diverse function from at least three kingdoms. These include recently discovered fatty acid primary amines that induce sleep in mammals (6), ω-1 hydroxyl fatty acids such as 11-hydroxy-jasmonic acid involved in defense gene signaling in plants (7), and small quorum-sensing regulators such as hydroxybutyryl homoserine lactones, which regulate social interaction in bacteria (8). The overall construction of the molecule is somewhat reminiscent (with respect to the polar head and less polar fatty acid tail) of the family of Nod factors, powerful developmental regulators produced by nitrogen-fixing plant symbionts (9).

Some of the structural features necessary for volicitin function in maize have been identified. The L-enantiomer of glutamine is indispensable for biological activity; volicitin containing D-glutamine is completely inactive (4). These molecules will almost inevitably be valuable tools in the search for volicitin receptors in plants.

How then could volicitin act to control the release of volatiles from maize leaves? Some clues may come from a recent study showing that jasmonic acid, a 12-carbon cyclopentanone fatty acid regulator derived from the plant octadecanoid pathway (7), can stimulate the release of cocktails of volatile compounds from a wide variety of plants, including maize (10). Alborn et al. (4) suggest that volicitin may interact with the octadecanoid signal pathway to effect volatile release. To test this it will be interesting to see whether plants incapable of responding to jasmonate can still respond to volicitin.

Why should a herbivorous insect make a potent molecule that betrays its presence and may ultimately lead to its demise? Presumably because the molecule has an important function in the insect. Alborn et al. (4) speculate that volicitin itself may play a hormonal role in beet armyworm, but the fact that it is present in insect oral secretions may imply that it has functions in the process of herbivory. At this stage many scenarios appear possible. For example, one could speculate that volicitin may be useful to the herbivore by disrupting part of a plant defense signal pathway, such as the jasmonate-octadecanoid pathway leading to antidigestive proteinase inhibitor production (11). In this way mechanical damage–induced defense gene expression would be reduced, favoring the insect. A function for volicitin in the physiology of the insect gut microflora cannot yet be ruled out.

Whatever its role in beet armyworm, volicitin must be the focus of intense selection pressure and in this respect is reminiscent of another class of elicitors that derive from plant pathogens. These are the avirulence gene products of fungi, bacteria, viruses, and nematodes (as well as a limited number of insects) that trigger the hypersensitive response of defense-related cell death in host plants of cognate genotype. Avirulence elicitors differ greatly in structure, ranging from proteins to low molecular mass fatty acid derivatives (12). As with volicitin, the functions of most avirulence gene products in pathogens remain unclear. Could the comparison of avirulence elicitors and volicitin be useful? The hypersensitive response leads to an easily visible phenotype (necrosis) and to powerful resistance. Are there parallels in less easily observed phenomena such as volatile release in tritrophic systems? To address this question and to better understand selection pressures acting on volicitin production, it will be interesting to know whether all maize genotypes respond in the same way to this molecule, whether the molecule is a general elicitor of volatile release in various plant species, and whether different insects produce different volicitins. This work will not only extend our knowledge of plant-insect interaction but will also provide fascinating comparisons with chemical distress signaling in other warring organisms, including vertebrates (13).

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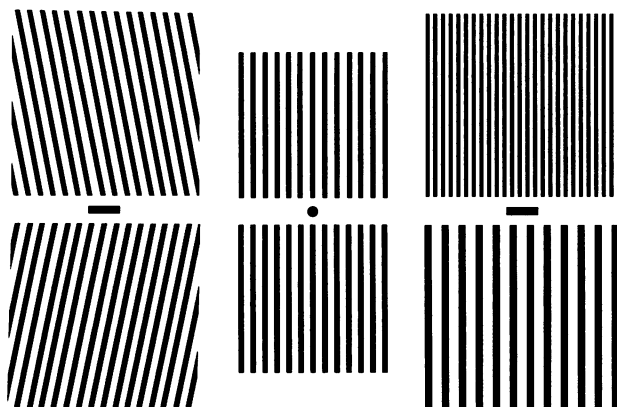
# Adaptation by Hyperpolarization

## Horace Barlow

**Y**ou can get used to anything. This aphorism may not always hold, but it is true that humans (and other animals) have a powerful ability to adapt to stimuli from the outside— a noxious odor, immediately repulsive, becomes hardly noticeable, the sound of traffic no longer annoying. The physiological reflection of this ability is the property of sensory neurons to "adapt" to a sensory stimulus maintained at a high value, giving a response that declines with time. This adaptation is prominent in neurons of the cerebral cortex, and new results reported by Carandini and Ferster (page 949) reveal the surprising way in which these neurons accomplish this adjustment to the environment (1).

Adaptation in the neurons of the visual cortex gives rise to optical illusions such as that shown in the figure (see next page). The distortions in the grating occur only in the region of the visual field exposed to the grating and persist for a few seconds or longer if the adaptation period is prolonged. Many similar aftereffects are known, perhaps the most familiar being the "waterfall phenomenon," in which the observer first gazes for a

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time at the downward stream of water, then looks at the surrounding scenery and sees it apparently drifting upward. These adaptive changes are specific for the pattern. The distortions can be understood in terms of a selective and persistent desensitization of those members of an array of cortical neurons that have been most vigorously excited by the adapting stimulus. Such desensitization has been shown to occur by recording the extracellular currents that accompany the action potentials transmitted by a cortical neuron to other neurons in the brain.

