MSU-2; a trend also borne out by physical evidence of the retreat of tropical alpine glaciers and ice caps[20] and changes in freezing levels[21]. Although the MSU-2R trend has significant uncertainties and should be interpreted with caution, we conclude that the satellite record is otherwise excellent for examining interannual variability of tropospheric temperature. □

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1. Houghton, J. T. *et al.* (eds) *Climate Change 1995: The Science of Climate Change* (Cambridge Univ. Press, 1996).
2. Hansen, J., Ruedy, R., Sato, M. & Reynolds, R. *Geophys. Res. Lett.* **23,** 1665–1668 (1996).
3. Christy, J. R., Spencer, R. W. & McNider, R. T. *J. Clim.* **8,** 888–896 (1995).
4. Spencer, R. W., Christy, J. R. & Grody, N. C. *J. Clim.* **3,** 1111–1128 (1990).
5. Spencer, R. W. & Christy, J. R. *J. Clim.* **5,** 858–866 (1992).
6. *Economist* 23 March, 83–85 (1996).
7. Michaels, P. J. *Forging Consensus: Climate Change and the United Nations* (George C. Marshall Inst., Washington DC, 1996).
8. Michaels, P. J. (ed.) *World Climate Report* Vol. 1, No. 17 (1996).
9. Singer, S. F. *Science* **271,** 581–582 (1996).
10. Spencer, R. W. in *State of the Climate Report A World in Perspective* (ed. Michaels, P. J.) 9–11 (Western Fuels Assoc. Arlington, VA, 1996).
11. Shah, K. P. & Rind, D. *J. Geophys. Res.* **100,** 13841–13874 (1995).
12. Hurrell, J. W. & Trenberth, K. E. *J. Clim.* **9,** 2222–2232 (1996).
13. Hansen, J. *et al. Clim. Change* **30,** 103–117 (1995).
14. Christy, J. R. & McNider, R. T. *Nature* **367,** 325 (1994).
15. Jones, P. D. *Geophys. Res. Lett.* **21,** 1149–1152 (1994).
16. Smith, T. M., Reynolds, R. W., Livezey, R. E. & Stokes, D. C. *J. Clim.* **9,** 1403–1420 (1966).
17. Reynolds, R. W. & Smith, T. M. *J. Clim.* **7,** 929–948 (1994).
18. Kiehl, J. T. *et al. NCAR Tech. Note NCAR/TN-420+STR* (National Center for Atmospheric Research, Boulder, CO, 1996).
19. Jones, R. H. & Dey, I. *Chem. Phys. Lipids* **76,** 1–6 (1995).
20. Thompson, L. G. *et al. Science* **269,** 46–50 (1995).
21. Diaz, H. F. & Graham, N. E. *Nature* **383,** 152–155 (1996).

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Coding of intention in the posterior parietal cortex

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To look at or reach for what we see, spatial information from the visual system must be transformed into a motor plan. The posterior parietal cortex (PPC) is well placed to perform this function, because it lies between visual areas, which encode spatial information[1,2], and motor cortical areas. The PPC contains several subdivisions, which are generally conceived as high-order sensory areas[3,4]. Neurons in area 7a and the lateral intraparietal area fire before and during visually guided saccades. Other neurons in areas 7a and 5 are active before and during visually guided arm movements[5–10]. These areas are also active during memory tasks in which the animal remembers the location of a target for hundreds of milliseconds before making an eye or arm movement. Such activity could reflect either visual attention[11–15] or the intention to make movements[16–22]. This question is difficult to resolve, because even if the animal maintains fixation while directing attention to a peripheral location, the observed neuronal activity could reflect movements that are planned but not executed[22]. To address this, we recorded from the PPC while monkeys planned either reaches or saccades to a single remembered location. We now report that, for most neurons, activity before the movement depended on the type of movement being planned. We conclude that PPC contains signals related to what the animal intends to do.

Neurons in the PPC were recorded from three hemispheres of two adult macaque monkeys during interleaved delayed-saccade and delayed-reach trials (Fig. 1). Delay activity (measured 150–600 ms after target extinction) was significantly modulated by direction of movement during either or both tasks in 373 of 652 neurons for which complete data was collected (Student's *t*-text, $P < 0.05$). Of these, 68% were motor-intention specific: 21% were significantly modulated before eye but not arm movements, while 47% were significantly modulated before arm but not eye movements. Surprisingly, activity during the cue interval (50 ms before, to 150 ms after, extinction) was intention specific in 44% of the 443 active neurons. Specificity so early after target presentation suggests that these findings apply during saccades and reaches made without delays, that is, during saccades and reaches to visible targets.

