Supplementary File 1

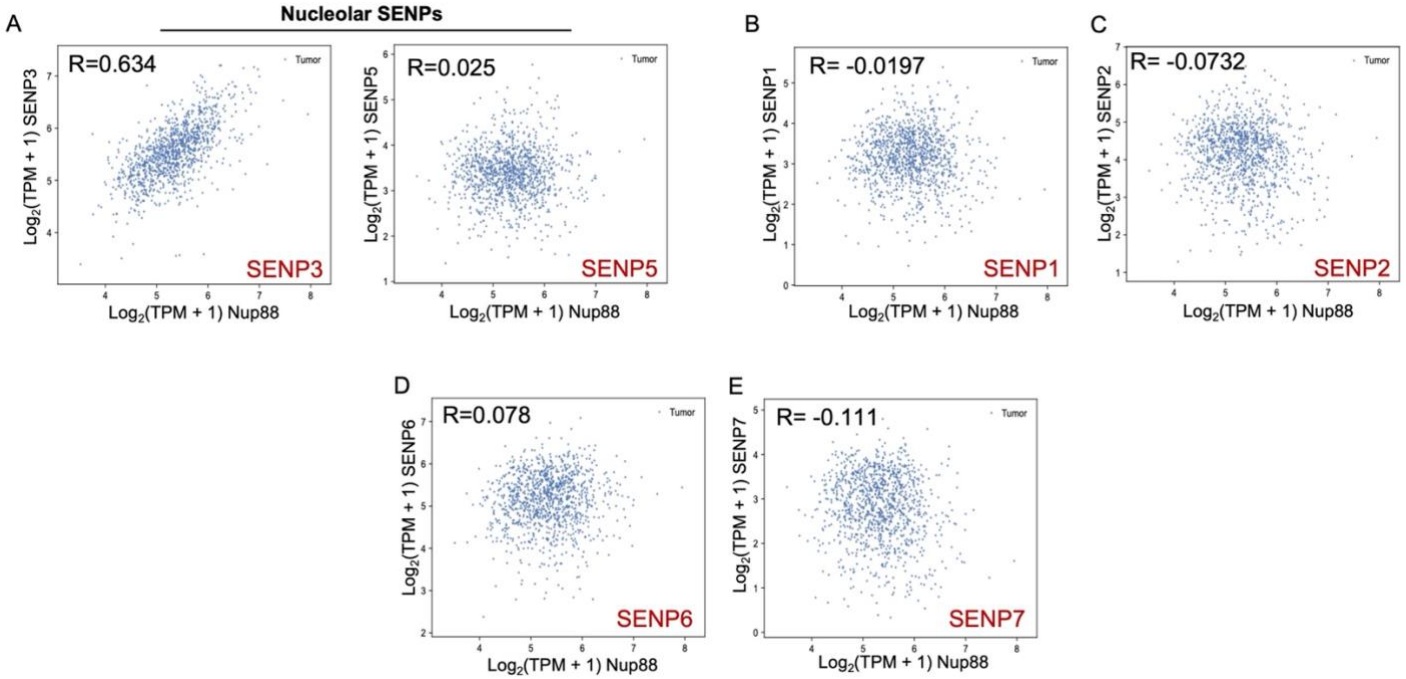


Figure S1. Scatter plots analysing correlation between Nup88 and different SENP homologs in breast cancer samples using the GEPIA3 database.

