interacting neuronal networks make adaptive behavioral decisions. Further new avenues opened up by recent work on *Lymnaea* may lead to an understanding of how learning and homeostatic processes interact, to the building of sophisticated computer models of functional connectivity and interacting networks, and even to the development of applications to artificial complex systems.

At a different level, the discovery and structural analysis of a glia-derived acetylcholine-binding protein that modulates synaptic transmission between identified neurons of the *Lymnaea* respiratory CPG has provided important cues to the structural basis of ligand binding in nicotinic acethylcholine receptors in vertebrates. This pioneering work in *Lymnaea* opened the way for new research that might lead to the development of new drugs involving nicotinic acetylcholine receptor-associated diseases.

A recently initiated pilot sequencing project is expected to result in a cDNA sequence database from the CNS, and in turn, will potentially lead to a full genomic sequencing project of *Lymnaea stagnalis*.

Where can I find out more?

Benjamin, P.R. (2008). *Lymnaea*. Scholarpedia *3(1)*, 4124. http://www.scholarpedia.org/article/Lymnaea.

Benjamin, P.R., Kemenes, G., and Kemenes, I. (2008). Non-synaptic neuronal mechanisms of learning and memory in gastropod molluscs. Front. Biosci. *13*, 4051–4057.

Kemenes, G. (2008). Molecular mechanisms of associative learning in *Lymnaea*. In J. David Sweatt (Ed.), Molecular Mechanisms of Memory. Vol. 4 of Learning and Memory: A Comprehensive Reference, 4 vols. ed. J. Byrn. (Oxford: Elsevier), pp. 133–148.

Lymnaea stagnalis Sequencing Consortium: http://www.lymnaea.org/.

Nikitin, E.S., Vavoulis, D.V., Kemenes, I., Marra, V., Pirger, Z., Michel, M., Feng , J., O'Shea, M., Benjamin, P.R., and Kemenes, G. (2008). Persistent sodium current is a non-synaptic substrate for long-term associative memory. Curr. Biol. *18*, 1221–1226.

Smit, A.B., Syed, N.I., Schaap, D., van Minnen, J., Klumperman, J., Kits, K.S., Lodder, H., van der Schors, R.C., van Elk, R., Sorgedrager, B., *et al.* (2001). A glia-derived acetylcholine-binding protein that modulates synaptic transmission. Nature *411*, 261–268.

Staras, K., Kemenes, I., Benjamin, P.R., and Kemenes, G. (2003). Loss of self-inhibition is a cellular mechanism for episodic rhythmic behavior. Curr. Biol. *13*, 116–124.

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Primer

Visual aftereffects

Peter Thompson[1] and David Burr[2]

In a now classic paper, Robert Addams (1834) wrote:
"During a recent tour of the Highlands of Scotland, I visited the celebrated Falls of Foyers on the border of Loch Ness, and there noticed the following phaenomenon. Having steadily looked for a few seconds at a particular part of the cascade, admiring the confluence and decussation of the currents forming the liquid drapery of waters, to observe the vertical face of the sombre age-worn rocks immediately contiguous to the waterfall, I saw the rocky surface as if in motion upwards, and with an apparent velocity equal to that of the descending water, which the moment before had prepared my eyes to behold this singular deception."
After 175 years of research we now have some doubts as to whether the 'aftereffect' has an apparent velocity equal to that of the adapting stimulus, but otherwise Addams' description has not been bettered. For more than a century the effect Addams described, variously know as the Waterfall illusion, motion aftereffect or movement aftereffect, was investigated in laboratories around the world; subjects 'adapt' to a moving stimulus — typically a pattern of stripes or moving dots — for a period of perhaps 60 seconds and then, when the adapting pattern stops, they experience an aftereffect of perceived movement in the opposite direction. The generally accepted explanation that emerged is that there exist detectors tuned to different directions of motion and that our perception of movement — or the lack of it when things are stationary — would be mediated by the relative responses of detectors tuned for

