Our observations strongly support Janzen's[4] and Huxley's[8,9] hypotheses of mutualism between *D. major* and *Philidris*. In exchange for shelter, ants provide significant amounts of two limiting resources: carbon dioxide and nitrogen. Both features could either expand the realized niche of *D. major*, enabling it to colonize hotter, drier habitats, or could provide *D. major* with a competitive edge over other epiphytes in this nutrient-poor ecosystem.

Finally, epiphytes in other tropical regions (including those of Central and South America, Papua New Guinea, the Philippines and Australia) have various structures occupied by ants[4,8,9,17–23]. In a facultative myrmecophytic relationship involving an ant-occupied orchid from the neotropics, Fisher *et al.*[24] have used stable carbon isotopes to quantify the extent to which ants may forage on their own host plant. We may eventually be able to combine the two approaches and examine reciprocal benefits between plants and ants. □

Received 12 August 1994; accepted 28 March 1995.

1. Anderson, J. A. R. *Gdns Bull. Singapore* **20,** 131–228 (1963).
2. Brünig, E. F. W. O. *UNESCO Symp. Ecol. Res. Humid Trop. Vegn, Kuching* **1963,** 289–313 (1965).
3. Ashton, P. S. *Malayan Nature J.* **24,** 151–162 (1971).
4. Janzen, D. H. *Biotropica* **6,** 237–259 (1974).
5. Shattuck, S. O. *Sociobiology* **21,** 1–181 (1992).
6. Kerr, A. F. G. *Br. Scient. Proc. R. Dubl. Soc.* **13,** 293–309 (1912).
7. Griffith, W. *Trans. Linn. Soc. Lond.* **20,** 387–390 (1846).
8. Huxley, C. R. *Biol. Rev.* **55,** 321–340 (1980).
9. Huxley, C. R. in *Insects and the Plant Surface* (eds Juniper, B. & Southwood, R.) 255–282 (Arnold, London, 1986).
10. Winter, K., Wallace, B. J., Stocker, G. C. & Roksandic, Z. *Oecologia* **57,** 129–141 (1983).
11. Treseder, K. K. thesis, Univ. Utah (1994).
12. Farquhar, G. D., Ehleringer, J. R. & Hubick, K. T. *A. Rev. Pl. Physiol. molec. Biol.* **40,** 503–537 (1989).
13. Keeling, C. D., Mook, W. G. & Tans, P. P. *Nature* **277,** 121–123 (1979).
14. DeNiro, M. J. & Epstein, S. *Geol. Soc. Am. Abstr. Prog.* **8,** 834–835 (1976).
15. DeNiro, M. J. & Epstein, S. *Geochim. cosmochim. Acta* **42,** 495–506 (1978).
16. Schoeninger, M. J. & DeNiro, M. J. *Geochim. cosmochim. Acta* **48,** 625–639 (1984).
17. Davidson, D. W. & Epstein, W. W. in *Vascular Plants as Epiphytes* (ed. Lüttge, U.) 200–233 (Springer, Heidelberg, 1989).
18. Rico-Gray, V., Barber, J. T., Thien, L. B., Ellgaard, E. G. & Toney, J. J. *Am. J. Bot.* **76,** 603–608 (1989).
19. Benzing, D. H. *Bull. Torrey Bot.* **97,** 109–115 (1970).
20. Madison, M. *Selbyana* **5,** 107–115 (1979).
21. Gay, H. *Biol. J. Linn. Soc. Lond.* **50,** 221–233 (1993).
22. Huxley, C. R. *New Phytol.* **80,** 231–268 (1978).
23. Rickson, F. R. *Am. J. Bot.* **66,** 87–90 (1979).
24. Fisher, B. L., Sternberg, L. S. L. & Price, D. *Oecologia* **83,** 263–266 (1990).
25. Ehleringer, J. R. & Osmond, C. B. in *Plant Physiological Ecology* (eds Pearcy, R. W., Ehleringer, J. R., Mooney, H. A. & Rundel, P. W.) 281–300 (Chapman & Hall, London, 1991).

ACKNOWLEDGEMENTS. We thank D. R. Sandquist for advice, discussions and critical reading of the manuscript; B. E. Howlett for help in the field; E. Medina and T. G. Whitham for comments on the manuscript; and C. S. Cook, A. J. Leffler and C. F. Kitty for technical assistance. This work was supported by grants from the Explorer's Club, Sigma Xi, NSF, and the HHMI Undergraduate Research Program (University of Utah).