A dissociation task was introduced to control for the possibility that the animal planned both a reach and a saccade to a target even when only a single movement was instructed (Fig. 2). In fact, in a previous pair of studies[12,23] which reported no specificity for saccades compared to reaching movements in the PPC, animals trained to reach towards targets without looking at them nonetheless looked towards the target at the end of the trial. It is likely that plans for both eye and arm movements to the target were formed simultaneously, with the execution of the eye movement delayed until the end of the trial. Delayed and even entirely unexecuted plans for movement may influence firing in the lateral intraparietal (LIP) area[22]. Similarly, in a delayed "go/no-go" task, neurons in area 5 code target location regardless of whether or not a

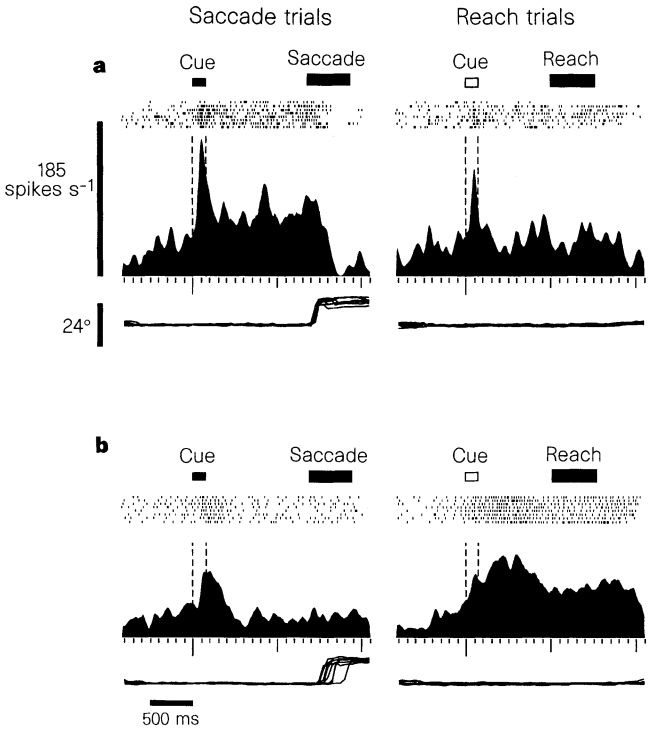

Figure 1 Responses of two intention-specific neurons in the delayed-saccade (left) and delayed-reach (right) tasks. Each panel shows timing of peripheral flash ('Cue': red flashes indicated by filled bars, green flashes by open bars) and response ('Saccade' or 'Reach'); eight rows of rasters corresponding to every third action potential recorded during each of eight trials; a spike density histogram of neuronal activity, generated by convolution with a triangular kernel[27] aligned on cue presentation, with cue onset and offset indicated by dashed lines; and eight overlaid traces showing vertical eye position. Neuronal responses in the cue interval (50 ms before to 150 ms after cue offset) were nonspecific. However, during the delay interval (150–600 ms), firing depended specifically on motor intent. **a**, A cell showing elevated delay period firing before a saccade (left) but not before a reach (right). For illustration purposes, data for this cell were collected using a fixed delay interval. **b**, A second cell showing reach rather than saccade specificity during the delay interval.

