however, rather small because the inactive motor units were very much in the minority. Their substantially larger sizes would be balanced by a smaller change in size among a larger population of active motor neurons. Moreover, detection of any such difference entails a comparison of motor-unit tensions scaled to the maximum direct tension — without using the within-animal normalization we employed to circumvent the significant individual variability in average motor unit sizes found even in normal animals. Nonetheless, a small bias in the appropriate direction was reported in our initial sample. Including the additional data obtained in the long-term recovery experiments (see above) makes this difference statistically significant ($P < 0.05$).

In summary, we feel we have now demonstrated a clear effect of differential activity on neuromuscular competition, and we hope that we have adequately addressed any residual doubts. We concur with Ribchester that differential activity is not the only factor contributing to the selective stabilization of synapses but it is likely to be important, given that the effects we observed were quite substantial (> 45 per cent), and particularly because of the short time for which differential activity was maintained. Effects of this magnitude, if integrated over the full period of synapse elimination, could in principle make an important contribution to sculpting motor-unit size distributions during normal maturation.

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Sequence patterns in protein kinases

SIR—Following Brenner's report[1] on phosphotransferase homologies, we have designed two sequence patterns that can be used to identify protein kinases and to discriminate them from other proteins. The first of these, derived from the one proposed by Brenner, is (LIV)(HY)XD (FILMVY)XXXNX(FILMV)(FILMV).

The second, much shorter and less degenerate, is (LIV)GXGX(FY)GX(LIV) and is based on alignments of protein kinase sequences in the vicinity of a lysine residue that has been shown[2] to be involved in ATP binding in both oncogenic tyrosine kinases and cyclic (c)AMP-dependent protein kinases.

(For both patterns the amino acids in parentheses are alternatives; X stands for any amino acid.)

```
FPHDWDLFKLMGIGSGGFGPVFESGFIEIL    TMS1 (424–453)
::..  ..:. .:::.:: :.:.. ..::
FPREKLTLRLL-LGSGAFGEVYEGTAVDIL    Human ros (14–42)
```

Sequence comparison of TMS1 and human *ros*. Identical amino acids are indicated by :, different but conserved amino acids by ., and the sequence pattern is underlined.

It must be noted that Brenner's pattern, while identifying most known phosphotransferase sequences, only flags a subset of all known protein kinases. For instance, it fails for cAMP- and cGMP-dependent kinases, protein kinases C and the *abl*, *fes*, *fps*, *ros* and *trk* oncogene products.

The second pattern is a generalization of the LGXGXFGXV pattern first pointed out by Barker and Dayhoff as a conserved element between the *src* gene product and the bovine cAMP-dependent protein kinase[3].

These patterns have been designed and their specificity tested by scanning the SWISS-PROT 5.1 databank (5,300 protein sequences, 1,400,000 residues) using the PC/Gene sequence analysis package (developed by A. Bairoch and D. Gavin, University of Geneva, and distributed by Genofit SA and IntelliGenetics Inc.), as well as the PseqIP 3.0 databank[4] (6,487 protein sequences, 1,700,000 residues) using the SASIP sequence analysis package[5]. A 'reverse translation' of the second pattern was also used to scan GenBank 50.0 (ref. 6) and the EMBL 10.0 nucleotide sequence data library[7].

Table 1 Sequence patterns used to identify protein kinases and discriminate them from other proteins

Protein family	Pattern 1	Pattern 2	Kinase type
cAMP-dependent PK	+	+	ST*
cGMP-dependent PK	+	+	ST
Phosphorylase *b* kinase	+	−	ST
Protein kinase C	+	+	ST
CDC2/CDC28	+	+	?
CDC7	+	−	?
PK-25/YKR	+	+	?
HSV kinase related	+	−	?
Myosin light chain kinase	−	+	?
EGF receptor (*erb-B*, *neu*)	+	+	Y†
Insulin receptor	+	+	Y
abl/dash	+	+	Y
fes/fps	+	+	Y
fgr	+	+	Y
fms	+	+	Y
kit	+	+	Y
LSK-T/*tck*	+	+	Y
met	+	+	Y?
mil/mht/raf	+	+	ST
mos	+	+	ST
pim-1	+	+	?
ros	+	+	Y
src/yes/syn	+	+	Y
trk	+	+	Y

* Serine/threonine; † tyrosine. References to all sequences are available from the authors.

As shown in the accompanying table, the first pattern found every known protein kinase except the myosin light-chain kinases, while the second, shorter pattern flagged all but the gamma chain of phosphorylase *b* kinase, the yeast CDC7 gene product and a kinase-related protein from herpes simplex virus type 1.

Whereas the first pattern did not detect any sequence other than known protein kinases, the second pattern pointed out two other sequences: the hexon-associated protein IX from adenovirus types 7 and 3 (but not the homologous protein from adenovirus types 5 and 12) and the TMS1 protein coded on the Ti plasmid from *Agrobacterium tumefaciens*.

Because of its involvement in plant tumorigenesis, the latter case was investigated further. Using the Fastp program[8] a good but very localized similarity was detected between the TMS1 protein and the *ros* oncogene product (as well as the mouse *pim*-1 oncogene) around the second pattern, as shown in the figure.

We tested other patterns from different conserved regions of protein kinases, but none was as specific as those reported here, which thus seem to be bona fide sequence signatures for protein kinases. Accordingly, it is tempting to propose that the *A. tumefaciens* TMS1 tumorigenic gene product exhibits protein kinase activity.

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Scientific Correspondence

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