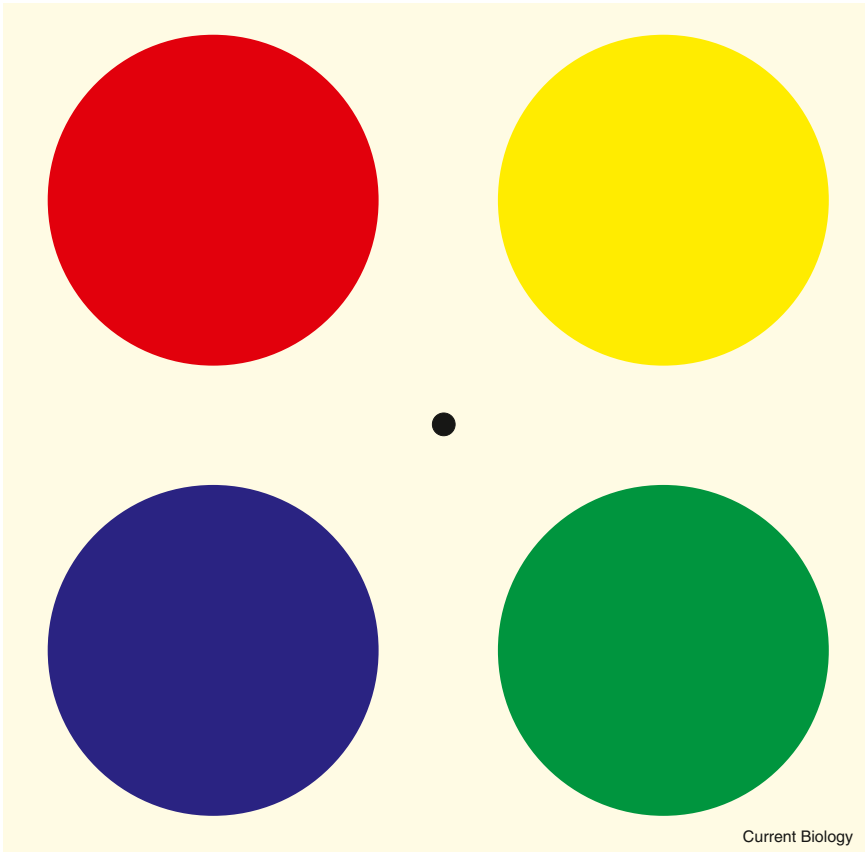

Figure 1. Colour aftereffects.
Stare at the central black spot for 30 seconds and then divert your gaze to a blank white surface. Complementary coloured afterimages will be seen. The red and green circles will have appeared to have switched places as have the yellow and blue.

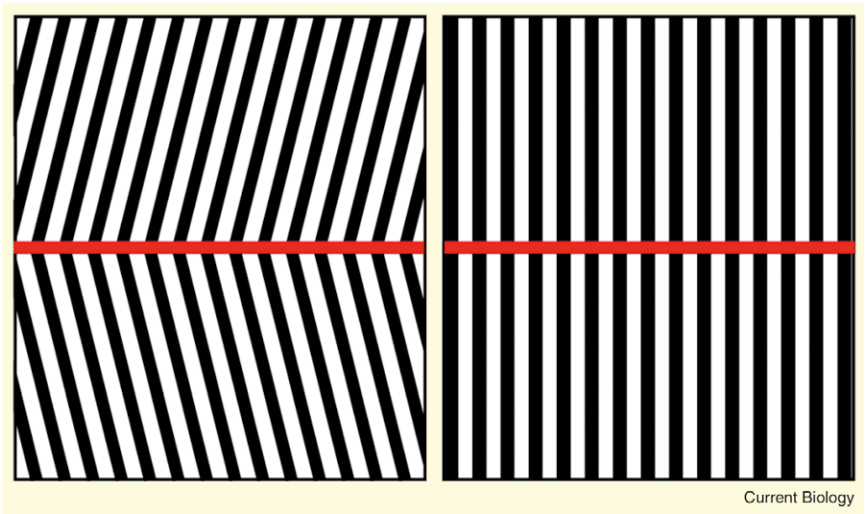

Figure 2. The tilt aftereffect.

