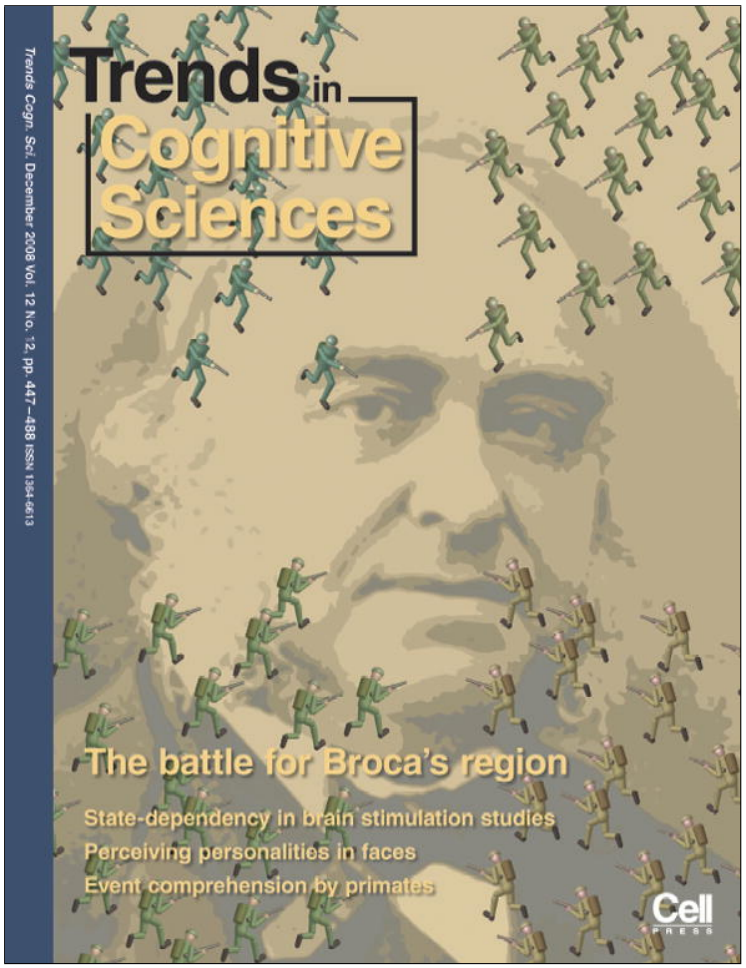

Review

The motion aftereffect reloaded

George Mather[1], Andrea Pavan[2], Gianluca Campana[2] and Clara Casco[2]

[1] Department of Psychology, University of Sussex, Falmer, Brighton, BN1 9QH, UK
[2] Department of General Psychology, University of Padova, Via Venezia 8, 35131 Padova, Italy

The motion aftereffect is a robust illusion of visual motion resulting from exposure to a moving pattern. There is a widely accepted explanation of it in terms of changes in the response of cortical direction-selective neurons. Research has distinguished several variants of the effect. Converging recent evidence from different experimental techniques (psychophysics, single-unit recording, brain imaging, transcranial magnetic stimulation, visual evoked potentials and magnetoencephalography) reveals that adaptation is not confined to one or even two cortical areas, but occurs at multiple levels of processing involved in visual motion analysis. A tentative motion-processing framework is described, based on motion aftereffect research. Recent ideas on the function of adaptation see it as a form of gain control that maximises the efficiency of information transmission at multiple levels of the visual pathway.

Introduction

After prolonged adaptation to a visual scene moving in a certain direction, observation of a stationary scene evokes an experience of motion in the opposite direction. This ancient perceptual effect, called the motion aftereffect (MAE) [1,2], is easy to generate and very robust. Research on the MAE has had a crucial role in the development of theories relating motion perception to neural activity in the brain. Sutherland [3] was the first to suggest a simple neural explanation of the MAE, inspired by Hubel and Wiesel's [4] discovery of direction-selective cortical cells in the cat:

> '. . .the direction in which something is seen to move might depend on the ratios of firing in cells sensitive to movement in different directions, and after prolonged movement in one direction a stationary image would produce less firing in the cells which had just been stimulated than normally, hence movement in the opposite direction would be seen to occur' (p.227 in Ref. [3]).