Visual motion aftereffect in human cortical area MT revealed by functional magnetic resonance imaging

Roger B. H. Tootell, John B. Reppas, Anders M. Dale*†, Rodney B. Look, Martin I. Sereno*, Rafael Malach, Thomas J. Brady & Bruce R. Rosen

Massachusetts General Hospital Nuclear Magnetic Resonance Center, 149 13th Street, Charlestown, Massachusetts 02129, USA
* Department of Cognitive Sciences, University of California at San Diego, La Jolla, California 92093-0515, USA
† Department of Neurophysiology, University of Oslo, Oslo, Norway

FUNCTIONAL magnetic resonance imaging (fMRI)[1–3] was used to measure local haemodynamic changes (reflecting electrical activity) in human visual cortex during production of the visual motion aftereffect, also known as the waterfall illusion[4,5]. As in previous studies[6–9], human cortical area MT (V5) responded much better to moving than to stationary visual stimuli. Here we demonstrate a clear increase in activity in MT when subjects viewed a stationary stimulus undergoing illusory motion, following adaptation to stimuli moving in a single local direction. Control stimuli moving in reversing, opposed directions produced neither a perceptual motion aftereffect nor elevated fMRI levels postadaptation. The time course of the motion aftereffect (measured in parallel psychophysical tests) was essentially identical to the time course of the fMRI motion aftereffect. Because the motion aftereffect is direction specific, this indicates that cells in human area MT are also direction specific. In five other retinotopically defined cortical areas, similar motion-specific aftereffects were smaller than those in MT or absent.

Prolonged viewing of a stimulus moving in one direction (as when staring fixedly at a waterfall) makes stationary stimuli appear to move in the opposite direction immediately afterwards[4,5]. It is not yet known which areas of human brain

TABLE 1 fMRI activation during the motion aftereffect

Cortical area	MAE excitation (%)	MAE motion selectivity (%)
MT	70	100
VP	29	53
V3a	26	48
V2	23	40
V3	~0	~0
V1	~0	~0

The maximum averaged difference in amplitude produced following single- minus reversing-direction rings was measured (as in Fig. 3) in MT and in five additional cortical visual areas. In the middle column, the amplitude of this fMRI MAE difference amplitude is expressed as a percentage of the averaged amplitude produced by the moving stimulus itself (moving minus initial fixation conditions), for each visual area. In the right column, the MAE amplitudes are expressed relative to the motion selectivity of each visual area (moving minus stationary conditions). The visual cortical areas were functionally labelled in the same scanning sessions in which the MAE data were taken, using retinotopically specific stimuli[9,20–22]. Cortical area names are generalized from apparently homologous areas in macaque. Human area V1 (primary visual cortex) was defined as the area containing a complete representation of left or right hemifield buried within the calcarine fissure, extending superiorly and inferiorly along the medial bank. Area V2 is that area surrounding the vertical meridian representation of V1, containing a mirror-image representation of that same hemifield, split into an inferior and a superior region, located mostly along the medial bank. Area V3 was the mirror-symmetrical representation of the inferior visual quarter-field, bordering superior V2, extending from the horizontal meridian representation at the superior V2/V3 border to the vertical meridian representation at the V3/V3a border, representing only the inferior quarter-field. Human area V3 is proportionately wider in humans than in macaques. Area V3a borders V3 on its inferior/posterior aspect, and it again contains a complete representation of the left or right hemifield. Area VP is retinotopically mirror-symmetrical to area V3, bordering inferior V2, containing a quarter-field representation of the superior visual field. Area MT (V5) is a small oval area on the lateral surface near the occipito-temporal-parietal junction, which is motion selective and has very high contrast sensitivity[9]. Area MST can be distinguished from MT using certain tests involving eye movements and motion coherence, described elsewhere.

mediate this visual motion aftereffect (MAE), although the motion-specific area MT[6–11] (also known as V5) is one likely candidate. In non-human primates, most area MT neurons are both motion- and direction specific, and direction-specific cells have been presumed to underlie the motion aftereffect, either in monkey MT[12] or in non-primates[13 18].

