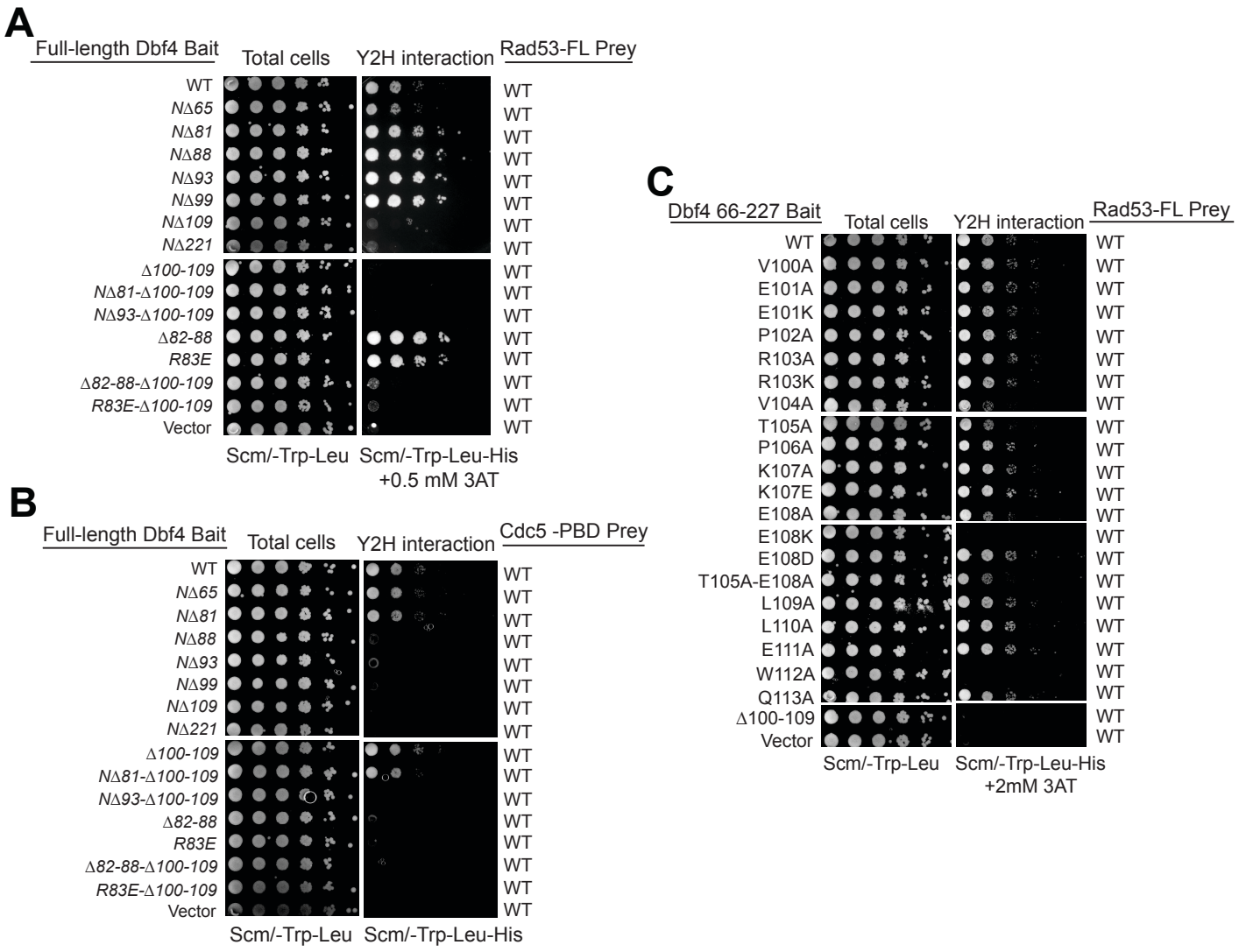

Figure S6 Dbf4 sequences important for binding full length Rad53 and the Cdc5 PBD. (A) A series of deletion in full-length Dbf4 was assayed by two-hybrid for interaction with full length Rad53 (panel A) or with the Cdc5 Polo-box domain (PBD) (panel B). The *dbf4-Δ100-109* deletion caused a loss of Rad53 binding, but still allowed interaction with the Cdc5-PBD. The *dbf4-Δ82-88* deletion caused loss of Cdc5 binding but not Rad53. An N-terminal deletion through residue 81 (NΔ81) or disruption of the Cdc5 binding site (Δ82-88 and R83E) caused increased Rad53 binding compared to full length Dbf4. (C) Dbf4 point mutations were assayed for their two-hybrid interaction against full length Rad53. The Δ100-109 deletion caused a loss of the two-hybrid signal similar to the vector control. The V104A, T105A, E108A mutations resulted in a diminished Rad53 interaction.