In 1963, Barlow and Hill [5] reported adaptation-induced changes in responsiveness in single cells in the rabbit retina, and Sutherland's [3] ratio account of the effect gained wide acceptance. Later discoveries of adaptation effects in cat and primate cortex encouraged the general view that the origin of the MAE was probably adaptation in motion-selective cells in primary visual cortex. The essential principle of population coding in the MAE is still universally accepted, but discoveries made possible with the introduction of new experimental techniques indicate that important changes to theoretical explanations of the MAE are required. These discoveries include work in human psychophysics [6–24], primate physiology [25–28], human neuroimaging [29–38], human electrophysiology (Visual Evoked Potentials [VEPs]), magnetoencephalography (MEG) [39–44] and transcranial stimulation [45,46]. Results indicate that the MAE is an amalgam of neural adaptation at several visual cortical sites. This short review offers a fresh appraisal of the MAE and its neural basis, based on this recent research.

Psychophysical evidence: how many aftereffects?

The classical MAE seen in natural viewing conditions involves a static test pattern; after one observes movement for a while, such as a waterfall or the view from a moving vehicle, subsequently viewed stationary objects seem to move. We shall refer to this effect as the static MAE or SMAE. In the late twentieth century, laboratory researchers began using dynamic test patterns such as dynamic visual noise or counter-phase flicker to study the aftereffects of motion adaptation. A dynamic visual noise (DVN) pattern contains a dense field of randomly positioned dots which are replaced by a completely new set of random dots at pre-defined time intervals, typically up to 100 times every second. DVN has the appearance of a de-tuned television display. Counter-phase flicker is created by reversing the contrast of a luminance sine-wave grating repetitively – black bars become white and white bars become black – at a pre-defined frequency (exactly the same effect can be created by spatially superimposing two identical gratings drifting in opposite directions). The properties of MAEs obtained using these dynamic test patterns, which we shall call dynamic motion aftereffects (DMAE), are markedly different from those obtained using stationary patterns and have led to the conclusion that the two aftereffects are mediated by different populations of cells. The contrasting effects produced by first-order motion and second-order motion are particularly important. First-order motion involves patterns defined by variations in the luminance of single image points, such as drifting luminance gratings or dot patterns. Second-order patterns contain features defined by variations in the luminance of pairs of image points, such as variation in texture contrast, size, orientation or binocular disparity. In moving second-order patterns the texture elements defining the pattern are usually replaced by new texture in each animation frame, so the pattern does not contain point-by-point correspondences over time. Adaptation to second-order motion does not produce a SMAE, but it does produce a DMAE [15,16]. Furthermore, first-order and second-order adapting patterns differ in terms of their inter-ocular

Corresponding author: Mather, G. (g.mather@sussex.ac.uk).

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transfer (IOT). It has long been known that when the adapting stimulus is presented to only one eye, and the test stimulus to the alternate eye, an aftereffect is still reported. This IOT relates to the binocularity of the underlying visual neurons. The SMAE shows only partial IOT [13], indicating that at least some of the cells involved are monocular, but the DMAE shows complete IOT [15], indicating that all the cells involved are binocular. These and other results led to the idea that the SMAE reflects adaptation in lower level, first-order motion sensors, whereas the DMAE reflects adaptation in higher level second-order sensors [15,30].

Recent research reveals that the distinction between SMAEs and DMAEs is not as simple as was once believed. Early studies of DMAEs tended to use dynamic patterns that changed at a relatively slow rate or temporal frequency. For example, Nishida and colleagues [15,16] used DVN at a frequency of 2 Hz (each pattern was replaced twice each second), and a pattern that contained features which could be tracked by attention. Their DMAE from adaptation to second-order motion decreased progressively at higher temporal frequencies. Moreover, their findings showed that the IOT was perfect when DMAE was measured in the central visual field and when observers paid attention to the adapting stimulus, but transfer was partial when DMAE was measured with the nulling method (a technique in which the MAE seen in counterphase flicker is cancelled out by increasing the contrast of the component drifting opposite to the MAE direction [17]), when the adaptation stimulus was presented in the peripheral visual field and when subjects were distracted by an interfering task. These results support the hypothesis that DMAEs tap at least two levels of motion analysis; the motion integration level (binocular) and a low-level stage of motion detection (monocular). More recent studies have measured DMAEs using first-order adapting stimuli and much higher frequency dynamic test patterns. Verstraten and colleagues [22–24] used test patterns that changed at rates of between 10 and 90 Hz. Some of their experiments [23,24] involved adaptation to two transparently moving sets of dots, one at high velocity and the other at low velocity. In these stimuli two sets of dots drifting in different directions are spatially superimposed, and can be seen passing through each other. The direction of the resulting aftereffect depended on the temporal properties of the test; stationary tests seemed to move in the direction opposite to that of the slow adapting stimulus, and dynamic tests flickering at 90 Hz seemed to move in the direction opposite to the faster adapting stimulus. Other recent work using dynamic test patterns [6,47] has concluded that two low-level populations of motion sensitive cell are involved in motion aftereffects, one maximally sensitive to flicker at 2 Hz and the other maximally sensitive at 8 Hz or higher (Figure 1).

