Electrophysiological evidence for independent speed channels in human motion processing

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A variety of psychophysical studies suggests that motion perception in humans is mediated by at least two speed-tuned channels. To study the neurophysiological underpinnings of these channels in the human visual cortex, we recorded visual evoked potentials (VEPs) to motion onset. We applied an adaptation paradigm that allowed us (a) to isolate and extract direction-specific cortical responses and (b) to assess cross-adaptation in the speed domain. VEPs resulting from the onset of left- or rightward motion at either low or high speeds were recorded from three occipital recording sites in 11 subjects. For each of these test stimuli, responses were collected after adaptation to one of five different conditions: a static adaptation pattern (baseline), adaptation to low-speed motion (3.5°/s) either in the same or in the opposite direction as the test, or adaptation to high-speed motion (32°/s) either in the same or in the opposite direction as the test. We report considerable direction-specific adaptation for same adaptation and test speeds (by 28–37% of baseline response; $p < .002$), whereas there was no direction-specific adaptation across speeds. We supplement these electrophysiological data with corresponding psychophysical results. The lack of direction-specific cross-adaptation in the speed domain demonstrated with physiological and psychophysical techniques supports models of at least two speed-tuned channels in the human motion system

Keywords: motion, speed, human, visual cortex, direction specificity, adaptation, VEP, MAE

Introduction

Do independent temporal channels feed into the motion system? This is a crucial question to understand speed coding in the motion system. Psychophysical investigations of human visual motion perception point at two or more broadly tuned channels (Anderson & Burr, 1985; Smith & Edgar, 1994; Thompson, 1984); the underlying neurophysiological mechanisms, however, are yet unknown.

A key tool in the psychophysical analysis of independent motion mechanisms is the motion after-effect (MAE; reviewed in Mather, Verstraten, & Anstis, 1998; Wade, 1994). After adaptation to a moving pattern, a stationary pattern appears to move in the opposite direction (static MAE). This aftereffect is also evident when dynamic random noise instead of a stationary pattern is viewed (dynamic MAE) (Hiris & Blake, 1992). Remarkably, static and dynamic test patterns seem to tap different speed channels – the static MAE is more likely to arise after adaptation to slow motion, whereas the dynamic MAE is more likely to arise after adaptation to fast motion (Verstraten, van der Smagt, Fredericksen, & van de Grind, 1999; Verstraten, van der Smagt, & van de Grind, 1998). Simultaneous adaptation to one fast and one slow speed even results in a *transparent* MAE when tested with a combination of a dynamic and static test pattern (van der Smagt, Verstraten, & van de Grind, 1999). This psychophysical finding suggests that there are at least two temporal channels in the human motion system, which can adapt independently. The physiological basis of this independent adaptation, however, is unknown (Müller, Göpfert, Breuer, & Greenlee, 1999).

There is ample evidence that a component in the motion-onset visual evoked potential (VEP), called N2, which is a negative deflection with a latency of around 150-200 ms, reflects cortical motion processing in humans (Bach & Ullrich, 1994; Hoffmann, Dorn, & Bach, 1999; Kubova, Kuba, Spekreijse, & Blakemore, 1995; Niedeggen & Wist, 1998 [review]; Probst, Plendl, Paulus, Wist, & Scherg, 1993). The key evidence is provided by adaptation studies, which helped to uncover two mechanisms that contribute to N2: one that adapts in a direction-specific manner and another that adapts independent of motion direction (Bach & Hoffmann, 2000; Heinrich & Bach, 2003; Hoffmann,

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Unsöld, & Bach, 2001). The part of N2 that adapts in a direction-specific manner is closely associated with mechanisms that underlie motion detection, as these are defined to be direction-specific. The isolation of the direction-specific part of N2 is therefore a powerful approach to investigate the properties of the neural substrate of motion perception in humans.

To test the prediction from psychophysical studies that adaptation with slow motion leaves fast motion mechanisms unaffected and vice versa, we investigated the direction-specific adaptation of N2 in a speed cross-adaptation paradigm. The hypothesis is that N2 to fast and slow test stimuli resemble static and dynamic MAEs in their property to selectively tap the adaptation of low- and high-speed motion mechanisms, respectively. Adaptation of low-speed mechanisms should therefore selectively affect motion-specific VEP responses to slow test stimuli and leave motion-specific responses to fast test stimuli unaffected, and vice versa for adaptation of high-speed mechanisms. We did indeed observe this independent adaptation to two extreme stimulus speeds, which supports the notion that the human motion system comprises at least two independent speed-tuned channels.

