



Figure S3 Dbf4 residues V104, E108, and L109 were critical for binding the Rad53 FHA domains. (A) The Dbf4 biotinylated peptide pThr105-FHA1 interaction was competed by the non-biotinylated T105-phosphorylated Dbf4 peptides (pThr105), but not by the same Dbf4 peptide with an E108A substitution, or by an unrelated phospho-serine peptide (pSpc72). (B) The pThr105-V104A and pThr105-L109A peptides were also defective in competing the biotinylated pThr105-FHA1 interaction.