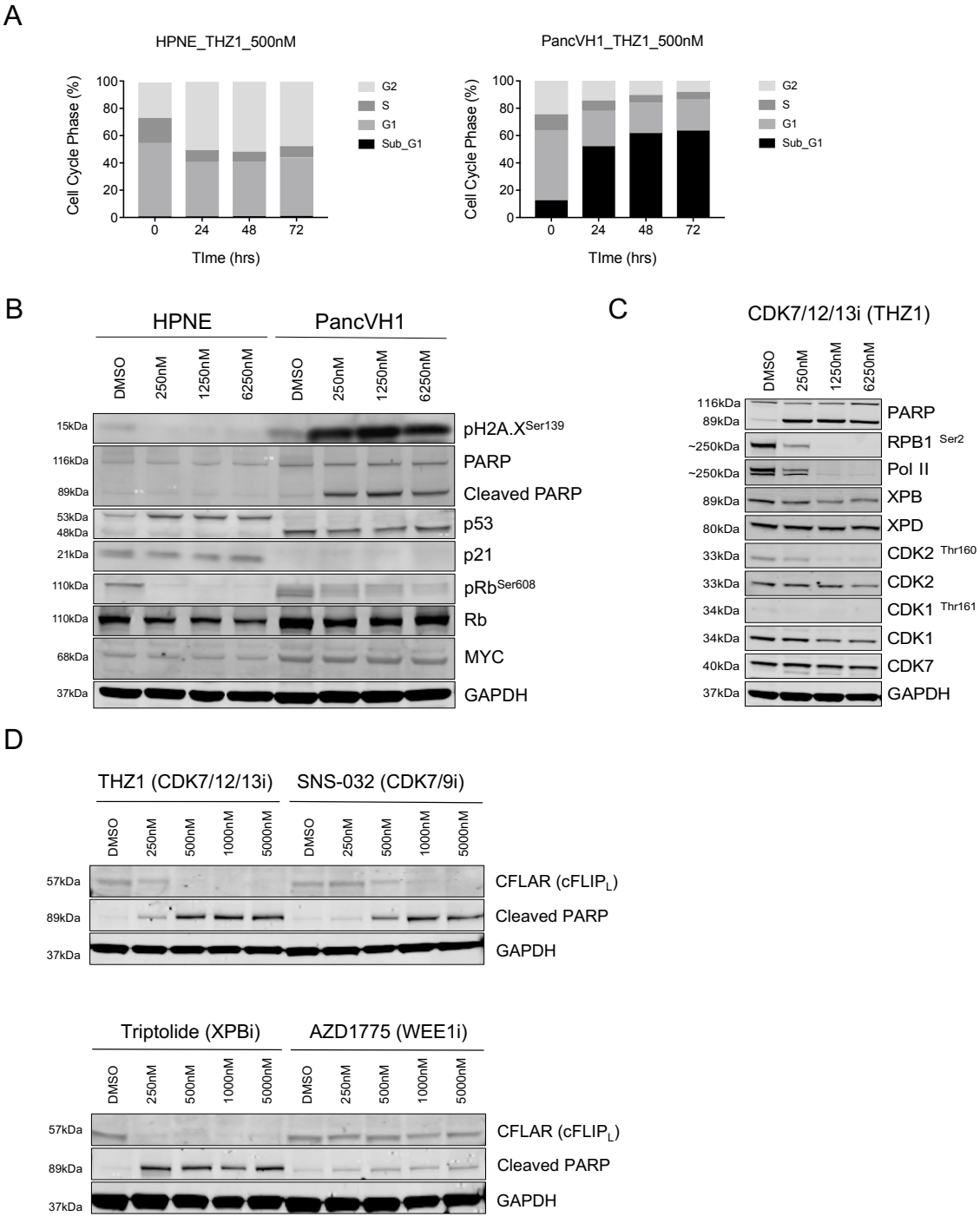

Figure S5. Extended Data. A. THZ1 initiates a cell death/apoptotic response at the G1/S transition in PancVH1, while initiating a G2 phase cell cycle arrest in HPNE cells; cell cycle analysis, time course post-treatment; bar graph of percentage of cells in subG1, G1, S, G2 phase assessed by DNA content. **B**. THZ1 induces DNA damage (pH2A.XSer139) and apoptosis (cleaved PARP) in PancVH1 but not in HPNE cells; immunoblot of pH2A.XSer139, PARP, p53, p21, pRBSer608, RB, MYC, GAPDH loading control. **C**. THZ1 treatment shows a dose-dependent inhibition of phospho-substrates of CDK7: CDK1Thr161, CDK2Thr160, and RPB1Ser2, and protein expression of RPB1, and cleavage of PARP; immunoblot to PARP, RPB1Ser2, RPB1/Pol II, XPB, XPD, CDK2 Thr160, CDK2, CDK Thr161, CDK1, CDK7, GAPDH loading control. **D**. PancVH1 treatment with TFIIH inhibitors THZ1, SNS-032, and Triptolide all show a dose-dependent reduction in CFLAR (cFLIP$_L$) protein expression with a concomitant cleavage of PARP indicating a CFLAR-mediated shared mechanism of action; equipotent inhibitor AZD1775 to the WEE1 kinase; immunoblot to CFLAR, PARP, GAPDH loading control.