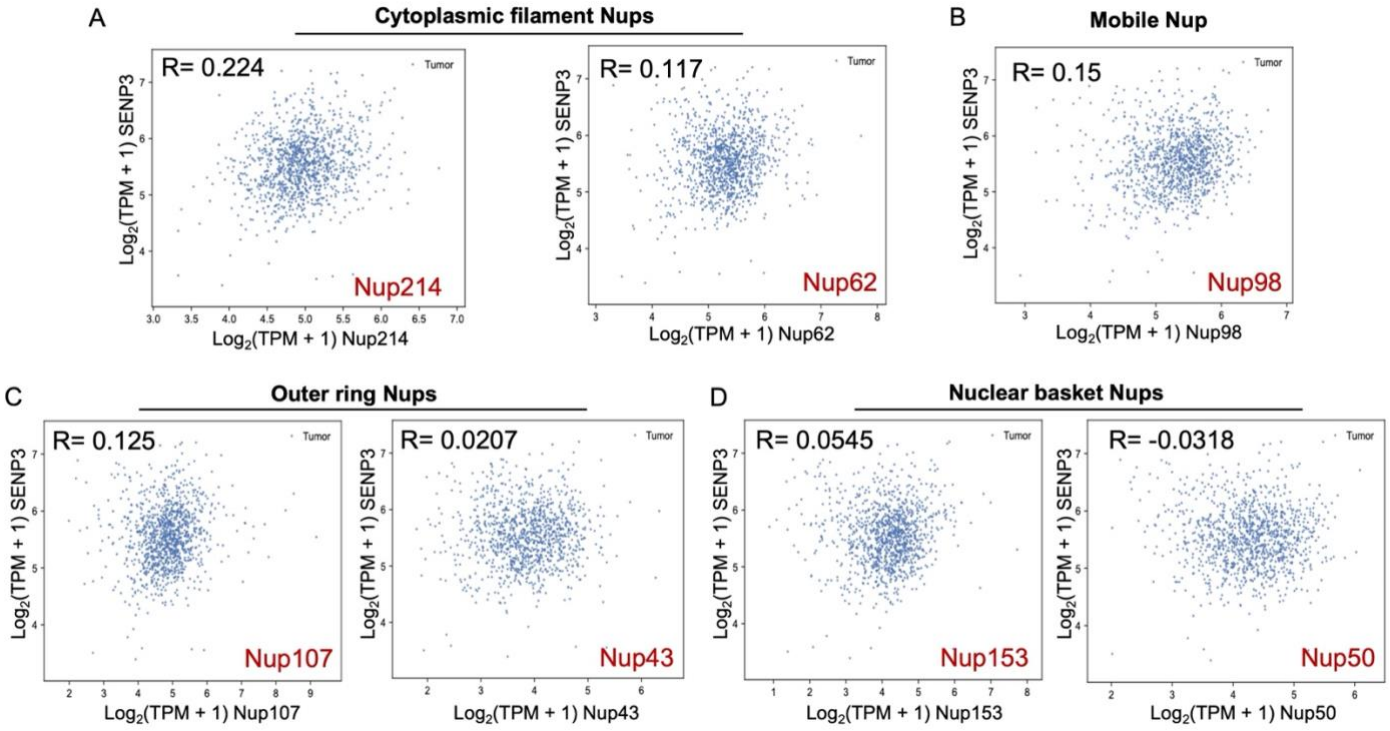


Figure S2. Scatter plots analysing correlation between SENP3 and different nucleoporins in breast cancer samples using the GEPIA3 database.