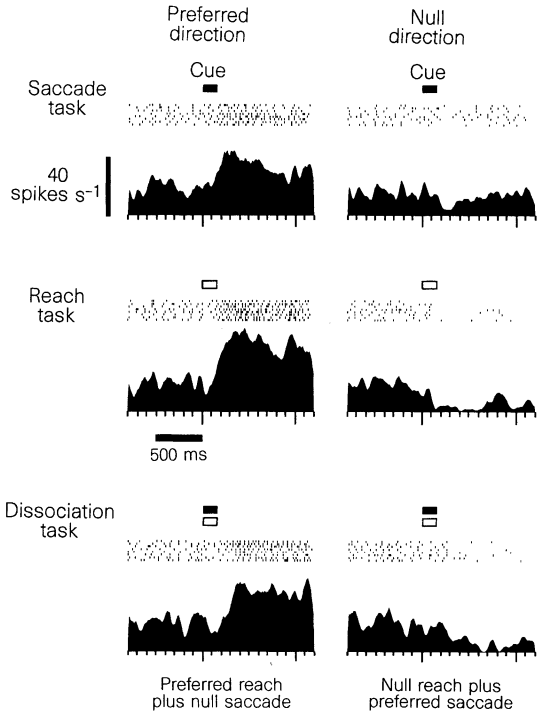

Figure 2 An intention-specific neuron whose motor specificity was revealed by the dissociation task. Delay activity was greater before movements towards the receptive field ('Preferred direction', left column) compared to away ('Null direction', right column) in both delayed saccade (top row) and reach (middle row) tasks. Thus in single-movement tasks, the neuron appears to code remembered target location independent of motor intent. However, motor specificity was revealed in the dissociation task (bottom row). Firing was vigorous before a preferred reach combined with a null saccade (bottom left), but nearly absent before a preferred saccade plus null reach (bottom right). Thus when both a reach and a saccade were planned, delay activity reflected the intended reach and not the intended saccade. Panel formats are similar to Fig. 1. Every other action potential is indicated by one raster mark.

movement is made[24]. The dissociation task eliminates plans for movements that will not be executed by explicitly instructing eye and arm movements in opposite directions. Of neurons with non-specific delay activity in the single-movement tasks (delayed reach or delayed saccade) that were tested in the dissociation task, 62% were revealed to be intention specific, bringing the total percentage of specific neurons to 84% (Table 1). In the cue interval, corresponding percentages were 45% and 63%.

Neurons specific for eye and arm movements were anatomically segregated (Table 1 and Fig. 3). While cells throughout the PPC showed motor-specific responses, cells in two subregions (area LIP and a reach area medial and posterior to LIP) tended to have strong, prolonged delay activity. In the middle third of the longitudinal extent of the intraparietal sulcus, intended-eye-movement cells outnumbered intended-arm-movement cells by 5:1. Of 47 cells active during the delay period, 28 were eye specific and only 5 arm specific in the simple tasks, with an additional 4 eye-specific and only 1 arm-specific cell revealed by the dissociation task. In a second area, medial and posterior to LIP, arm-specific cells outnumbered eye-specific cells 9:1. Of 95 active cells, 68 were arm specific and only 9 eye specific in the simple tasks, with an additional 12 arm-specific cells revealed by the dissociation task. This anatomical segregation argues against chromatic tuning as a basis for our results, as clustering of red- or green-preferring neurons over many square millimetres of cortex is unlikely. The dissociation task revealed neurons specific for saccades as well as reaches. This helps to rule out the possibility that different activity levels reflect differential allocations of attention; if reaching, for example, required greater attention, then all cells should have appeared reach specific.

Our results reveal separate intended-reach and intended-saccade pathways in the PPC. This demonstrates that the decision of how to utilize a particular sensory stimulus is reflected in PPC firing. Attention may still be present in the PPC[11,12,14,25] encoded by the small number of cells that are not specific for one type of movement, or in the nonspecific cue responses of the cells that are movement-specific in the delay period, or in the weak response of some specific neurons before their non-preferred movement. Alternatively, nonspecific neurons may reflect plans for moving body parts other than the eyes or arms (for example, pinna movements) that

Table 1 Summary of neurons

	All areas	Area LIP		Reach area	
	Both	M1	M2	M1	M2
Cue interval (100–300 ms from cue onset)					
Saccade specific	104 (23%)	17 (40)	12 (39)	17 (24)	1 (4)
Plus dissociation	161 (36%)	26 (62)	22 (71)	18 (26)	1 (4)
Reach specific	91 (21%)	4 (10)	1 (3)	13 (19)	13 (50)
Plus dissociation	119 (27%)	4 (10)	1 (3)	17 (24)	17 (65)
Nonspecific	163 (37%)	12 (29)	8 (26)	35 (50)	8 (31)
Total active cells	443	42	31	70	26
Delay interval (300–750 ms from cue onset = 150–600 ms from cue offset)					
Saccade specific	79 (21%)	17 (59)	11 (61)	9 (13)	0 (0)
Plus dissociation	87 (23%)	18 (62)	14 (78)	9 (13)	0 (0)
Reach specific	175 (47%)	3 (10)	2 (11)	47 (68)	21 (81)
Plus dissociation	227 (61%)	4 (14)	2 (11)	55 (80)	25 (96)
Nonspecific	59 (16%)	7 (24)	2 (11)	5 (7)	1 (4)
Total active cells	373	29	18	69	26

