interact with a 'flickering cluster' size distribution which has a greater abundance of smaller clusters, thereby causing decomposition.

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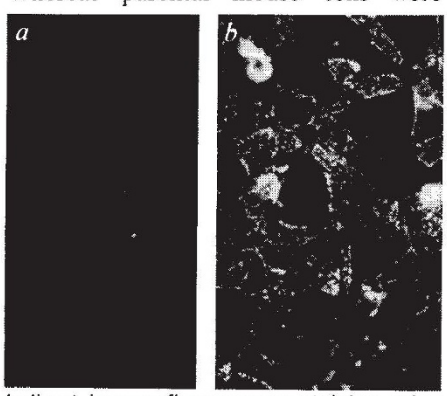
1. Baverstock, K.F. & Cundall, R.B. *Nature* **332**, 312–313 (1988).
2. Colligiani, A., Pinzino, C. & Angelone, R. *Molec. Phys.* **62**, 97–107 (1987).
3. Burns, W.G., Sims, H.E. & Goodall, J.A.B. *Int. J. Radiat. Phys. Chem.* **23**, 143–163 (1983).
4. Clifford, P., Green, N.J.B., Pilling, M.J., Pimblott, S.H. & Burns, W.G. *Int. J. Radiat. Phys. Chem.* **30**, 125–132 (1987).
5. Mozumder, A. & Magee, J.L. *J. chem. Phys.* **45**, 3332–3341 (1966).
6. Schwarz, H.A. *J. phys. Chem.* **75**, 1928 (1969).
7. Erdey-Gruz, T. *Transport Phenomena in Aqueous Solutions* (Akadémiai Kiadó, Budapest, 1974) transl. Ruff, I., rev. Egyed, Y. (Adam Hilger, London).
8. Green, N.J.B., Pilling, M.J., Clifford, P. & Burns, W.G. *J. chem. Soc. Faraday Trans.* **80(1)**, 1313–1322 (1984).
9. Jonah, C.D. & Miller, J.R. *J. phys. Chem.* **81**, 1974 (1977).
10. Burns, W.G., May, R., Buxton, G.V. & Wilkinson-Tough, G.S. *J. chem. Soc. Faraday Trans.* **77(1)**, 1543 (1981).
11. Brenner, D.J. *Int. J. Radiat. Phys. Chem.* **32**, 157–162 (1988).
12. Burns, W.G. & Sims, H.E. *J. chem. Soc. Faraday Trans.* **77(1)**, 2803–2813.

# Unmasking N-CAM

SIR—Using heterokaryon analysis, Pavlath *et al.*[1] recently demonstrated the restricted expression of the antigen reactive with the monoclonal antibody 5.1H11 (ref. 2) to localized regions of the plasma membrane defined by the position of active nuclei. We wish to point out that the antigen recogized by 5.1H11 is human N-CAM, an adhesion molecule originally discovered in neural cells.

Since its initial identification, the molecular nature of the 5.1H11 antigen has remained undefined due to the detergent-sensitive nature of the reactive epitope. We have, however, recently cloned human complementary DNAs encoding the major alternatively spliced N-CAM isoforms of skeletal muscle[3–5]. In the course of expression studies of these human N-CAMs we examined 3T3 fibroblasts stably transfected with human N-CAM cDNAs for reactivity with 5.1H11 using indirect immunofluorescence. Whereas parental mouse cells were unreactive with 5.1H11, all transfected cell lines were strongly reactive. The figure shows an example. As the antibody reacts with a species-specific epitope[6], artefactual or indirect activation of a previously silent mouse gene is excluded. Thus, the 5.1H11 antigen is unequivocally identified as human N-CAM. Similar results were found with many independently derived clones and reactivity was also found with different isoforms of N-CAM, including transmembrane, glycosyl-phosphatidylinositol-linked and secreted forms. Therefore, previous data indicating that a diffusable *trans*-activator regulates 5.1H11 expression in muscle cells[6] in fact relates to regulation of the gene for N-CAM.




Indirect immunofluorescence staining using monoclonal antibody 5.1H11 with *a*, 3T3 cells or *b*, 3T3 cells transfected with a human N-CAM cDNA.