At least three populations of cell are required to explain the diverse empirical properties of the aftereffects reviewed so far. One low-level population mediates the classical SMAEs from first-order adaptation seen using static test patterns, and perhaps DMAEs seen in very low temporal frequency dynamic test patterns. A second low-level population mediates DMAEs from adaptation to rapid first-order motion seen using high temporal frequency test patterns. A third, 'higher level' population mediates DMAEs from second-order motion seen using

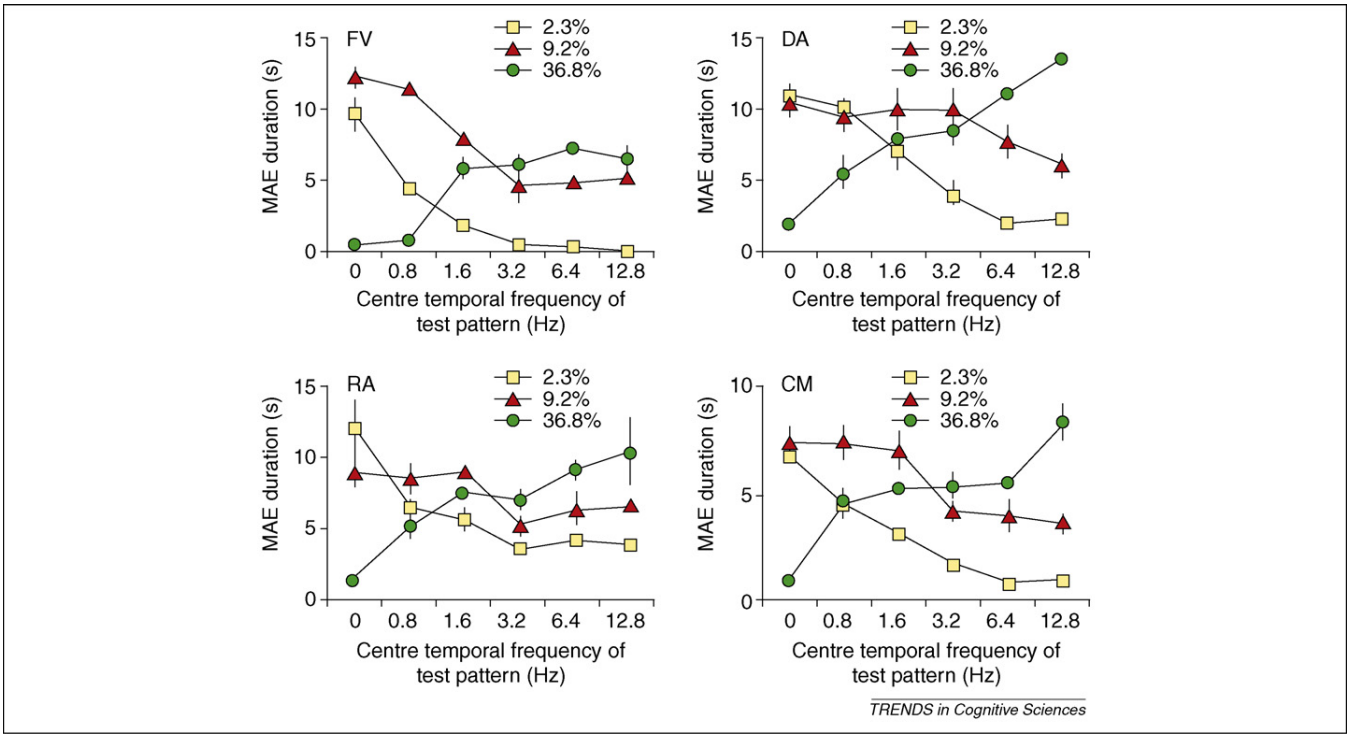

Figure 1. Motion aftereffect duration as a function of the temporal frequency of the test pattern (abscissa) and the speed of the adapting stimulus (different plot symbols). Results are shown for four subjects. For the slowest adapting speed (2.3 ° sec^{-1}, squares), MAE duration is maximal for stationary tests and absent at the highest test temporal frequency; for the fastest adapting speed (36.8 ° sec^{-1}, circles), the MAE is absent for stationary tests and maximal at the highest test temporal frequency (Reproduced, with permission, from Ref. [6]).