Most active cells were intention specific in both cue and delay intervals. Data for the cue interval (during and immediately after stimulus presentation) and for the delay interval (after stimulus presentation but well before movement) are shown. Columns show cell counts and percentages for cells in all areas, LIP cells in the first and second monkey (M1 and M2), and reach-area cells in M1 and M2. Effects of movement intention were assayed by comparing activity before movements in the best and opposite directions (determined in a previous block of trials). If activity was modulated only by the intention to saccade in opposite directions (Student's two-tailed t-test, $P < 0.05$), the cell was classified as saccade specific. If activity was modulated only by the intention to reach in opposite directions, the cell was classified as reach specific. If activity was modulated by either movement, the cell was classified as nonspecific. Rows labelled 'plus dissociation' include cells active in both the simple saccade and reach tasks whose firing depended on movement direction in the dissociation task. Inactive cells and cells without directionally selective activity were excluded. Not all cells were tested in the dissociation task.

we did not test[26]. Some saccade-specific neurons are active before arm movements and some reach-specific neurons are active before saccades. We propose that this activity reflects plans for movements not explicitly called for by the task, but formed automatically in response to target appearance. When these default plans are countermanded by explicit instructions, as in the dissociation task, intention specificity is revealed. The mechanism may involve inhibition between neurons in the same motor pathway coding different directions. Functionally, this coupling of saccade and reach activity may reflect the fact that these movements are often coupled.

The idea that the PPC plays a role in motor planning is consistent with previous experiments in area LIP. In a delayed double-saccade task, animals memorized two flashed locations and then, after a delay, saccaded to them sequentially. Most LIP delay activity coded the goal of the first saccade (target 1) rather than the location of the most recently presented stimulus (target 2). At the time of the first saccade, firing changed to code the goal of the second saccade (target 2). These two observations, taken together, rule out a strictly sensory role for area LIP, and support the motor planning hypothesis[20–22]. The appearance of activity coding target 2 after the first saccade can alternatively be explained as sensory remapping of a remembered stimulus in retinal coordinates. The observation that this activity sometimes anticipates the completion of the first saccade has been proposed to support the sensory remapping hypothesis[13]. However, predictive behaviour is as likely to occur in motor planning as in sensory pathways, and so the observation of anticipatory activity does not favour either hypothesis. In contrast, we now have shown directly that the majority of the delay activity in LIP as well as in the neighbouring reach area is related to specific motor intention and not to either sensory stimuli or spatial attention.

Other instances of task requirements influencing PPC responses have been reported, and have been ascribed to attentional processes[11,12]. In the dorsal visual stream, posited to be dedicated to action[2,6], attention and intention can be difficult to distinguish. Even when a given task does not require an action, plans for movements not explicitly required by the task can nonetheless be formed, producing potentially deceptive results. The results reported here indicate that it is important to rule out intention-related signals (using controls like the dissociation task) before concluding that task-dependent modulation in the PPC reflects an attentional process. □

Methods

Animals faced an array of nine buttons 3.7 cm in diameter at 28 cm distance. Each button contained a red and a green light-emitting diode (LED) side by side behind a 1.2-cm translucent lens. The animal pressed buttons illuminated green using its contralateral arm, and fixated buttons illuminated red. No other lights were present. All trials began with fixation (± 2.7°) and depression of the illuminated central button. After 750 ms, a red (saccade task) or green (reach task) peripheral LED was flashed for 150 ms. After a 1–1.6 s delay, the central LEDs were extinguished and the monkey saccaded (latency mean ± s.d.,

