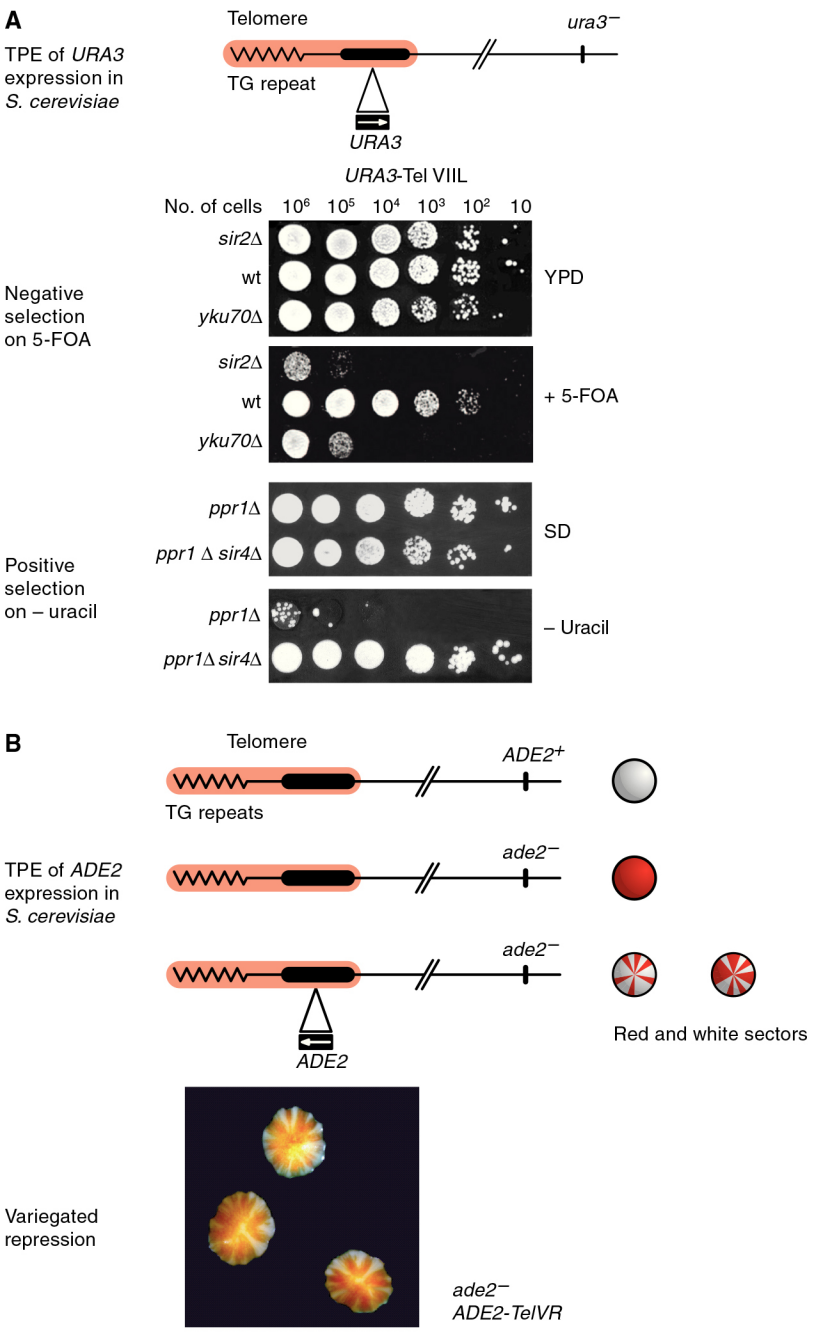

Figure 1. Silencing and TPE in yeast. (*A*) The *URA3* gene, inserted near the telomeric simple TG-rich repeat at the left arm of Chr VII, is silenced by telomeric heterochromatin in this yeast strain. In normal rich media (YPD) no growth difference can be detected between wild-type (wt) cells that repress the subtelomeric *URA3* gene, and silencing mutants that lose telomeric heterochromatin and express *URA3*. In media containing 5-FOA (*middle* panel), on the other hand, cells that repress *URA3* (e.g., wt cells) can grow, whereas cells that express it (*sir2*Δ and *yku70*Δ) cannot, because the *URA3* gene product converts 5-FOA to the toxic intermediate 5-flourouracil. The serial dilution/drop assay allows detection of silencing in as few as 1 in 10^6 cells. In cells deleted for the *URA3* activator, Ppr1 (*ppr1*Δ), one can screen for repression by plating on synthetic dextrose (SD) medium, lacking uracil. In this case, silencing the gene inhibits colony growth. (*B*) Cells containing the wild-type *ADE2* gene produce a colony that is white, whereas those containing mutant *ade2* appear red, because of the accumulation of a reddish intermediate in adenine biosynthesis. When the *ADE2* gene is inserted near the telomere at the right arm of Chr V it is silenced in an epigenetic manner. The silent *ADE2* state and the active *ADE2* state in genetically identical cells are both inherited creating red and white sectors in a colony.