RESEARCH ARTICLE

Igor Riečanský · Alexander Thiele · Claudia Distler
Klaus-Peter Hoffmann

Chromatic sensitivity of neurones in area MT of the anaesthetised macaque monkey compared to human motion perception

Abstract We recorded activity from neurones in cortical motion-processing areas, middle temporal area (MT) and middle posterior superior temporal sulcus (MST), of anaesthetised and paralysed macaque monkeys in response to moving sinewave gratings modulated in luminance and chrominance. The activity of MT and MST neurones was highly dependent on luminance contrast. In three of four animals isoluminant chromatic modulations failed to activate MT/MST neurones significantly. At low luminance contrast a systematic dependence on chromaticity was revealed, attributable mostly to residual activity of the magnocellular pathway. Additionally, we found indications for a weak S-cone input, but rod intrusion could also have made a contribution. In contrast to the activity of MT and MST neurones, speed judgments and onset amplitude of evoked optokinetic eye movements in human subjects confronted with equivalent visual stimuli were largely independent of luminance modulation. Motion of every grating (including isoluminant) was readily visible for all but one observer. Similarity with the activity of MT/MST cells was found only for motion-nulling equivalent luminance contrast judgments at isoluminance. Our results suggest that areas MT and MST may not be involved in the processing of chromatic motion, but effects of central anaesthesia and/or the existence of intra- and inter-species differences must also be considered.

Keywords Chromatic sensitivity · Macaque monkey · Area MT/MST · S cones · Optokinetic eye movements · Speed judgment

I. Riečanský · A. Thiele · C. Distler · K.-P. Hoffmann (✉)
Department of General Zoology and Neurobiology,
Ruhr University Bochum, Bochum, Germany
E-mail: kph@neurobiologie.rub.de

A. Thiele
Psychology, Brain, and Behaviour, University of Newcastle upon Tyne, Newcastle upon Tyne, United Kingdom

Introduction

Detection and analysis of motion is one of the fundamental abilities of the visual system. Motion processing provides valuable information about changes in the environment, plays a role in image segmentation, perceptual grouping, depth encoding and locomotion. Motion is represented in the visual system explicitly through direction- and speed-selective neurones. These neurones occur predominantly in areas of the dorsal (parietal) stream of the cortical visual information processing pathway (Mishkin et al. 1983; for review see e.g. Desimone and Ungerleider 1989), especially in the middle temporal area (MT, also termed V5), which has been attributed a central role in the motion processing system (for review see e.g. Orban 1997). This extrastriate visual area has been identified in all primates studied so far including humans (Kaas 1997).

Numerous studies have revealed a close relationship between the activity of MT neurones and an animal's performance in direction discrimination tasks (Newsome et al. 1989; Britten et al. 1992, 1996; Shadlen et al. 1996; Thiele et al. 1999a) or the generation of smooth eye movements (Newsome et al. 1988; Ilg 1997; Lisberger and Movshon 1999; Churchland and Lisberger 2001). The behavioural significance of the MT area in motion processing was additionally demonstrated by experimental lesions (Newsome et al. 1985; Newsome and Pare 1988; Dursteler et al. 1987; Dursteler and Wurtz 1988; Cowey and Marcar 1992; Marcar and Cowey 1992; Schiller 1993; Pasternak and Merigan 1994; Orban et al. 1995) and by microstimulation studies (Komatsu and Wurtz 1989; Salzman et al. 1990, 1992; Groh et al. 1997; Born et al. 2000).

MT neurones are able to signal the direction of motion defined by a large variety of cues. These include luminance defined motion (first order) cues as well as non-luminance defined motion cues (second order; for review see Sperling and Lu 1998) such as contrast modulation (O'Keefe and Movshon 1998) or temporal

structure (Albright 1992; O'Keefe and Movshon 1998). Consequently, it has been argued that area MT might be a stage to process form-cue independent motion information and thus the universal hub for motion perception (Albright and Stoner 1995). However, recent evidence using specific higher-order motion stimuli suggests that this popular view might be an oversimplification (Churan and Ilg 2001; Ilg and Churan 2004).

Despite a large body of research, the role of area MT in the processing of chromatic (colour-defined) motion remains controversial. On the one hand, several reports state that area MT contains neurones that are able to signal motion of stimuli defined by pure colour contrast (Saito et al. 1989; Gegenfurtner et al. 1994; Dobkins and Albright 1994; Seidemann et al. 1999). Dobkins and Albright (1994) demonstrated that MT neurones can use the chromatic sign when luminance cues failed to provide information about the motion direction. On the other hand, Gegenfurtner et al. (1994) found that chromatic contrast sensitivity of MT neurones was not sufficient to explain performance in a direction discrimination task unless tested at high temporal frequencies.

So far only one study has simultaneously recorded the sensitivity of MT neurones and the animal's perception to chromatically defined motion (Thiele et al. 2001). They employed the equivalent luminance contrast (EqLC) paradigm of Cavanagh and Anstis (1991). Here the contribution of a colour grating to motion was quantified by its ability to override the motion of a luminance grating moving in the opposite direction. Based on the observation that EqLC was relatively uninfluenced by luminance contrast added to the chromatic modulation. Cavanagh and Anstis (1991) proposed that human chromatic motion perception is mediated by the activity of colour-opponent P-LGN cells. Thiele et al. (1999b) measured the EqLC of individual MT neurones and found that only a subset of units showed this property. Moreover, psychophysical EqLC measurements in macaque monkeys (Thiele et al. 2001) revealed that the EqLC of chromatic gratings decreased when luminance contrast was increased—a result different to humans. Performance of monkeys was more in line with a magnocellular hypothesis for the origin of chromatic motion processing. Interestingly, these psychophysical results were well matched by the simultaneously recorded activity of MT neurones. The difference in performance between macaques and humans raised the possibility of substantial inter-species differences in the processing of chromatic motion. In humans, activation of MT/V5 complex by colour-defined motion was found in several functional imaging studies (PET: Ffytche et al. 1995; fMRI: Tootell et al. 1995; Chawla et al. 1998, 1999; Wandell et al. 1999) though reports on the magnitude of activation by chromatic as compared to luminance motion differed greatly.

The aim of the present study was to (re-) address the controversial topic of MT involvement in chromatic motion processing. We tested responses of single neurones in area MT and adjacent area MST to a wide range of chromoluminant modulations. Equivalent stimuli were used to measure the human perceptual and oculomotor performance to allow a direct comparison to MT/MST neuronal activity.

Methods

Visual stimulation

In the electrophysiological experiments, visual stimuli were generated by an IBM-compatible PC using an ELSA Winner 2000 X Pro graphic board with a resolution of 800×600 pixels and 75 Hz refresh rate. Stimuli for behavioural experiments were created by an SGI Octane computer with a pixel resolution of 1,280×1,024 and a 72 Hz refresh rate. Stimuli were displayed on a SONY GDM-500T9 CRT video monitor (19.8 in.), which ensured the generation of equivalent stimuli in both experiments. Monitor output was calibrated (Wandell 1995; Robson 1999) using a spectrophotometer PR-650 (Photo Research, USA) and controlled for channel additivity. The calibration was repeatedly checked during data collection but no significant changes were registered. Chromatic CIE XY coordinates of the monitor primaries at maximum intensity had the following values: red gun [0.625; 0.344], green gun [0.284; 0.609], blue gun [0.149; 0.066]. Details regarding the stimuli and their cone excitations are given in Table 1.

Visual stimuli were moving sinusoidal gratings at a constant spatial and temporal frequency: spatial frequency (SF) = 0.4 cycles/deg, temporal frequency (TF) = 4 Hz. MT neurones generally respond well to such stimuli (Perrone and Thiele 2001), which was also confirmed during initial measurements of directional tuning. The monitor distance was 57 cm yielding an angular speed of 10°/s. Stimuli were modulated in the chromatic and luminance domain (chromoluminant modulation); a total of 56 modulations were used.

In the chromatic domain, gratings were modulated from a central white (grey) point [0.298; 0.280] in eight directions defined in the CIE chromaticity diagram (Fig. 1). The chromatic modulation was always the maximal possible, limited by the CRT. Modulations of colours, no. 1, 3, 5 and 7, roughly corresponded to modulations in cardinal directions of isoluminant plane of the Derrington–Krauskopf–Lennie (DKL) colour space (Derrington et al. 1984), and were termed as azimuths, 0, 90, 180 and 270°. Colours no. 2, 4, 6 and 8 were defined as intersections of monitor triangle with axes lying in the geometrical centre between the cardinal chromatic directions. We term these modulations as azimuths, 45, 135, 225 and 315°. Azimuths of the DKL chromatic space (calculated using 10° cone fundamentals (Stockman and Sharpe 2000)) were 343, 69, 89, 100,

Table 1 CIE xy coordinates of the chromatic modulation, relative cone excitation and Michelson cone contrast of L, M, and S cones at $V_{\lambda 10°}$ isoluminance

Colour azimuth (deg)	CIE x	CIE y	Le (0.10e-3)	Lcc	Me (0.10e-3)	Mcc	Se (0.10e-3)	Scc
0	0.412	0.228	5.68	0.080	−9.67	−0.195	1.01	0.022
45	0.549	0.399	4.43	0.066	−9.56	−0.202	−20.79	−0.873
90	0.392	0.519	0.84	−0.013	−1.73	−0.030	−20.69	−0.847
135	0.244	0.440	−2.69	−0.043	2.43	0.039	−14.06	−0.453
180	0.210	0.307	−1.98	−0.031	4.72	0.074	−0.08	−0.002
225	0.192	0.235	−2.84	−0.044	5.34	0.080	12.11	0.204
270	0.217	0.114	−0.14	−0.002	4.06	0.059	62.85	0.557
315	0.315	0.164	2.13	0.029	−6.33	−0.111	21.99	0.307

Cone excitation was computed by integration of 10 *deg* cone fundamentals (Stockman and Sharpe 2000) with spectral power distribution of emitted light. Excitation elicited by the white (grey) part of the grating was then subtracted from the excitation elicited by the azimuthal (coloured) part of the grating. Positive values indicate an increase in excitation with respect to the white/grey and the negative values indicate the opposite case

163, 250, 268, and 280°. Table 1 summarizes Commission Internationale de l'Eclairage (CIE) xy chromatic coordinates as well as values of relative cone excitations at $V_{\lambda 10°}$ isoluminance.

Seven values of Michelson luminance contrast were used for the modulation in luminance domain: $+10$, $+5$, $+2.5$, 0, -2.5, -5, -10% (positive sign indicates "coloured stripes" brighter than "grey stripes"). Luminance was computed using a CIE 1924 2° photopic luminous efficiency function ($V_{\lambda 2°}$) in recordings from three animals. In one animal, we used a CIE 1964 10° photopic luminous efficiency function ($V_{\lambda 10°}$). The spatially averaged luminance was always constant (26.8 cd/m^2, $V_{\lambda 10°}$). Retinal illuminance in the animal experiments was at least ~530 photopic trolands corresponding to pupil diameter of ~5 mm ensured by the use of mydriatics. Gratings were displayed on a background corresponding to the central grey point ([$x = 0.298$, $y = 0.280$], 26.8 cd/m^2, $V_{\lambda 10°}$), which was permanently present on the screen (including during the interstimulus intervals).

Animal preparation

All experiments had been approved by the local ethics committee and were carried out in accordance with an European Communities Council Directive from the 24 November 1986 (86 609 EEC) and NIH guidelines for care and use of animals for experimental procedures. Four adult male macaque monkeys under central anaesthesia were used in these experiments. After initial anaesthesia with ketamine hydrochloride (10 mg/kg i.m.), an intravenous catheter was placed and the animals were intubated orally. Following additional local anaesthesia with bupivacaine hydrochloride 0.5% or prilocaine hydrochloride 0.5%, the animals were placed into a stereotaxic apparatus. In two animals, 3 µg/kg bolus of fentanyl was applied i.v. for additional analgesia, and was followed by continuous infusion (3 µg/kg/h) for the whole recording session. During surgery the animals received doses of pentobarbital as needed. After completion of surgical procedures, the animals were paralysed with alcuronium chloride. During the whole session the monkeys were artificially ventilated with nitrous oxide:oxygen ratio of 3:1 containing 0.3–1% halothane. Heart rate, SPO$_2$, blood pressure, body temperature, and endtidal CO$_2$ were continuously monitored and kept at physiological levels. Two animals had been used in behavioural studies before and thus had surgical implants at the time of the experiment. The other two animals were not chronically implanted. Therefore, in these animals, the skin overlying the skull was cut and craniotomies were performed according to stereotaxic coordinates based on the previous MRI scans to allow access to the superior temporal sulcus. Tropicamide was used to prevent changes in accommodation and pupil size. Corneae were protected with
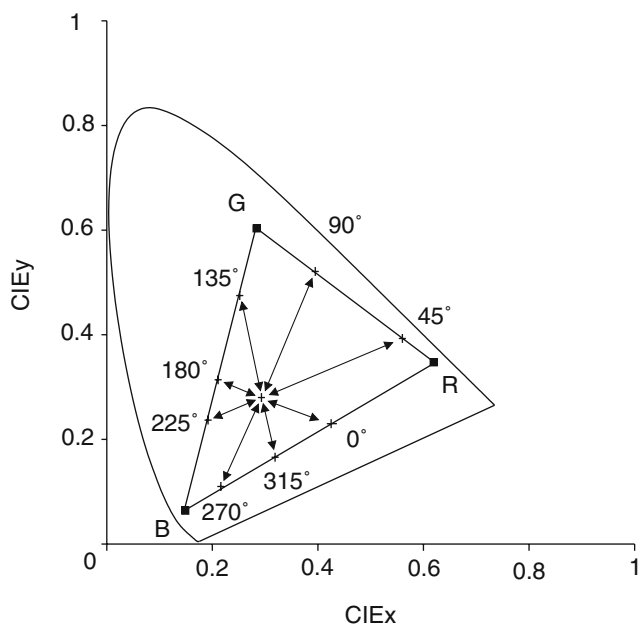

Fig. 1 CIE xy 1931 chromaticity diagram of the colour modulation used in the experiments. Eight unidirectional modulations (azimuths) with the origin in the central white/grey point were used. Modulation in every direction was always the maximal possible allowed by the monitor. *R, G, B* CRT monitor primaries

contact lenses which were chosen with a refractometer (Rodenstock) to focus the animals' eyes at the distance of the monitor screen used for visual stimulation.