The stimuli used in this study were concentric rings (0.5 cycles deg^{-1}, duty cycle = 0.2), either moving (7 deg s^{-1}) or stationary (see Fig. 1*a*). Human area MT was activated much more by moving than by stationary stimuli (see Figs 1*b* and 2), as described previously[6–9]. In addition, we found a clear increase in magnetic resonance (MR) signal amplitude during viewing of stationary stimuli when they were preceded by an adaptation stimulus moving continuously in a single direction. During these times the stationary stimuli appeared to be moving owing to a prominent motion aftereffect. Such an elevated MR signal did not occur following adaptation to otherwise identical stimuli that continually reversed direction (0.5 Hz), and neither did such stimuli induce a perceptual motion aftereffect. One obvious interpretation is that the signal remained high in area MT because the stimulus appeared to be moving, even though it was in fact stationary. Because this MR increase is direction specific, and because it was accompanied by a perceptual motion aftereffect, we refer to it as a fMRI motion aftereffect.

The fMRI motion aftereffect was isolated by subtracting its signal from that of control conditions not showing a perceptual motion aftereffect (see Fig. 3). The peak amplitude of that averaged motion aftereffect reached 1.9%, equal to the mean steady-state difference in MR signal produced by rings that were actually moving versus those that were stationary.

If the fMRI motion aftereffect is related to the perceptual motion aftereffect, the time course of the two aftereffects is expected to be similar. To test this we showed subjects the MAE stimulus used in the magnet (e.g. expanding rings for 40 s, followed by stationary rings), with modifications to allow measurement of the psychophysical motion aftereffect (two-alternative forced choice motion-nulling staircase procedure[19]). The time course of the decay of fMRI and psychophysical motion aftereffects were very similar, with best-fit exponents of 8.3 s and 9.2 s, respectively (see Fig. 3).

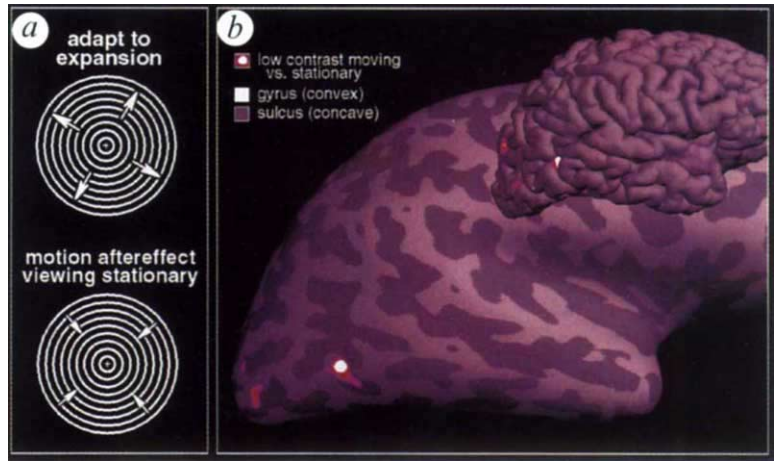


FIG. 1 Stimulus used in this experiment, and human cortical visual area MT (V5) activated by that stimulus. *a*, A stationary view of the stimulus used in these experiments. *b*, A three-dimensional reconstruction of the brain of one subject (M.S.), derived entirely from magnetic resonance data. The brain is shown in both normal and 'inflated' format. The inflated version was made by relaxing curvature while approximately preserving local area and local angles[22]. Sulcal cortex (concave) is dark magenta and gyral cortex (convex) is lighter magenta. The fMRI activity produced by moving minus stationary rings is coded in a pseudocolour scale varying from saturated magenta (threshold) to white (maximum activity). The prominent white patch on the bottom right lateral surface is area MT (V5), a region that responds selectively in fMRI experiments to moving visual stimuli, as compared with stationary stimuli[6–9]. For greater functional specificity the stimulus used to produce *b* was low in luminance contrast[9].