low temporal frequency test patterns. A corollary of this conclusion is that DMAEs do not tap a single population of cells, but different populations depending on the properties of the adapting and test stimuli. Maruya and colleagues [48] provided further evidence that the SMAE taps low-level stages and DMAE taps both low- and high-level stages of motion analysis, using a technique called binocular suppression: visual awareness of the adapting stimulus is suppressed, resulting in adaptation to invisible motion. Observers view moving stimuli with one eye, while the other eye receives flickering gratings varying by contrast, size or position [49]. Maruya *et al.*'s [48] observers were adapted and tested using invisible motion to the same eye, or adapted using one eye and tested using the other eye (IOT), using both static and flickering test patterns. Results showed that binocular suppression reduces both the SMAE and the DMAE in the same manner when the same eye is tested, indicating that low-level motion detectors adapt to some extent even when the moving stimulus does not reach awareness. However, in the IOT condition that prevented low-level adaptation, no DMAE at all was obtained following exposure to suppressed adapting motion. These results indicate that invisible motion cannot produce adaptation at a high level of motion analysis, and that DMAEs reflect adaptation both at low- and high-level stages of motion processing.

Recent psychophysical results have implicated two further sites of adaptation in motion aftereffects. Several studies have found evidence for adaptation at a relatively late stage in the motion pathway, in which global movements such as rotation and expansion are computed. Aftereffects have been reported using adapting and test patterns of varying complexity [8,10,18]. Bex *et al.* [8], for example, found that adaptation to radial and rotational patterns produced stronger MAEs than adaptation to translating patterns. Several papers report so-called 'phantom' MAEs, which appear when the test stimulus is projected onto a region of the retina that was not exposed to the adapting stimulus, and that did not seem to contain motion during adaptation (e.g. Refs [50–52]). Meng *et al.* [51], for instance, found phantom MAEs only when the adapting pattern contained radial expansion rather than translation. The presumed cortical location of phantom aftereffects is middle temporal area (MT) or medial superior temporal area (MST), where receptive fields are very large and sensitive to large-scale rotary or radial motion.

Culham *et al.* [11] have argued that apparent motion mediated by attentional tracking can also generate an aftereffect. During adaptation subjects viewed an ambiguous counter-phase grating, and were instructed to '...use attention to mentally track the bars of a radial grating in one of the two ambiguous directions...'. Tests on a static pattern showed no aftereffect, but tests on a 2 Hz counter-phase grating did reveal an aftereffect. Culham *et al.* [11] argued that their DMAE from attentive tracking arose in relatively late cortical areas, perhaps MST. Their adapting stimulus offered equal and opposite signals for motion sensors, so it is possible that attention served to modulate these signals rather than generate its own motion signal. The fact that their effects were confined to DMAEs might indicate the site at which the attentional modulation occurred.

So far, the psychophysics indicates that up to five populations of cells all potentially contribute to motion aftereffects. Are these populations functionally distinct? Do they occupy different cortical locations? Perhaps recent electrophysiological and brain imaging can clarify these fundamental questions.

Physiological evidence: how many sites?

Single-unit recordings

Important recent studies by Kohn and Movshon [25,26] measured adaptation-induced changes in the response of direction-selective cells in macaque MT (previously reported in Refs [27,28]). One of their aims was to determine whether adaptation effects occur at the level of MT, or are inherited in responses fed forward from V1 cells. In the latter case, the spatial extent of adaptation in MT should be limited by the smaller size of receptive fields in V1. Kohn and Movshon [25] did indeed find spatially specific adaptation within MT receptive fields, consistent with adaptation ascending from V1 (Figure 2). Other results reported by Kohn and Movshon [25] indicate a particular role for MT responses in DMAEs. They found that adaptation to the null direction of an individual MT cell (opposite to its preferred direction) enhanced its response to a balanced counter-phase flickering grating, a neural correlate of the DMAE. Adaptation apparently weakened the opponent input to the MT cell, enabing an enhanced response to motion balanced stimuli. Kohn and Movshon [25] do not rule out the possibility that adaptation can also occur in MT neurons themselves.

Human brain imaging

Results from recent functional magnetic resonance imaging (fMRI) studies of human motion processing support a functional distinction between at least two populations of motion sensors, responsive respectively to first- and second-order motion, but these populations do not seem to occupy anatomically segregated locations. Ashida *et al.* [29], for instance, employed a fMRI adaptation paradigm: when repeated presentation of similar stimuli reduced the blood oxygen level dependent (BOLD) response, their inference was that the change in response (fMRI adaptation) reflected changes in the responsiveness of cortical cells activated by all stimuli; when there was little or no reduction in BOLD response they inferred that different cells were activated by the different stimuli. Using this technique they found evidence for separate populations of cells sensitive to first-order and second-order motion in several visual areas including V3A, MT and MST (technical issues could have prevented adequate examination of responses in V1). Nishida *et al.* [35] and Seiffert *et al.* [36] had previously found fMRI responses to both first-order and second-order motion in several visual areas including V1, V2, V3, VP, V3A, V4 v and MT.