The experiments were performed as acute and terminal in three animals. In one animal several recording sessions were carried out before the acute terminal recording. In this case the paralytic infusion was stopped after recordings, and the effect of paralysis was antagonised by pyridostigmine bromide (0.3 mg/kg), if needed. After recovery of spontaneous breathing, the animal was returned to its room and observed until full recovery from anaesthesia had occurred. In the terminal experiments, the animals were sacrificed with an overdose of pentobarbital, and were perfused transcardially with 0.9% NaCl solution followed by paraformaldehyde–lysine periodate containing 4% paraformaldehyde.

Electrophysiological recordings

Extracellular recordings of neuronal action potentials were carried out using custom-made glass-coated tungsten microelectrodes with impedance 1.5–3 MΩ at 1 kHz. The microelectrode was advanced using a Burleigh microdrive. A tiny perforation in the dura mater was made before the penetration in order to enable microelectrode insertion into the brain tissue. The signal from the microelectrode was amplified, filtered, and passed into a two-amplitude spike window discriminator. The signal was in parallel displayed on an analogue oscilloscope and audio monitor. Single units were isolated by the window discriminator based on the amplitude of action potentials. In addition to single cells, we occasionally recorded multi-unit activity. Positively identified spikes triggered digital pulses which were sampled at 1 kHz and stored on a data acquisition computer disc. Stimulus presentation and data acquisition were accomplished by the software REC2 (developed by A. Thiele and A. Wachnowski) running under MS-DOS and RTKernel (4.0).

We recorded from the area MT and adjacent area MST in the superior temporal sulcus (STS). The electrode penetrations were vertical in three animals, in one animal a posterior approach in the sagittal plane was employed. The area MT can be identified at a typical penetration depth by the appearance of characteristic receptive field properties (directional selectivity, receptive field size, position in the contralateral hemifield, topographical organization). Typically, when performing the approach from posterior, upon deeper penetration into the brain the electrode leaves the MT in the posterior bank of the STS, crosses the sulcus, and enters area MST lying in the anterior bank of the STS. This is accompanied with a characteristic vanishing and reappearance of neuronal activity. The MST can then be recognized by a shift in receptive field location with respect to the previous position and by changes in response properties (larger

receptive fields, occurrence of responses to expansive and contractive visual motion).

Receptive fields of the recorded neurones were located within the central 30° of the visual field. Receptive fields were initially mapped using a hand-held projector. Next, the directional tuning was determined based on sensitivity to motion of a black–white sinewave grating (SF = 0.4 cycles/deg, TF = 4 Hz) moving in eight different directions evenly covering 360° of the vertical plane (45° steps). Circumference of stimuli was spatially optimised to fit the "classical" receptive field of the cell. Thereafter, we determined the ability of a neurone to process chromatic motion by presenting chromatic gratings (see above) drifting in the neurone's preferred and opposite (non-preferred) direction for 1,000 or 500 ms. Stimuli were displayed in a random order within the presentation cycles. The usual number of cycles was 6–7 (minimum number of cycles was 4).

Data analysis

For each modulation the average firing rate of the neurone beginning 50 ms after stimulus onset and terminating 50 ms after stimulus offset was computed. Spontaneous firing rate was determined from the first 300 ms of the pre-stimulus interval. Response to a given chromoluminant modulation was regarded significant if the mean activity exceeded the 98th percentile of the distribution of the spontaneous firing rate (spontaneous activity of a neurone was recorded before every stimulus presentation). This measure was chosen (rather than methods based on data dispersion) due to the relatively low number of repetitions in stimulus presentation, which was the necessary consequence of the high number of modulations tested. Spontaneous activity was subtracted from the mean firing rate in the light-adaptation experiment in order to exclude any unspecific effect of experimental manipulation. In order to be able to include multi-units and to pool recordings from several animals neuronal activity was normalised to the maximal mean firing rate that occurred with any of the modulations tested.

Azimuthal minimum (also termed residual responsivity) was defined as the mean minimal activity for the given azimuth for motion in PD. Activity at azimuthal minima was submitted to a linear regression analysis. Neuronal activity was modelled from the linear combination of cone excitations:

$$\text{activity} = e + w_{\text{LM}} \cdot (|\text{Le}| + |\text{Me}|) + w_{\text{S}} \cdot |\text{Se}|$$

where e, w_{LM}, w_{S} were estimated parameters, and Le, Me, Se were relative cone excitations at $V_{\lambda 10°}$ isoluminance. The regression was computed for every unit and also for the neuronal sample as a whole. Data for the regression of responsivity of individual units consisted of every response to stimulus presentation (i.e. all single trials). Individual neuronal average responses were used for the regression for the population of neurones. Sig-

nificance level for the regression and contribution of regressors was set to 0.05. In the alternative model, which assumed persistence of rod sensitivity, relative rod excitation at $V_{\lambda 10^\circ}$ isoluminance replaced the excitation of S-cones in the regression equation and w_R was the estimated parameter.

Light adaptation

Recordings of neuronal activity after white light adaptation ensuring rhodopsin bleaching were performed as a control experiment. The method was similar to that used by Dobkins and Albright (1993). Two reflectors illuminated a white board placed approximately 30 cm in front of the animal. The reflected light had an intensity of ~100,000 cd/m². The animal was exposed to this light for 2 min. Prior to the experiment, the time course of the absolute threshold for visual sensitivity after the 2-min light exposure was determined in two human subjects (including the author I.R.). They adjusted the intensity of a 7.5×7.5° wide square flickering at 2 Hz placed 10° below or above the fixation point on a black monitor screen, to make it just visible. We found that after an initial decrease (~2 min) caused by cone re-sensitisation the threshold was fairly stable for about 10 min (the rod-bleached cone plateau phase). After that time, the threshold began to decrease indicating rod

re-sensitisation. Recordings of neuronal activity were thus carried out in the interval 2–10 min after the adapting light was switched off. After 10 min the recording was stopped and the reflectors were turned on again. The procedure was repeated until the necessary number of trials was obtained.

Histological reconstruction

For the reconstruction of cortical recording sites cerebral hemispheres were cut in the frontal (three cases) or in the parasagittal plane (one case). Serial sections were cut in five alternate series and stained for Nissl, Klüver-Barrera, neutral red, myeloarchitecture (Gallyas 1979; as modified by Hess and Merker 1983), SMI 32 (Hof and Morrison 1995) and Wisteria floribunda agglutinin (WFA) (Brückner et al. 1994). Cortical penetration tracks were reconstructed from serial sections with the aid of the penetration scheme and electrolytic microlesions made at identified recording sites. Recording sites were marked on these tracks according to the location of microlesions and depth reading of the microdrive during the experiment. Two-dimensional cortical reconstructions were made by bending wires along layer IV of enlarged drawings of Nissl-stained sections through the entire hemisphere spaced 2 mm apart. Landmarks as lip and fundus of sulci were marked on

Fig. 2 Flat maps of the posterior part of the superior temporal sulcus of monkeys P, F, Z, and W. All maps are presented as left hemispheres to facilitate comparison, i.e. anterior is to the left, posterior to the right. *Thick solid lines* lip of sulcus, *thick dashed lines* fundus of sulcus, *thin dashed lines* myeloarchitectonic borders of areas MT, FST, V4t, and the densely myelinated zone (DMZ) of MST, *circles* recording sites, *scale bars* = 5 mm
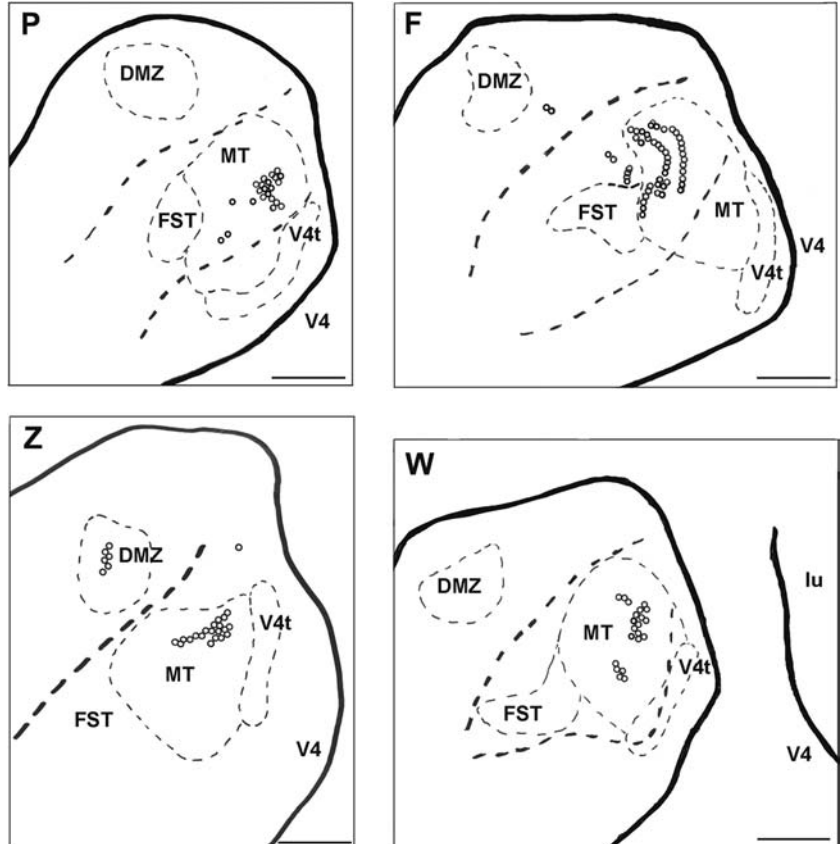
the wires, which were then soldered together appropriately to form three-dimensional models. These models were unfolded into two-dimensional maps of the hemispheres (Van Essen and Maunsell 1980). Borders of cortical areas were determined based on the myeloarchitecture, SMI-32, and WFA staining as described in the literature (summarised in Distler et al. 1993; Cusick et al. 1995; Hof and Morrison 1995). These areal borders together with the identified recording sites were then transferred onto the two-dimensional maps of the cortex (Fig. 2).

Behavioural experiments

Chromaticity and luminance of stimuli used in human behavioural experiments were identical to those used in animal recordings with the difference that isoluminance was set individually for each observer. In all experiments gratings subtended 7.5×7.5° and moved in horizontal direction. In order to eliminate the potential luminance artefacts borders of the stimuli were smoothed with a cosine contrast envelope. Software for the visual stimulation was written by I. Riečanský based on a specialised programme for presentation and control of psychophysical experiments developed by B. Krekelberg.

Observers were undergraduate and graduate students, collaborators, and members of the department staff. The age ranged from 23 to 37 years. One of the authors (I.R.) participated as subject in all experiments. Except for him all observers were not aware of the aims of the study. Most of the subjects had previous experience with psychophysical experiments. Seven subjects participated in speed judgments, seven in eye movement recordings, six in EqLC measurement. All observers had normal or corrected-to-normal visual acuity, and had no symptoms of colour vision deficiency as assessed by Ishihara colour plates and Lanthony's desaturated 15 hue test.

Subjects were seated in a darkened room in a comfortable armchair with a head stabilizer. Distance to the monitor was 57 cm. In the measurements of perceived speed and EqLC, observers viewed the display monocularly with the dominant eye while the non-dominant eye was covered by a mask. Test stimuli were presented in the upper visual field centred at 6° vertically, the fixation point was presented in the centre of the screen. In the eye movement recordings, the viewing was binocular, and the gratings were displayed in the centre of the screen.

Isoluminance setting

We used two photometric methods to equal the luminance of the stimuli individually for each observer: the minimal-motion technique of Cavanagh et al. (1987) and the heterochromatic flicker photometry.

In the minimal-motion paradigm a chromatic grating was interleaved at the same spatial location with an achromatic grating of identical spatial frequency (0.4 cycle/deg) and 10% luminance contrast. The chromatic grating was presented sequentially at phases 0° and 180°, the achromatic grating at 90° and 270°. Thus, due to the phase reversal each grating gives no net motion, if presented separately. If the chromatic grating is contaminated by luminance contrast, summation of luminance cues from both gratings provide motion information with the direction depending on the sign of luminance contrast in the chromatic grating. Each grating/phase was displayed for four frames (56 ms), which yielded a temporal frequency of 4.5 Hz. Observers fixated the central fixation point and adjusted the luminance contrast of the chromatic grating with a key press until they perceived no net motion. The judgment was repeated four times for each stimulus and the values were averaged. All stimuli were presented within a single block; the order of presentation was randomised. If needed, the judgments were repeated for selected stimuli.

Many subjects found the judgments with the minimal-motion technique difficult, and some were unable to perform them. In some observers, the variability among the trials was too large to yield a reliable estimation of the isoluminance point. Therefore, we alternatively determined the isoluminance by heterochromatic flicker photometry (HFP). Counterphase sinewave gratings which had the same spatio-temporal parameters as the moving test gratings (SF = 0.4 cycles/deg, TF = 4 Hz) were used, since the isoluminance point depends on the spatial and temporal frequencies of the stimulus (Cavanagh et al. 1987; Dobkins et al. 2000a). Subjects adjusted the luminance ratio of the grating by a key press until the percept of flicker was minimal. Chromatic gratings were displayed in a random order. Each grating was presented six times, the mean of the six judgments determined the isoluminance point. In subjects who had no problems to make minimal-motion judgments both minimal-motion method and HFP yielded equivalent values of estimated isoluminance. In three (of the seven) subjects we additionally determined the HFP isoluminance points at 12 Hz, and used both isoluminance points for the speed matching paradigm (see below), thus resulting in two speed matching curves for each of these three subjects.

