



Fig. 2 The regression of corrected RIDA on the square of body weight in single 4-day old male flies from the Montgomery inbred line (homozygous for the *Adh^S* allele). The dotted line represents the overall regression ($y = 1.915 - 0.144x$, $P < 0.001$) which is estimated for comparison with Fig. 1. The measurements were made in two separate generations, represented by open and closed circles. The difference between the generations, due to uncontrolled environmental differences, is very clear. The methods were identical to those given in Fig. 1 legend, except that individual flies were homogenised, and the homogenates centrifuged cold at 13,000g for 2 min, and that the gels contained 3% sheep antiserum. Each point represents the mean of two replicate gels.

approximately proportional to the cube of the weight ($b = 3.13$), and once more there is a fourfold range. The significant difference between the slopes of the regressions shown in Figs 1 and 2 suggests that the lines differ in their responses to the environmental factor that increases the quantity of ADH, but this remains to be confirmed.

Because the weight of adult flies is known to be influenced by the conditions of larval growth, we introduced newly hatched first instar Kaduna *SS* larvae into 2.5-cm shell vials containing 10 ml of dead-yeast-cornmeal-molasses medium with various quantities of dead yeast. Four vials had 50 g of yeast per l of medium, four had 10 g, four had 2 g and four had no yeast at all. The quantities of ADH in single 4-day-old adult males that emerged from these vials were measured by radial immunodiffusion. The results, given in Table 1, show that although the progressive addition of yeast increased the mean body weight by about 30%, it more than doubled the average quantity of ADH. Within the treatment groups there was only a slight association between body weight and ADH. It did not depart from linearity, and was significant in only one group (10 g yeast per l). Between the groups, however, the association is very striking, and the differences are highly significant ($P < 0.001$).

Table 1 The effects of different concentrations of dead yeast on the amount of ADH protein

Amount of dead yeast (g per l medium)	No. of vials	No. of 4-day male flies measured	Mean weight mg (s.e.)	Mean RIDA mm ² (s.e.)
0	4	18	0.719(0.015)	30.13(1.37)*
2	4	17	0.791(0.021)	47.88(2.89)*
10	4	18	0.901(0.015)	81.83(2.02)
50	4	20	0.906(0.018)	80.70(1.73)

1st instar larvae were obtained from eggs deposited by Kaduna *Adh^S* homozygotes on laying dishes, and were grown in 2.5-cm shell vials containing 10 ml of medium (50 larvae per vial) at 25 °C. The methods were otherwise identical to those given in Fig. 2 legend. The asterisks represent means based on RIDAs that were significantly heterogeneous between vials ($P < 0.05$). Despite this heterogeneity the differences in RIDA between treatments are highly significant ($P < 0.001$).

These findings have several implications. The amount of enzyme is 'allometric' with body weight, rather than strictly proportional to it. This means that conventional ways of correcting for body size (dividing the activity or quantity of enzyme either by the body weight itself or by the total soluble protein, which is proportional to body weight) may give seriously misleading results. Supposed genetic differences in the activity or quantity of enzyme may merely reflect persistent disparities of body weight and/or the conditions of culture. This uncertainty applies to the report of a twofold difference in the quantity of ADH between genotypes⁷, and to the recent claim that a 30% difference between a line selected for resistance to ethanol and an unselected control was due to the action of 'regulatory' genes¹⁴. It may also apply to work on other *Drosophila* enzymes, for which the effects of variations in the medium have not been studied. Our results suggest that screening for activity mutants may be greatly improved by a careful control of the medium. We note that disparities between lines in the conditions of culture may seem to be genetic because micro-organisms can be passed on from the medium of the parents to that of their offspring.

The causes of the changes in the quantity of ADH are unknown. They could be due to an increased synthesis of enzyme in favourable conditions, to a decreased breakdown, or to changes in the time course of synthesis or breakdown. These possibilities are being studied, but in any event there is evidence that the precipitating factor is some part of the larval environment correlated with the quantity of yeast. A constituent or product of the yeast is the most obvious candidate. Because the flies were raised on dead yeast the factor is unlikely to be ethanol. Two per cent ethanol added to medium containing 2 g yeast per l does not increase the quantity of ADH (unpublished observation). If the changes prove to be a case of induction, and if the inducing substance can be identified, these observations will offer interesting possibilities for the study of eukaryotic gene control.

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Erratum

In the letter 'Influence of leaves on sporophore production by fungi forming sheathing mycorrhizas with *Betula* spp.' by F. T. Last *et al.*, *Nature* **280**, 168–169, in paragraph 2 line 5, for '*Betula pendula* collected by Roth', read '*Betula pendula* Roth collected from . . .'

Corrigendum

In the letter 'Non-selective isolation of human somatic cell hybrids by unit-gravity sedimentation' by P. L. Chang *et al.*, *Nature* **278**, 168–169, in paragraph 4 lines 9–10 the letters A and B referring to Fig. 1 have been transposed. It should read '... 2.4 units (A) ... compared to 4.1 units (B) ...'