Adapt to the left-hand part of the figure by letting your eyes move along the central horizontal line. After 30 seconds transfer your gaze to the central horizontal line on the right-hand part of the figure. Briefly the vertical lines will appear tilted in the opposite direction.

opposite directions of motion. Thus when Addams looked at the waterfall in Scotland he 'adapted' his downwards detectors so that when looking at the stationary sombre age-worn rocks there was now an imbalance between the adapted downwards detectors and the unadapted upwards detectors, resulting in the perception of upwards motion. This motion adaptation doesn't affect just the appearance of stationary patterns; generally, patterns moving in the same direction as the adaptation stimulus will be significantly slowed, even to the point where they can appear stationary.

It is not just adaptation to movement that can produce aftereffects. Perhaps the most common aftereffects are experienced after looking at colours (Figure 1). Note that, again, the aftereffect experienced is from the opposite end of some notional continuum. In colour vision it is believed that colours are represented along orthogonal opponent axes: red to green; and yellow to blue. Because of this opponency we cannot experience a reddish green or a yellowish blue but we can envisage yellowish green or a reddish blue, for example. Many examples of aftereffect demonstrations can be found at www.viperlib.com.

A century after Addams, J.J. Gibson showed that after adapting to curved lines, subsequently viewed straight lines appeared curved in the opposite direction, and that adapting to straight lines tilted a few degrees from vertical led to a 'tilt aftereffect' in which a truly vertical pattern now appeared tilted in the opposite direction. In all these cases the perception of some 'null point' — a stationary stimulus, straight lines or vertical lines — is shifted by adaptation (Figure 2). This led Gibson to propose:

"If a sensory process which has an opposite is made to persist by a constant application of its appropriate stimulus conditions, the quality will diminish in the direction of becoming neutral, and therewith the quality evoked by any stimulus for the dimension in question will be shifted temporarily towards the opposite or complementary quality."

This was the first attempt to explore the possibility that aftereffects might be beneficial to the visual system, rather than an unfortunate failure of the system to represent the world accurately.

Research into aftereffects, and our understanding of them, accelerated in the 1960s as a result of neurophysiological studies that provided support for the mechanisms proposed by psychophysics. The visual cortex of cat and monkey was shown to contain cells that respond to a narrow range of orientations, and could also be direction-selective, precisely the mechanisms that could support the 'fatigue' model of adaptation. This position was convincingly supported by a report by Barlow and Hill who showed that direction-selective ganglion cells in the rabbit adapted during prolonged stimulation and remain adapted for some seconds thereafter, a duration conveniently similar to the duration of psychophysically observed aftereffects.

Psychophysical studies in the next decade demonstrated new aftereffects. One was an elevation in contrast threshold following adaptation to sinusoidal gratings which was both orientation selective and spatial frequency selective. A spatial frequency

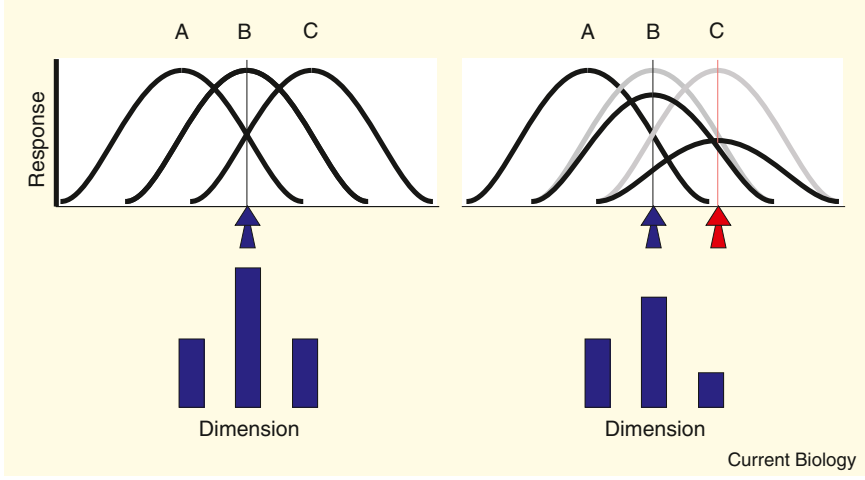

Figure 3. Explanation of aftereffects.