Speed matching

Speed of the chromatic test grating (set to isoluminance of the individual subjects) was matched to the speed of a black–white sinewave grating of identical spatial frequency moving in the lower visual field at the same eccentricity (6°) in opposite direction. Luminance contrast of the match grating was 100%, mean luminance and chromaticity was equal to the grey background. The opposite directions of motion were chosen for the two stimuli to ensure that judgments were based on the

perceived speed, not on some other (e.g. positional) cues. Observers fixated the central point and indicated by a key press the stimulus that moved faster (two alternative forced choice). The test grating drifted at 4 Hz. Temporal frequency of the match grating was varied according to the subject's response in a simple staircase procedure: the frequency was increased after the test had been judged to move faster and decreased otherwise. Theoretically, the staircase converges to the exact value of the perceived speed of the chromatic test grating. Staircase parameters were as following: initial TF step = 1 Hz, final step = 0.2 Hz, required reversals to stop the procedure = 15. The last six reversals were averaged to yield an estimation of perceived speed. This value was then corrected for potential hemifield difference (Smith and Hammond 1986) by referring to perceived speed of a match-identical black–white grating moving in the upper visual field. Perceived speed of this grating was regarded to equal 4 Hz. In all experiments, the gratings were presented for 700 ms and the next trial started 980 ms after the response had been given. The time interval for the response was not limited, but all subjects responded immediately after the stimuli disappeared. The fixation point and the grey background were displayed during the whole interstimulus interval. Eight stimuli (all chromaticities with identical values of luminance contrast) or nine stimuli (when the black–white grating was additionally used as a test to assess the hemifield differences in perceived speed) were presented in one block in a random order. One session contained maximally two blocks.

Eye movement recordings

The measurements were performed using an EyeLink eye-tracking system (SensoMotoric Instruments, Germany). The system is head-mounted, eyeballs are illuminated by infrared LEDs, and the image is scanned by miniature cameras at a sampling rate of 250 Hz. We ensured that the position of the cameras did not restrain a free view on the monitor screen. Viewing was binocular, the position of the dominant eye was recorded. Each stimulus appeared 25 times moving to the right and 25 times moving to the left. Presentation was randomised, eight stimuli were presented in one block/session. In two observers the whole range of chromoluminant combinations was tested (seven sessions). Five subjects were tested at isoluminance only (one session). Start of each trial was determined by the experimenter. After a random time interval of 840–1,260 ms a moving grating appeared for 350 ms.

Subjects were given a passive instruction ("keep the stimulus sharply visible") in order to invoke passive optokinetic eye movements (optokinetic response—OKR). A break in the experiment was introduced whenever needed (e.g. in case of subjective discomfort). Stimulus onset was registered as an external event by the eye-tracking system, and the sequence of stimuli was reconstructed from the timing files created by the stimulation programme. Eye position data were digitally filtered with a two-pole Butterworth filter with 25 Hz frequency cut-off. The traces were aligned to the time of stimulus onset and to the position at the time of stimulus onset. Eye position data for the leftward visual stimulation were sign-inverted and data for a single chromatic stimulus were aligned. The traces were inspected, outliers and trials contaminated by saccades were manually removed, and the average eye position was computed. Eye speed was calculated by the differentiation of eye position data. The onset of eye movements was determined from the mean eye velocity and the eye position, by inspection as the time of the start of a consistent eye position deviation and speed increase. We measured the amplitude of the early open-loop period of eye movements (Tychsen and Lisberger 1986) , which was defined as the average gain of eye movements in the interval 80–120 ms after the OKR onset.

EqLC measurement

To measure the EqLC of the chromatic gratings we used a motion-cancellation method introduced by Cavanagh and Anstis (1991). A chromatic test grating and an achromatic luminance match grating of identical spatio-temporal frequencies were superimposed (optically fused by the presentation in alternate frames) and they drifted in opposite directions. Perceived motion direction in this display is determined by the grating with the higher effective contrast. The chromatic gratings were isoluminant; the achromatic grating had the chromaticity and mean luminance equal to the neutral grey background. Observers performed two-alternative forced choice judgements of motion direction. Based on the response given, luminance contrast of the achromatic grating was adjusted by a staircase rule: if motion in direction of the chromatic grating was seen, the luminance contrast of the achromatic grating was increased; if motion in direction of the achromatic grating was indicated, the reverse change was made. When both gratings were equally powerful perceptually, no consistent motion direction was seen and thus the probability to indicate either directions was equal (50%). At the start of the staircase, the achromatic grating had 0% luminance contrast, the initial step size of luminance increases was 10%, the final step size was 0.5%. 25 reversals were required to complete the procedure. Stimuli were presented in a random order within one block. The EqLC of the chromatic grating was calculated as the mean contrast of the achromatic grating from the last five reversals. Stimuli were presented for 700 ms, the response time was not restricted, but all observers responded immediately after the stimulus disappeared.

Fig. 3 Typical example of an MT single-neurone activity evoked by moving chromatic gratings (all 56 modulations are shown). *Circles* and *solid lines*: mean activity for stimulus motion in PD, *crosses* and *dotted line*: stimulation in ND, *error bars*: ±SEM. Spike plots and histograms depict activity in PD. *Asterisks* indicate modulations that failed to activate the neurone significantly, i.e. the mean PD activity did not exceeded 98th percentile of the distribution of spontaneous firing rate
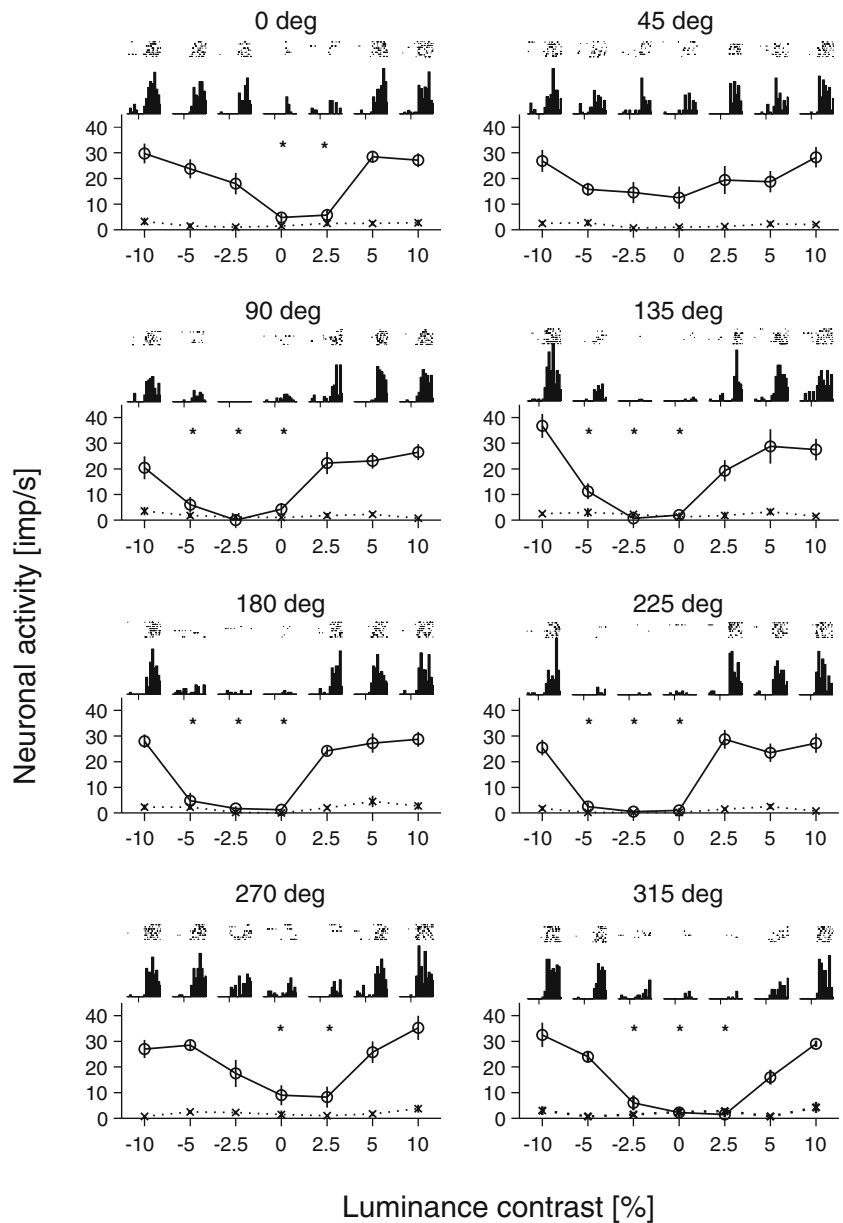
The next stimulus appeared 700 ms after the response had been given. In the inter-trial interval a fixation point and the grey background were presented.

Results

Electrophysiological experiments

Activation of MT/MST neurones by chromatic motion

We recorded from area MT and to a smaller extent from area MST in five hemispheres of four male macaque monkeys. Responses to chromatic visual stimulation were obtained from 207 MT neurones (163 single and 44 multi- units) and 19 MST neurones (14 single and 5 multi-units). A typical example of single-neurone activity in area MT is shown in Fig. 3. Discharge rate to stimulus motion in preferred (PD) and opposite non-preferred direction (ND) is depicted as a function of luminance contrast for all eight chromatic azimuths. At all colour azimuths the activity in PD was dependent on luminance contrast. Responses dropped with decreasing luminance contrast and reached minima close to $V_{\lambda 10°}$ isoluminance. The reduction of the response was often complete. At the modulations marked by asterisks the activity in PD was below the criterion which was set to the 98th percentile of the distribution of spontaneous firing rate (see Methods). The response to motion in ND was virtually absent at all modulations and thus could not be affected by luminance contrast. As evident from Fig. 3 minima in neuronal

512
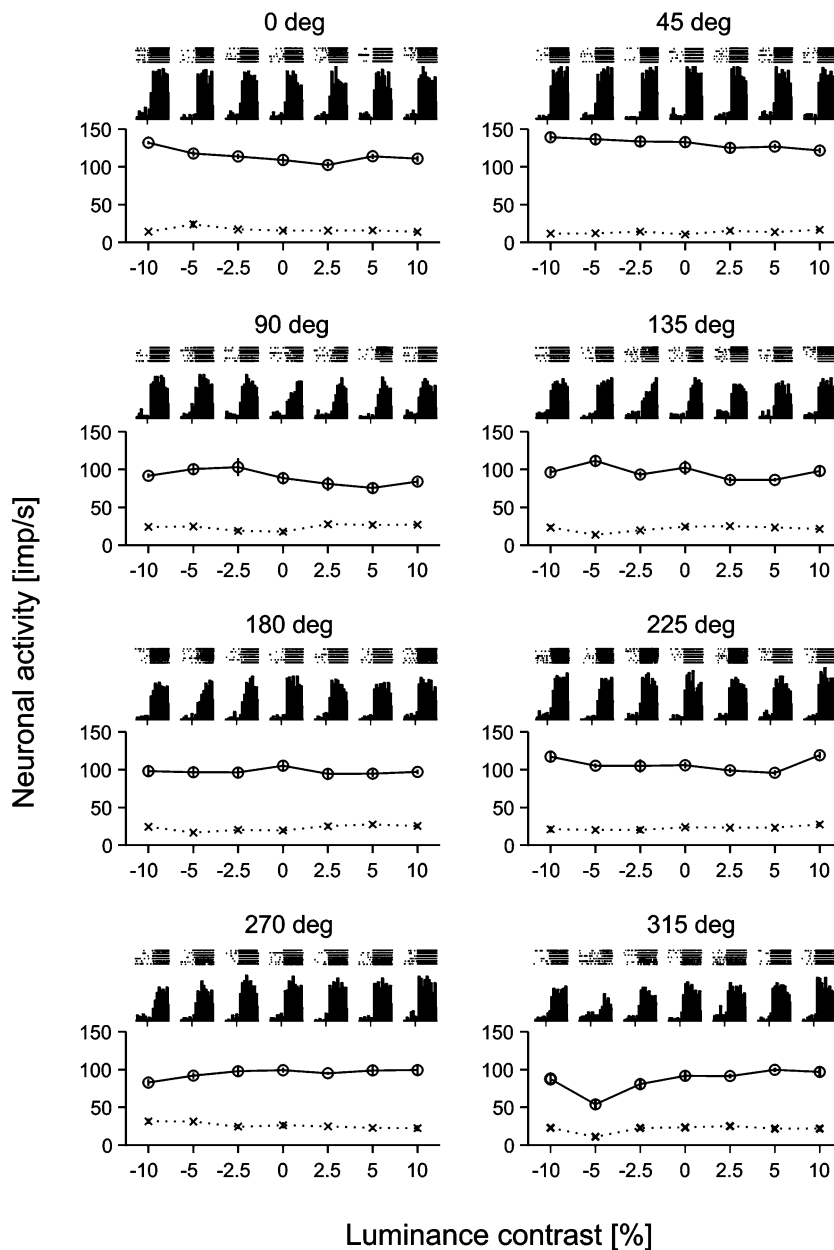
activity were not necessarily reached at $V_{\lambda 10°}$ isolumi-
nance. Factors contributing to this fact include the
physiological scatter of isoluminance points among MT
neurones (Gegenfurtner et al. 1994; Dobkins and Al-
bright 1995), inter-species (Dobkins et al. 2000b), and
inter-individual differences (see e.g. Chichilinsky et al.
1993), which may shift the true isoluminance point
away from zero as defined by the standard human
observer (CIE 1964 $V_{\lambda 10°}$ function).

Although not all units showed such regular pattern of
minima, this tendency was clearly seen for the vast
majority of neurones. MST units responded in the same
way and were thus pooled with the MT sample. We
found only 7 of 226 neurones (3%, five single and two
multi-units, all in MT) activated by every chromolumi-

nant modulation. Responses of one such neurone are
depicted in Fig. 4. The neurone was clearly direction-
selective at all modulations tested including isolumi-
nance. These units were found in one animal only.

Figure 5 shows the number of responsive neurones in
the MT/MST sample from one monkey. Neuronal re-
sponsivity was dependent on luminance contrast and
was lowest near $V_{\lambda 10°}$ isoluminance. Two chromolumi-
nant modulations did not activate any neurone and only
very few cells gave a significant response to several other
modulations. Modulations that failed to activate any
neurone were found in three of four animals (Fig. 6, see
below). This strongly indicates that area MT was not
able to provide information about motion of these
(isoluminant) stimuli.