These data are important because they are not consistent with early brain imaging studies of the MAE that implicated area MT so strongly [38]. Indeed other recent imaging studies have disputed the primacy of MT in MAEs [31,33], and indicate that several brain areas are activated

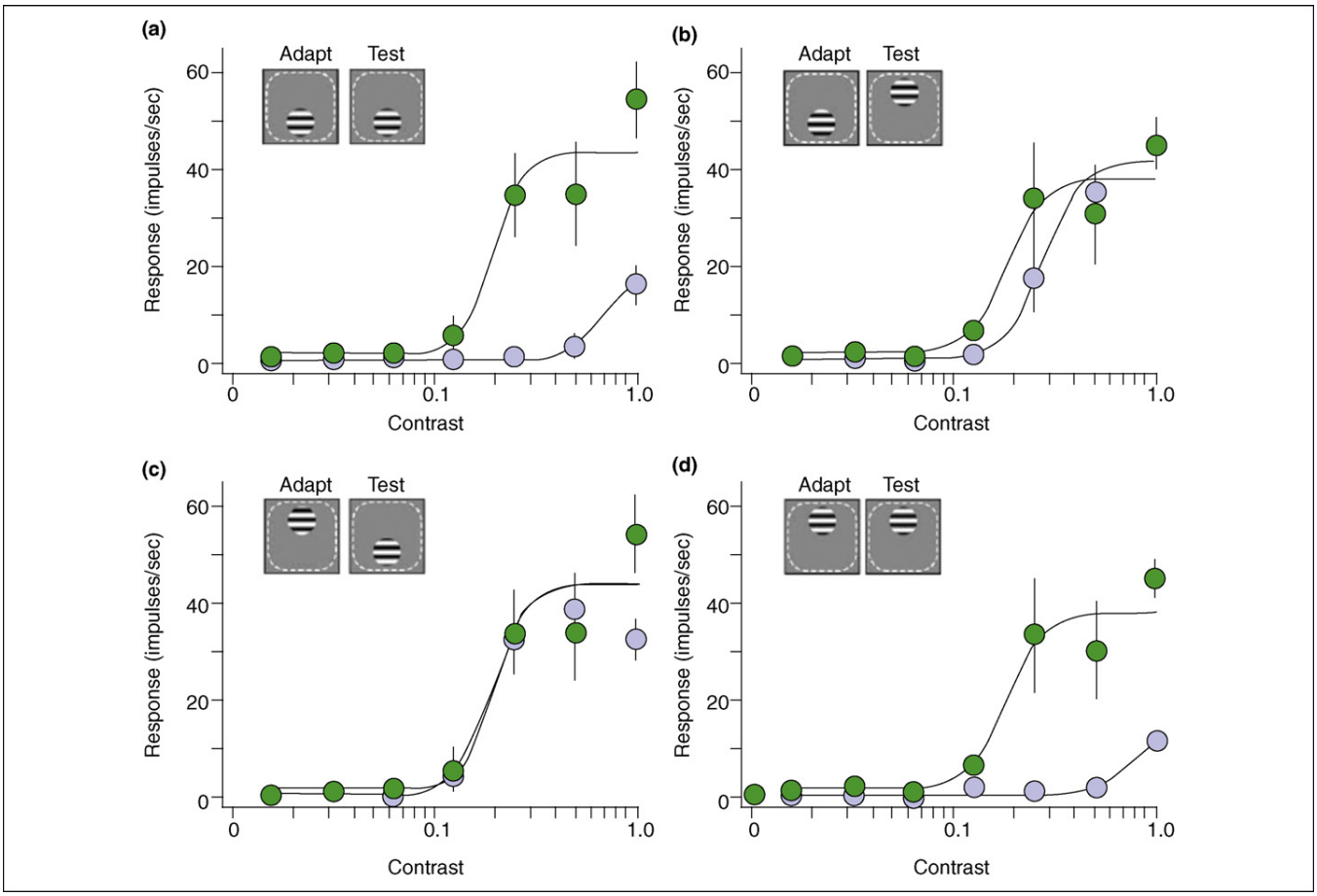

Figure 2. Contrast response functions of an MT neuron measured before adaptation (green circles) and after adaptation (blue circles). The inset in each graph shows the spatial arrangement of adapting and test stimuli in the receptive field of the cell (broken lines). When adapting and test locations overlapped, **(a)** and **(d)**, the response of the cell was strongly reduced; when the adapting and test locations differed, **(b)** and **(c)**, response was largely unaffected by adaptation. (Reproduced, with permission, from Ref. [25]).