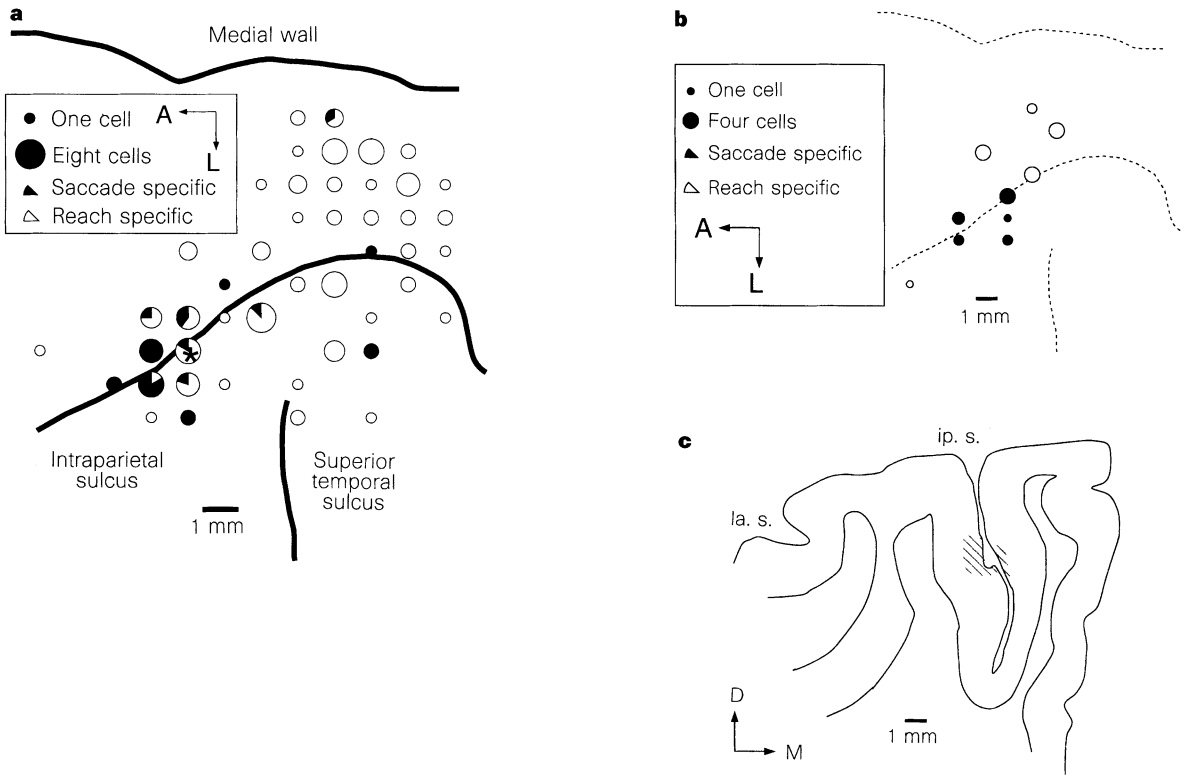

Figure 3 Neurons specific for the intention to make eye and arm movements were anatomically segregated. **a**, Surface map showing the locations of motor-specific cells relative to the intraparietal and superior temporal sulci in the left hemisphere of one animal. The area of each pie chart corresponds to the number of cells (between one and eight) with saccade-specific (filled) and reach-specific (open) delay activity at each location. Only cells with robust delay activity are shown (≥ 16 spikes per s in one or both task delay intervals). The excluded cells are primarily located in and around area 7a, the ventral intraparietal area and the medial superior temporal area. Because the *x*-*y* positioning apparatus for single neuron recording and for dye injection were not identical, the placement of tracks may be misaligned by up to ± 1 mm. **b**, Same format for robust motor-specific cells from the right hemisphere of the second animal. Anatomy is not available, so the sulcal pattern of the first animal (dotted lines) is superimposed to indicate roughly where these neurons may lie. Regardless of how different the sulcal patterns in the two animals may be, the clustering of reach and saccade cells is quite similar. The sulcal pattern and recording sites have been reflected onto the left hemisphere to facilitate comparisons with **a**. **c**, Coronal section showing a nuclear yellow dye injection made into the centre of the cluster of eye-specific cells in the first animal (indicated by an asterisk in **a**). The injection (hatching in **c**) was visualized midway down the lateral bank of the intraparietal sulcus (ip.s.), in area LIP (la.s., lateral sulcus). Arrows indicate anterior, lateral, dorsal and medial directions (A, L, D, M).