Chromatic dependence of MT/MST responses

We did not find colour-selective MT and MST neurones, i.e. neurones with high responses restricted to certain chromatic azimuth(s) and independent of luminance contrast. However, among colour azimuths we observed systematic differences in responsivity at low luminance contrast. The neurone in Fig. 3 remained responsive to all luminance modulations at 45° despite response reduction around isoluminance. However, at other azimuths the cell was completely silenced. The chromatic preference of MT and MST units at low luminance contrast is evident from Fig. 5 (data from monkey Z). Figure 6 depicts the minimal percentage of responsive neurones for every animal at a given azimuth (x-axis). Thus, for monkey Z (Fig. 5) the following data points were selected: 0% $V_{\lambda 10°}$ luminance contrast (LC) was taken for 0 and 45°, −5% LC for 90°, −2.5% LC for 135, 180, and 225°, and +2.5% LC at 270 and 315°. The Fig. 5 demonstrates that reduction of responsivity at/ around isoluminance was in general prominent and often complete at 90, 135, and 180°, but much less dramatic at 0, 45, and 270°.
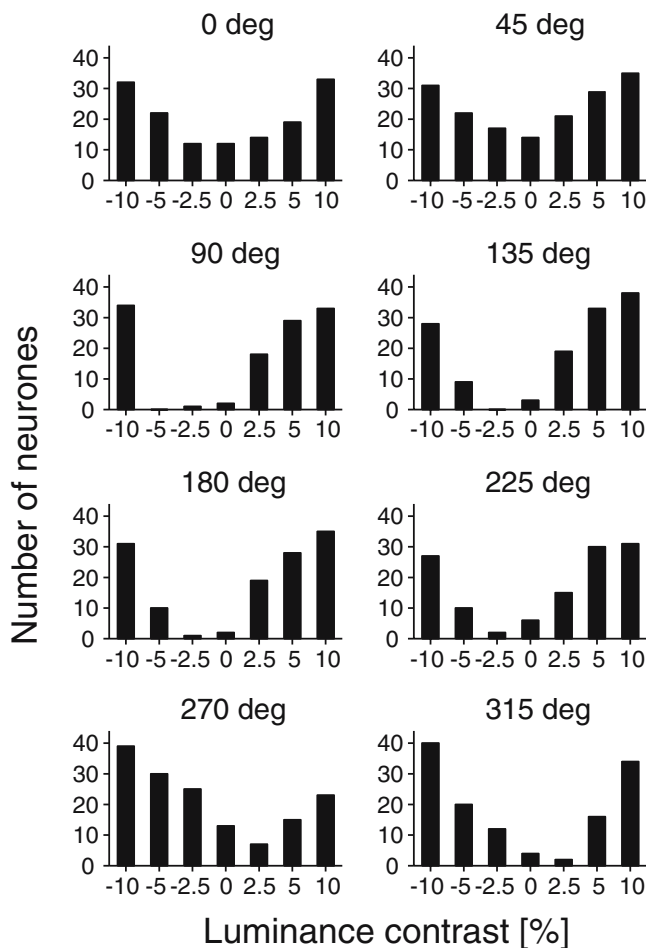
In order to study the chromatic dependence of MT/ MST activity in detail we selectively analysed the minimal (residual) responses at each azimuth (termed azimuthal minima) which enabled us to minimise the consequences of isoluminance point scatter among MT and MST neurones (Gegenfurtner et al. 1994; Dobkins and Albright 1995). To reveal the origin of chromatic preference the residual responses were modelled from cone activation. We considered linear summation of cone signals and calculated linear regression of neuronal activity on relative cone excitation at $V_{\lambda 10°}$ isoluminance (see Methods). The variables in the model included (1) the sum of L- and M-cone activation, and (2) S-cone activation. Summed excitation of L and M cones was taken due to the substantial overlap in absorption spectra that would result in detrimental collinearity in the regression model.

In 63 of 226 (24%) units the regression was significant ($P < 0.05$) on the activation of L + M cones only (Fig. 7). Responses of 26 of 226 (10%) were modelled from the excitation of S cones only. In 43 of 226 (20%) units both L + M- and S-cone excitation significantly contributed to the response model. For these units, L + M-cone weight (w_{LM}, see the regression equation in Methods) was on an average of 4.4 times higher than S-cone weight (w_S). The combination of L + M- and S-cone activation significantly predicted the mean activity of MT/MST neurones at azimuthal minima (Fig. 8, thick solid line, $R^2_{adj} = 0.085$, $P < 0.001$) yielding $w_{LM}/w_S = 5.4$.

In summary, our analysis suggests that signals from all cone classes reach areas MT and MST. However, the input is uneven—the contribution from S cones is lower than from L + M cones by about factor 5.

The fact that absorption spectra overlap between S-cone and rods raised the possibility that the S-cone contribution might have been artificially enhanced by rod intrusion. Although there is no reason to expect a high rod influence at the retinal illuminance from our visual stimulation (Lee et al. 1997), this was in fact below the value ensuring full rod bleaching (Wyszecki and Stiles 1982). A comparatively good fit of the mean MT/ MST neuronal activity was obtained from the combination of L + M and rod excitation (Fig. 8, thick dashed line). The model yielded w_{LM}/w_R ratio equal to 1.9. Therefore, we carried out a control experiment, in which we recorded neuronal responses before and after a high-intensity light adaptation (see Methods). If rod activation contributed to the chromatic preference of MT/ MST neurones, we would expect a decrease in neuronal activity correlating with the magnitude of rod excitation.

The experiment was analysed using a three-way RM ANOVA (three within-subject factors: 'light adaptation', 'direction of motion', 'colour azimuth') with subsequent fractionation of the complex statistical model using the Bonferroni–Holm sequential method for correction of significance level (Holm 1979; cited in Havránek 1993). Only the activity in PD was affected by the experimental manipulation and is presented in Fig. 9. The effect of light adaptation on activity in PD de-

Fig. 5 Number of MT/MST neurones that gave significant responses to motion in PD in animal Z (n = 88)

Fig. 6 Percentage of MT/MST neurones that gave significant responses to motion in PD at different colour azimuths (*x*-axis). In every animal the luminance modulations are selected that yielded the lowest number of significantly activated units at a given chromatic azimuth (for procedures see text)
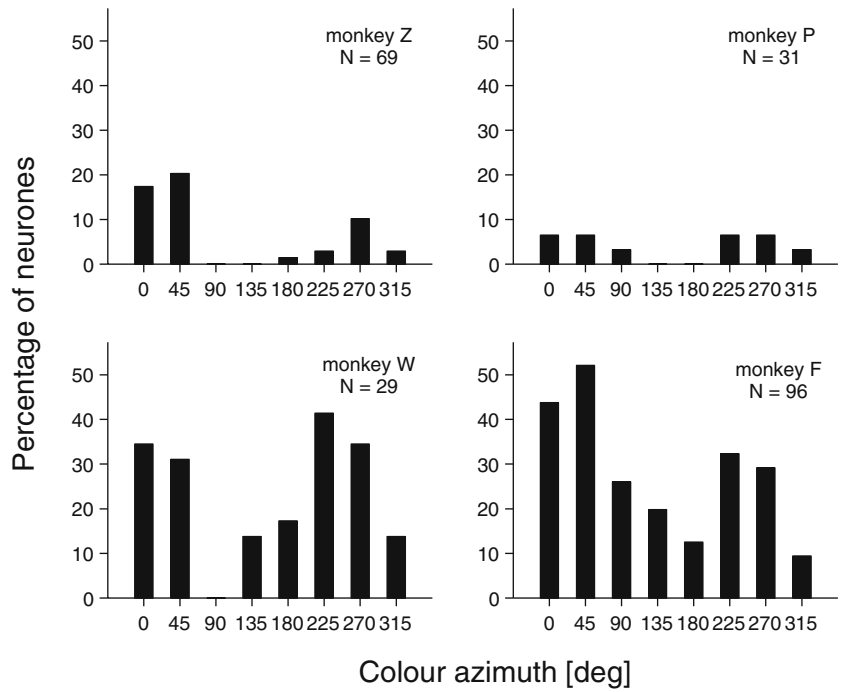
pended on stimulus chromaticity—the interaction of factors 'light adaptation' and 'colour azimuth' was significant ($P < 0.05$, two-way RM ANOVA, sequential Bonferroni–Holm significance level correction). Light

adaptation removed the significant difference in activation among the colour azimuths (one-way RM ANOVA, $P < 0.05$, sequential Bonferroni–Holm significance level correction). All 11 neurones decreased their responsivity at the azimuth with the highest presumed rod activation (45°). The effect of light adaptation on the mean activity was significant at 45° and 0° (paired *t* test, $P < 0.05$, sequential Bonferroni–Holm significance level correction).
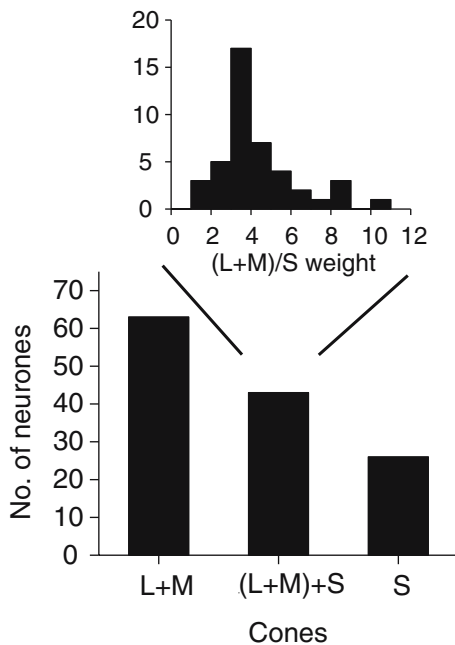

Fig. 7 Number of MT/MST neurones ($n = 226$) whose residual responsivity could be significantly ($P < 0.05$) fitted from the linear combination of cone signals. $L + M$ units with responses correlating with $L + M$-cone excitation only, S units with responses correlating with S-cone excitation only, $(L + M) + S$ units with significant contribution of both $L + M$- and S-cone excitation. Plot above the latter column depicts the distribution of w_{LM}/w_S ratio in those units
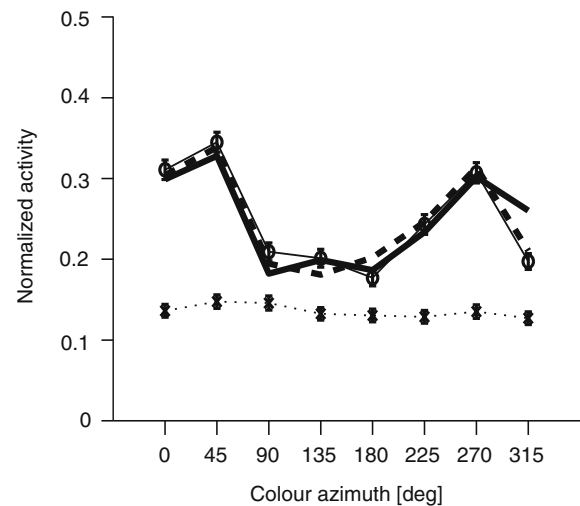

Fig. 8 Mean normalised activity of MT and MST neurones at azimuthal minima. Included are cells from all monkeys, $n = 226$. *Circles* and *thin solid line* mean response to stimulation in PD, *crosses* and *dotted line* stimulation in ND, *error bars* ± SEM. *Thick solid line* response fit from a linear regression model assuming linear summation of cone signals ($w_{LM}/w_S = 5.4$). *Thick dashed line* linear regression model assuming the persistence of rod sensitivity ($w_{LM}/w_R = 1.9$)

Assuming the rod intrusion to MT/MST responses we could additionally expect light adaptation to cause a shift in the values of luminance contrast at which the azimuthal minima occur. Therefore, between chromatic azimuths we compared the number of neurones that shifted their minima following light adaptation. Due to the small sample size we simplified the design and compared the occurrence of shifts between azimuths with high (0, 45, 90, 270°) and low (135, 180, 225, 315°) rod activation. Response minima shifted more often for azimuths with high expected rod excitation but the probability only approached statistical significance (χ^2 test, $P = 0.064$). This indicates that the changes in MT/MST neuronal responsivity following the light adaptation might not be attributed solely to rod desensitisation.

In summary, the dependence of MT/MST activity at azimuthal minima on $L + M$ cone excitation can be attributed to residual responsivity of the magnocellular pathway at isoluminance (Kaiser et al. 1990; Valberg et al. 1992). Our data indicate in general no input from the colour-opponent parvocellular pathway as the responsivity to certain modulations activating P-LGN was very unreliable and consistently present in only one animal. An input from K-LGN seems likely since we did find some indication of contribution from S cones (Dacey 2000; Hendry and Reid 2000; Sun et al. 2004; Sincich et al. 2004), although this could partly be explained by rod intrusion.

Behavioural experiments

In order to make inference about the role of area MT in the processing of chromatic motion we carried out a series of behavioural experiments on human subjects confronted with the same visual stimuli used in the electrophysiological recordings. For human observers, however, isoluminance was determined photometrically
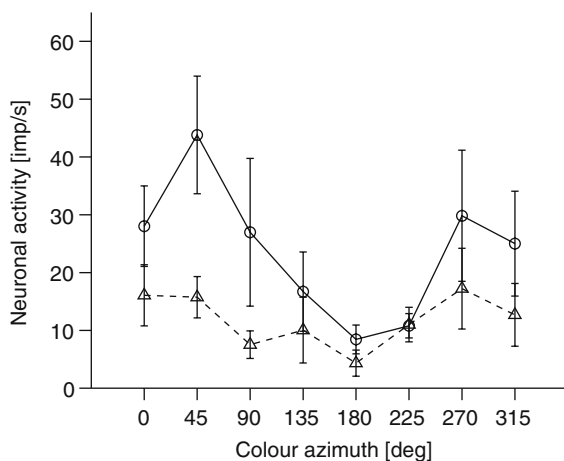

Fig. 9 Effect of light adaptation on MT activity at azimuthal minima ($n = 11$). *Circles* and *solid lines* original (mesopic-photopic) conditions, *triangles* and *dashed lines* activity after the light adaptation (pure photopic), *error bars* $\pm$ SEM

(see Methods) in order to remove the variability resulting from inter-individual differences in isoluminanance points. The following variables that relate to motion processing were measured: (1) apparent speed of the chromatic gratings, (2) amplitude of evoked passive optokinetic eye movements, and (3) motion-cancellation equivalent luminance contrast at isoluminance.