during the perception of MAEs. In Taylor *et al.*'s [37] study, subjects were adapted for 21 s to drifting bars or to reversing bars (the control condition). Immediately after adaptation, stationary bars were presented for 21 s, and subjects were instructed to press a button once the subjective experience of a SMAE ceased. During perception of the SMAE, statistically significant activation was indeed found in MT, but also in a network of posterior and anterior cortical sites (Figure 3). In particular there was consistent activation in the anterior cingulate gyrus (CG), BA47 and BA40. Taylor and colleagues [37] argued that these brain regions could be candidates for mediating awareness of the MAE. Correlation analysis showed that two different neural networks are involved in the MAE; a posterior network mainly involved in motion analysis and an anterior network involved in the experience of the MAE. The posterior network includes V1, V2, V3 and MT; the anterior network includes BA37, BA40, BA44, BA46, BA47 and CG. As shown in Figure 3, the most posterior region of the anterior network is the BA37. There is evidence that this area belongs to the anterior network and is anatomically and functionally distinct from area MT. Therefore, BA37 can be considered a bridge between the anterior and the posterior network. The joint activity of the two networks might constitute the neural basis of MAE perception.

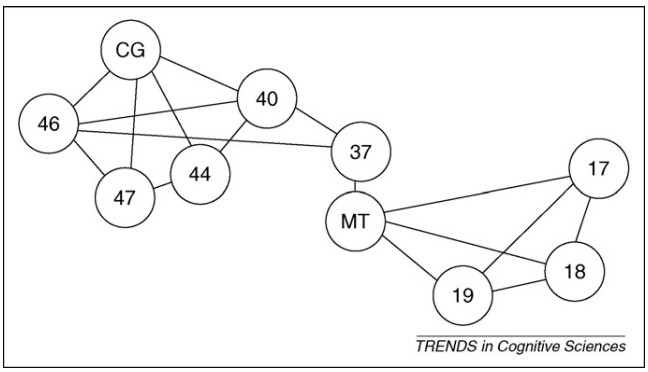

Figure 3. The posterior and anterior neural networks active during the perception of MAE; the connections between sites are derived from the correlation coefficients of the activation time courses. The lines join cortical sites that have cross correlations of at least r = 0.5. Numbers refer to Brodmann areas. (Adapted, with permission, from Ref. [37]).

The same message emerges from Hautzel *et al.* [32], who measured regional cerebral blood flow (rCBF) with positron emission tomography (PET) during MAEs. They found increased rCBF in areas V2, V3a and MT. In addition, when subjects perceived the MAE, an increase in rCBF was also seen in the lateral parietal cortex (BA40) predominantly on the right side, in the right dorsolateral prefrontal cortex (DLPFC), in the anterior cingulate and in the left cerebellum. These results are in broad agreement with

those of Taylor *et al.* [37], and provide evidence of activation in multiple cortical areas during perception of the MAE. The increased rCBF in BA40 and DLPFC might represent activation of cognitive functions during perception of the MAE, such as alertness, attention and working memory.

The participation of attentional networks raises the question of whether activity detected in visual areas such as MT might actually reflect attention to the MAE. Huk and colleagues [34] addressed this question. In previous imaging studies of the MAE there was no control of attentional state, so subjects were free to allocate and shift attention differentially between MAE and control conditions, and attention could have enhanced the MT response. Huk *et al.* [34] initially replicated previous findings using a paradigm similar to that of He *et al.* [33]: there was a larger increase in MT response in the MAE condition (after adapting to unidirectional motion) than in the control condition (adaptation to motion reversal at 2 Hz). To equate attention in both MAE and control conditions, subjects had to perform a sequence of two-alternative forced-choice speed discriminations during 5 s test period. Results showed no difference in BOLD signal between MAE and control conditions.

Huk *et al.* [34] performed another experiment to test direction-selective adaptation (adaptation is direction-selective if it is larger for test stimuli moving in the same direction as the adapting stimulus than for test stimuli moving in the opposite direction). Subjects adapted to a given motion direction and then viewed test stimuli moving either in the adapted direction or in the opposite direction. Direction-selective adaptation was observed in V1, V2 and MT. Thus, there are no grounds for claiming that MT has any unique status in terms of MAE locus.

