ing of the northbound tidal wave by the islands and shallow bank to the southwest.

The mean currents are predicted to be of the order of 0.1 m s^{-1} in deep water and up to 3.0 m s^{-1} on the narrow ridge (50–100 m deep) between Lofotodden and Røst (Fig. 2). A lack of data from this area hampers validation of the model; however, we have made comparisons with measurements from channels between the islands east of Lofotodden at Napp, Sundklakk and Gimsøy. Current shifts are predicted to occur about 2 h before high water, with a northwards current at high water and a southwards current at low water, in agreement with observations[10].

A weak, roughly 6-km diameter, clockwise eddy appears in the simulations, centred about 5 km southwest of Lofotodden at the time of current shift on a rising sea. A similar sized anticlockwise eddy appears nearer Lofotodden at the time of current shift on falling sea. The current speed in these eddies is roughly 0.1 m s^{-1}, no comparison to the eddy in ancient literature. The eddies owe their existence to sea-floor topography and friction. West and east of Mosken the current vector rotates clockwise in nearly circular ellipses which may have been interpreted as a large eddy by early observers.

The semi-diurnal components, S_2 and N_2, show a similar amplitude pattern to M_2. The diurnal component, K_1, which interacts with shelf wave modes, produces dominant diurnal currents in Sortlandsundet between stations 7 and 8 (Fig. 1), where the M_2 current is weak, matching observations (Norwegian Hydrographic Service, 1994). The generation and advection of small-scale eddies in the strong tidal jet remain a subject for future studies but these local effects are not likely to alter the large-scale patterns of sea-level variations reported here.

B. Gjevik, H. Moe
A. Ommundsen
Department of Mathematics, University of Oslo, 0316 Blindern, Oslo, Norway
e-mail: bjorng@math.uio.no

1. Peterson, R. G., Stramma, L. & Kortum, G. *Progr. Oceanogr.* **37,** 1–115 (1996).
2. Wahl, T. *Nordic Space Activities* **2–3,** 22–23 (1995).
3. Guerber, H. A. *The Norsemen* 128–130 (Senate Studio Editions, London, 1994).
4. Magnus, O. *Historia om de Nordiska Folken* Swedish edn, 89–90 (Gidlunds, Stockholm, 1982; first published Rome, 1555).
5. Schønnebøl, E. H. in *Historisk-Topografiske Skrifter om Norge og Norske Landsdele* (ed. Storm, G.) 178–218 (A. W. Brøggers Bogtrykkeri, Christiania, Oslo, 1895; original from c. 1590).
6. Dass, P. *Nordlands Trompet* (ed. Seip, D. A.) 94–98 (Aschehoug, Oslo, 1947).
7. Schelderup, A. *Kongelige Norske Videnskabersselskabs Skrifter* **2,** 1, 78–85 (Trondheim, Norway, 1824).
8. Pontoppidan, E. *Norges Naturlige Historie* 123–140 (Reprint by Rosenkilde og Bagger, Copenhagen, 1977; first printed Copenhagen, 1752; English edn, London, 1755).
9. Mabbott T. O. *Collected works of Edgar Allan Poe* **II,** 574–597 (Belknap, Cambridge, MA, 1978).
10. *Den Norske Los* **5,** 6th edn, 241–242 (Norwegian Hydrographic Serv., Stavanger, 1987).
11. Gjevik, B. in *Modeling Marine Systems* (ed. Davies, A. M.) **I,** 187–219 (CRC Press Inc, Boca Raton, Florida, 1990).
12. Gjevik, B., Nøst E. & Straume T. *J. Geophys. Res.* **99,** C2, 3337–3350 (1994).

Morbillivirus in monk seal mass mortality

We have identified a morbillivirus in organs of Mediterranean monk seals (*Monachus monachus*), lost during a recent mass die-off. About half of the population of this highly endangered species, which inhabits the Mauritanian coast of West Africa (Cap Blanc), were killed. The outbreak is reminiscent of several recent morbillivirus outbreaks in aquatic mammals.

