CORRIGENDA

Mutational analysis of a transcriptional activation region of the VP16 protein of herpes simplex virus
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The authors wish to apologize for incorrect versions of Figures 2, 3, 4 and 5 that were published in the above paper. The correct figures and corresponding legends are presented below.

Figure 2. Relative activities in yeast of alanine-scanning mutants of the VP16C subregion. Alanine substitutions were constructed at each position of VP16C except glycines and existing alanines. The activities of Gal4–VP16C fusion proteins bearing these substitutions were assayed using a β-galactosidase reporter gene as described in the legend to Figure 1. The position of each mutation is represented by the amino acid sequence of VP16C along the horizontal axis of this figure. Bars indicate mean activity (with standard deviation) of β-galactosidase in yeast cell extracts from at least three parallel cultures, expressed relative to the activity of the wildtype Gal4–VP16C fusion protein (indicated by +). β-galactosidase activities in extracts lacking the Gal4–VP16C fusion protein (indicated by –) were negligible. The activity of a double substitution of alanine for both F473 and F475 is shown at the right end of the figure.

Figure 3. Relative activities of Gal4–VP16C alanine-scanning mutants in mammalian cells. Each of the alanine-scanning mutations described in Figure 2 was recloned into a mammalian expression plasmid and transfected into mouse L cells with a reporter gene that expresses chloramphenicol acetyltransferase under control of Gal4 binding sites (pGbcAT). The CAT enzyme activities in cell extracts were assayed by the fluor-diffusion method, adjusted to ensure that measurements were within the linear range of the assay. Bars indicate mean activities (with standard deviations) of CAT activity from at least three plates of cells transfected with a given Gal4–VP16C mutant expression plasmid, relative to the activity of the wildtype Gal4–VP16C plasmid (indicated by +).
Figure 4. Relative activities in yeast of Gal4–VP16C mutants bearing various amino acid substitutions at specific positions. Mutant Gal4–VP16C proteins with substitutions at F473 (A), F475 (B), E476 (C), M478 (D) or F479 (E), were assayed for their ability to activate expression of a β-galactosidase reporter gene as described in Figure 1. The individual substitutions are indicated along the horizontal axis of each panel in descending order of activity from left to right, with the mean activity (with standard deviations) from at least three cultures for each mutant being represented by a vertical bar. (B) also indicates the activity of one mutant with leucine substitutions at both F473 and F475.

Figure 5. Relative activities in mammalian cells of Gal4–VP16C fusion proteins with various substitutions at specific positions. Each of the VP16C mutants described in Figure 4 was recloned into a mammalian expression vector and assayed for the ability to activate expression of a CAT reporter gene as described in Figure 2. The individual substitutions are indicated along the horizontal axis of each panel in descending order of activity from left to right, with the mean activity (with standard deviations) from at least three plates of transfected cells for each mutant being represented by a vertical bar.