ligands may also participate in the regulation of PER or SIM function in *Drosophila*.

Although more PAS mutants need to be analysed, the result of the PERL (V243→D243) change may explain how this affects circadian rhythms, namely by interfering with proper protein–protein interaction. The data shown in Fig. 4*b* indicate that these could include homotypic interactions which might be a molecular basis for the semi-dominance of most *per* mutations[1,9,14]. PER may also engage in heterotypic interaction with unknown protein partners, for example by sequestering one or more bHLH–PAS proteins through a PAS-mediated protein–protein interaction, as in the mechanism proposed for the function of the HLH proteins ID (inhibitor of MyoD) and EMC (extra macrochaetae)[15,16]. Such a mechanism could underlie the feedback loop involving PER and the transcription of its own messenger RNA[6–8], and pertain to the temporal regulation of gene expression in other organisms that is an important feature of the circadian oscillator (for example, see refs 17–21). This would be supported by the identification of a protein 'partner' that interacts with PER through its PAS domain. □

Received 23 February; accepted 28 April 1993.

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ACKNOWLEDGEMENTS. We thank K. Curtin and K. Kreutter for help with site-directed mutagenesis; S. Crews and O. Hankinson for cDNAs encoding SIM and ARNT, respectively; H.V. Colot, J. C. Hall and R. Sen for comments on the manuscript; and T. Tishman for secretarial assistance. The work was supported by a grant from the NIH to M.R.

ERRATUM

Virus persistence in acutely infected immunocompetent mice by exhaustion of antiviral cytotoxic effector T cells

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Nature **362**, 758–761 (1993)

DURING the production process the top and bottom panels of Fig. 3 in this letter were accidentally switched. The correct figure is shown below; the legend remains the same.