Methods

Subjects

VEPs were recorded binocularly from 14 human observers with normal or corrected-to-normal visual acuity (>1.0) A subset of 9 of these observers participated in the psychophysical part of this study. The subjects gave their informed written consent to participate in the experiment. The procedures followed the tenets of the Declaration of Helsinki, and the protocol was approved by the ethics committee of the University of Freiburg, Germany.

Stimuli

Stimuli were generated by a Power Macintosh G4 with a program based on the Apple Game sprockets (Bach, 1999) and presented binocularly on a CRT with a frame rate of 90 Hz at a distance of 57 cm. The stimulus pattern consisted of a random pixel array (pixel size = 0.04°) moving within a circular mask of 24° diameter. Pixels were either light or dark with equal probability. The space-averaged mean luminance of the pattern was 18.5 cd/m^2. The contrast was set at 73%. A relatively large fixation target of 3° diameter to reduce optokinetic nystagmus was centered on the pattern.

The stimulus patterns could move either left- or rightwards at two speeds, namely at 3.5 or 32°/s. These stimulus speeds were chosen on the basis of the speed-dependence of static and dynamic MAEs (Verstraten et al., 1998). Whereas motion adaptation at 32°/s hardly elicits any static MAE, the dynamic MAE is still reasonably strong (van de Grind, van Hof, van der Smagt, & Verstraten, 2001; Verstraten et al., 1998). The reverse holds for adaptation at 3.5°/s. This relatively low speed elicits a near optimal static MAE, whereas the dynamic MAE for this speed is suboptimal.

Stimulus trials were presented in a cyclic design. A stimulus trial of a total duration of 3000 ms comprised three epochs: 2200 ms adaptation; 500 ms stationary pattern; and 300 ms of motion either right- or leftwards and either slow or fast, selected randomly. During this 300-ms test epoch, the motion-onset potentials were recorded. For baseline measurements, the pattern remained stationary during the 2200-ms adaptation epoch. For the adaptation measurements, the pattern moved either right- or leftwards at either 3.5 or 32°/s. Within one block, the same stimulus speed and direction were used for adaptation in all trials. The cyclic design resulted in a stable adaptation state after the first few trials. For each combination of test direction and test speed, three adaptation conditions can be distinguished: stationary (baseline), same speed (uncrossed adaptation), and different speed (crossed adaptation).

Procedure

Motion-VEPs were recorded in six blocks presented in a counter-balanced blocked design (adaptation fast leftwards, adaptation slow rightwards, baseline, baseline, adaptation slow leftwards, and adaptation fast rightwards). Adaptation blocks were followed by a 2-min recovery break during which the subjects were allowed to look around freely. Each block contained at least 230 artifact-free trials, except the baseline block, which contained 115 trials, as left- and rightwards test motions are equivalent (Hoffmann et al., 2001). Responses for the same adaptation and test-speed combinations were averaged in groups of same and opposite adaptation and test-stimulus directions.

Electrophysiological recordings

Potentials were recorded from three scalp electrodes referenced to linked ears: O_z (occipital pole) according to standard nomenclature (American Encephalographic Society, 1994) and O_{tl} and O_{tr} (occipito-temporal left and right, at 5 cm left and right from O_z). The ground electrode was attached to the right wrist. Signals were amplified, filtered (first-order bandpass, 0.3–70 Hz, Toennies Physiologic Amplifier), and digitized to a resolution of 12 bits at a sampling frequency of 500 Hz with a Macintosh 7200 computer. Using LabView (National Instruments), signals were streamed to disk and also averaged online (across all stimuli) to monitor the recording.

Data analysis

Trials were analyzed off-line with Igor Pro (Wavemetrics, Inc.) for an interval that began 100 ms prior to motion onset and ended 500 ms after motion onset. Trials with blinks, detected with a threshold criterion of 100 μV, were discarded. Sweeps were pooled according to stimulus con-

ditions and digitally filtered (0–40 Hz) before being averaged. The zero level was defined as the mean value of the averaged trace from 100 ms before to 50 ms after stimulus onset and used as reference for peak measurements.