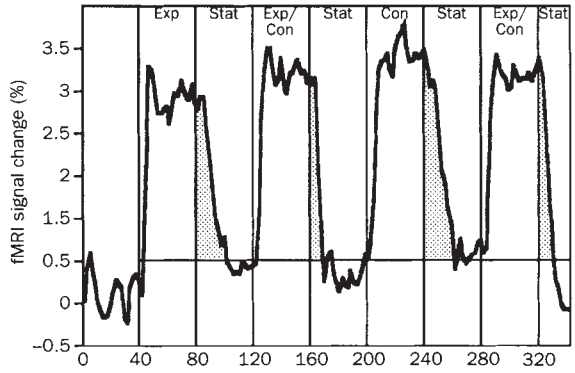
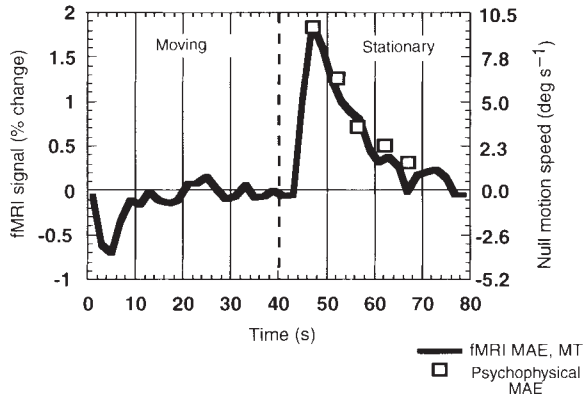


FIG. 2 Averaged MR time course showing responses during real and illusory visual motion. Subjects (12) were scanned in a 1.5 T GE MR scanner using echo-planar imaging (Advanced NMR) and a 5″ radial surface coil positioned over visual cortex. Five or six slices (4–6 mm thick) were oriented approximately perpendicular to the calcarine fissure. In-plane resolution was 3.1 × 3.1 mm. In most subjects, head motion was greatly minimized by the use of a bite bar. We tested for motion artefact by presenting all images within a scan as a movie, or by analysis of difference images taken from different time periods. Any scans in which head motion within a scan exceeded approximately 2 mm were discarded. Asymmetric spin echo sequences (repetition time, 2,000 ms, echo time, 80 ms, 180° refocusing pulse offset by −25 ms) were used to measure 'activation' (local increases in blood flow and oxygenation). Subjects were shown a pattern of concentric rings (50° diameter) surrounding a fixation spot, with 1 image every 2 s. In different periods (typically 40″ long) during each scan (5′ 40″), the moving rings were either (1) continuously expanding (Exp), (2) continuously contracting (Con), or (3) reversing direction (expanding or contracting, Exp/Con) at 0.5 Hz. Radially symmetric stimuli (rings) were used to induce the motion aftereffect because such stimuli do not induce optokinetic nystagmus, a potential confound. However, within each local visual region, the direction of stimulus movement was either unidirectional (conditions 1 and 2) or bidirectional (condition 3). Following every period of moving rings, stationary rings, otherwise equal (Stat), were presented. Following the periods of continuous unidirectional local motion (the expanding or contracting stimuli), a profound visual motion aftereffect was seen in the (physically stationary) rings. Following the periods of reversing direction, no motion aftereffect was reported. The averaged MR signal from all 38 scans in human area MT/V5 is shown. The MR response amplitudes to both the single- and reversing-direction stimuli were approximately equal in amplitude. The response to stationary stimuli was only slightly greater than that to a blank field. However, the fMRI response immediately following the single-direction stimuli (i.e. when the motion aftereffect was visible) remained high for some time after stimulus offset, significantly longer than that predicted by the normal temporal response of the fMRI signal[1,3,27]. MR responses following reversing-direction stimuli (when motion aftereffects were absent) instead returned promptly to the steady-state response level produced by stationary stimuli. One interpretation is that area MT remained activated because the stimulus appeared to be moving, even though it was physically stationary.