N-CAM expression increases in association with myotube formation and there is also a switch from a splicing pathway generating a transmembrane N-CAM to one producing a glycosyl-phosphatidylinositol-linked isoform[3,4]. Isoform-specific antibodies or cDNAs might thus be used to determine which isoforms are produced in the heterokaryon model and which is localized over active nuclei.

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1. Pavlath, G.K., Rich, K., Webster, S.G. & Blau, H.M. *Nature* **337**, 570–573 (1989).
2. Walsh, F.S. & Ritter, M.A. *Nature* **289**, 60–64 (1981).
3. Dickson, J.G. *et al. Cell* **50**, 1119–1130 (1987).
4. Barton, C.H. *et al. Development* **104**, 165–173 (1988).
5. Gower, H.J. *et al. Cell* **55**, 955–964 (1988).
6. Blau, H.M. *et al. Science* **230**, 758–766 (1985).

# Sequence identity

SIR—We wish to draw attention to the complete identity between the C-terminal sequence of the cytosolic calcium-binding protein (CaBP) p14 and the N terminus of two small neutrophil-immobilizing-factor proteins, NIF-1 and NIF-2.

The two associated cytosolic CaBPs[1], p8 and p14 (also known as CF antigen[2], LI molecule[3], or MRP8 and MRP14 (ref. 4)) are members of a larger family of CaBPs that may be involved in intracellular signal transduction. All the proteins have a molecular mass of about 10,000 with the exception of p14 which has a longer C-terminal sequence following the second calcium-binding domain, prompting speculation that this region of the molecule performs a unique function[5]. By searching the OWL database[6] with the sequence of p14 from position 89–108, we found that it is identical to the N-terminal 20 amino-acid residues of NIF-1 and NIF-2 (refs. 7 and 8), with the exception of an extra alanine residue at the N terminus of NIF-2. We suggest that residues 89–108 of p14 contain a sequence that performs an extra-cellular function of immobilizing neutrophils engaged in chemotaxis. It is also possible, however, that the common sequence of p14 and NIF serves some other function and that the neutrophil-immobilizing activity actually resides in the C-terminal half of the NIF peptides.

A further possibility is that the NIF peptides are formed by proteolytic cleavage of p14 after the tryptophan residue at position 88. If this is so, then two features of the NIF peptides require further explanation. First, cleavage of p14 at position 88 would yield a C-terminal peptide of 26 amino acids in length, whereas NIF-1 and NIF-2 are predicted from their amino-acid compositions to contain 41 and 38 amino acids respectively[8]. But recalculation of the amino-acid composition of NIF-1 for a peptide of 26 residues produces a composition almost identical to that of the C-terminal tail of p14 with the exception of 0.4 mol $mol^{-1}$ of cysteic acid. A similar analysis of NIF-2 could not be accounted for.

It was originally reported that p8 and p14 are associated with a separate macrophage-migration inhibitory factor (MIF)[9]. We now suggest that a neutrophil immobilizing activity is part of the p14 protein itself. Our previous work indicates that the p8, p14 complex (or some component part) is released by myeloid cells during their interaction with vascular endothelium[1]. Therefore p14 could be involved in immobilizing myeloid cells at the endothelial surface both during an inflammatory response and during the normal exudation of myeloid cells into tissues.

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1. Hogg, N., Allen, C. & Edgeworth, J. *Eur. J. Immun.* (in the press).
2. Dorin, J.R. *et al. Nature* **326**, 614–617 (1987).
3. Dale, I., Fagerol, M.K. & Naesgaard, I. *Eur. J. Biochem.* **134**, 1–6 (1983).
4. Odink, K. *et al. Nature* **330**, 80–82 (1987).
5. Lagasse, E. & Clerc, R.G. *Molec. cell. Biol.* **8**, 2402–2410 (1988).
6. Akrigg, D. *et al. Nature* **335**, 745–746 (1988).
7. Goetzl, E.J. & Austen, K.F. *J. exp. Med.* **136**, 1564–1580 (1972).
8. Watt, K.W.K., Brightman, I.L. & Goetzl, E.J. *Immunology* **48**, 79–86 (1983).
9. Burmeister, G., Tarcsay, L. & Sorg, C. *Immunobiology* **171**, 461–474 (1986).

## Scientific Correspondence

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