172 ± 36 ms) or reached (269 ± 45 ms) to the remembered peripheral location in complete darkness. The animal maintained central fixation during reach trials, and maintained central button depression during saccade trials. Eight delayed-saccade and eight delayed-reach trials were performed in each of eight directions. For most neurons, the best direction was determined from this first block of trials and then a second block of interleaved delayed-saccade, delayed-reach and delayed-dissociation movements was performed in the best and the opposite directions (8 or 16 trials per task per direction). The dissociation task was similar to the simple tasks, but now simultaneous red and green flashes were delivered on opposite sides of the fixation point, and the animal responded with a near-simultaneous reach and saccade in opposite directions when the fixation light was extinguished. The animal typically performed over 90% of the trials successfully. Results (Table 1) were based primarily on data from the second block of trials.

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1. Ungerleider, L. G. & Mishkin, M. in *Analysis of Visual Behavior* (eds Ingle, D. J., Goodale, M. G. & Mansfield, R. J. W.) 549–586 (MIT Press, Cambridge, MA, 1982).
2. Goodale, M. G. & Milner, A. D. *Trends Neurosci.* **15,** 220–225 (1992).
3. Hyvärinen, J. & Poranen, A. *Brain* **97,** 673–692 (1974).
4. Gross, C. G. *Cereb. Cortex* **5,** 455–469 (1994).
5. Hartje, W. & Ettlinger, G. *Cortex* **9,** 346–354 (1974).
6. Mountcastle, V. B., Lynch, J. C., Georgopoulos, A., Sakata, H. & Acuna, C. *J. Neurophysiol.* **38,** 871–908 (1975).
7. Seal, J. & Commenges D. *Exp. Brain Res.* **58,** 144–153 (1985).
8. Shibutani, H., Sakata, H. & Hyvarinen, J. *Exp. Brain Res.* **55,** 1–88 (1984).
9. Andersen, R. A., Essick, G. K. & Siegel, R. M. *Exp. Brain Res.* **67,** 316–322 (1987).
10. Murata, A., Gallese, V., Kaseda, M. & Sakata, H. *J. Neurophysiol.* **75,** 2180–2186 (1996).
11. Robinson, D. L., Goldberg, M. E. & Stanton, G. B. *J. Neurophysiol.* **41,** 910–932 (1978).
12. Bushnell, M. C., Goldberg, M. E. & Robinson, D. L. *J. Neurophysiol.* **46,** 755–772 (1981).
13. Duhamel, J.-R., Colby, C. & Goldberg, M. E. *Science* **255,** 90–92 (1992).
14. Steinmetz, M. A., Conner, C. E., Constantinidis, C. & McLaughlin, J. R. *J. Neurophysiol.* **72,** 1020–1023 (1994).
15. Colby, C. L., Duhamel, J. R. & Goldberg, M. E. *Cereb. Cortex* **5,** 470–481 (1995).
16. Gnadt, J. W. & Andersen, R. A. *Exp. Brain Res.* **70,** 216–220 (1987).
17. Andersen, R. A. *Annu. Rev. Neurosci.* **12,** 377–403 (1989).
18. Thier, P. & Andersen, R. A. *Proc. Natl Acad. Sci. USA* **93,** 4962–4967 (1996).
19. Shadlen, M. N. & Newsome, W. T. *Proc. Natl Acad. Sci. USA* **93,** 628–633 (1996).
20. Mazzoni, P., Bracewell, R. W., Barash, S. & Andersen, R. A. *J. Neurophysiol.* **76,** 1439–1456 (1996).
21. Andersen, R. A. *Cereb. Cortex* **5,** 457–469 (1995).
22. Bracewell, R. W., Mazzoni, P., Barash, S. & Andersen, R. A. *J. Neurophysiol.* **76,** 1457–1464 (1996).
23. Goldberg, M. E. & Bushnell, M. C. *J. Neurophysiol.* **46,** 773–787 (1981).
24. Kalaska, J. F. & Crammond, D. J. *Cereb. Cortex* **5,** 410–428 (1995).
25. Lynch, J. C., Mountcastle, V. B., Talbot, W. H. & Yin, T. C. T. *J. Neurophysiol.* **40,** 362–389 (1977).
26. Bon, L. & Lucchetti, C. *Exp. Brain Res.* **102,** 259–271 (1994).
27. Scott, D. W. *Ann. Statist.* **13,** 1024–1040 (1985).