Human transcranial stimulation studies

Stewart and colleagues [43] were the first to succeed in reducing the duration of SMAE (but not of the colour aftereffect) with magnetic stimulation over MT, indicating a role for MT in the SMAE. Théoret *et al.* [46] applied repetitive transcranial magnetic stimulation (rTMS) over MT during a storage period in between MAE adaptation and testing. Stimulation shortened the duration of the subsequent MAE, compared to a control condition without rTMS. There was little effect of stimulation to V1 on storage. In a second experiment rTMS was applied to MT during perception of the MAE, and was found to reduce MAE duration, but stimulation to DLPFC and to posterior parietal cortex had no effect on aftereffect duration. It should be noted, however, that Théoret *et al.* [46] used complex radial and rotational stimuli. Therefore, it is not surprising that they found a specific involvement of the MT complex. Antal *et al.* [45] used a relatively new technique, namely transcranial direct current stimulation (tDCS), to explore the role of MT in MAEs. They found that stimulation of MT significantly decreased MAE duration. Stimulation of V1 did not affect MAE duration, although the authors admit that its retinotopic organization might have spared the relevant region of cortex from disruption. Overall, TMS and tDCS studies clearly implicate MT in the MAE, but the role of other areas is still unclear.

VEPs and MEG

Which components of the VEP reflect activity related specifically to the MAE? Human electrophysiological studies have shown that the amplitude of a negativity peak at ~200 ms (N2) is affected by motion adaptation [40], but it is not clear whether this effect is direction selective. More recently, Kobayashi *et al.* [41] found a significant bilateral increase of a positive component at ~160 ms (P160) in the occipitotemporal region after motion adaptation. They also observed a laterally biased effect in the right posterior temporal region, perhaps related to the engagement of attentional circuits.

Neural gamma-band activity (GBA, high-frequency neural activity in the range 40–100 Hz) seems to be associated with synchronization among different brain regions, which is thought to be important for visual feature binding and motion perception [53]. Tikhonov *et al.* [44] investigated GBA associated with the MAE using MEG (a non-

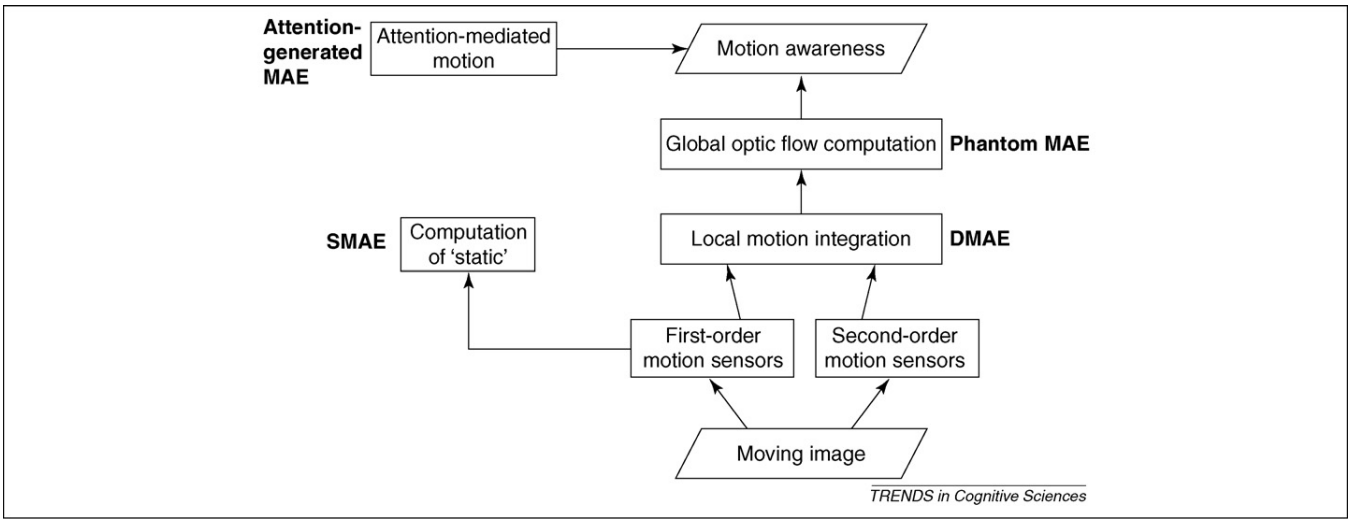

Figure 4. Functional diagram relating the main stages of motion processing in the human brain to MAE adaptation sites. SMAEs are mediated by motion sensors that contribute to computation of 'static', whereas DMAEs are mediated by motion sensors that contribute to motion integration computations. Phantom MAEs involve sensors contributing to the computation of optic flow.

Box 1. Where do SMAEs and DMAEs arise?