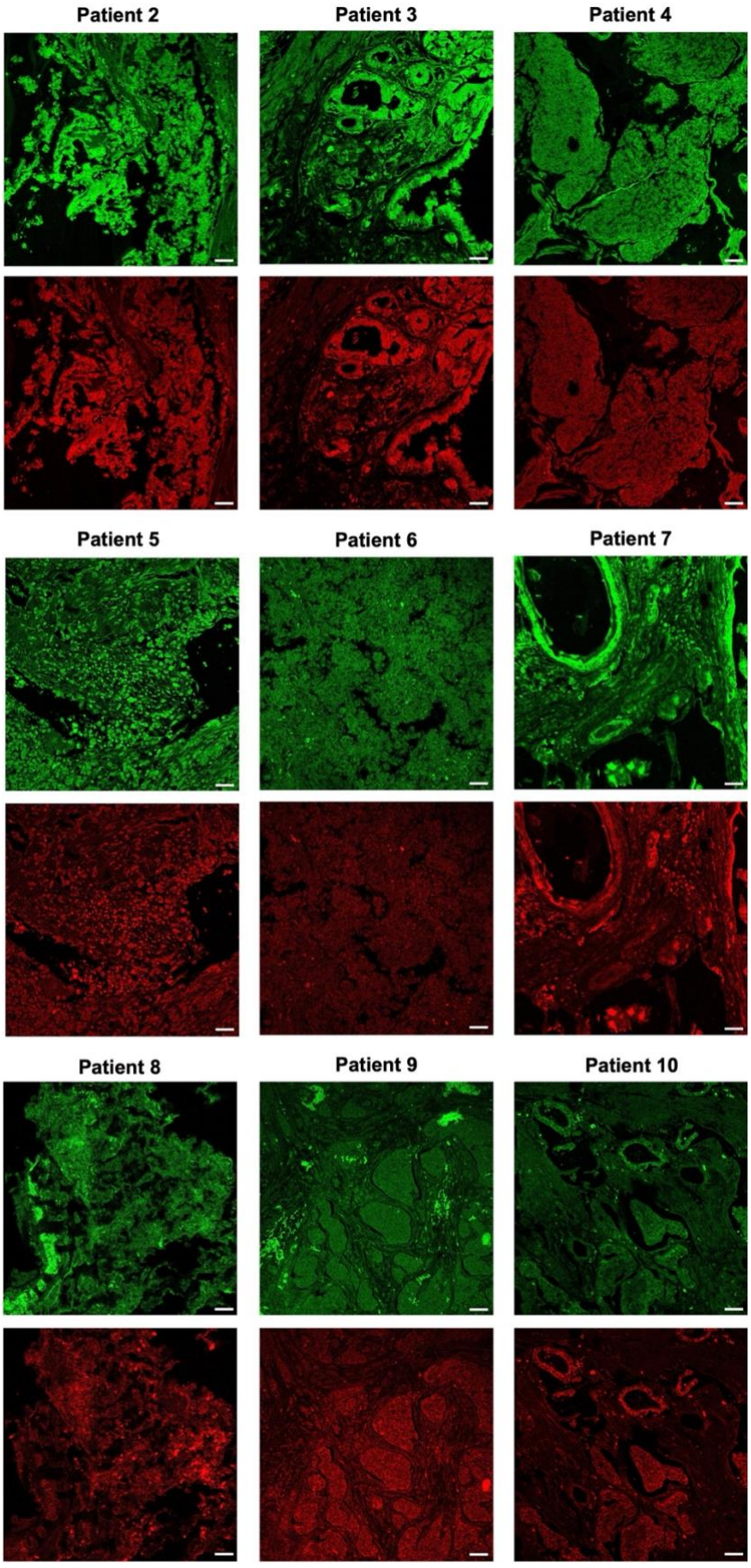


Figure S3. Nup88 (green) and SENP3 (red) expression observed in tumor sections of breast cancer patients via immunofluorescence analysis. Scale bar, 50 μm.