In 1988 there was a mass die-off of the two indigenous seal species of northwestern Europe, eventually leading to the deaths of about 20,000 animals. We and others identified a previously unrecognized morbillivirus, phocine distemper virus (PDV), as its primary cause. In subsequent years, several other mass mortalities of pinniped and cetacean species could be attributed to infections with known and newly identified morbilliviruses[1,2]. This prompted us to speculate about the possible threat of morbillivirus infections to highly endangered pinnipeds like the Mediterranean monk seal (*Monachus monachus*)[3]. Two subpopulations of this species exist, one living off the Mediterranean coasts and the other off the Mauritanian coast of West Africa (Cap Blanc), with estimated population sizes of about 500 and 270 animals, respectively.

We and others have observed significant mortality in the West African population since May this year. After finding about 40 dead animals in and around the two caves that they predominantly inhabit, it became clear that a serious disease outbreak was ongoing, which eventually killed more than half of the total population. We autopsied several animals that had washed ashore during the outbreak. Most of these were in a state of advanced decomposition, probably because of the long period before washing ashore and the high ambient temperature. It was clear however, that most of these animals had suffered from respiratory distress, as emphysema and congestion of the lungs were among the most frequent macroscopic findings, consistent with previous morbillivirus infections of seals[2].

In an enzyme-linked immunosorbent assay[3] for the detection of serum antibodies to canine distemper virus (CDV)[1], seven of 17 samples collected from the hearts of dead animals scored positive. We carried out virological analysis using organ samples from the 14 least decomposed carcasses. Using standard procedures[2] we isolated a cytopathic virus in Vero cells from lung and other organ samples of three of these animals. This virus also proved to be infectious for ferrets on parenteral infection. Our preliminary characterization of the virus with polyclonal and monoclonal antibodies[3] showed that the virus is a morbillivirus, probably different from CDV and PDV (data not shown).

We isolated total nucleic acids from lung samples with the guanidinium method[4] and analysed them with a reverse transcriptase polymerase chain reaction (RT–PCR) using random hexanucleotides for first-strand synthesis. We used group-specific primers for the genus *Morbillivirus*, which recognize the *N* gene (primer 1: 5′-ACAAACCA-NGGATTGCTGAAATGAT-3′ and primer 2: 5′-CTGAAYTTGTTCTGAAYTGAGT-TCT-3′) for amplification[5]. A semi-nested PCR using primer 2 and primer 3 (5′-ATC-GARACWATGTACCCGGC-3′) increased the PCR signal for direct sequencing.

Sequencing allowed us to make a phylogenetic comparison with other morbilliviruses, using the Phylogeny Interference Package (provided by J. Felsenstein). We generated a maximum-likelihood tree, and assessed its stability using a bootstrap method (Fig. 1). We found the virus to be most closely related (mean distance, 0.030) to the previously described dolphin morbillivirus (DMV)[2]. The antigenic difference between CDV and the isolated virus, the suboptimal state of the samples, and the fact that morbillivirus infections cause profound immunosuppression, may collectively explain why not all the dead monk seals tested had developed antibodies to CDV.

Further studies, preferably of live and freshly autopsied animals, are needed to confirm that this outbreak, reminiscent of many recent morbillivirus outbreaks in aquatic mammals, is indeed caused by infection with this newly identified monk seal morbillivirus (MSMV). In this light it is noteworthy that we could not detect the