FIG. 3 Isolated fMRI motion aftereffect and its relation to psychophysics. This time course shows MR amplitudes during and after single-direction conditions, minus amplitudes during and after reversing-direction conditions. All data are included. During the first 40 s (20 images), the figure indicates no steady-state difference between the activation produced by single-direction versus reversing-direction stimuli. Thus the subsequent fMRI aftereffect cannot be attributed to unequal levels of activation. Thereafter the subjects viewed stationary stimuli, but the MR signal remained high following single-direction stimuli (relative to reversing-direction stimuli) for approximately 20 s. The time course of the psychophysical motion aftereffect was measured in response to the fMRI stimuli, with modifications. As in the fMRI experiments, subjects viewed rings moving in a single local direction (e.g. expanding) for 40 s, followed by stationary rings. After a delay of either 0, 4.5, 9.5, 14.5 or 19.5 s, the stationary rings were replaced by a blank field of uniform grey for 0.5 s. This was followed by a pattern of sinusoidal rings (same spatial frequency) of low (~5%) contrast, presented for 0.5 s. The subjects then indicated (using a two-alternative forced-choice procedure) which (illusory) direction the rings were moving and the speed and direction of the rings was adjusted opposite to the indicated direction in small increments at every button press. Thus the ring speed converged to a velocity and direction equal but opposite to that of the illusory motion, independently for every delay measured (0, 5, 10, 15 and 20 s). Data from all four subjects were averaged. The average standard deviation at plateau was 0.37 deg s $^{-1}$. The psychophysical data are shown as five squares. To facilitate comparisons between fMRI and psychophysical motion aftereffects, the psychophysical time course was shifted to the right by 7 s to compensate for the known delay in the fMRI haemodynamics[1,3,27], and peak fMRI and psychophysical amplitudes were normalized on different *y*-axes (psychophysical, right). There is an excellent fit between the time courses of the psychophysical and the fMRI motion aftereffects. Previous models of the motion aftereffect predict a downward deflection of fMRI signal rather than the positive inflection obtained. Single-unit studies in non-primate species, from areas other than MT, suggest that direction-specific cells tuned to the adapting stimulus direction habituate (decrease firing) during and after



the adapting period[13–18]. Thus the population firing of cells preferring opposing directions would predominate in response to a stationary stimulus after single-motion adaptation, leading to an illusory motion in the opposing direction, and perhaps a decrease in MR amplitude. However, other evidence and a different model are consistent with the present fMRI findings. In the only electrophysiological study in monkey area MT[12] on the motion aftereffect, cells preferring directions opposite to the adapting direction increased firing postadaptation. As in other studies, cells preferring the adaptation direction also decreased their firing rate. Together, these indicate that cells tuned to opposing directions tonically inhibit each other. Tonic, reciprocal, direction-specific inhibition of monkey MT cells is suggested by other evidence as well[28–30]. Because cortical single units typically have low baseline rates, this model (and our fMRI data) suggests that poststimulus excitation overcomes poststimulus inhibition, because inhibition cannot decrease below zero spikes per second, whereas excitation is not so limited. However, because the physiological basis underlying fMRI is incompletely understood, such a model is obviously speculative.

Presumptive MT (V5) has been shown to be motion selective [6–9] (see Fig. 1*b*). However, the prominent feature of single units in monkey MT is that they are direction specific as well as motion specific. To our knowledge, the direction-specific motion aftereffect revealed here is the first physiological evidence for direction specificity in human cortical visual area MT. This is important because the human area chosen to be 'MT' or 'V5' is only one of several motion-selective cortical areas[7–9], and many visual neurons lacking direction specificity will respond better to moving than to stationary stimuli.

The high spatial resolution of the fMRI technique, coupled with retinotopically specific visual stimulation, made it possible to localize accurately other cortical visual areas[9,20–22] during the same experiments (see Table 1). This allowed us to investigate which of those other areas, if any, also show a fMRI motion aftereffect, and to measure its relative amplitude.

These data are shown in Table 1. As expected, area MT showed the highest MAE activation ratio of all the measured areas. Smaller fMRI motion aftereffects were seen in areas V2 and V3a. This is compatible with the presence of a minority percentage of direction-selective single units in V2 and Va, and with reciprocal connections between MT, V2 and V3a in monkey (see ref. 23). A robust MAE appeared to be present in presumptive human MST as well, but more data are required.

Because the perceptual effect depends logically on the presence of direction specificity, a trivial explanation for our results is that the amplitude of the direction-specific fMRI MAE is simply proportional to the motion specificity of the area. However, when this correction was applied (Table 1, middle column), the MAE values still differed markedly across the different cortical areas. The fMRI motion aftereffect was largest in MT even when corrected for the greater motion selectivity of that area.

These results suggest that MT and certain areas 'upstream' from it are selectively activated during the perceptual motion aftereffect. Other stimuli, not tested here, produce a motion aftereffect which (based on the extent of binocular transfer) appears to tap higher stages of motion processing than those tested here[24,25]; such stimuli might produce a correspondingly more specific fMRI localization as well. The illusory motion reported here may also be related to other reports of illusory motion[26] or illusory lack of motion at equiluminance[9]. □

Received 19 December 1994; accepted 18 April 1995.

