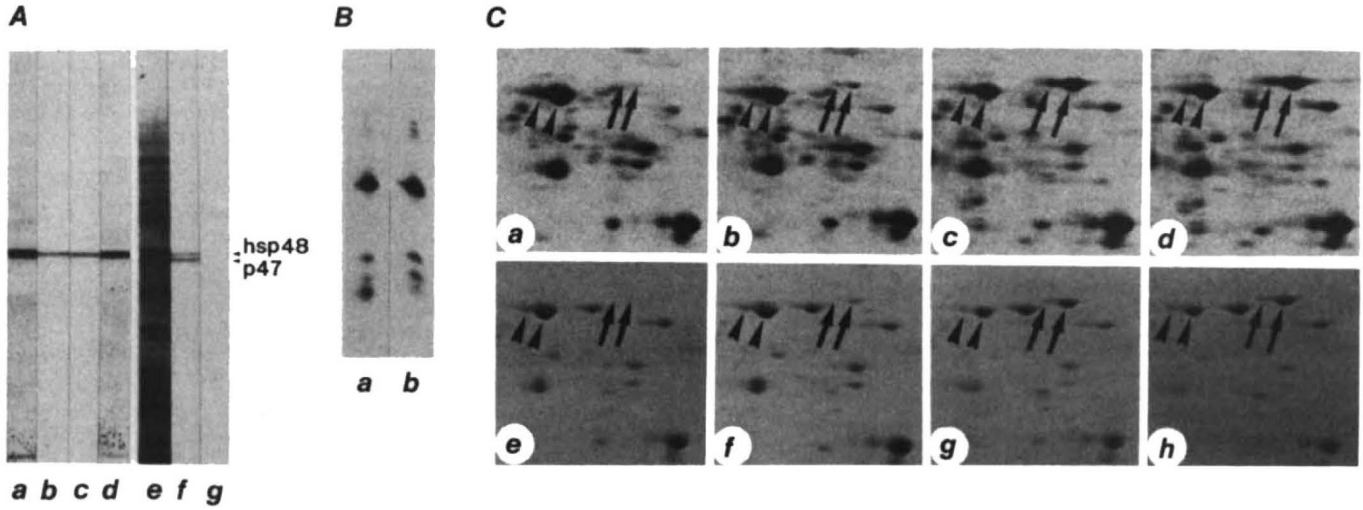

Fig. 3 *A*, Immune blotting of yeast whole-cell extracts and authentic yeast enolase with anti-yeast HSP48 antibody. Purified HSP48 (lanes *a*, *b*), yeast enolase (lanes *c*, *d*) and extracts of X2180-1A cells in the stationary phase (lanes *e*–*g*) were resolved in SDS-PAGE and electrophoretically transferred to a nitrocellulose sheet. The sheet was stripped and strips were separately incubated with 1:500 diluted rabbit yeast anti-HSP48 antiserum (lanes *b*, *c*, *f*) and preimmune rabbit serum (lane *g*), followed by incubation with peroxidase-conjugated goat anti-rabbit IgG (Cappel). A strip was stained with Auro Dye (Janssen) (lanes *a*, *d*, *e*). *B*, Peptide mapping of HSP48 and p47. The spots of ^{35}S-methionine-labelled HSP48 (lane *a*) and p47 (lane *b*) were subjected to peptide mapping with *S. aureus* V8 protease as described in Fig. 1 legend. *C*, synthesis rate and content of HSP48 and p47 in heat-shocked or G_0-arrested yeast cells. *a*, *e*, Exponentially growing X2180-1A cells at 23 °C in synthetic complete medium; *b*, *f*, heat-shocked cells at 36 °C for 1 h; *c*, *g*, cells kept in stationary phase for 24 h; *d*, *h*, cells starved of sulphur for 24 h by incubation in sulphur-free medium[8]. The cells in each of the above cultures were pulse-labelled with ^{35}S-methionine and chased as described in Fig. 1 legend. The whole proteins (25 μg each) extracted from these labelled cells were analysed in two-dimensional gel electrophoresis as described for Fig. 1. Protein spots were revealed by autoradiography (*a*–*d*) and staining (*e*–*h*). A pair of arrowheads indicate p47A (left; acidic form) and p47B (right; basic form). A pair of arrows indicate HSP48A (left) and HSP48B (right).

during differentiation and maturation of neurones and teratocarcinoma and neuroblastoma cell lines[15–18].

The expression of the enolase genes seems to be regulated by many systems. First, it depends on carbon sources in the media[14]. Second, we have shown previously that the *HSR1* gene negatively regulates the synthesis of six proteins—HSP48A, HSP48B, two G_0-induced proteins (p73 and p56) and two unidentified proteins (p63 and p60)[8]. Third, exposure of *HSR1* cells to mild heat shock increases the synthesis rates of the two HSP48s and of other HSPs[7]. Finally, *hsr1* cells starved of sulphur or arrested in the stationary phase synthesize HSP48A, HSP48B, p73 and p56 at higher rates than do exponentially growing *hsr1* cells[8]. These results suggest that the synthesis rate of HSP48 (enolase 1) is affected by at least three factors, the *HSR1* gene, growth conditions and heat shock, although these factors may partly overlap. In agreement with our results, McAlister and Holland have noted that the content of enolase 1, but not of enolase 2, is significantly increased in yeast cells grown to the stationary phase[14].

Finally, the question arises of whether HSP48 functions solely as a glycolytic enzyme when it is involved in growth control and in protecting yeast cells from lethal heat shock. The synthesis of other glycolytic enzymes linked to enolase, such as glyceraldehyde 3-phosphate dehydrogenase or pyruvate kinase, is not significantly altered in *hsr1* cells or when *HSR1* cells are preheated by 36 °C (ref. 19 and our unpublished observation). This result is consistent with the hypothesis that the heat-shock resistance acquired by the *hsr1* mutation and heat pretreatment may be ascribed to some function associated with HSP48 other than enolase activity. Analysis of the intracellular distribution and molecular interactions of HSP48 (enolase 1) with other molecules, especially in comparison with p47 (enolase 2), should provide more direct evidence.

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Erratum

Colour, albedo and nucleus size of Halley's comet

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Nature **315**, 122–124 (1985).

ON page 122, column 1, line 3 of the first paragraph should contain the values J (1.25 μm) and V (0.55 μm).