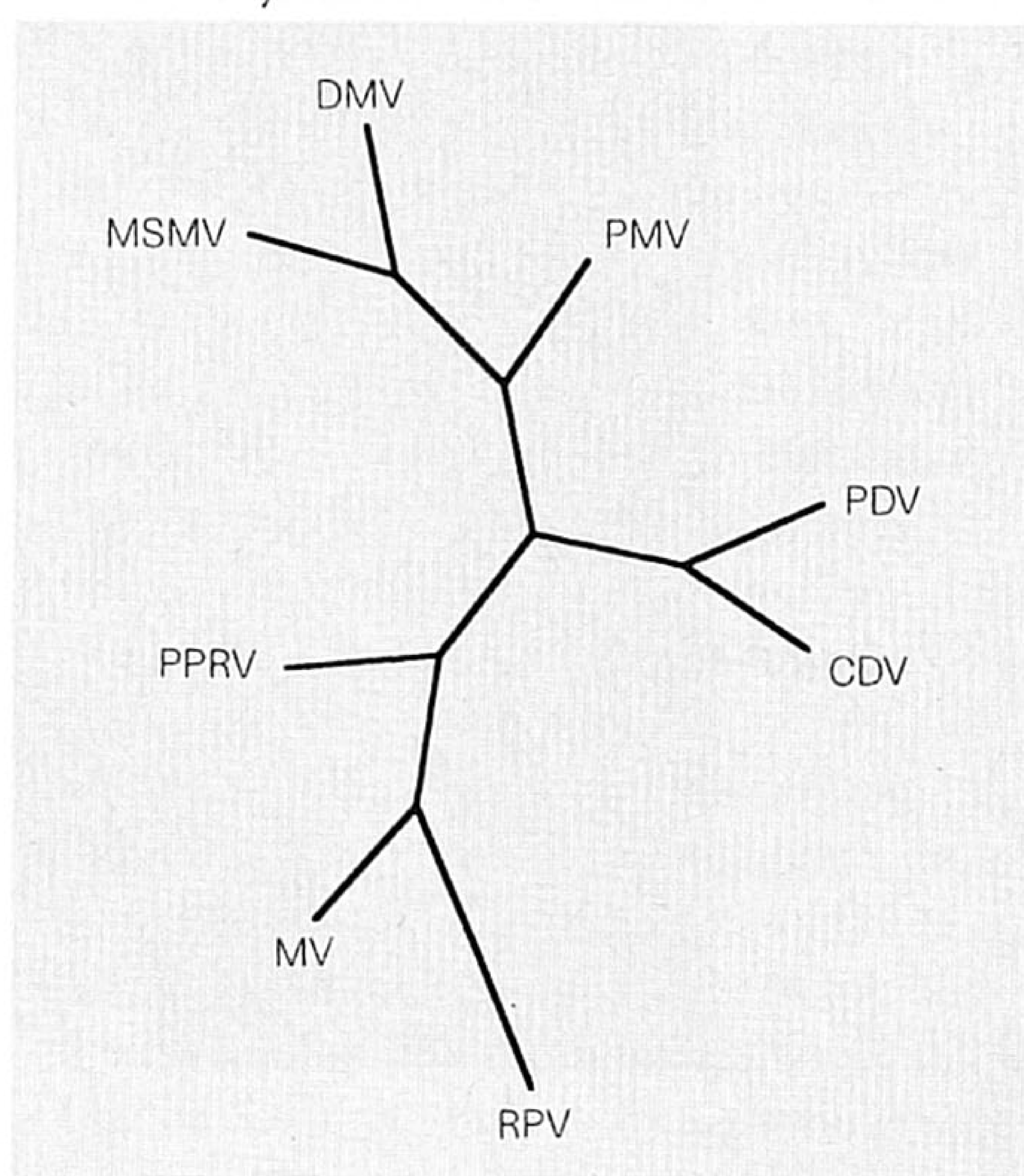

Figure 1 A maximum likelihood tree of N protein fragment sequences (121 nucleotides) of different morbilliviruses. DMV, dolphin morbillivirus; PMV, porpoise morbillivirus; PDV, phocine distemper virus; CDV, canine distemper virus; RPV, rinderpest virus; MV, measles virus; PPRV, peste des petits ruminants virus; MSMV, monk seal morbillivirus.

presence of paralytic shellfish poison in seal carcasses and in a composite mussel sample from the area. Further characterization of the virus as well as investigation of its origin are ongoing, and a rescue and rehabilitation programme for the remaining animals has been initiated by an international collaborative effort.

Albert Osterhaus, Jan Groen
Hubert Niesters
Erasmus University Hospital Rotterdam, Institute of Virology, dr. Molewaterplein 50, 3015 GE Rotterdam, The Netherlands
Marco van de Bildt, Byron Martina
Lies Vedder
Seal Rehabilitation and Research Centre, Hoofdstraat 94A, 9968 A6 Pieterburen, The Netherlands
Joseph Vos, Hans van Egmond
National Institute of Public Health and The Environment, PO Box 1, 3720 BA Bilthoven, The Netherlands
Ba Abou Sidi, Mohamed Ely Ould Barham
Centre National de Recherches Oceanographiques et des Peches, B.P. 22, Nouadhibou, Mauritania

1. Osterhaus, A. D. M. E. & Vedder, E. J. *Nature* 335, 20 (1988).
2. Barrett, T. *et. al. Vet. Microbiol.* 44, 261–265 (1995).
3. Osterhaus, A. D. M. E. *et. al. Vet. Rec.* 130, 141–142 (1992).
4. Chomczynski, P. & Sacchi, N. *Anal. Biochem.* 162, 156–159 (1987).
5. Baron, M. D. & Barrett, T. *J. Gen. Virol.* 76, 593–602 (1995).