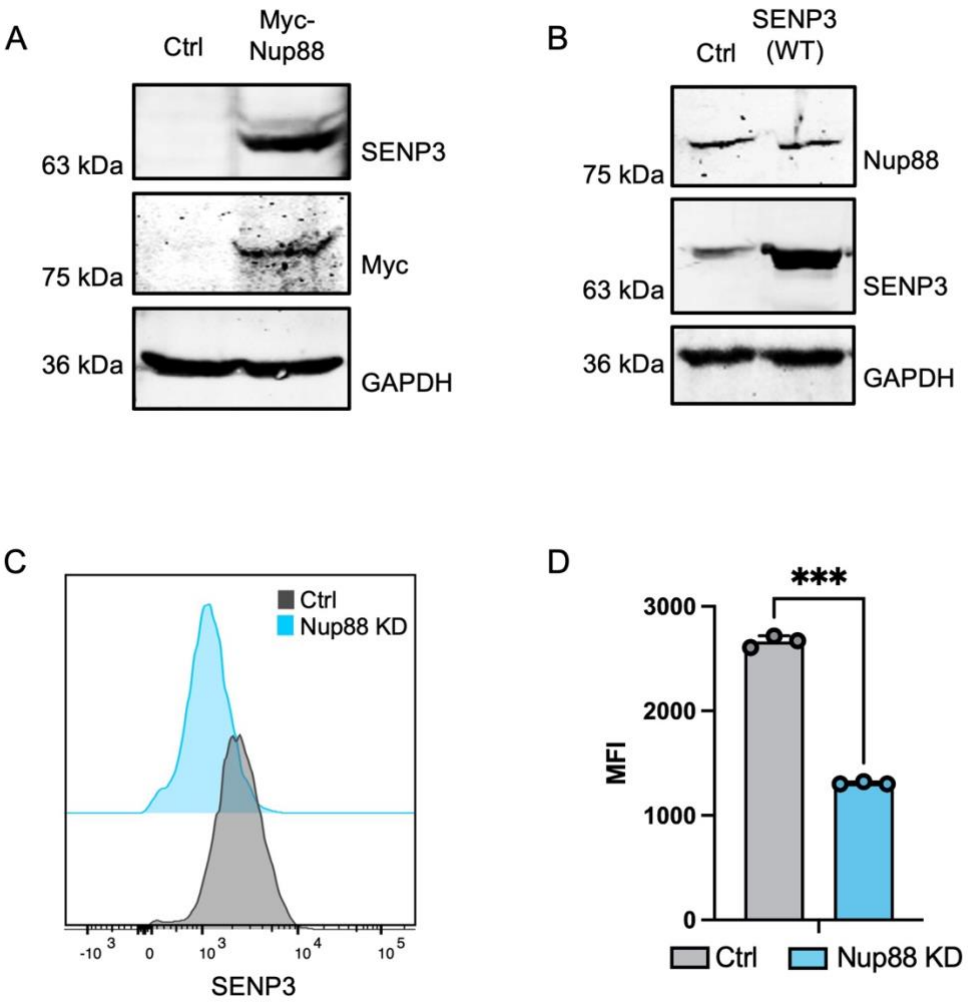


Figure S4. A, Immunoblot analysis showing SENP3 levels upon Myc-Nup88 overexpression, confirming tag-independent effects. B, Immunoblot analysis of endogenous Nup88 levels upon overexpression of RGS-SENP3 or its catalytic mutant (C532A). C, Flow cytometry histogram showing SENP3 expression upon Nup88 knockdown as compared to control. D, Quantification of MFI of SENP3 under indicated conditions.