Perceived speed of chromoluminant gratings

Judgements of speed of the chromatic gratings were inter-individually very variable (Fig. 10). For only one subject (SD) perceived speed was directly related to luminance contrast and was clearly underestimated near isoluminance. In some trials SD completely failed to see motion of the stimuli and reported to see them stationary. To all other subjects motion of every grating was clearly visible. In one subject the judgements for
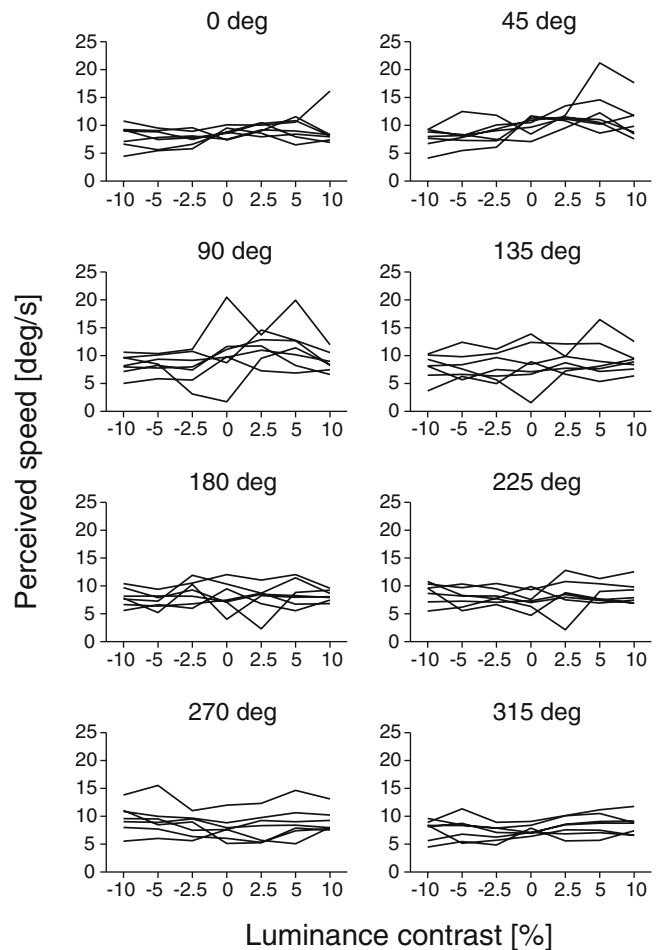

Fig. 10 Perceived speed of chromatic gratings in seven subjects. Speed of the chromatic test gratings moving in the upper visual field was matched to the speed of a black–white comparison grating moving in the lower visual field in the opposite direction. Speed judgements were corrected to the perceived speed of match-identical black–white reference test grating moving in the upper visual field (for details see Methods)
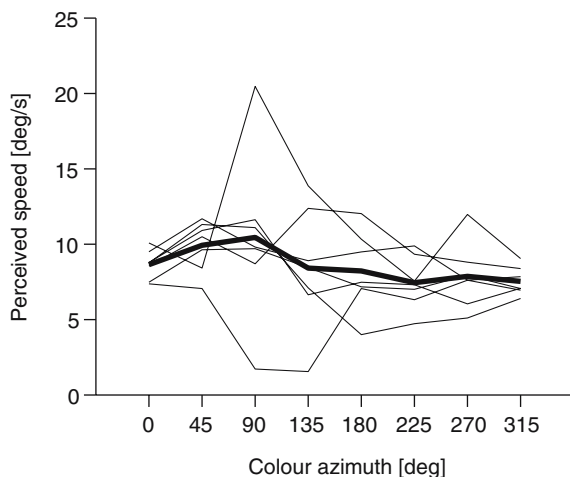
Fig. 11 Perceived speed of chromatic gratings at isoluminance. Data are replotted from the previous figure. *Thick line* indicates the mean performance

isoluminant stimuli were higher than for grating with non-zero luminance contrast. Perceived speed of the remaining 5 observers could vary considerably as a function of position in chromaticity space, but no regular dependence on luminance contrast was observed. This result contrasted with the neuronal activity in macaque MT/MST which was directly related to luminance contrast and often vanished entirely at certain (isoluminant) modulations.

Speed judgements for the isoluminant gratings are replotted in Fig. 11. Despite great intersubject differences the performance was determined by factors other than the residual activity of macaque MT/MST neurones (Fig. 8). Neither any individual (Fig. 11, thin lines) nor the group mean performance (thick line) correlated significantly (Pearson correlation coefficient, $P > 0.05$) with the mean normalised activity of macaque MT and MST neurones at azimuthal minima (Fig. 8). Unlike the MT/MST neuronal activity the chromatic preference of speed judgments could not be modelled from the linear combination of photoreceptor excitation at $V_{\lambda 10°}$ isoluminance (neither $L + M-$ and S-cone nor $L + M$ cones and rods).

In the experiments described above we used isoluminance points determined by the minimum motion technique (at 4.5 Hz) or by HFP at 4 Hz. Traditionally, determination of HFP has been performed at higher temporal frequencies (> 10 Hz). Individual isoluminance settings depend on temporal frequency (Cavanagh et al. 1987; Dobkins et al. 2000a) and it was thus important to determine the individual isoluminance points at the temporal frequency that was also used for the behavioural (and neuronal) testings. Despite this necessity, we nevertheless tested for the possibility that our behavioural results could be dependent on which isoluminance settings were used. Therefore, we additionally determined the individual isoluminance points in three of our subjects at 12 Hz using HFP, and used these isoluminance points for the speed matching para-

digm. In accordance with the previous measurements (Fig. 10), isoluminant stimuli determined by this method did not compromise perception of motion for any of the chromaticity axes or any of the luminance values that were added to the grating. Using isoluminance settings determined at 12 Hz HFP we also found high-speed judgements for the 90° chromatic direction. Thus, speed-matching results obtained with 4 Hz HFP isoluminance points and obtained with 12 Hz HFP isoluminance points yielded very similar results, and both were in contrast to the activity of MT neurones.

We therefore conclude that human perceptual experience of motion of chromoluminant gratings does not correspond to neuronal activity in areas MT and MST in the anaesthetised macaque monkey.

OKR to moving chromoluminant gratings

In addition to measurements of perceived speed we investigated the amplitude of the early open-loop phase (Tychsen and Lisberger 1986) of optokinetic eye movements (OKR). Such passive reflex-like behaviour is expected to be relatively free of voluntary, attentive, and decisional factors inherent to measurements of perceived speed but obviously absent in the anaesthetised monkeys as used for the electrophysiological recordings. Due to the variation of the isoluminance point with retinal position we used a small-field grating (rather than a traditional wide-field stimulus) that could be set to isoluminance (see Methods). Amplitude of OKR in two subjects is shown in Fig. 12. All stimuli readily elicited the optokinetic reflex. Latencies of OKR ranged from 80 to 152 ms and were mildly affected by luminance contrast in one observer. In one subject the eye speed seemed to be influenced by the luminance contrast at some chromaticities (e.g. at 90 and 135°). However, this tendency was far less consistent than the regular dependence of macaque MT/MST neurones on luminance modulation. In the second subject no clear relationship of OKR amplitude to luminance contrast was seen.

Figure 13 depicts OKR in seven subjects at isoluminance. There was no significant correlation (Pearson correlation coefficient, $P > 0.05$) between the OKR amplitude [of individual data (Fig. 13, thin lines) and the group mean (Fig. 13, thick line)] with the mean activity of macaque MT/MST neurones at azimuthal minima (Fig. 8).

As for the previous paradigm, the linear regression model was computed to analyse the mechanism of chromatic preference. In one subject the eye speed significantly ($P < 0.05$) correlated with the excitation of $L + M$ cones. OKR of another subject was successfully ($P < 0.05$) modelled from the linear combination of $L + M-$ and S-cone excitation. w_{LM}/w_S equalled 5.1, which is similar to the value resulting from the model of the mean MT/MST activity. Nevertheless, as already mentioned, performance of this subject did not signifi-
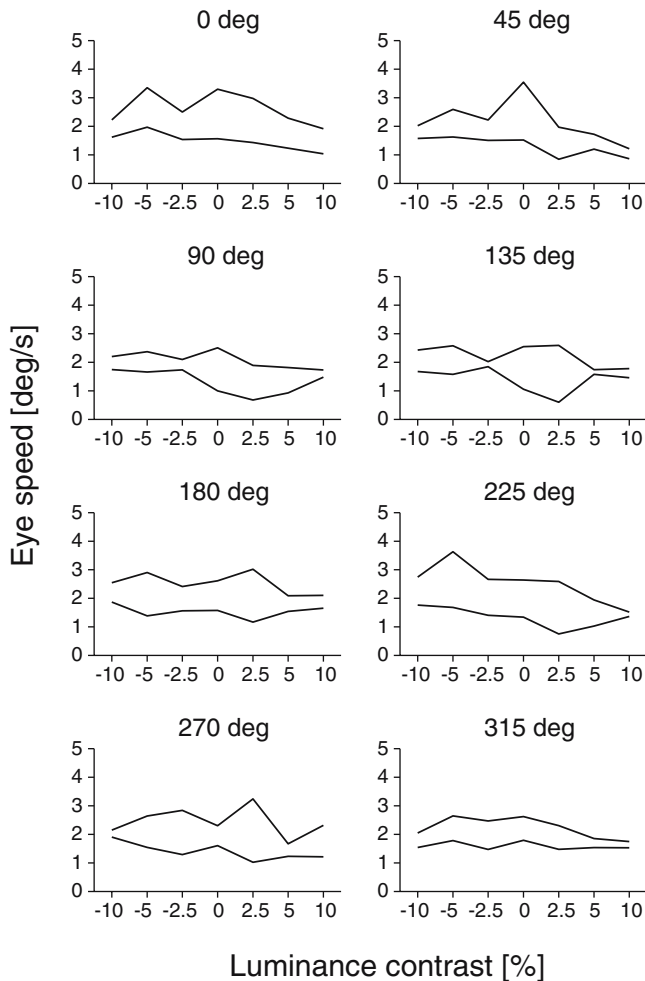
Fig. 12 Amplitude of OKR evoked by chromatic gratings in two subjects. Data represent the mean gain of eye movements in the interval 80–120 ms following the OKR onset (see Methods)

Fig. 13 Amplitude of OKR at isoluminance in seven subjects. *Thick line* indicates the mean performance

cantly correlate with the activity of MT/MST neurones (Pearson correlation coefficient, $R = 0.611$, $P = 0.108$). Regression on the combination of $L + M$ and rod excitation was insignificant in this observer. In all other subjects, the linear model failed to fit the eye speed data.

In summary, similarly to the measurement of perceived speed, there was no clear correspondence between the OKR amplitude in human observers and the activity in areas MT and MST in anaesthetised macaques.

EqLC at isoluminance

Equivalent luminance contrast as derived from the motion-nulling paradigm (Cavanagh and Anstis 1991) was the measured variable in two previous studies investigating the role of area MT in the processing of chromatic motion (Thiele et al. 1999b, 2001). Our recordings revealed chromatic dependence of neuronal activity at isoluminance so that we set out to explore the dependence of EqLC on chromaticity, which has not been studied before.
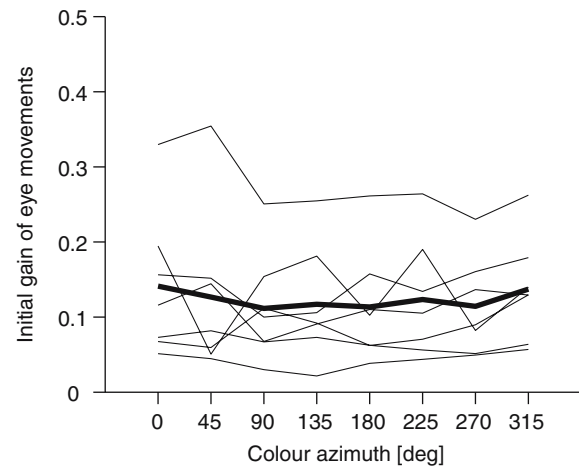
EqLC of the isoluminant gratings in six subjects is plotted in Fig. 14. Judgements of all observers were fairly similar. Unlike perceived speed and OKR amplitude EqLC corresponded quite well to the residual MT/MST activity (Fig. 8). Performance of four subjects was significantly correlated (Pearson correlation coefficient, $P < 0.05$) with the mean normalised activity of macaque MT/MST neurones at azimuthal minima and the correlation of the group mean EqLC was significant as well (Pearson correlation coefficient, $R = 0.795$, $P = 0.018$).

Linear regression on cone excitation at $V_{\lambda 10°}$ isoluminance significantly modelled EqLC from the combined activation of $L + M$ and S cones in three subjects. Behaviour of the observers was modelled from $L + M$-cone excitation only. Performance of the remaining subject was not significantly fitted with any regressor. Regression of the group performance yielded a significant contribution from all cones ($R^2_{adj} = 0.543$, $P < 0.001$) with the ratio $w_{LM}/w_S = 8.5$. The alternative
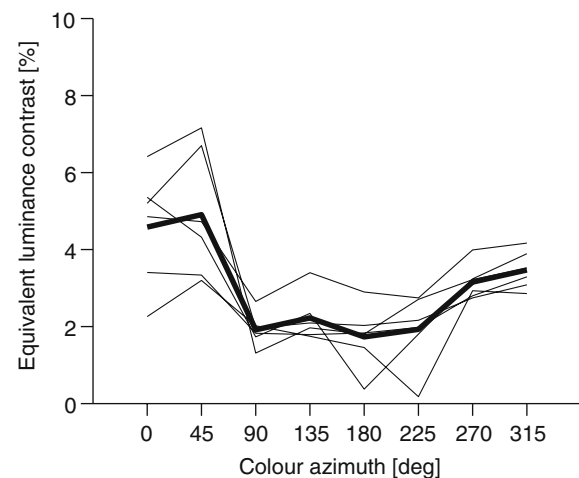

Fig. 14 Motion-nulling EqLC of isoluminant gratings in six subjects. *Thick line* indicates the mean performance

model, assuming persistence of rod sensitivity ((L + M) + rods), also yielded a significant fit of the mean group EqLC. In contrast to the cone model, however, the regression was significant for only one subject.