In left-hand diagram, A, B and C represent tuned mechanisms ('channels') along some dimension, for example, direction of movement, tilt, size, disparity, colour and so on. A stimulus on the dimension (marked by blue arrow) will produce the greatest response in channel B, with lower but equal responses in A and C. Adaptation at some point along the dimension (shown by red arrow in right-hand diagram) results in greatly reduced response in channel C, a slight reduction in the response of B, and no change in A. The stimulus at the blue arrow will now produce a greater response in A than C, resulting in a shift away from the adaptation stimulus.

aftereffect was described which showed that the perceived spatial frequency of a grating could be shifted away from its true value by adaptation; adapting to a slightly lower spatial frequency increased the perceived frequency of the test stimulus and adapting to a slightly higher frequency decreased it.

Soon there were reports that cortical neurons exhibit adaptation which might underlie the psychophysically observed aftereffects, and even attempts to show that the amount of interocular transfer of an aftereffect — the size of the effect when the adaptation and test stimuli are presented to the same eye compared with that when adaptation is presented to one eye and the test to the other — could be related to the percentage of monocular and binocular neurons in primary visual cortex. Figure 3 outlines a simple explanation of how adapting these mechanisms could lead to aftereffects.

Two further aspects of the movement aftereffect seemed to anchor it firmly as a distortion encountered early in visual processing: it appeared not to be affected by attention; and an adaptation pattern rendered invisible by binocular rivalry still acted as an effective adaptation pattern.

In the heady days of the 1970s, visual aftereffects became the 'psychologist's microelectrode'. If physiologists found single cells tuned for stereoscopic disparity (and they did) then psychophysicists would find a disparity aftereffect (and they did). John Mollon in a review of aftereffects at the time wrote "if you can adapt it, it's there".

But despite the seemingly cosy correspondence between low-level physiology and psychophysics, there were storm clouds gathering. The notion that aftereffects resulted from 'fatiguing' cells in the visual pathway became untenable and evidence mounted that inhibitory processes are responsible for the reduced response of cells during adaptation. And there was increasing interest in what appeared to be a radical new type of 'contingent' aftereffect, the McCollough effect (Figure 4). Adaptation to a red and black vertical grating alternating with a green and black horizontal grating produced a colour aftereffect in which black and white verticals were tinged with green and horizontals were tinged with pink. This effect might superficially be squeezed into the adaptation-of-single-neurons model, but it had some