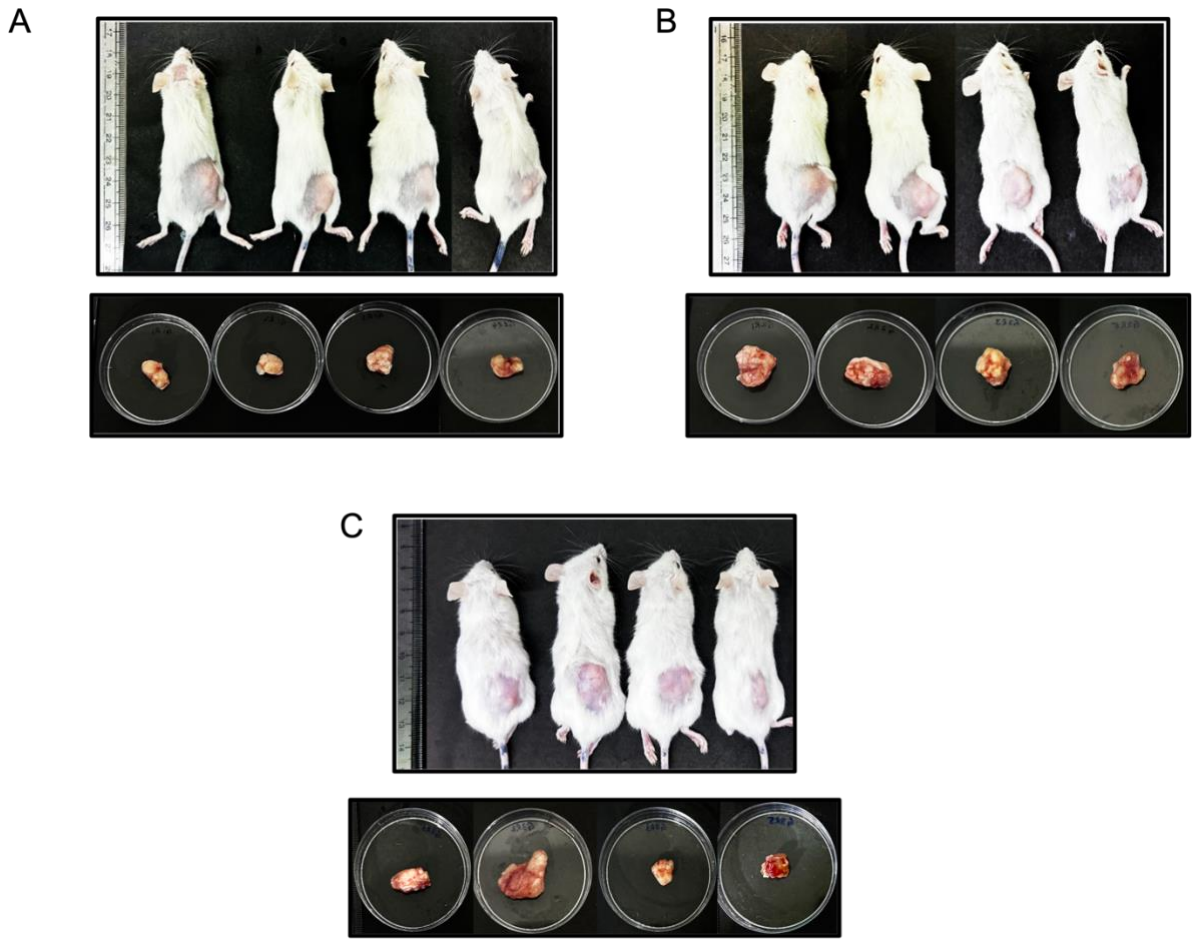


Figure S5. A-C, Subcutaneous tumor growth in NOD/SCID mice injected with MDA-MB-231 cells either expressing GFP (A), GFP-Nup88 (B), or GFP-Nup88 with SENP3 knockdown (C).

