pmn mice. The traditional dogma that loss of motor neuron somata represents the primary lesion in ALS has never been supported by convincing data; on the contrary, it has been seriously questioned for both ALS[5–8] and spinal muscular atrophy[9]. Fewer myelinated nerve fibres are present in distal compared to proximal segments of the phrenic nerves of ALS patients[10], arguing in favour of a 'dying back' process as observed in *pmn* mice. Moreover, the pronounced axonal degeneration and regeneration in the ventral roots of ALS patients[11] is not compatible with the assumption of primary degeneration of motor neuron somata. Indeed, electron micrographs of degenerating motor endplates[7] strikingly resemble the initial changes detected in *pmn* mice.

We conclude from our data that CNTF not only prevents the loss of motor neuron somata, but also maintains axonal integrity and supports axonal regeneration. A recent report on the effects of CNTF on sprouting of motor neurons from endplates and nodes of Ranvier[12] is in agreement with this interpretation. The consequence of these effects is functional restoration of the motor innervation of the paralytic skeletal musculature. Thus, that CNTF interferes with pathological manifestations at both functional and morphological levels appears relevant to the potential usefulness of CNTF in the treatment of ALS patients.

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1. Vrbová, G., Greensmith, L. & Sieradzan, K. *Nature* **360**, 216 (1992).
2. Sendtner, M. *et al. Nature* **358**, 502–504 (1992).
3. Schmalbruch, H. *et al. J. Neuropath. exp. Neurol.* **50**, 192–204 (1991).
4. Cork, L. C. *et al. Lab Invest.* **61**, 69–76 (1989).
5. Hirano, A. *et al.* in *Motor Neuron Diseases* (eds Norris, F. H. & Kurland, L. T.) 51–60 (Grune & Stratton, New York, 1969).
6. Ghatak, N. R. *et al. J. Neuropath. exp. Neurol.* **45**, 385–386 (1986).
7. Festoff, B. W. in *Amyotrophic Lateral Sclerosis* (ed. Davidoff, R. A.) Handbook of the Spinal Cord Vol. 4, 607–684 (Dekker, New York, 1987).
8. Festoff, B. W. in *Handbook of Amyotrophic Lateral Sclerosis* (ed. Smith, R. A.) 661–685 (Dekker, New York, 1992).
9. Hausmanowa-Petrusewicz, I. in *Spinal Muscular Atrophy Infantile and Juvenile Type*, 1–180 (National Center for Scientific, Technical and Economic Information, Warsaw, 1978).
10. Bradley, W. G. *et al. Ann. Neurol.* **14**, 267–277 (1983).
11. Hanyu, N. *et al. J. neurol. Sci.* **55**, 99–115 (1982).
12. Gurney, M. E., Yamamoto, H. & Kwon, Y. *J. Neurosci.* **12**(8), 3241–3247 (1992).

Per — no link to gap junctions

SIR — It has recently been determined that the results of certain experiments originally performed in the laboratory of one of the authors (D.C.S.), and described in our paper[1] entitled "The *Drosophila* clock gene *per* affects intercellular junctional communication", cannot be reproduced. This paper reported changes in gap-junction-mediated intercellular communication in salivary glands from *D. melanogaster period* (*per*) mutants. Three classes of coupling measurements were performed in the original study: (1) measurement of dye spread among cells in per^0, per^s and per^+ salivary glands; (2) determination of electrotonic coupling in intact salivary glands of these genotypes; and (3) determination of electrical coupling between isolated cell pairs from dissected salivary glands of these genotypes. For all three classes of experiments, measurements were performed by D.C.S. on material supplied by T.A.B. and M.K.B. This work led to a central conclusion put forth in the paper — that by all three criteria strong differences in intercellular coupling distinguished the *per* genotypes.

A reinvestigation of dye coupling[2] has concluded that no consistent differences in intercellular coupling in salivary glands can be attributed to *per* locus mutations. This new report[2] was initiated by K. Flint, M. Rosbash and J. C. Hall, who communicated their unpublished results to us. Similar data were then obtained by D.C.S. and K. Siwicki, and these two sets of data were pooled. The new paper, and the absence of any indication as to the cause of the discrepancy between the two studies, causes us to question the validity of the original measurements of dye and electrical coupling. We also wish to point out that these data were essential for support of a model proposed in our paper: that *per* might control circadian rhythms by modulating intercellular junctional communication in the nervous system.

In addition to the studies of intercellular communication described above, our paper included data supporting two other conclusions: (1) that antibodies against the *per* product can detect an antigen of unexpectedly high molecular mass, which is reduced in size by treatment with heparinase II; and (2) that *per* expression can be detected in developing *Drosophila* salivary glands. These experiments were carried out in the laboratory of M.W.Y. We note that our detection of antigen with proteoglycan properties was consistent with an earlier, related biochemical study of *per* antigens by others[3], and we know of no studies that further directly address this issue. We have no reason to question the data presented in support of this aspect of our study. Indirect evidence does suggest that such modification of *per* may not be required for expression of circadian rhythms[4]. As has been previously acknowledged[5], *per* expression in developing salivary glands has not been confirmed by studies from another laboratory[6,7], while, for example, work in M.W.Y.'s laboratory indicates consistent labelling of these glands with *per* antibodies and with strand-specific *per* RNA probes following *in situ* hybridization. More direct support for *per* expression in developing salivary glands has come from blot analysis of RNA from hand-dissected larval tissue[8]. The latter analysis also shows that hybridizing transcripts from the glands are indistinguishable in size from *per* RNA extracted from heads[8]. It may also be important that evidence for *per* expression in *adult* salivary glands has been presented by others[6].

In conclusion, we wish to retract those portions of our paper reporting evidence for the involvement of *per* in gap-junction-mediated intercellular communication. We acknowledge the shared responsibilities for that work as coauthors of a collaborative effort. We deeply regret any difficulties our original report may have caused other researchers working in this field.

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1. Bargiello, T. A. *et al. Nature* **328**, 686–691 (1987); correction *Nature* **360**, 610 (1992).
2. Siwicki, K. K., Flint, K., Hall, J. C., Rosbash, M. & Spray, D. C. *Biol. Bull.* (in the press).
3. Reddy, P., Jacquier, A. C., Abovich, N., Petersen, G. & Rosbash, M. *Cell* **46**, 53–61 (1986).
4. Rosbash, M. & Hall, J. C. *Neuron* **3**, 387–398 (1989).
5. Saez, L. & Young, M. W. *Molec. cell. Biol.* **8**, 5378–5385 (1988).
6. Liu, X., Lorenz, L., Yu, Q., Hall, J. C. & Rosbash, M. *Genes Dev.* **2**, 228–238 (1988).
7. Siwicki, K. K., Eastman, C., Petersen, G., Rosbash, M. & Hall, J. C. *Neuron* **1**, 141–150 (1988).
8. Baylies, M. K., Weiner, L., Vosshall, L. B., Saez, L. & Young, M. W. in *Molecular Genetics of Biological Rhythms* (ed. Young, M. W.) 123–154 (Dekker, New York, in the press).