α-Synuclein in Lewy bodies

Lewy bodies, a defining pathological characteristic of Parkinson's disease and dementia with Lewy bodies (DLB)[1–4], constitute the second most common nerve cell pathology, after the neurofibrillary lesions of Alzheimer's disease. Their formation may cause neurodegeneration, but their biochemical composition is unknown. Neurofilaments and ubiquitin are present[5–8], but it is unclear whether they are major components of the filamentous material of the Lewy body[9,10]. Here we describe strong staining of Lewy bodies from idiopathic Parkinson's disease with antibodies for α-synuclein, a presynaptic protein of unknown function which is mutated in some familial cases of the disease[11]. α-Synuclein may be the main component of the Lewy body in Parkinson's disease. We also show staining for α-synuclein of Lewy bodies from DLB, indicating that the Lewy bodies from these two diseases may have identical compositions.

We studied formalin- or ethanol-fixed, paraffin-embedded tissue sections of substantia nigra from six patients with idiopathic Parkinson's disease, and from four patients with DLB (all clinically and neuropathologically confirmed cases), as well as cingulate cortex from the DLB patients (one with additional Alzheimer's disease pathology). We stained the tissue with affinity-purified, anti-α-synuclein serum PER2 (diluted 1:200)[12]. This antibody, raised against a synthetic peptide corresponding to residues 116–131 of human α-synuclein, specifically recognizes α-synuclein on immunoblots of human cerebral cortex extracts and does not cross-react with the related β-synuclein[12]. We also stained the sections with anti-β-synuclein serum PER3 (diluted 1:200) raised against a peptide corresponding to residues 99–111 of human β-synuclein[12]. As an absorption control we pre-incubated diluted PER2 overnight at 4 °C with 10 μM recombinant human α-synuclein. For immunohistochemistry we used avidin–biotin, with diaminobenzidine as the chromogen[6,12].