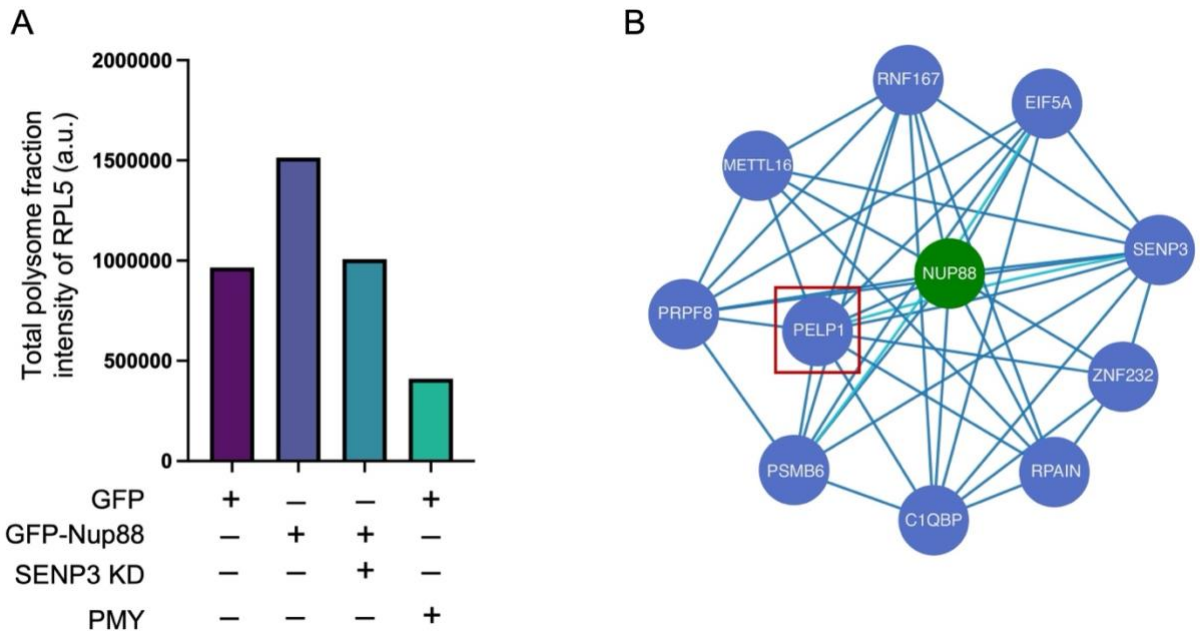


Figure S6. A, Quantification of RPL5 expression in polysomal fractions (Fractions 11, 14) under mentioned conditions obtained from sucrose density gradient based polysome profile. B, Network analysis of Nup88-correlated genes in breast cancer (BRCA), highlighting PELP1 as a significantly associated node.

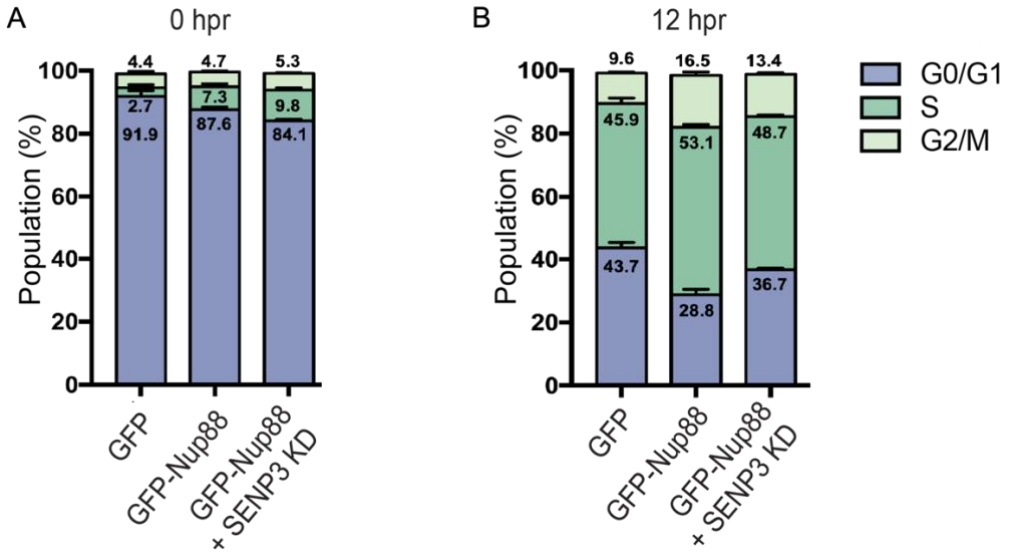


Figure S7. A,B, Quantification of cell cycle progression analysis by flow cytometry upon Palbociclib mediated synchronization-release of MCF7 cells under indicated conditions at different time points, 0 hpr (A) and 12 hpr (B). Bars indicate mean ± S.E.M. from three independent experiments.