Discussion

Dependence of MT/MST responsivity on luminance contrast

Our results largely confirm earlier reports on response properties of directionally selective MT and MST neurones. Dependence of activity in areas MT and MST of the macaque and human V5/MT complex on luminance contrast has been found in numerous single-unit and functional imaging studies (Saito et al. 1989; Sclar et al. 1990; Cheng et al. 1994; Gegenfurtner et al. 1994; Dobkins and Albright 1994, 1995; Tootell et al. 1995; Wandell et al. 1999; Thiele et al. 2000). In accordance with these, we observed a prominent decrease in responsivity with decreasing luminance contrast in the vast majority of neurones. On the other hand, Saito et al. (1989) and Gegenfurtner et al. (1994) reported examples of MT neurones with constantly high responsivity to chromatic stimuli apparently unaffected by the magnitude of luminance contrast. We encountered only few such cells and, interestingly, all were found only in one of four monkeys.

A part of the variability may be attributed to inter-animal differences in isoluminance point. In the electrophysiological experiments we used a standardised luminous efficiency function so that inter-individual variability in the isoluminance point would become apparent. Moreover, in monkeys P, W, and F (but not in monkey Z) we used $V_{\lambda 2°}$ also for parafoveal recordings. It cannot be ruled out that we did not "hit" the isoluminance point of the monkey F at any chromatic azimuth. It is possible that by using finer steps in luminance contrast or scaling the stimuli individually according to the perceptual (behavioural) isoluminance we could have found lower numbers of neurones responding near isoluminance in this animal. However, the occurrence of neurones apparently unaffected by luminance modulation cannot be entirely explained this way.

The difference between animals cannot be attributed to a specific anaesthetic agent such as fentanyl. In monkey F, fentanyl was used for additional analgesia as was in monkey Z (but not in monkeys P and W). Inter-animal differences in the exposure to environmental factors (e.g. daylight) can also be ruled out. Great differences were also found between the human subjects which we shall proceed to discuss.

Dependence of MT/MST responsivity on chromaticity

Our recordings from area MT are in accordance with previous studies that reported neurones in this area to be lacking colour selectivity (Zeki 1974, 1983; Maunsell and Van Essen 1983). In fact, in addition to directional selectivity the absence of colour preference is regarded as a characteristic MT property and one of the basic arguments supporting the theory of functional specialization in the primate extrastriate cortex (for review see Zeki 1993). We did not observe true colour-specific responses independent of luminance contrast, but azimuthal differences in responsivity became apparent near/at isoluminance. Our analysis suggests that these are largely explained by residual responsivity of M-LGN neurones (Kaiser et al. 1990; Valberg et al. 1992). Several previous studies concluded that area MT receives predominantly magnocellular input (Maunsell et al. 1990; Dobkins and Albright 1995; Seidemann et al. 1999; Thiele et al. 1999b, 2001). Moreover, the fact that in some animals we found no neurones responding significantly to certain (isoluminant) modulations indicates a potential total absence of colour-opponent P-LGN input.

Additionally, our data suggest the presence of S-cone signal in areas MT and MST. Because unequivocal evidence of S-cone input to M-LGN cells is lacking (Chatterjee and Callaway 2002; but see Sun et al. 2004) we suggest that this S-cone input to MT/MST is mediated by K-LGN cells. Such a proposal is supported by data from Sincich et al. (2004) who revealed a direct projection from K-LGN neurones to area MT in the macaque. S-cone input to MT neurones has already been reported by Seidemann et al. (1999). These authors estimated the weight of the S-cone signal to MT/MST responses to be about ten times lower than the weight of the summed L + M signal. Our analysis suggests an even higher contribution (on average ~20%) but could have been influenced by rod intrusion as our stimulation did not reach the level of retinal illuminance required for complete rhodopsin saturation (~2,000 scotopic trolands; Wyszecki and Stiles 1982). The discrepancy between our study and that by Seidemann et al. (1999) could be due to their determination of S-cone contributions in awake animals using 2° cone fundamentals, while we used an anaesthetised preparation and 10° fundamentals (the latter was more appropriate for the eccentricities of receptive fields from our cells).

Although Lee et al. (1997) reported a low rod intrusion at retinal illuminance above 200 photopic trolands, they were able to detect rod signals at higher levels in some magnocellular neurones. Sensitivity of human V5/MT complex to scotopic visual stimulation was confirmed by Hadjikhani and Tootell (2000). Additionally, Elfar and Britten (1998) reported responses of MT neurones being influenced by rod activation in chromatic visual stimulation similar to ours.

Our model, assuming rod mediated MT/MST activity at azimuthal minima, yielded a significant fit of the data. It should be stated, however, that our results of the rod-bleaching experiment remained inconclusive. On the one hand, light adaptation attenuated responses at azimuths with high rod activation. On the other hand, at these azimuths light adaptation did not shift the response

minima to different values of $V_{\lambda10°}$ luminance contrast. Therefore, it is possible that cones were affected by the light adaptation too, potentially due to anaesthesia, as an inhibitory effect of halothane (the anaesthetic we used) on cone adaptation was reported by van Norren and Padmos (1975, 1977).

Perceived speed of chromatic gratings

Human behavioural measurements employed the same stimuli used in macaque electrophysiological recordings. This allowed a direct comparison of behavioural variables with neuronal activity. The only difference was the calibration of luminance contrast—isoluminance was individually set in order to ensure equal stimulation conditions for every observer. Surprisingly, perceived speed of chromatic gratings was very variable. Differences in the value of the perceived speed could be partially attributed to hemifield (upper vs. lower) differences in apparent speed (Smith and Hammond 1986). These were evident from the speed matching of a match-identical black–white test grating moving in the upper visual field. Therefore, speed of this grating served as a reference for speed of the chromatic stimuli. This hemifield difference was not sufficient to explain the variable effect of luminance contrast on perceived speed observed among the subjects.

In all but one observer, reduction of luminance contrast did not reduce perception of motion (i.e. the apparent speed). The motion of all stimuli including isoluminant was readily visible and speeds were generally perceived veridically. Perception of (speed) motion thus sharply contrasted with neuronal activity in macaque areas MT and MST that showed a high dependence on luminance contrast and a complete response absence for certain isoluminant stimuli. The speed-matching results may at first glance appear at odds with the traditionally cited phenomenon of motion-slowing at isoluminance (Cavanagh et al. 1984). Motion slowing was generally observed at low speeds (temporal frequencies), in general below 4 Hz (Cavanagh et al. 1984; Teller and Lindsey 1993; Hawken et al. 1994; Gegenfurtner and Hawken 1996; Farell 1999; Dougherty et al. 1999). Perceived speed at higher temporal frequencies is not affected by changes in luminance contrast (Hawken et al. 1994; Gegenfurtner and Hawken 1996). Therefore, it was not really a surprise that most observers judged the speed of isoluminant gratings veridically. For a single subject (SD) we found a clear motion-slowing at isoluminance. For this subject the temporal frequency (speed) was possibly not high enough to reach the zone where perceived speed of chromatic and luminance stimuli does not differ.

We were also surprised to find a subject who saw isoluminant gratings to move faster than luminant gratings. From her response profile we can rather state that the speed of the isoluminant targets was perceived veridically, and that the gratings away from isolumi-

nance were seen to move slow. Although it has been reported that addition of chromatic contrast to a luminance grating decreases its apparent speed (Cavanagh et al. 1984; Farell 1999), the case when a pure chromatic grating is perceived to move substantially faster than a chromoluminant combination has not been reported to the best of our knowledge. On the other hand, Chawla et al. (1998, 1999) observed in some subjects higher activation of MT/V5 complex by moving isoluminant chromatic stimuli than by moving high-luminance contrast stimuli. Based on the arguments of Wandell et al. (1999), the perceived speed of chromatic stimuli for these subjects should be maximal at isoluminance.

The fact that the maximal slowing at azimuths 180° and 225° occurred in subject SD slightly away from isoluminance could question our calibration of isoluminance for this subject (isoluminance was determined by HFP at 4 Hz). It is conceivable that the isoluminance point derived from HFP using these settings did not exactly correspond to the isoluminance point for the motion system. HFP at low temporal frequencies is more difficult to perform than at higher temporal frequencies, and the above described result may mirror this fact. However, as already discussed in connection to inter-animal differences, even if we assume a discrepancy between the "used" and the "real" isoluminance point for this subject, we cannot explain the systematic difference in speed matching behaviour of SD compared to speed matching behaviour in the other observers.

We found high inter-individual variability of perceived speed at isoluminance as a function of the chromatic axis of the motion stimuli. This, again, could cast doubt on the method of isoluminance determination. However, the speed judgements did not depend on the method of isoluminance calibration. In a subset of observers we measured speed matching at isoluminance points determined by HFP at 4 Hz as well as using isoluminance points determined by a "classic" HFP (a square flickering at a higher frequency of 12 Hz). Speed-matching measurements using the latter isoluminance points were in agreement with those using the former, and neither yielded the pattern evident in MT/MST neurones.

Optokinetic eye movements

To the best of our knowledge the onset of optokinetic eye movements to isoluminant stimuli has not been investigated so far. Our experimental design differs from the conditions used to elicit so-called ocular following responses (Miles et al. 1986; Gellman et al. 1990; Kawano 1999). The paradigms for evoking ocular following response used a sudden movement of previously visible large stationary scene. Our stimuli were not visible before the motion was initiated and were restricted to the central portion of the visual field in order to largely parallel conditions of MT/MST recordings.

We would have expected to find a dependence of OKR on luminance contrast based on previous reports. Crognale and Schor (1996) reported a lower efficiency of isoluminant stimuli in the generation of optokinetic nystagmus (OKN) compared to luminant stimuli. Isoluminant chromatic stimuli were also less effective in eliciting "early suppressed ocular following response" (reflexive eye movements to moving background during fixation of a stationary target) in macaques (Guo and Benson 1999). Von Campenhausen and Kirschfeld (1999) measured spectral sensitivity of OKN and concluded that OKN is generated by a luminance-based system lacking the S-cone input. In addition, we found that the MT units projecting to subcortical nuclei involved in oculomotor control (NOT-DTN, DLPN) did not exhibit response properties different from the rest of the MT population—all were highly sensitive to luminance contrast (unpublished data). Contrary to our expectation, the amplitude of OKR was independent of luminance contrast. Moreover, OKR latency showed no clear dependence on luminance contrast either.

Inter-individual differences in OKR amplitude at isoluminance were high so that a generalization is difficult. Nevertheless, our results contradict earlier reports (Crognale and Schor 1996; von Campenhausen and Kirschfeld 1999) that the optokinetic system is lacking input from S cones. Correspondence with chromatic preference of MT/MST neuronal responses was absent in every subject. Our results thus do not support the notion that OKR onset in humans is based on the neuronal substrate equivalent to MT and MST of the anaesthetised macaque.

Equivalent luminance contrast

EqLC of isoluminant gratings measured by motion-nulling was the only behavioural parameter that significantly correlated with the residual activity of macaque MT/MST neurones. Previously, Thiele et al. (2001) reported a discrepancy between human and macaque EqLC when luminance contrast was added to chromatic grating. In macaques colour-mediated motion signals depended on luminance contrast of the stimulus and animal performance corresponded well with the activity of MT neurones. In humans, on the contrary, contribution of colour was constant and independent of luminance contrast added to the chromatic modulation (Cavanagh and Anstis 1991; Thiele et al. 1999b, 2001). Following prediction from a hypothetical model, chromatic motion signal in humans has been attributed to a colour-opponent parvocellular mechanism (Cavanagh and Anstis 1991). However, chromatic component of the stimuli has never been systematically varied in previous studies. Our data suggest, in contrast, that human motion-nulling judgements were primarily based on the activity of the magnocellular system as were also the responses of macaque MT/MST neurones. However, this suggestion needs to be treated with caution. We only

measured the EqLC when the chromatic grating was at isoluminance (due to the large number of chromatic directions investigated). The discrepancy between the human and macaque EqLC in previous studies became apparent only when luminance was added to the chromatic grating. Upon such manipulation monkey psychophysical EqLC and neuronal EqLC decreased (Thiele et al. 2001), while human psychophysical EqLC did not (Cavanagh and Anstis 1991; Thiele et al. 1999b, 2001). Thus, further experiments are necessary to determine the range of EqLC at different chromaticities and luminance additions in humans.

Multiple mechanisms may interact in motion-cancellation judgements. The rationale of the paradigm supposes an independent processing of colour and motion signals at an early stage and a linear summation of the two signals at a later stage that finally governs directional decision (Cavanagh and Anstis 1991; Agonie and Gorea 1993). In fact, however, the structure of the perceptual system may deviate from this expectation. For example, (1) at higher temporal frequencies, both P-LGN and M-LGN neurones are able to detect pure chromatic modulation at perceptual threshold (Lee et al. 1990); (2) many neurones in the striate cortex signal, the presence of both chromatic and luminance borders equally well (Johnson et al. 2001).

Our data show that chromatic preference varies with the motion paradigm tested. This suggests that different paradigms might employ different neuronal substrates. Our results provide support to arguments of Agonie and Gorea (1993). The authors demonstrated great differences in EqLC as derived from various motion paradigms and concluded that colour contribution to motion signal depends on processing stage.

Does area MT subserve chromatic motion processing?

