



Figure S4 The synthetic lethality between *dbf4-NΔ109* and *rad53-1* or *rad53Δ* was not due to either loss of Cdc5 interaction or increased Dbf4 stability, but requires sequences between residues 82-109. Wild type and various *dbf4* mutants were cloned in low-copy number (*ARS/CEN/LEU2*) vectors, driven by the *DBF4* endogenous promoter. Plasmids were transformed into M1589 (*rad53-1 dbf4Δ::kanMX6 [pDBF4-URA3]*) or M3581 (*rad53Δ::TRP1 sml1Δ::HIS3 dbf4Δ::kanMX6 [pDBF4-URA3]*) and the wild-type *DBF4-URA3* plasmids were selected against on FOA. Cells that could not grow on FOA plates were scored as having a synthetic lethal interaction. The NΔ65 deletion causes increased Dbf4 stability by deleting sequences important for ubiquitin-mediated proteolysis. The Δ82-88 deletion prevented the Cdc5 interaction with Dbf4, while the Δ100-109 deletion prevented the interaction with Rad53 (see Figure S6).