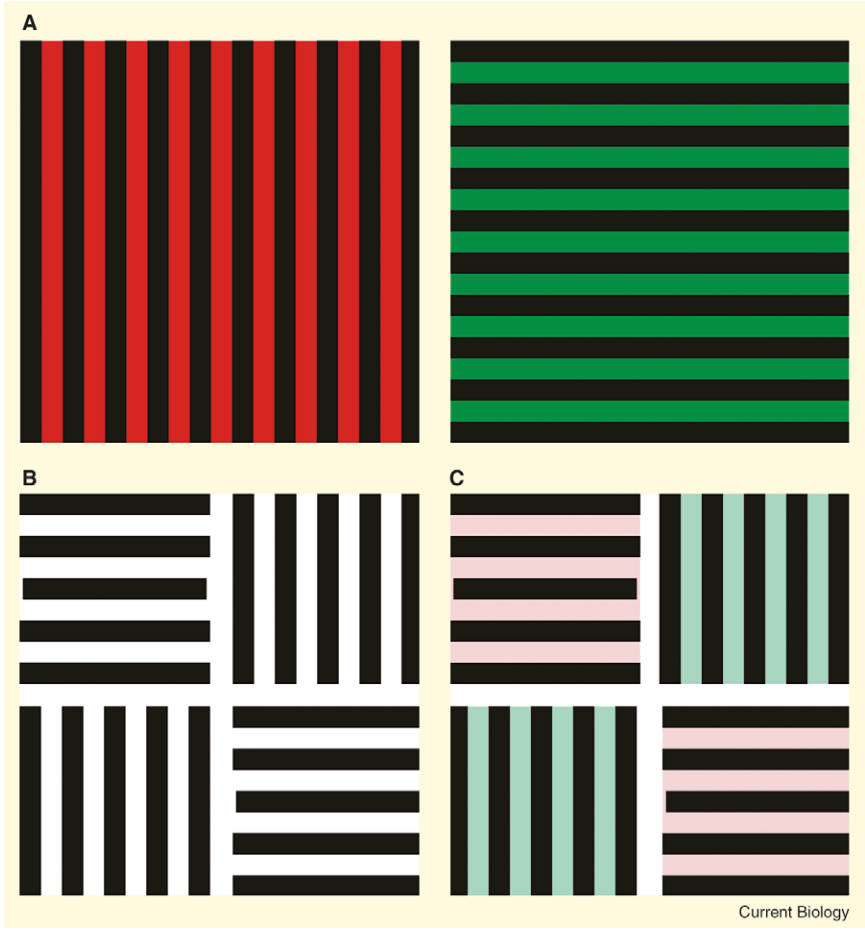

Figure 4. The McCollough effect.
Adapting to alternating patterns of red vertical and green horizontal gratings (A) results in a black and white test pattern (B) appearing coloured in complementary colours (C).

puzzling characteristics, chiefly that a few minutes of adaptation could produce an effect that lasts for days and weeks if not months, that seem to defy an explanation in terms of the short-lived adaptation reported in single cells. It became clear that the movement aftereffect itself could be demonstrated at least 20 hours after the adaptation exposure, and furthermore it exhibited 'storage' — if, after adaptation, the subject closed his eyes for a period of time longer than the expected duration of the aftereffect, upon opening his eyes the aftereffect was still seen. These effects all suggested that recovery from the adaptation was not a simple physiological recovery from fatigue or release from inhibition.

The final nagging doubt about any such simple explanation of aftereffects was, like Sherlock Holmes' silent dog, the lack of an expected event; why, if 30 seconds adaptation in a laboratory can produce a vivid movement aftereffect, do we so seldom experience these effects as we move through the world? Very often motion on the retina is associated with our movement through the environment rather than the artificial movement of a laboratory stimulus while we are stationary. This suggests that the movement aftereffect may result in part from some recalibration process. There is some evidence to support this idea beyond the simple observation that an hour's driving along a motorway doesn't produce the expected aftereffect given the persistent expanding optic flow we've been experiencing. If in the laboratory subjects move forwards while adapting to an expanding visual stimulus, we should expect a reduced movement aftereffect, and this is what is found.

The advent of functional magnetic resonance imaging (fMRI) has opened a new chapter on visual aftereffects generally and the movement aftereffect in particular. Increased activity in human area MT (V5) during aftereffect motion has been taken as evidence that this

Figure 5. The face-identity aftereffect.

Adapting to the photograph of John Mollon (left) will result in the central ambiguous figure being perceived as more saintly whereas adapting to the Archbishop of Canterbury (right) will result in the central image appearing more secular. (Courtesy of John Mollon.)

extrastriate area, known to be central to motion processing, is important for the movement aftereffect as well. But fMRI studies have opened one area of controversy; there is now good imaging evidence that attention can affect activity in primary visual cortex and even the lateral geniculate nucleus, a site so early in the visual pathway that it precedes direction-selectivity in cells in primates. Why then should attention not appear to have an influence on the movement aftereffect? This is an area of current debate but what seems clear is that the once generally accepted view that attention has no effect on the movement aftereffect has been overthrown and attentional effects have been shown in a number of studies.

So while the realm of 'top-down' processing descends to ever-more peripheral sites in the visual system, the visual areas implicated in aftereffects expand in the opposite direction. In the psychophysical (and physiological) certainty of the seventies, it seemed that all aftereffects might be explained in terms of activity in primary visual cortex. Now we know that V1 is only part of the story; a variant of the movement aftereffect, the dynamic movement aftereffect, has been shown to be more consistent with activity in extrastriate area MT (V5), and there has recently been an explosion of reports of adaptation of cells in temporal cortex that respond selectively to faces. In one area it may be that the adaptation is to the direction of gaze of the face and in another the face identity regardless of whether a full frontal or profile view is seen (Figure 5). It may even be that adaptation to a low level feature (an upturned curve) can alter the perceived facial expression of a face from being happy to being sad and any perceived aftereffect is the result of adaptation at multiple sites within the visual pathway.