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Self-centring activity of cytoplasm

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Fish melanophore cells aggregate pigment granules at the centre or redisperse them throughout the cytoplasm. The granules move along radial microtubules by means of molecular motors[1–3]. Cytoplasmic fragments of melanophores organize a radial array of microtubules and aggregate pigment at its centre[4–7]. Here we report self-centring in microsurgically produced cytoplasmic fragments of black tetra melanophores. We observed rapid (10 min) formation of a radial microtubule array after stimulation of aggregation. Arrangement of microtubules in the fragments returned to random during pigment redispersion. Apparently, formation of the radial array does not depend on a pre-existing microtubule-organizing centre. The array did not form in granule-free fragments nor in fragments treated with inhibitors of the intracellular motor protein cytoplasmic dynein. We conclude that formation of the radial microtubule array is induced by directional motion of pigment granules along microtubules and present evidence that its position is defined by interaction of microtubules with the surface.

Nascent fragments of cultured black tetra melanophores, which had been induced to aggregate, initially formed the aggregate at the cut edge. Remarkably, after 2–3 min delay, the aggregate relocated to the centre (Fig. 1a) with kinetics approximated by a single declining exponential ($t_{1/2} = 150 \pm 55$ s, mean ± s.d; $n = 10$). Fragments induced to aggregate after a period of incubation (30–300 min) showed irregular granule motion which resulted in aggregation at the centre (Fig. 1a). Immunostaining with anti-tubulin antibody confirmed that nascent fragments, like intact cells, displayed an array of microtubules directed towards the parental cell centre, whereas in incubated fragments microtubules were randomly arranged (Fig. 1a). Remarkably, upon inducing aggregation, both nascent and incubated fragments formed a radial array of microtubules focusing on the granule aggregate situated at the fragment centre (Fig. 1a). Fragments responded to caffeine treatment by dispersing pigment granules indistinguishably from intact cells. As dispersion depends on a microtubule plus-end kinesin-like motor[8], we may infer that the polarity of microtubules in the radial array was also similar to that of intact cells: minus ends at the aggregate and plus ends towards the periphery. The microtubule-stabilizing drug taxol did not interfere with pigment aggregation in intact cells, but blocked redistribution of the pigment aggregate to the centre in nascent fragments (Fig. 1a), and fusion and centring of local clusters in incubated fragments (Fig. 1a); it also prevented formation of a radial array of microtubules (not shown). Thus, fragments of black tetra melanophores were able to form and centrally locate a radial array of microtubules. Centring required microtubule dynamics and, contrary to previous observations[5–7], was very rapid.

To test whether the radial microtubule array in the fragments was organized by a centrosome, we tested for molecular components of microtubule-nucleating structures, γ-tubulin[9] and pericentrin[10]. Double immunostaining showed that γ-tubulin was at the focus of the microtubule array in parental cells (Fig. 1b), but could not be detected in the fragments even after incubation (300 min) (Fig. 1b) or after a cycle of aggregation–redispersion. Immunostaining for pericentrin was positive in the parental controls but negative in the cytoplasmic fragments (result not shown). Thus, we found no evidence that the fragments either retained or reconstituted a centrosome after severing from their parent cell.

Direct live observations of labelled microtubules showed that the random distribution of microtubules in incubated fragments transformed into a radial array during aggregation and returned back to a random arrangement during the course of redispersion (not shown). Thus, microtubule distribution seems to be coupled to the aggregation state of pigment granules. To test whether the attainment of a radial microtubule array depends on the interaction of microtubules with granules, we prepared fragments devoid of pigment granules by dissecting them from the cells with aggregated pigment. Adrenaline treatment of pigment-free fragments failed to induce microtubule reorganization (Fig. 1c), suggesting that formation of the radial array depends on the motion of granules along microtubules.

To test further the role of granule motion in microtubule reorganization, we used inhibitors of cytoplasmic dynein and kinesin, microtubule-dependent motors responsible for pigment aggregation and dispersion[2,3,8]. The cytoplasmic dynein inhibitor sodium orthovanadate[11,12], which prevents pigment aggregation in intact chromatophores[13,14], blocked pigment aggregation and formation of radial microtubules in the fragments (Fig. 2). Other dynein inhibitors erythro-9-[3-(2-hydroxynonyl)] adenine[11,12] (EHNA; 2 mM) and AMP-PNP[11,12] (100 mM in the needle), though not entirely specific for pigment aggregation, had the