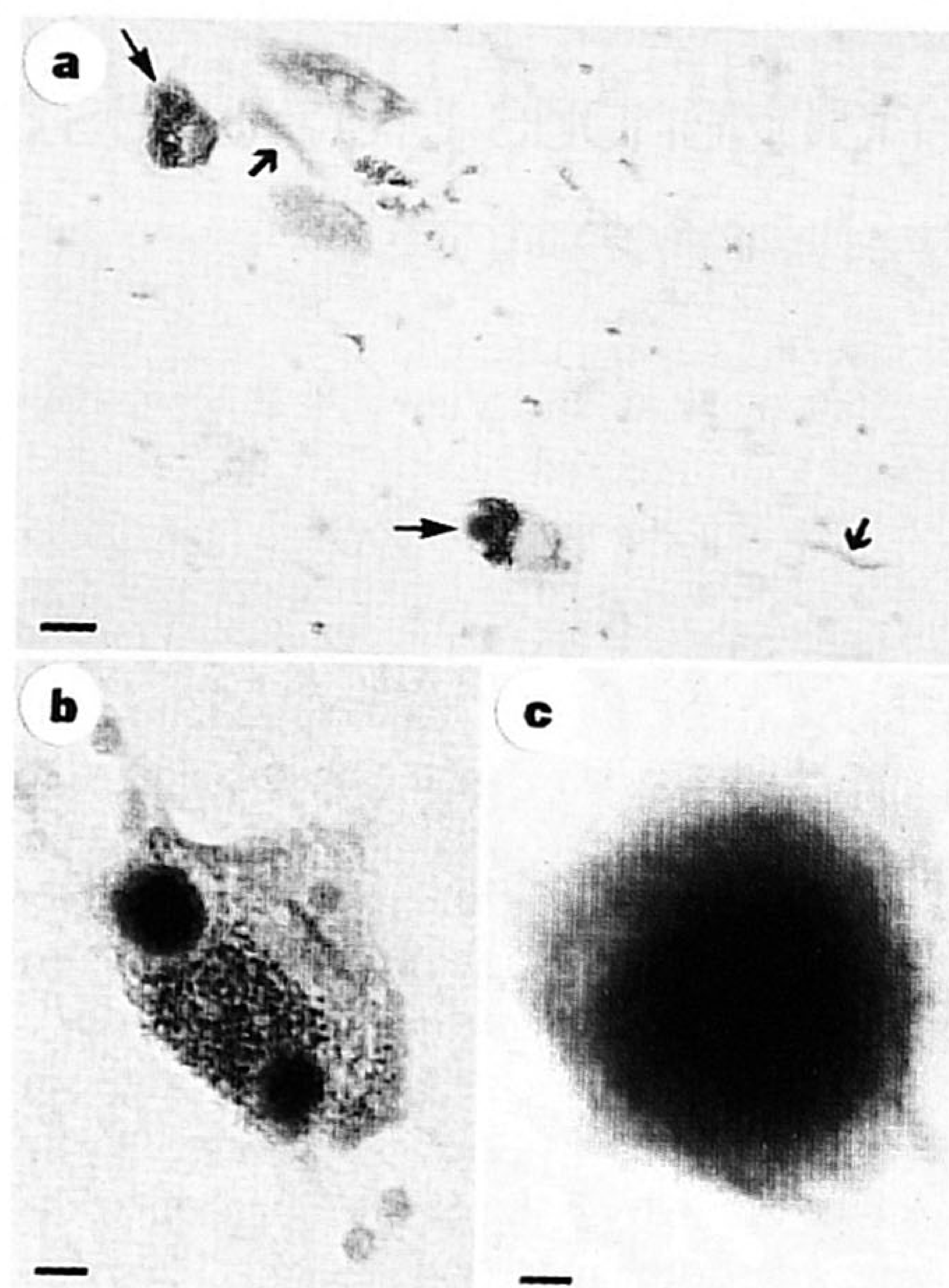

Figure 1 Substantia nigra from patients with Parkinson's disease (from the MRC Cambridge Brain Bank) immunostained for α-synuclein. **a,** Two pigmented nerve cells, each containing an α-synuclein-positive Lewy body (thin arrows). Lewy neurites (thick arrows) are also immunopositive. Scale bar, 20 μm. **b,** A pigmented nerve cell with two α-synuclein-positive Lewy bodies. Scale bar, 8 μm. **c,** α-Synuclein-positive, extracellular Lewy body. Scale bar, 4 μm.

Substantia nigra sections from Parkinson's disease and DLB, and cingulate cortex sections from DLB and DLB with Alzheimer's disease incubated with the α-synuclein antibody PER2 showed staining of numerous brainstem-type and cortical Lewy bodies (Figs 1 and 2). Lewy neurites, which are dystrophic processes with the same immunohistochemical staining profile as Lewy bodies, were also reactive (Figs 1a, 2a). The strong staining made it difficult to distinguish between the core and the corona of the brainstem-type Lewy bodies (Figs 1, 2b). The staining was specific, and did not occur after pre-adsorption of the primary antibody with recombinant α-synuclein.

We obtained similar results with antibody PER1, raised against a synthetic peptide corresponding to residues 11–34 of human α-synuclein, indicating that full-length α-synuclein may be present in the Lewy body (data not shown). We found no specific staining of Lewy bodies or Lewy neurites with the β-synuclein antibody PER3.

We also stained tissue sections from Parkinson's disease and DLB with the ubiquitin monoclonal antibody 1510 (Chemicon, diluted 1:500)[13]. Double-staining of substantia nigra sections from Parkinson's disease with PER2 and 1510, showed staining of similar numbers of Lewy bodies and neurites with each antibody. Similarly, in adjacent tissue sections of substantia nigra and cingulate cortex from DLB, we found comparable numbers of Lewy bodies and neurites stained with PER2, antibody 1510 or undiluted neurofilament antibody RMO32, a monoclonal antibody to a phosphorylated epitope in the mid-sized neurofilament subunit which