One can infer from Kohn and Movshon's [25] single-unit recording study that the SMAE reflects adaptation-induced changes in the response of first-stage cells in V1 which contribute to a computation of static, whereas the DMAE reflects changes in the response of second-stage MT neurons that receive opponent inputs from V1 cells. The psychophysical study of Morgan *et al.* [12] concurs with this inference. They argue that SMAEs represent a shift in the population response of cells tuned to relatively slow velocities (we see 'static' when the outputs of low-velocity units are balanced), whereas DMAEs arise from adaptation-induced dis-inhibition in higher level cells tuned to higher velocities (see also Ref. [23]). Why do some adapting stimuli, such as second-order patterns, only produce aftereffects on dynamic tests? Perhaps the cells activated by such stimuli are not themselves involved in the computation of 'static', so an adaptation-induced imbalance does not lead to a SMAE, but their responses nevertheless feed forward to influence later motion computations and cause DMAEs.

Box 2. Outstanding questions

Several important issues remain to be resolved, including:
- How do the different adaptation components combine to determine the observed aftereffect?
- Why do static and dynamic tests tap different adaptation processes (see Box 1)?
- Can attention generate its own aftereffect, or does it only modulate lower level adaptation?
- What is the functional significance of multiple adaptation sites (see Box 3)?

Box 3. What is the mechanism and function of adaptation?

Adaptation can be viewed as a form of automatic gain control, in which a unit attenuates its own response to continuous intense stimulation. Van de Grind *et al.* [19,20] developed a detailed computational model of MAE adaptation that uses divisive feed-forward inhibition as the gain control mechanism. During adaptation, a leaky integrator in the gain control circuit of active cells charges up, and during testing the charge leaks out to cause an imbalance in output between adapted and unadapted units. The results of psychophysical experiments conducted by van de Grind and colleagues were consistent with their model. Other psychophysical [12] and electrophysiological [25] results are also consistent with this kind of gain control mechanism.

What function does gain control serve, and why should it be present at several different sites in the processing hierarchy? One idea is that gain control improves the efficiency of encoding by striving to maximize the information about the stimulus conveyed in neural spike trains [54]. Visual motion analysis involves the transmission of information between multiple processing stages. Gain control at each stage of transmission should serve to optimize the efficiency of coding in the system as a whole. According to this view, there should be as many sites of adaptation for MAEs are there are processing stages in the motion analyzing system. MAE research indicates that adaptation at higher level sites is weaker than adaptation at lower level sites because the aftereffects tend to be short-lived. Why should this be so? The persistence of adaptation can be viewed as a prediction by the visual system that the current pattern of stimulation will continue into the future [55]. Perhaps the complex motions signalled at high-level sites are less predictable and persistent than the simpler motions signalled at lower levels, and the difference in adaptation strength is an expression of this difference in predictability.

invasive technique used to measure magnetic fields generated by the electrical activity in neurons). Adaptation and test patterns were presented either in the right or left visual hemifield. Results showed GBA reflecting the MAE in channels over two locations (indicating the presence of two dipole sources), providing evidence that MAE depends on the synchronization of different brain regions. The first location was in parieto-occipital cortex (in the region of area MT). The second location was more posterior, but it was not possible precisely to locate the origin of this source. Two possibilities discussed were striate cortex and the cerebellum.

Conclusions

Figure 4 is a simple functional diagram that attempts to summarize the main stages of visual motion processing from the perspective of the motion aftereffect research reviewed here. Motion sensors in the earliest cortical areas (V1, V2 and V3) feed into a computation underlying the perception of 'static' and also into a local motion integration stage. First-order motion sensors tuned to slow velocities contribute to 'static' computations, whereas first-order sensors tuned to higher velocities and second order sensors both feed into motion integration. As explained in Box 1, adaptation in sensors contributing to the static computation leads to the SMAE; adaptation in sensors contributing to motion integration leads to a DMAE, probably in area MT. Adaptation of cells involved in computation of optic flow in area MST mediates phantom MAEs. Attention-mediated motion aftereffects and subjective awareness of motion aftereffects involve more anterior cortical areas such as parietal and cingulate cortex. The varieties of motion aftereffect reviewed here tend to be regarded as a cognate group of effects but this framework highlights the fact that, in functional terms, they are distinct and separate. The SMAE, for example, involves an entirely different population of neurons and separate computations from those involved in phantom MAEs, in the same way that the tilt aftereffect involves different processes from those underlying the size aftereffect.

This emerging framework is very much a work-in-progress, and several issues remain to be resolved (see Boxes 1, 2 and 3).

Acknowledgements

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