So, do these aftereffects serve any functional purpose, other than to entertain readers of *Current Biology*? Almost certainly. It is now generally agreed that aftereffects, and adaptation in general, are not mere by-products of 'fatiguing neurons', but reflect neural strategies for optimising perception, including calibration (discussed earlier), gain control and 'decorrelation'. All neurons have a limited working range where their response increases with stimulus strength: low stimulus intensities fail to activate the neuron and higher intensities cause saturation. Gain control allows maximum use of this limited range. For example, staring at a bright field desensitizes retinal neurons, thereby avoiding saturation, but also produces brightness aftereffects, akin to those illustrated in Figure 1, under suitably contrived conditions. Indeed, this is the main mechanism that allows the visual system to operate over a range of some ten orders of magnitude (variation in pupil size accounts for only one order of magnitude).

In principle, similar mechanisms could work for more complex perceptual tasks, such as face perception. For example, much evidence suggests that faces are encoded in a multi-dimensional space centred around a norm, which reflects the average of all faces. Adaptation could ensure the sensitivities of these dimensions are tuned to ensure use of the whole response range, without saturation. Although firm evidence is still lacking, the notion of an adaptable face-space accounts both for the vivid aftereffects (like those of Figure 5), and for many other phenomena, like the relative ease of distinguishing faces of ones own racial group (to which we continually adapt) compared with another.

Another possibility, suggested by Horace Barlow, is that aftereffects can increase efficiency by a process of 'decorrelation'. Coding in multidimensional space is most efficient when the dimensions are orthogonal, uncorrelated with each other. If correlations exist, the dimensions will not be orthogonal, and thus less efficient. For example, if there is a tendency for red to be associated with vertical and green with horizontal — as in the McCollough effect illustrated in Figure 4 — the visual system could exploit this correlation by adjusting sensitivities to restore orthogonality between these dimensions (decorrelation). A by-product of this process would be the contingency aftereffect observed in Figure 4 when the correlation is removed (by replacing red and green with white).

In the 35 years since John Mollon's review we have discovered an increasing range of aftereffects; if adapting it means it is there then there must be a lot more things in our brains than we suspected.

Further reading

Addams, R. (1834). An account of a peculiar optical phaenomenon seen after having looked at a moving body etc. London Edinburgh Philosoph. Magaz. J. Sci. 3rd series *5*, 373–374.

Blakemore, C., and Campbell, F.W. (1969). On the existence of neurons in the human visual system selectively sensitive to the orientation and size of retinal images. J. Physiol. *203*, 237–260.

Barlow, H.B., and Földiák, P. (1989). Adaptation and decorrelation in the cortex. In The Computing Neuron. R. Darbin, C. Miall and G. Mitchison. (Wesley Publishers Ltd.), pp. 54–72.

Barlow, H.B., and Hill, R.M. (1963). Evidence for a physiological explanation of the waterfall phenomenon and figural after-effects. Nature *200*, 1345–1347.

Clifford, C.W.G., and Rhodes, G. Eds. (2005). Fitting the mind to the world; adaptation and after-effects in high-level vision. In Advances in Visual Cognition (Oxford: Oxford University Press).

Greenlee, M.W., and Heitger, F. (1988). The functional role of contrast adaptation. Vision Res. *28*, 791–797.

Krekelberg, B., Boynton, G.M., and Van Wezel, R.J. (2006). Adaptation: from single cells to BOLD signals. Trends Neurosci. *29*, 250–256.

Maffei, L., Fiorentini, A., and Bisti, S. (1973). Neural correlate of perceptual adaptation to gratings. Science *182*, 1036–1038.

Mollon, J.D. (1974). After-effects and the brain. New Scientist *61*, 479–482.

Movshon, J.A., and Lennie, P. (1979). Pattern-selective adaptation in visual cortical neurons. Nature *278*, 850–852.

Wohlgemuth, A. (1911). On the aftereffect of seen movement. Br. J. Psychol. Mon. Suppl. *1*, 1–117.

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