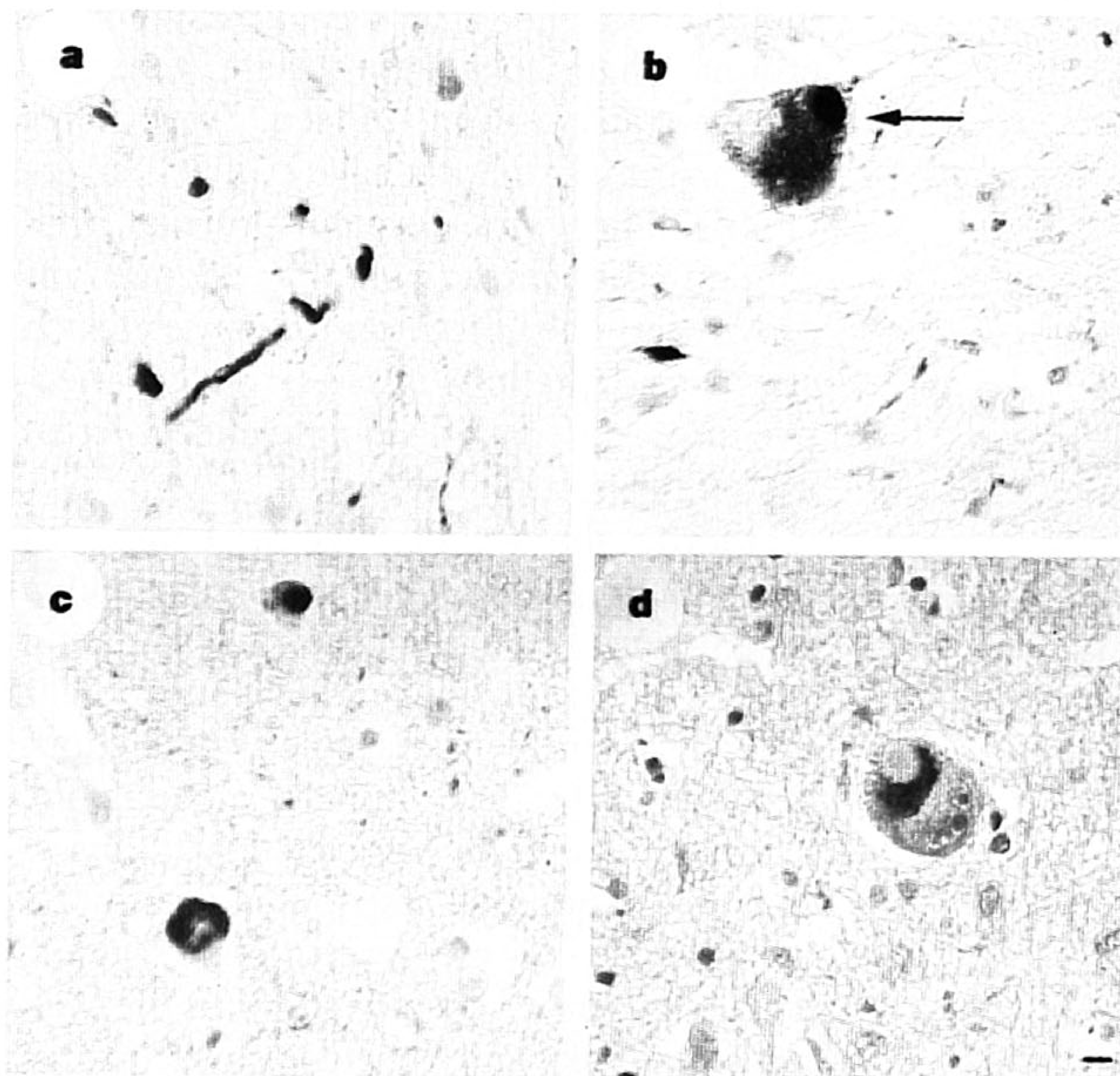

Figure 2 Tissue from patients with DLB (from the tissue collection of the Department of Pathology and Laboratory Medicine, University of Pennsylvania) immunostained for α-synuclein. **a,** α-Synuclein-positive Lewy neurites in the substantia nigra. **b,** α-Synuclein-positive Lewy body (arrow) in pigmented nerve cell of the substantia nigra. **c,** Two α-synuclein-positive Lewy bodies in the cingulate cortex. **d,** Haematoxylin and eosin-stained section of substantia nigra with a pigmented nerve cell containing a Lewy body. Scale bar for **a**–**d**, 10 μm.