The great discrepancy between human perceptual performance and MT neuronal activity evoked by equivalent visual stimuli suggests that MT does not contribute to motion processing of purely chromatic stimuli. However, for several reasons it is not possible to make general and final conclusions about the role of area MT in colour motion perception. The following factors must be considered:

Central anaesthesia There are several indications that the responsivity of MT (and other cortical) neurones under general anaesthesia might be different from the awake state. Gegenfurtner et al. (1994) recorded from anaesthetised macaques and reported about 80% of MT neurones being completely unresponsive at isoluminance. Contrary to this, Dobkins and Albright (1994) recording from awake animals found a substantial reduction of activity in only 10% of MT cells. A depressive effect of central anaesthesia might have led to a far lower proportion of MT neurones responsive to second-order motion in the study of O'Keefe and

Movshon (1998) in comparison to experiments of Albright (1992). Pack et al. (2001) reported a lower ability of MT neurones to integrate local motion signals under central anaesthesia in comparison to the awake state.

The effect of central anaesthesia might be, at least partially, attributed to the elimination of attentional factors. It has been argued that higher-level attentional mechanisms such as feature-tracking (Cavanagh 1992) or selective attention to colour (Blaser et al. 1999; Lu et al. 1999) play a crucial role in chromatic motion perception (but see Thiele et al. 2002).

Spatio-temporal parameters of the visual stimulation The spatio-temporal parameters of the stimuli were constant in all experiments. From this it follows that neurones may not have been confronted with their individual preferred stimulus configuration. However, the stimulation was chosen to be in a range where most MT and MST neurones give good responses (Perrone and Thiele 2001) and all neurones in our sample gave good and strong direction-selective responses to chromatic stimuli at higher luminance contrasts. It is still possible that responses at low luminance contrasts would not have dropped so dramatically, if the stimulus had been optimal for each cell. On the other hand, constant parameters of visual stimulation were chosen deliberately in order to provide a better picture of neuronal activity from a population perspective to allow for comparison to human behavioural results.

There are numerous indications that different systems may be involved in the perception of chromatic motion depending on the dynamic properties of the stimulus. It has been argued that chromatic motion at high temporal frequencies (above ~4 Hz) is processed similarly to luminance motion and is based on the activity of the magnocellular pathway (Hawken et al. 1994; Gegenfurtner and Hawken 1995, 1996; Burr et al. 1998; Seiffert and Cavanagh 1999; McKeefry 2001, 2002). At higher temporal frequencies, the magnocellular system may participate in detection of chromatic isoluminant targets (Lee et al. 1990). Experiments by Gegenfurtner et al. (1994) suggest that MT neurones are able to signal motion of chromatic gratings at perceptual threshold for stimuli moving at 8 Hz but not at 1 Hz. Our data support the hypothesis that MT is not involved in chromatic motion processing at low temporal frequencies (Gegenfurtner et al. 1994) in the anaesthetised monkey. We cannot exclude that the minimal responses of MT and MST neurones would have been higher provided we had stimulated with gratings moving at a higher temporal frequency.

Inter-individual differences We found substantial inter-individual variability in both macaque electrophysiological and human behavioural experiments. Remarkable differences between observers in a perceptual task employing chromatic motion have already been reported by Seiffert and Cavanagh (1999). This inter-individual heterogeneity indicates a possibility of difference in neuronal substrates. We note that isoluminance is a highly artificial condition hardly present in the natural environment. The visual system has not been confronted with isoluminant stimuli to a large degree during phylogenetic or ontogenetic development. This makes the isoluminant conditions potentially sensitive for revelation of inter-subject differences not seen in common perceptual tasks. The fact that largely different substrates can support identical behaviour has been repeatedly confirmed by lesion studies, supported by the finding that lesions of area MT yield only temporal deficits in motion processing of simple visual stimuli (Newsome et al. 1985; Dursteler and Wurtz 1988; Yamasaki and Wurtz 1991; Pasternak and Merigan 1994; Rudolph and Pasternak 1999).

Inter-species differences In fact, we cannot be sure that macaques would actually have been able to see the motion of all of the presented isoluminant gratings in the awake state because we did not perform behavioural tests on these animals. Although the similarity of human and macaque perception is traditionally stressed, contradictory findings especially regarding the perception and processing of chromatic motion have been reported. Thiele et al. (2001) reported a difference in chromatic motion processing between humans and macaque monkeys. In their experiments, performance of macaques was in agreement with the magnocellular model of origin of motion signal, whereas human performance indicated a colour-opponent parvocellular input. The magnocellular hypothesis of chromatic motion signal in macaques is further supported by results of Schiller et al. (1991) who found that isoluminant colour motion detection had not been affected by lesion of the P-LGN. Inter-species difference in chromatic signals in area MT is indicated also by fMRI experiments of Chawla et al. (1998, 1999). They reported higher activation of human MT/V5 complex by iso- or low-luminant chromatic stimuli than by high-luminant isochromatic stimuli. Given the correlation of the human BOLD signal with the spiking activity in area MT (Heeger et al. 1999; Seidemann et al. 1999; Rees et al. 2000; Ulbert et al. 2001) this finding would contradict the results of macaque single-unit literature and our results as well. Findings of Chawla and co-workers, however, were not supported by the fMRI study of Wandell et al. (1999). Moreover, iso- or low-luminant stimuli may fail to activate inhibitory mechanisms in area MT (Thiele et al. 2000), such that the population spike rate exceeds the spike rate to luminance stimuli, thereby yielding larger BOLD signals.

Indications for macaque–human inter-species difference in motion processing systems also come from the experiments that employed 'theta motion'—a kind of second-order motion stimulus. Such motion is visible to macaque monkeys but it does not activate MT and MST neurones. Thus, monkey's perception must rely on activity in different visual area(s) (Churan and Ilg 2001; Ilg and Churan 2004). In humans, on the other hand, scalp distribution of cortical activation evoked by theta

motion and first-order motion, respectively, does not differ (Patzwahl et al. 1994), which suggests a common neuronal substrate.

Alternative substrates for colour motion processing

Various areas apart from MT can be regarded as a potential substrate for colour motion processing. Gegenfurtner et al. (1997) proposed area V3, based on their finding of a remarkable proportion of cells tuned to both colour and direction of motion. Other candidates include areas of the temporal stream of visual information processing. Directional selectivity has been reported in areas V4 and TE (Ferrera et al. 1994; Vanduffel et al. 2001), which are involved in colour perception (Walsh et al. 1993; Schiller 1993; Heywood et al. 1995; Cowey et al. 2001). Neurones tuned to both colour and direction of motion were found also in areas V1 and V2 (Burkhalter and Van Essen 1986; Leventhal et al. 1995; Gegenfurtner et al. 1996; Tamura et al. 1996).

Conclusions

Our data suggest that area MT might not support the processing of motion cued by pure colour contrast. We found significant inter-animal differences in responsivity of MT neurones to chromatic motion and great inter-subject differences in perception of motion of chromatic gratings in humans. We found an exceptional human observer with no contribution of colour to motion and also an exceptional macaque monkey with MT neurones that were able to respond in direction-selective manner to all stimuli tested. It is tempting to speculate that the highly artificial condition of isoluminance is susceptible to reveal subtle inter-individual and inter-species differences in motion processing.

References

Agonie C, Gorea A (1993) Equivalent luminance contrast of red-green drifting stimuli: dependency on luminance-color interactions and on the psychophysical task. J Opt Soc Am A 10:1341–1352

Albright TD (1992) Form-cue invariant motion processing in primate visual cortex. Science 255:1141–1143

Albright TD, Stoner GR (1995) Visual motion perception. Proc Natl Acad Sci USA 92:2433–2440

Blaser E, Sperling G, Lu ZL (1999) Measuring the amplification of attention. Proc Natl Acad Sci USA 96:11681–11686

Born RT, Groh JM, Zhao R, Lukasewycz SJ (2000) Segregation of object and background motion in visual area MT: effects of microstimulation on eye movements. Neuron 26:725–734

Britten KH, Shadlen MN, Newsome WT, Movshon JA (1992) The analysis of visual motion: a comparison of neuronal and psychophysical performance. J Neurosci 12:4745–4765

Britten KH, Newsome WT, Shadlen MN, Celebrini S, Movshon JA (1996) A relationship between behavioral choice and the visual responses of neurons in macaque MT. Vis Neurosci 13:87–100

Brückner G, Seeger G, Brauer K, Härtig W, Katza J, Bigl V (1994) Cortical areas are revealed by distribution patterns of proteoglycan components and parvalbumin in the Mongolian gerbil and rat. Brain Res 658:67–86

Burkhalter A, Van Essen DC (1986) Processing of color, form and disparity information in visual areas VP and V2 of ventral extrastriate cortex in the macaque monkey. J Neurosci 6:2327–2351

Burr DC, Fiorentini A, Morrone C (1998) Reaction time to motion onset of luminance and chromatic gratings is determined by perceived speed. Vision Res 38:3681–3690

von Campenhausen M, Kirschfeld K (1999) Visual attention modifies spectral sensitivity of nystagmic eye movements. Vision Res 39:1551–1554

Cavanagh P (1992) Attention-based motion perception. Science 257:1563–1565

Cavanagh P, Anstis S (1991) The contribution of color to motion in normal and color-deficient observers. Vision Res 31:2109–2148

Cavanagh P, Tyler CW, Favreau OE (1984) Perceived velocity of moving chromatic gratings. J Opt Soc Am A 1:893–899

Cavanagh P, MacLeod DI, Anstis SM (1987) Equiluminance: spatial and temporal factors and the contribution of blue-sensitive cones. J Opt Soc Am A 4:1428–1438

Chatterjee S, Callaway EM (2002) S cone contributions to the magnocellular visual pathway in macaque monkey. Neuron 35:1135–1146

Chawla D, Phillips J, Buechel C, Edwards R, Friston KJ (1998) Speed-dependent motion-sensitive responses in V5: an fMRI study. Neuroimage 7:86–96

Chawla D, Buechel C, Edwards R, Howseman A, Josephs O, Ashburner J, Friston KJ (1999) Speed-dependent responses in V5: a replication study. Neuroimage 9:508–515

Cheng K, Hasegawa T, Saleem KS, Tanaka K (1994) Comparison of neuronal selectivity for stimulus speed, length, and contrast in the prestriate visual cortical areas V4 and MT of the macaque monkey. J Neurophysiol 71:2269–2280

Chichilnisky EJ, Heeger D, Wandell BA (1993) Functional segregation of color and motion perception examined in motion nulling. Vision Res 33:2113–2125

Churan J, Ilg UJ (2001) Processing of second-order motion stimuli in primate middle temporal area and medial superior temporal area. J Opt Soc Am A 18:2297–2306

Churchland M, Lisberger S (2001) Shifts in the population response in the middle temporal visual area parallel perceptual and motor illusions produced by apparent motion. J Neurosci 21:9387–9402

Cowey A, Marcar VL (1992) The effect of removing superior temporal cortical motion areas in the macaque monkey: I. Motion discrimination using simple dots. Eur J Neurosci 4:1219–1227

Cowey A, Heywood CA, Irving-Bell L (2001) The regional cortical basis of achromatopsia: a study on macaque monkeys and an achromatopsic patient. Eur J Neurosci 14:1555–1566

Crognale MA, Schor CM (1996) Contribution of chromatic mechanisms to the production of small-field optokinetic nystagmus (OKN) in normals and strabismics. Vision Res 36:1687–1698

Cusick CG, Seltzer B, Cola M, Griggs E (1995) Chemoarchitectonics and corticocortical terminations within the superior temporal sulcus of the rhesus monkey: evidence for subdivisions of superior temporal polysensory cortex. J Comp Neurol 360:513–535

Dacey DM (2000) Parallel pathways for spectral coding in primate retina. Annu Rev Neurosci 23:743–775

Derrington AM, Krauskopf J, Lennie P (1984) Chromatic mechanisms in lateral geniculate nucleus of macaque. J Physiol 357:241–265

Desimone R, Ungerleider LG (1989) Neural mechanisms of visual processing in monkeys. In: Boller F, Grafman J (eds) Handbook of neuropsychology, vol 2. Elsevier, New York, pp 267–299

Distler C, Boussaoud D, Desimone R, Ungerleider LG (1993) Cortical connections of inferior temporal area TEO in macaque monkeys. J Comp Neurol 334:125–150

Dobkins KR, Albright TD (1993) What happens if it changes color when it moves?: psychophysical experiments on the nature of chromatic input to motion detectors. Vision Res 33:1019–1036

Dobkins KR, Albright TD (1994) What happens if it changes color when it moves?: the nature of chromatic input to macaque visual area MT. J Neurosci 14:4854–4870

Dobkins KR, Albright TD (1995) Behavioral and neural effects of chromatic isoluminance in the primate visual motion system. Vis Neurosci 12:321–332

Dobkins KR, Gunther KL, Peterzell DH (2000a) What covariance mechanisms underlie green/red equiluminance, luminance contrast sensitivity and chromatic (green/red) contrast sensitivity? Vision Res 40:613–628

Dobkins KR, Thiele A, Albright TD (2000b) Comparison of red-green equiluminance points in humans and macaques: evidence for different L:M cone ratios between species. J Opt Soc Am A 17:545–556

Dougherty RF, Press WA, Wandell BA (1999) Perceived speed of colored stimuli. Neuron 24:893–899

Dursteler MR, Wurtz RH (1988) Pursuit and optokinetic deficits following chemical lesions of cortical areas MT and MST. J Neurophysiol 60:940–965

Dursteler MR, Wurtz RH, Newsome WT (1987) Directional pursuit deficits following lesions of the foveal representation within the superior temporal sulcus of the macaque monkey. J Neurophysiol 57:1262–1287

Elfar SD, Britten KH (1998) Chromatic contributions to motion processing in macaque extrastriate area MT. Soc Neurosci Abstr 24:1978

Farell B (1999) Color and luminance in the perception of 1- and 2-dimensional motion. Vision Res 39:2633–2647

Ferrera VP, Rudolph KK, Maunsell JH (1994) Responses of neurons in the parietal and temporal visual pathways during a motion task. J Neurosci 14:6171–6186

Ffytche DH, Skidmore BD, Zeki S (1995) Motion-from-hue activates area V5 of human visual cortex. Proc R Soc Lond B 260:353–358

Gallyas F (1979) Silver staining of myelin by means of physical development. Neurol Res 1:203–209