To assess adaptation that is not specific for motion direction, VEPs with *opposite* adaptation and test directions were compared to baseline. VEPs with *same* adaptation and test directions reflect this global adaptation effect in addition to the direction-specific adaptation effect. Consequently, VEPs with same adaptation and test directions were compared to the non-directional effect to assess direction-specific adaptation, which is indicative of motion-specific processing (Bach & Hoffmann, 2000; Hoffmann et al., 2001).

Motion-onset potentials are often strongly lateralized to the Ot_r or Ot_l derivation (Andreassi & Juszczak, 1982). To maximize the signal-to-noise ratio for N2 amplitudes, we selected for each subject the Ot derivation with the greater N2 amplitude (based on the mean of normalized N2 peaks of the responses to the baseline stimuli tested) and labeled it Ot*.

The adaptation experiments of this study pursued the investigation of a third-order effect. Therefore, the signal-to-noise ratio is a vital issue and only subjects with a mean N2 baseline amplitude that exceeded 4 μV at both Oz and OT* were included in the analysis. This left 11 subjects out of the 14 subjects who originally entered the study. Nine of these 11 subjects were naïve to the specific aim of the study and were not experienced in the assessment of static and dynamic MAEs.

Statistical analysis

Data were normalized with respect to the baseline response. The statistical significance of experimental effects was assessed with paired Student *t* tests and a subsequent sequential Bonferroni correction (Holm, 1979). Significance levels are indicated in the figures (*$p < .05$, ** $p < .01$, and *** $p < .001$).

Psychophysics

To determine whether slow and fast stimulus speeds elicit preferentially static or dynamic MAEs, respectively, we measured MAE durations after adaptation to motion stimuli with spatial features identical to those stimuli used in the electrophysiological experiments. We presented unidirectional motion at either 3.5°/s or 32°/s for a duration of 30 s of adaptation. This duration is based on pilot experiments, which showed that shorter adaptation epochs (i.e., with a duration of only 15 s) are not sufficient to obtain a reliable measure for the duration of the dynamic MAE. Subjects were prompted by a beep 5 s before motion adaptation to take up fixation. Further beeps indicated start and end of the adaptation epoch. After the adaptation epoch, a test pattern that was either static or dynamic (90-Hz refresh rate) was presented until the subject pressed either of two keys, one to indicate that the perception of the MAE had ceased, the other to indicate the absence of any MAE. The MAE duration measurement of the latter was set to zero seconds, which is necessary as the absence of an MAE (i.e., an MAE duration of zero seconds) can be judged only after a delay. To minimize build-up and crosstalk of adaptation, the next trial was delayed for another 45 s, during which a static pattern was presented. The subjects were instructed not to close their eyes during this epoch, and they were allowed to move their eyes and look around in the room. We obtained MAE-duration measures in two sessions, each lasting about 1.5 hr. To determine MAE durations for the different stimulus conditions, all four possible combinations of stimulus speed and test pattern were presented in a random sequence of 12 trials in a single block. In each session, three of these blocks were presented and the subjects were allowed to take a brief rest between blocks. The first of these three blocks was a practice block, and the results of this block were therefore discarded. The results of the remaining two blocks of each session yielded 12 MAE duration measurements for each condition. At the beginning of the first session, additional practice trials were inserted, allowing the subjects to collect experience in the judgement of MAE durations: During the course of testing, naïve subjects tend to change the criterion by which the end of the dynamic MAE is judged. After a short demonstration of static and dynamic MAEs, the subjects ran a block of 12 trials with motion adaptation at 32°/s and a dynamic test pattern to give them a chance to stabilize this criterion. We performed this psychophysical procedure in 9 of the 11 subjects that contributed to the electrophysiological results.

Results

The effects of motion adaptation on the motion-onset VEP are shown in Figure 1, where the grand mean VEP traces are depicted. Motion adaptation reduces N2 amplitude at both derivations (Oz and OT*) and for all stimulus conditions. The comparison of N2 amplitudes after same and opposite adaptation and test directions allows one to assess the degree of direction-specific adaptation. Such a comparison of the VEP traces already indicates that the direction-specific adaptation is strongest for same adaptation and test speeds (i.e., for the uncrossed adaptation conditions). Interestingly, not only N2 is differentially affected by direction-specific adaptation. An earlier positivity, P1, is also affected by motion adaptation. However, although P1 exhibits a dependence on adaptation direction, P1 itself does not appear to be a motion-specific component: In contrast to N2, P1 increases with increasing depth of motion adaptation. This has been demonstrated previously (Bach & Ullrich, 1994; Hoffmann et al., 1999) and is presumably associated with the fact that the motion-onset VEP is the summed potential of different processes leading to P1 and N2 (Bach & Ullrich, 1997; Kubova et al., 1995). Assuming that these processes overlap in time, they will reduce each other as they superimpose in the VEP. Conse-