In the new work, Carandini and Ferster used a technique called whole-cell patch recording, in which a fluid-filled electrode terminating in a small pore is pushed up to the membrane of the cell, the rim of the pore making a tight seal with the surface. Electrical contact with the cell's interior is then established by popping the membrane within the seal, enabling the cell's intracellular potential to be continuously recorded. The technique is difficult to perform in the cerebral cortex because tiny movements of the electrode relative to the cell break the seal with the cell surface, but it is possible to record for long enough to find out what happens during and after an adapting stimulus similar to the one in the figure.

**Adapt your cortical cells.** First, look at the dot between the central gratings and confirm that they are both vertical and have the same spacing. Then, place the figure about 30 cm in front of you and fix your eyes for 30 s on the black strip between the left gratings, moving them along the strip but not outside it. Next, transfer your gaze to the central dot and compare the upper and lower gratings; for a brief period the upper grating should appear tilted to the right and the lower one to the left. To produce an illusion of size, repeat the procedure by adapting to the right pair of gratings. [Adapted from (6)]

As expected, movement of a grating over the portion of the visual field that excites a neuron causes a modulated stream of action potentials that diminish in number during adaptation. The new result is that the stimulus-induced responses of the intracellular potential are barely attenuated by adaptation; instead, the reduction in the number of action potentials is caused by a slowly developing and persistent increase in the negative intracellular potential of the cell, so that a diminished fraction of each cycle of modulation rises above the cell's firing threshold, and fewer impulses occur with each cycle as adaptation progresses. They obtained no direct evidence about the cause of this hyperpolarization, but it was not accompanied by any significant change in the membrane resistance of the cell.

The biophysical mechanisms underlying this phenomenon are likely to be the subject of further experiment and argument, but the existence of a factor that slowly changes the membrane potential of a cortical neuron may tie in with other recent developments.

The increase in the cortical response that occurs when an animal is attending to a particular stimulus could be accomplished by such a tonic process that depolarizes the neuron (2). Although in the opposite direction, this could work through the same mechanism as the hyperpolarization of adaptation. There are also dramatic changes in the receptive fields of neurons in the primary visual cortex when regions outside the receptive field are stimulated, which could result from a change in the neurons' responsiveness (3). If so, this plasticity could also use the same mechanism.

There is little evidence to decide whether such a common modulatory system would work through tonic excitation that is increased by attention or surround stimulation and decreased by adaptation, or through tonic inhibition with the directions of change reversed.

Glutamate released into the synaptic cleft of hippocampal neurons can leak back to metabotropic glutamate receptors on the synaptic terminals, thereby inhibiting further release (4). Such negative feedback looks at first like a promising mechanism for adaptation, but if it were operative in Carandini and Ferster's experiment it would have decreased the modulated response and would only cause a persistent hyperpolarization if a high proportion of the neuron's tonic input were presynaptically inhibited; hence, it cannot explain adaptation at these neurons, but serves as a warning that adaptation in the cortex is a not a simple affair.

There are other adaptation experiments in the pipeline (5) showing that cortical neurons adapt and respond to the joint presentation of two patterned stimuli in a manner that cannot be accounted for by their adaptation to the stimuli presented separately, suggesting that a component of their adaptation is contingent on their joint presence during adaptation. The cerebral cortex, a part of the brain that first appeared in mammals and is largest in primates and humans, is thought to give its owner the power to adapt its behavioral responses in accordance with variations in enviromental circumstances. This in turn requires it to take account of the observable statistical contingencies and associations, for it is these that necessitate something beyond a stupid, invariant response. Neurophysiology has so far told us very little about how the cortex does this, so this ability to adapt to contingencies is particularly interesting, and the elucidation of adaptation mechanisms may ultimately give us insight into these broader problems of cortical function.

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**CLIMATE CHANGE**

# Are We Seeing Global Warming?

## K. Hasselmann

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The measured increase in global mean surface temperature since the last century is about 0.5°C. This value is consistent with the predictions of state-of-the-art climate models (see figure, top), but an order of magnitude smaller than the climate variations experienced year for year in any given region of the Earth (1). Regional climate fluctuations are largely due to shifts in air masses and tend to cancel when averaged over the globe or over a longer time period. Thus, attempts to detect anthropogenic global warming have focused on global scales and long-term trends. Despite considerable progress, the question of whether the observed gradual increase in global mean temperature over the

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last century is indeed caused by human activities or is simply an expression of natural climate variability on larger spatial and temporal scales remains a controversial issue.

To answer this question we need to (i) predict the anthropogenic climate change signal, (ii) determine the natural climate variability noise, and (iii) compute the signal-to-noise ratio and test whether the ratio exceeds some predefined statistical detection threshold. The last problem is the easiest one. It can be solved by generalizing standard signal analysis methods developed for

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