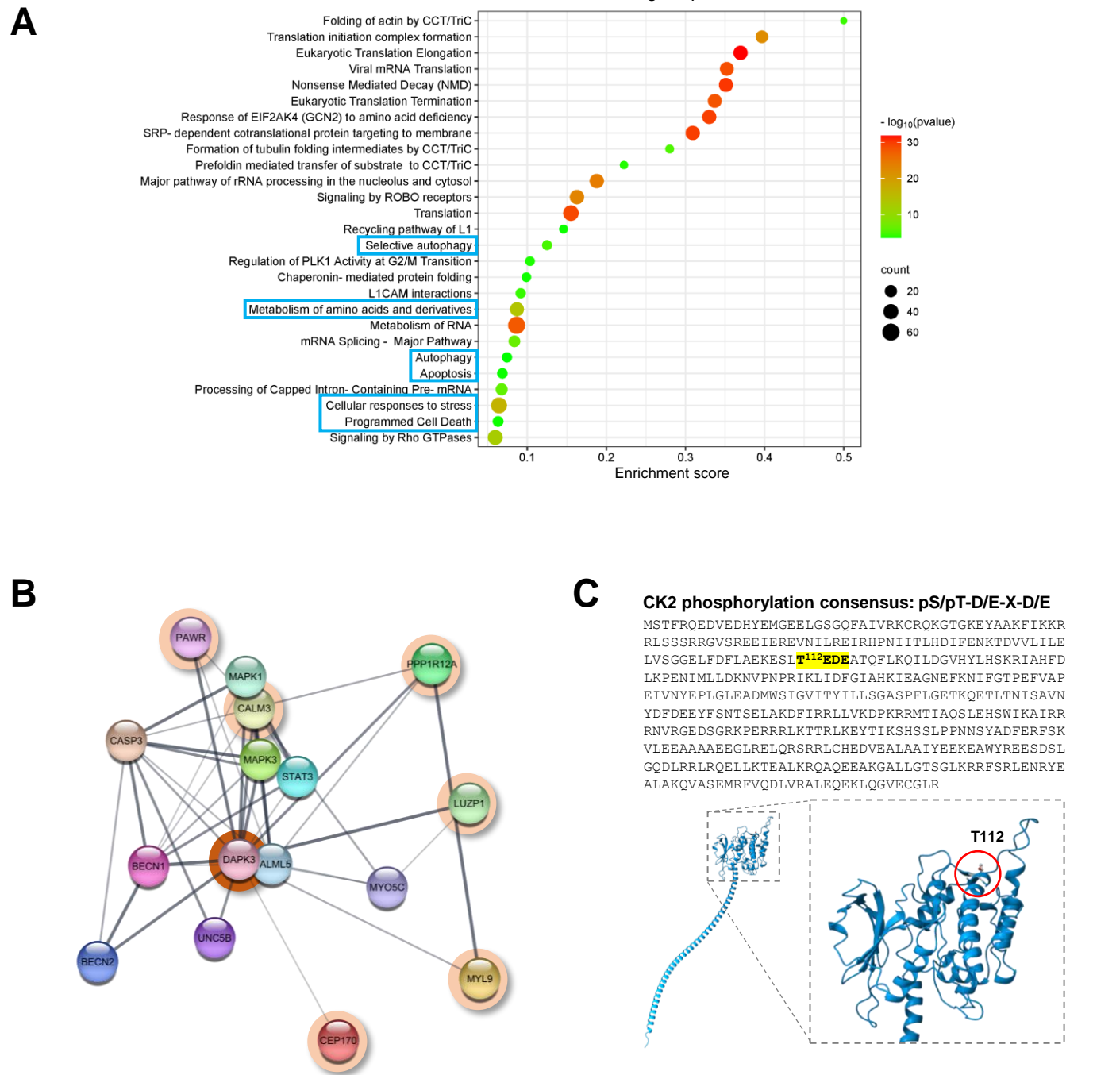
Supplementary Figure 1. Validation of quiescence induction by western blot. Expression of indicated proteins was analyzed in MDA-MB-231 in various timepoints corresponding to the transition between quiescence and proliferation. Timepoints were as follows: *A* – continuously growing cells; *B* – serum starved cells (48 hrs); *C* – serum starved cells (96 hrs); *D* - serum reactivated cells (+20 min); *E* - serum reactivated cells (+120 min). α -tubulin was used as loading control.

Supplementary Figure 2:



Supplementary Figure 2. CK2 inhibition potentiates doxorubicin-induced apoptosis in TNBC cells. Expression and cleavage of PARP was analyzed in MDA-MB-231, Hs578T and BT549 under indicated conditions (+/- CK2 inhibition, +/- Doxorubicin) by western blot. α -Tubulin was used as loading control. Numbers indicate percentage of cleaved PARP in corresponding conditions.

Supplementary Figure 3:



Supplementary Figure 3. Functional and structural analysis of DAPK3 (A) GO Biological Processes enrichment analyses showing enriched pathways identified through phospho-CK2 substrate pulldown analysis. Color indicates false discovery rate (FDR), circle size indicates number of identified hits in the pathway, X-axis shows enrichment score. (B) Network analysis of DAPK3 visualized using String database (<https://string-db.org/>) with highlighted proteins identified in the pulldown. (C) Primary amino acid sequence of DAPK3 with threonine 112 highlighted in red. Threonine 112 resides within a CK2 consensus motif (T¹¹²EDE), positioned on a solvent-accessible surface of the kinase domain.