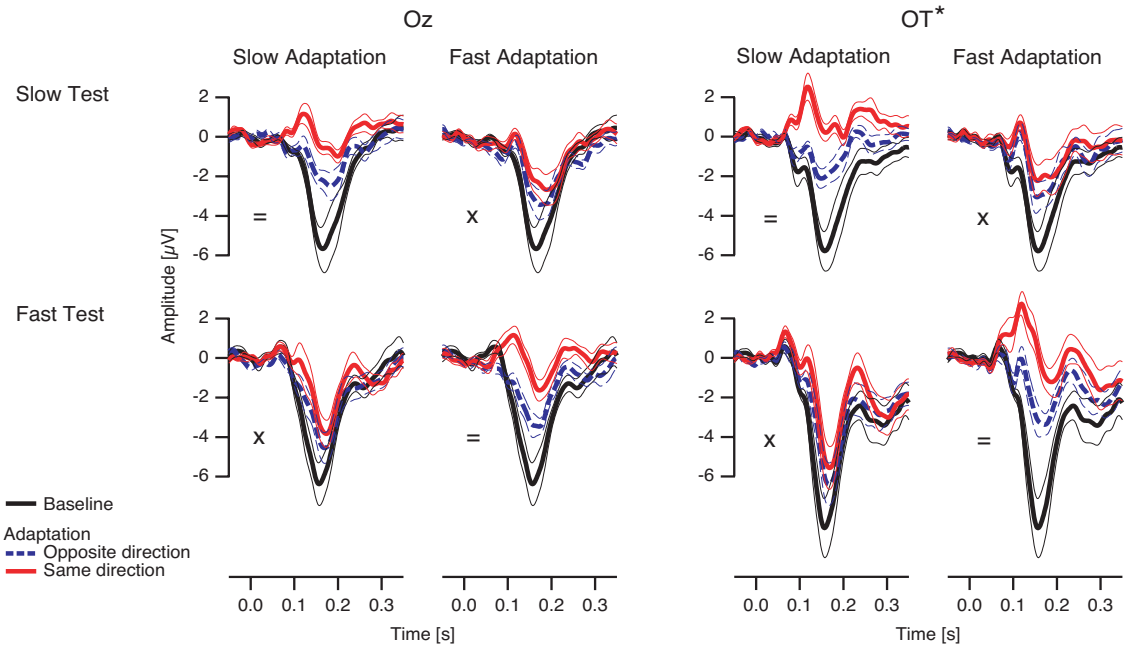

Figure 1. VEP traces (grand mean ± *SEM*, thick and thin traces, respectively; $n = 11$) in eight panels. Each of the eight panels depicts a triplet of VEP traces: response after adaptation to a stationary grating (baseline, black trace), after adaptation to motion in the opposite direction of the test stimulus (opposite adaptation direction, blue dashed trace), and after adaptation to motion in the same direction as the test stimulus (same adaptation direction, red trace). From these three traces, the direction specificity of the response in the cross-adaptation paradigm can be assessed. The eight panels are arranged as two quadruplets, one for each electrode (Oz and OT*) to depict speed specificity as assessed in a cross-adaptation paradigm of two speeds, slow (3.5°/s) and fast (32°/s), a total of four speed combinations. Uncrossed and crossed speed adaptation and tests are indicated by "=" and "x," respectively. Note that the baseline response for same test speeds is depicted twice in each row for better comparability. The main component of the motion VEP is a negative deflection at around 155 ms after stimulus onset, called N2. N2 amplitudes are reduced after adaptation to motion; direction-specific adaptation is only evident for the uncrossed adaptation conditions (i.e., in the top left and bottom right panel in each quadruplet). The reduction of N2 also uncovers a positive defection (P1) around 120 ms (see text for details).

quently, the reduction of N2 due to motion adaptation will result in an uncovering of P1, which appears at first sight as a paradoxical increase of its amplitude.