1. Kwong, K. K. *et al. Proc. natn. Acad. Sci. U.S.A.* **89,** 5675–5679 (1992).
2. Ogawa, S. *et al. Proc. natn. Acad. Sci. U.S.A.* **89,** 5951–5955 (1992).
3. Bandettini, P. A., Wong, E. C., Hinks, R. S., Tikofsky, R. S. & Hyde, J. S. *Magn. Reson. Med.* **25,** 390–397 (1992).
4. Adams, R. *Phil. Mag.* **5,** 373–374 (1834).
5. Wohlgemuth, A. *Br. J. Psychol.* (Suppl.) **1,** 1–117 (1911).
6. Zeki, S. *et al. J. Neurosci.* **11,** 641–649 (1991).
7. Watson, J. D. G. *et al. Cerebr. Cort.* **3,** 79–94 (1993).
8. Dupont, P., Orban, G. A., DeBruyn, B., Verbruggen, A. & Mortelmans, L. *J. Neurophysiol.* **72,** 1420–1424 (1994).
9. Tootell, R. B. H. *et al. J. Neurosci.* **15,** 3215–3230 (1995).
10. Clarke, S. & Miklossy, J. *J. comp. Neurol.* **298,** 188–214 (1990).
11. Tootell, R. B. H. & Taylor, J. B. *Cerebr. Cort.* **5,** 39–55 (1995).
12. Petersen, S. E., Baker, J. F. & Allman, J. M. *Brain Res.* **346,** 146–150 (1985).
13. Barlow, H. B. & Hill, R. M. *Nature* **200,** 1345–1347 (1963).
14. Hammond, P., Mouat, G. S. V. & Smith, A. T. *Expl Brain Res.* **60,** 411–416 (1985).
15. Marlin, S. G., Sohail, S. J., Hasan, J. & Cynader, M. S. *J. Neurophysiol.* **59,** 1314–1330 (1988).
16. Movshon, J. A. & Lennie, P. *Nature* **278,** 850–852 (1979).
17. Vautin, R. G. & Berkeley, M. A. *J. Neurophysiol.* **40,** 1051–1065 (1977).
18. Von der Heydt, R., Hanny, P. & Adorjani, C. *Archs ital. Biol.* **116,** 248–254 (1978).
19. Carney, T. & Shadlen, M. N. *Vision Res.* **33,** 1977–1995 (1993).
20. Schneider, W., Noll, D. C. & Cohen, J. D. *Nature* **365,** 150–153 (1993).
21. DeYoe, E. A. *et al. J. Neurosci. Meth.* **54,** 171–187 (1995).
22. Sereno, M. I. *et al. Science* (in the press).
23. Felleman, D. J. & Van Essen, D. C. *Cerebr. Cort.* **1,** 1–47 (1991).
24. Nishida, S., Ashida, H. & Sato, T. *Vision Res.* **34,** 2707–2716 (1994).
25. Nishida, S. & Sato, T. *Vision Res.* **32,** 1635–1646 (1992).
26. Zeki, S., Watson, J. D. & Frackowiak, R. S. *Proc. R. Soc.* B**252,** 215–222 (1993).
27. Friston, K. J., Jezzard, P. & Turner, R. *Hum. Brain Mapping* **1,** 153–171 (1994).
28. Born, R. T. & Tootell, R. B. H. *Nature* **357,** 497–499 (1992).
29. Allman, J. M., Baker, J. F., Newsome, W. T. & Petersen, S. E. in *Cortical Sensory Organization Vol. 2. Multiple Visual Areas* (ed. Woolsey, C. N.) 171–185 (Humana, Clifton, NJ, 1981).
30. Lagae, L., Gulyas, B., Raiguel, S. & Orban, G. A. *Brain Res.* **496,** 361–367 (1989).

ACKNOWLEDGEMENTS. This study was supported by an NEI grant to R.B.H.T. We thank several subjects, including L. Kobierski, W. Kennedy, W. Vanduffel and H. Aronen. We also thank T. Campbell, who ran the MR scanner on some of the scans, and many others in the MGH NMR Center.