Gegenfurtner KR, Hawken MJ (1995) Temporal and chromatic properties of motion mechanisms. Vision Res 35:1547–1563

Gegenfurtner KR, Hawken MJ (1996) Perceived velocity of luminance, chromatic and non-fourier stimuli: influence of contrast and temporal frequency. Vision Res 36:1281–1290

Gegenfurtner KR, Kiper DC, Beusmans JM, Carandini M, Zaidi Q, Movshon JA (1994) Chromatic properties of neurons in macaque MT. Vis Neurosci 11:455–466

Gegenfurtner KR, Kiper DC, Fenstemaker SB (1996) Processing of color, form, and motion in macaque area V2. Vis Neurosci 13:161–172

Gegenfurtner KR, Kiper DC, Levitt JB (1997) Functional properties of neurons in macaque area V3. J Neurophysiol 77:1906–1923

Gellman RS, Carl JR, Miles FA (1990) Short latency ocular-following responses in man. Vis Neurosci 5:107–122

Groh JM, Born RT, Newsome WT (1997) How is a sensory map read out? Effects of microstimulation in visual area MT on saccades and smooth pursuit eye movements. J Neurosci 17:4312–4330

Guo K, Benson PJ (1999) Grating and plaid chrominance motion influences the suppressed ocular following response. Neuroreport 10:387–392

Hadjikhani N, Tootell RB (2000) Projection of rods and cones within human visual cortex. Hum Brain Mapp 9:55–63

Havránek T (1993) Statistika pro biologické a lékařské vědy. Academia, Praha

Hawken MJ, Gegenfurtner KR, Tang C (1994) Contrast dependence of colour and luminance motion mechanisms in human vision. Nature 367:268–270

Heeger DJ, Boynton GM, Demb JB, Seidemann E, Newsome WT (1999) Motion opponency in visual cortex. J Neurosci 19:7162–7174

Hendry SH, Reid RC (2000) The koniocellular pathway in primate vision. Annu Rev Neurosci 23:127–153

Hess DT, Merker BH (1983) Technical modifications of Gallyas' silver stain for myelin. J Neurosi Methods 8:95–97

Heywood CA, Gaffan D, Cowey A (1995) Cerebral achromatopsia in monkeys. Eur J Neurosci 7:1064–1073

Hof PR, Morrison JH (1995) Neurofilament protein defines regional patterns of cortical organisation in the macaque monkey visual system: a quantitative imunohistochemical analysis. J Comp Neurol 352:161–186

Holm S (1979) A simple sequentially rejective multiple test procedure. Scand J Stat 6:65–70

Ilg U (1997) Responses of primate area MT during the execution of optokinetic nystagmus and afternystagmus. Exp Brain Res 113:361–361

Ilg U, Churan J (2004) Motion perception without explicit activity in areas MT and MST. J Neurophysiol 92:1512–1523

Johnson EN, Hawken MJ, Shapley R (2001) The spatial transformation of color in the primary visual cortex of the macaque monkey. Nat Neurosci 4:409–416

Kaas JH (1997) Theories of visual cortex organisation in primates. In: Rockland KS, Kaas JH, Peters A (eds) Cerebral cortex, vol 12. Plenum, New York, pp 91–125

Kaiser PK, Lee BB, Martin PR, Valberg A (1990) The physiological basis of the minimally distinct border demonstrated in the ganglion cells of the macaque retina. J Physiol 422:153–183

Kawano K (1999) Ocular tracking: behavior and neurophysiology. Curr Opin Neurobiol 9:467–473

Komatsu H, Wurtz RH (1989) Modulation of pursuit eye movements by stimulation of cortical areas MT and MST. J Neurophysiol 62:31–47

Lee BB, Pokorny J, Smith VC, Martin PR, Valberg A (1990) Luminance and chromatic modulation sensitivity of macaque ganglion cells and human observers. J Opt Soc Am A 7:2223–2236

Lee BB, Smith VC, Pokorny J, Kremers J (1997) Rod inputs to macaque ganglion cells. Vision Res 37:2813–2828

Leventhal AG, Thompson KG, Liu D, Zhou Y, Ault SJ (1995) Concomitant sensitivity to orientation, direction, and color of cells in layers 2, 3, and 4 of monkey striate cortex. J Neurosci 15:1808–1818

Lisberger S, Movshon J (1999) Visual motion analysis for pursuit eye movements in area MT of macaque monkeys. J Neurosci 19:2224–2246

Lu ZL, Lesmes LA, Sperling G (1999) The mechanism of isoluminant chromatic motion perception. Proc Natl Acad Sci USA 96:8289–8294

Marcar VL, Cowey A (1992) The effect of removing superior temporal cortical motion areas in the macaque monkey: II. Motion discrimination using random dot displays. Eur J Neurosci 4:1228–1238

Maunsell JH, Van Essen DC (1983) Functional properties of neurons in middle temporal visual area of the macaque monkey. I. Selectivity for stimulus direction, speed, and orientation. J Neurophysiol 49:1127–1147

Maunsell JH, Nealey TA, DePriest DD (1990) Magnocellular and parvocellular contributions to responses in the middle temporal visual area (MT) of the macaque monkey. J Neurosci 10:3323–3334

McKeefry DJ (2001) Visual evoked potentials elicited by chromatic motion onset. Vision Res 41:2005–2025

McKeefry DJ (2002) The influence of stimulus chromaticity on the isoluminant motion-onset VEP. Vision Res 42:909–922

Miles FA, Kawano K, Optican LM (1986) Short-latency ocular following responses of monkey. I. Dependence on temporospatial properties of visual input. J Neurophysiol 56:1321–1354

Mishkin M, Ungerleider LG, Macko KA (1983) Object vision and spatial vision: two cortical pathways. Trends Neurosci 6:414–417

Newsome WT, Pare EB (1988) A selective impairment of motion perception following lesions of the middle temporal visual area (MT). J Neurosci 8:2201–2211

Newsome WT, Wurtz RH, Dursteler MR, Mikami A (1985) Deficits in visual motion processing following ibotenic acid lesions of the middle temporal visual area of the macaque monkey. J Neurosci 5:825–840

Newsome W, Wurtz R, Komatsu H (1988) Relation of cortical areas MT and MST to pursuit eye movements. II. Differentiation of retinal from extraretinal inputs. J Neurophysiol 60:604–620

Newsome WT, Britten KH, Movshon JA (1989) Neuronal correlates of a perceptual decision. Nature 341:52–54

van Norren D, Padmos P (1975) Cone dark adaptation: the influence of halothane anesthesia. Invest Ophthalmol Vis Sci 14:212–227

van Norren D, Padmos P (1977) Influence of anesthetics, ethyl alcohol, and freon on dark adaptation of monkey cone ERG. Invest Ophthalmol Vis Sci 16:80–83

O'Keefe LP, Movshon JA (1998) Processing of first- and second-order motion signals by neurons in area MT of the macaque monkey. Vis Neurosci 15:305–317

Orban GA (1997) Visual processing in macaque area MT/V5 and its satellites (MSTd and MSTv). In: Rockland KS, Kaas JH, Peters A (eds) Cerebral cortex, vol 12. Plenum, New York, pp 359–434

Orban GA, Saunders RC, Vandenbussche E (1995) Lesions of the superior temporal cortical motion areas impair speed discrimination in the macaque monkey. Eur J Neurosci 7:2261–2276

Pack CC, Berezovskii VK, Born RT (2001) Dynamic properties of neurons in cortical area MT in alert and anesthetized macaque monkeys. Nature 414:905–908

Pasternak T, Merigan WH (1994) Motion perception following lesions of the superior temporal sulcus in the monkey. Cereb Cortex 4:247–259

Patzwahl DR, Zanker JM, Altenmuller EO (1994) Cortical potentials reflecting motion processing in humans. Vis Neurosci 11:1135–1147

Perrone JA, Thiele A (2001) Speed skills: measuring the visual speed analyzing properties of primate MT neurons. Nat Neurosci 4:526–532

Purpura K, Kaplan E, Shapley RM (1988) Background light and the contrast gain of primate P and M retinal ganglion cells. Proc Natl Acad Sci USA 85:4534–4537

Rees G, Friston K, Koch C (2000) A direct quantitative relationship between the functional properties of human and macaque V5. Nat Neurosci 3:716–723

Robson T (1999) Topics in computerized visual-stimulus generation. In: Carpenter R, Robson J (eds) Vision research: a practical guide to laboratory methods. Oxford University Press, New York, pp 81–105

Rudolph K, Pasternak T (1999) Transient and permanent deficits in motion perception after lesions of cortical areas MT and MST in the macaque monkey. Cereb Cortex 9:90–100

Saito H, Tanaka K, Isono H, Yasuda M, Mikami A (1989) Directionally selective response of cells in the middle temporal area (MT) of the macaque monkey to the movement of equiluminous opponent color stimuli. Exp Brain Res 75:1–14

Salzman CD, Britten KH, Newsome WT (1990) Cortical microstimulation influences perceptual judgements of motion direction. Nature 346:174–177

Salzman CD, Murasugi CM, Britten KH, Newsome WT (1992) Microstimulation in visual area MT: effects on direction discrimination performance. J Neurosci 12:2331–2355

Schiller PH (1993) The effects of V4 and middle temporal (MT) area lesions on visual performance in the rhesus monkey. Vis Neurosci 10:717–746

Schiller PH, Logothetis NK, Charles ER (1991) Parallel pathways in the visual system: their role in perception at isoluminance. Neuropsychologia 29:433–441

Sclar G, Maunsell JH, Lennie P (1990) Coding of image contrast in central visual pathways of the macaque monkey. Vision Res 30:1–10

Seidemann E, Poirson AB, Wandell BA, Newsome WT (1999) Color signals in area MT of the macaque monkey. Neuron 24:911–917

Seiffert AE, Cavanagh P (1999) Position-based motion perception for color and texture stimuli: effects of contrast and speed. Vision Res 39:4172–4185

Shadlen MN, Britten KH, Newsome WT, Movshon JA (1996) A computational analysis of the relationship between neuronal and behavioral responses to visual motion. J Neurosci 16:1486–1510

Sincich LC, Park KP, Wohlgemuth MJ, Horton JC (2004) Bypassing V1: a direct geniculate input to area MT. Nat Neurosci 7:1123–1128

Smith AT, Hammond P (1986) Hemifield differences in perceived velocity. Perception 15:111–117

Sperling G, Lu Z-L (1998) A system analysis of visual motion perception. In: Watanabe T (eds) High-level motion processing: computational, neurobiological, and psychophysical perspectives. MIT Press, Cambridge, pp 153–183

Stockman A, Sharpe LT (2000) The spectral sensitivities of the middle- and long-wavelength-sensitive cones derived from measurements in observers of known genotype. Vision Res 40:1711–1737

Sun H, Smithson H, Lee B, Zaidi Q (2004) A new technique for measuring cone inputs to visual neurons. Invest Ophthalmol Vis Sci 45, E-Abstract 4277

Tamura H, Sato H, Katsuyama N, Hata Y, Tsumoto T (1996) Less segregated processing of visual information in V2 than in V1 of the monkey visual cortex. Eur J Neurosci 8:300–309

Teller DY, Lindsey DT (1993) Motion at isoluminance: motion dead zones in three-dimensional color space. J Opt Soc Am A 10:1324–1331

Thiele A, Distler C, Hoffmann KP (1999a) Decision-related activity in the macaque dorsal visual pathway. Eur J Neurosci 11:2044–2058

Thiele A, Dobkins KR, Albright TD (1999b) The contribution of color to motion processing in macaque middle temporal area. J Neurosci 19:6571–6587

Thiele A, Dobkins KR, Albright TD (2000) Neural correlates of contrast detection at threshold. Neuron 26:715–724

Thiele A, Dobkins KR, Albright TD (2001) Neural correlates of chromatic motion perception. Neuron 32:351–358

Thiele A, Rezec A, Dobkins KR (2002) Chromatic input to motion processing in the absence of attention. Vision Res 42:1395–1401

Tootell RB, Reppas JB, Kwong KK, Malach R, Born RT, Brady TJ, Rosen BR, Belliveau JW (1995) Functional analysis of human MT and related visual cortical areas using magnetic resonance imaging. J Neurosci 15:3215–3230

Tychsen L, Lisberger S (1986) Visual motion processing for the initiation of smooth-pursuit eye movements in humans. J Neurophysiol 56:953–968

Ulbert I, Karmos G, Heit G, Halgren E (2001) Early discrimination of coherent versus incoherent motion by multiunit and synaptic activity in human putative MT+. Hum Brain Mapp 13:226–238

Valberg A, Lee BB, Kaiser PK, Kremers J (1992) Responses of macaque ganglion cells to movement of chromatic borders. J Physiol 458:579–602

Van Essen DC, Maunsell JHR (1980) Two-dimensional maps of the cerebral cortex. J Comp Neurol 191:255–281

Vanduffel W, Fize D, Mandeville JB, Nelissen K, Hecke PV, Rosen BR, Tootell RB, Orban GA (2001) Visual motion processing investigated using contrast agent-enhanced fMRI in awake behaving monkeys. Neuron 32:565–577

Walsh V, Carden D, Butler SR, Kulikowski JJ (1993) The effects of V4 lesions on the visual abilities of macaques: hue discrimination and colour constancy. Behav Brain Res 53:51–62

Wandell BA (1995) Foundations of vision. Sinauer Press, Sunderland

Wandell BA, Poirson AB, Newsome WT, Baseler HA, Boynton GM, Huk A, Gandhi S, Sharpe LT (1999) Color signals in human motion-selective cortex. Neuron 24:901–909

Wyszecki G, Stiles WS (1982) Color science. Wiley, New York

Yamasaki DS, Wurtz RH (1991) Recovery of function after lesions in the superior temporal sulcus in the monkey. J Neurophysiol 66:651–673

Zeki S (1974) Functional organization of a visual area in the posterior bank of the superior temporal sulcus of the rhesus monkey. J Physiol 236:549–573

Zeki S (1983) The distribution of wavelength and orientation selective cells in different areas of monkey visual cortex. Proc R Soc Lond B 217:449–470

Zeki S (1993) A vision of the brain. Blackwell, London