The direction-specific adaptation of N2 was analyzed more quantitatively for Figure 2, which depicts the mean N2 amplitudes for the various stimulus conditions and derivations. For this analysis, N2 amplitudes were normalized with respect to each subject's individual baseline, to reduce the effect of the inter-individual variability of N2 amplitudes. We observed non-direction–specific adaptation for all stimulus conditions at both derivations Oz and OT* (significant at alpha = 0.01, sequential Bonferroni adjustment; Holm, 1979). In contrast, direction-specific adaptation (i.e., the difference of opposite- and same-direction responses) is significant only for the uncrossed adaptation conditions (uncrossed direction-specific adaptation, mean ± *SEM* [% of baseline]: OT* slow, 29 ± 5 [$p = .0009$]; OT* fast, 37 ± 5 [$p = .0001$]; Oz slow, 32 ± 5 [$p = .0004$]; and Oz fast, 28 ± 4 [$p = .0016$]). This suggests a lack of cross-adaptation of motion-specific mechanisms in the speed domain.

Psychophysical results are depicted as individual MAE durations in Figure 3. MAE duration with a static test pattern is greatest after low-speed adaptation [$p = .008$, Wilcoxon signed rank test; medians slow vs. fast: 16.3 vs. 0 s], whereas MAE duration with a dynamic test pattern is greatest after high-speed adaptation [$p = .038$, Wilcoxon signed rank test (sequentially Bonferroni adjusted for multiple testing, Holm, 1979); medians slow vs. fast: 3.7 vs. 14.3 s].

Discussion

It is clear from our electrophysiological data that direction-specific low-speed adaptation is not reflected in the motion-onset VEP to high-speed motion and vice versa. Our data therefore demonstrate, for the first time, a neurophysiological correlate of the independent adaptation of mechanisms tuned to slow and fast motion in humans. Such independent adaptation mechanisms have previously been reported in psychophysical studies, which indicate that static and dynamic MAEs are evoked preferentially by slow and fast stimuli, respectively (van de Grind et al., 2001; van der Smagt et al., 1999; Verstraten et al., 1999; Verstraten et al., 1998). Indeed, we were able to replicate these psychophysical findings in the subset of subjects that participated in the electrophysiological part of this study.

