the calcium-influx signal in lymphocytes and in other tissues, particularly brain and testis where it is more abundantly transcribed. CAML may bind cyclophilin B present at the calciosome[22] (a subspecialized region of the endoplasmic reticulum involved in calcium homeostasis), and may regulate intracellular calcium release or generate the signal responsible for opening plasma membrane calcium channels. □

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CORRECTIONS

The genomic mutation rate for fitness in *Drosophila*

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Nature **359,** 58–60 (1992)

IT has come to our attention that an important stock used in this paper was probably contaminated during the experiments we reported. We accumulated spontaneous mutations in the virtual absence of natural selection on many copies of a single second chromosome of *Drosophila melanogaster*. To provide a control stock for these accumulation lines, we pooled six of the best performing lines, in two replicates, then maintained these stocks at a large population size to allow natural selection to purge any deleterious variation. In our paper we reported results from generation 44 of mutation accumulation. S. Nuzhdin and T. Mackay (personal communication) examined insertion sites of the transposable element *297* by *in situ* hybridization in the control stocks and found that about half of the insertion sites in both replicates were polymorphic. Our subsequent analyses of insertion sites of *297* and *roo* have confirmed this observation. This level of polymorphism is too high to be explained by transposition of these elements, but still far lower than that expected in an outbred population. The results are most consistent with contamination of one of the six lines originally used to found both replicates of the control stock. We have also examined transposition sites in six mutation lines extracted at generation 62 of mutation accumulation for *297* and *roo*, and S. Nuzhdin examined an additional six lines for *297*; none of these lines shows evidence for contamination (D. Houle *et al. Genetics*, in the press).

Consequently, we do not have a valid estimate of the rate of decline in mean fitness. Since our estimates of the genomic mutation rate and the average effect of a mutation are functions of this rate, our data cannot be used to make these estimates. On the other hand, our estimate of the increase in variance among replicate chromosomes is still valid. □

Mouse microcytic anaemia caused by a defect in the gene encoding the globin enhancer-binding protein NF-E2

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WE previously reported that the globin enhancer-binding protein NF-E2 was defective in the mouse hypochromic microcytic anaemia mutation *mk*. We based our conclusion upon the following. First, NF-E2 mapped to within 1 cM of the *mk* locus on mouse chromosome 15. Second, the *mk* phenotype, which includes decreased globin synthesis and defective iron uptake, made NF-E2 a logical candidate gene, especially once we established that NF-E2 was expressed in the proximal intestine. Third, the *mk* NF-E2 allele had a single base-pair difference at nucleotide 851 (T→C), predicting a valine-to-alanine substitution at amino acid 173, which was not present in DNA from its normal inbred strain of origin, DBA/2J (the *mk* mutation originally arose on C57BL/6J × DBA/2J F_2 mice; by Southern blot analysis, we determined that *mk* carried the DBA/2J NF-E2 gene). Since publication, however, S-J. Lu, S. Rowan, M. R. Bani and B.-D. Yaacov (*Proc. natn. Acad. Sci. U.S.A.*, in the press) have shown, and we have confirmed, that normal inbred BALB/c mice also have C at position 851 of the NF-E2 gene, suggesting that the valine-to-alanine change is polymorphic in nature and does not represent the primary gene defect producing the *mk* mutation. We conclude that NF-E2 is closely linked to *mk* on mouse chromosome 15 but must be further evaluated in regards to its role, if any, in iron metabolism and in producing the *mk* phenotype. □

ERRATUM

Correlated neuronal discharge rate and its implications for psychophysical performance

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THE ordinate axis in Fig. 3*a* of this Letter should run from 1.0 to 10, and not from 0.1 to 10 as published. □