Figure S1.—(A) Suppression of esal’s growth defect by rpd3 was not allele-specific. Serial dilutions of wild-type (LPY5), esal-L254P (LPY12160), esal-L254P rpd3 (LPY12164), and rpd3 (LPY12154) were plated on SC at the permissive and restrictive temperatures. (B) esal rpd3 carrying either wild-type RPD3 (LPY14359), catalytically inactive rpd3-H150A-H151A (LPY14360), or vector (LPY14356) on a HIS3 plasmid were plated at permissive and restrictive temperatures on SC-His to assay for growth. (C) Deletion of RPD3 did not bypass the need for ESA1. The following strains, wild-type (LPY12200), esal (LPY12204), esal rpd3 (LPY12206) and rpd3 (LPY12202), all carried a wild-type URA3/ESA1 plasmid (pLP796). These strains were subjected to a plasmid shuffle by counterselection on 5-FOA.