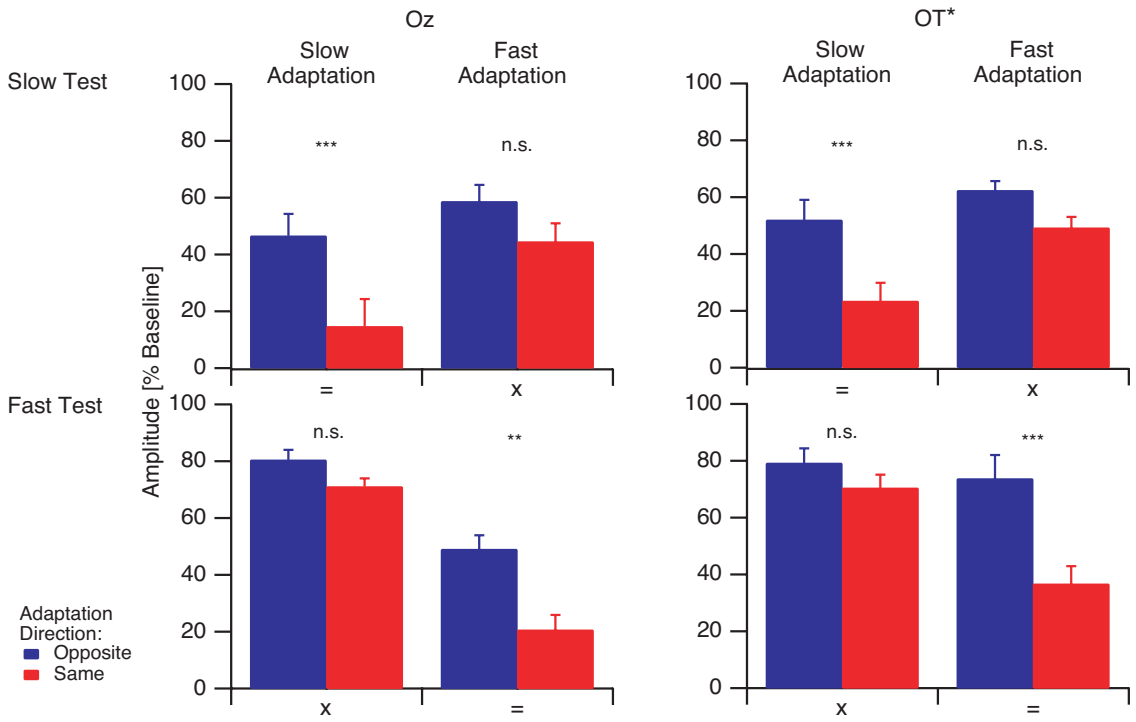

Figure 2. Normalized N2 amplitudes after motion adaptation in same and opposite direction of the test direction for Oz and OT* (mean of 11 subjects ±*SEM*; amplitudes are normalized to each subject's individual baseline amplitude). Quadruplets are arranged in accordance to Figure 1. It should be noted that small amplitudes indicate strong adaptation, whereas large amplitudes indicate weak adaptation. N2 amplitudes for all stimulus conditions are reduced after adaptation (i.e., they are smaller than 100% baseline). Direction-specific adaptation is only evident for same adaptation and test speeds (top left and bottom right panel in each quadruplet) (i.e., no direction-specific adaptation across speeds is evident).

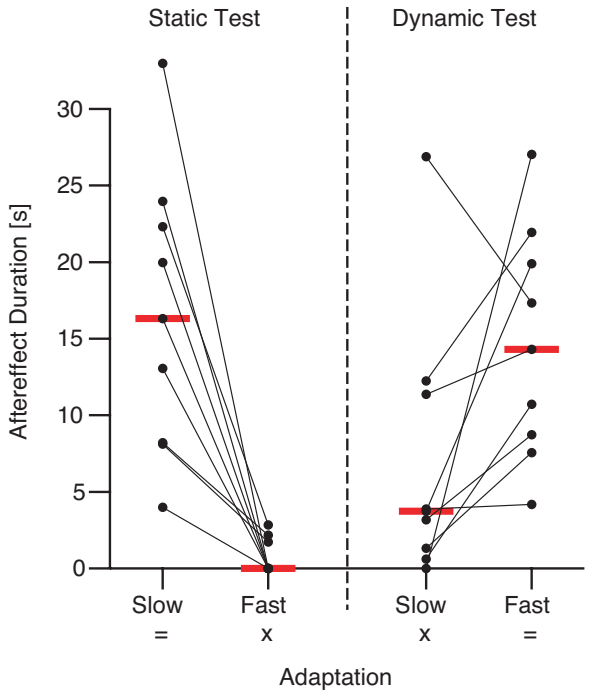

Figure 3. Individual MAE durations of nine subjects as measured with dynamic and static test patterns after adaptation with slow (3.5°/s) and fast (32°/s) stimulus speeds. Red bars indicate median values.

Therefore, our electrophysiological and psychophysical results concur with a model of two (or more) speed-tuned channels in human motion processing. Although we cannot infer the actual number of channels from the two speeds tested, previous psychophysical work (confirmed by our psychophysical experiment) suggests that we do not simply tap two ends of a continuous speed-sensitivity domain. Interestingly, we observed that adaptation that is *not specific* for stimulus direction is independent of stimulus speed. As a consequence, the independent adaptation of temporal channels appears to be specific to motion mechanisms and is not reflected by general phasic mechanisms that contribute to the adaptation effect not specific for direction.

In the psychophysical part of the study, static and dynamic MAEs are preferentially elicited by slow and fast adaptation speeds, respectively. While we observed a great uniformity of this selective adaptation across subjects for the static test pattern, we observed less uniformity for the dynamic test pattern. This is in accordance with the inter-individual variability of the speed-tuning curves obtained with dynamic test patterns in previous studies (van de Grind et al., 2001). On a subject-by-subject basis, the variability of the psychophysical data does not correspond to that of the electrophysiological data. In conclusion, we demonstrate for the first time a neurophysiological correlate in humans of two independent motion systems, one

tuned to lower and one to higher speeds. This result is in concurrence with previously reported temporal channels (Anderson & Burr, 1985; Smith & Edgar, 1994; Thompson, 1984) and predicted by psychophysical studies that have shown the independent adaptation of processing mechanisms for slow and faster motion (van de Grind et al., 2001; van der Smagt et al., 1999; Verstraten et al., 1999; Verstraten et al., 1998). Electrophysiological studies in monkeys (Gegenfurtner, Kiper, & Levitt, 1997; Lagae, Raiguel, & Orban, 1993) have also indicated the presence of at least two broadly tuned motion channels. Our neurophysiological findings of at least two independent speed-tuned motion channels in humans thus bridge the gap between monkey physiology and human psychophysics.

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