in a thin, brittle plate is much more complicated than what would be inferred from simple-minded two-dimensional theory.

How can all this be explained? The mathematics of the generation of acoustic waves by a moving crack can be said to be reasonably well understood, although the practical problem of actually computing the exact nature of such waves given a specific crack motion may be quite formidable. This problem is analogous to determining the exact nature of the electromagnetic waves radiated by a specific antenna array. The answer to the converse question, what is the response of a moving crack to a given acoustic wave, is quite unknown. In the electromagnetic case, the analogous question would be what is the response of, say, an electron to a radio wave, and the answer is well known. The problem in the case of the crack is that it is not really an object that can be separated from the medium in which it lies, as an electron is an entity that is separate from an electromagnetic wave. The crack tip is, rather, what is called a "singularity": a region within an elastic solid where stresses are greatly concentrated, much as electromagnetic fields are magnified at the tip of a lightning rod. At regions where stresses are so concentrated, Hooke's law, according to which the deformation of a solid is linearly proportional to the applied stress, ceases to be valid. This nonlinear behavior is a major complication, and it is at the root of the difficulties encountered by attempts at a theoretical understanding of what goes on with a moving crack in a Plexiglas plate. In electromagnetism, however, it is possible to understand the dynamics of such singularities (*8*), and borrowing ideas from that field may well prove helpful in understanding the dynamic behavior of singularities within an elastic solid.

The fracture of solids belongs, like turbulence in fluids, to that fascinating class of problems that are of common everyday occurrence, have major technological importance, and yet, their basic physics remains a mystery. The Boudet-Ciliberto experiment (*1*) has isolated a clean effect that significantly advances our understanding of dynamic fracture.

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NEUROBIOLOGY

Mapping the Sensory Mosaic

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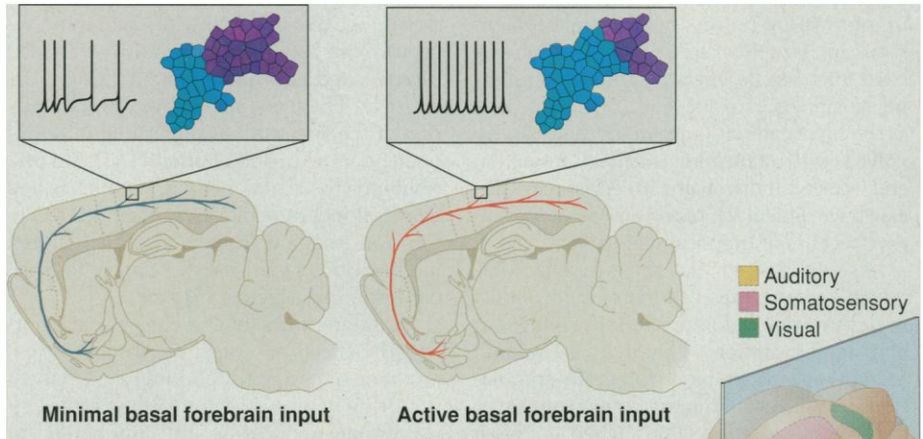
Neuroanatomy texts often illustrate the somatosensory cortex of the brain with a homunculus, a distorted human figure laying on the surface of the brain, each body part over the area of the cortex that responds when its skin is touched. In fact, the whole sensory cortex is a mosaic of such maps of sensory space. Once thought to be static, these maps are in fact dynamic structures (*1*). Changes in sensory input produced by an event as traumatic as the loss of a limb or something as routine as daily violin practice can cause long-term changes in these maps, reallocating a lost limb's cortical space to other body regions or devoting more cortical space to an often-used digit. Changes also can occur quickly, in minutes to hours, in response to inputs from other brain regions. In a report on page 1714 of this issue, Kilgard and Merzenich explore the modulation of auditory maps by input from the lower part of the forebrain and show that this input may be responsible for shaping much of the form of cortical maps, both long and short term (*1*).

The basal forebrain is a region of the brain most known for its participation in certain kinds of learning (*2*, *3*). But the projections from the basal forebrain also play a more global role in cortical processing. Their input signals the importance of sensory stimuli to the animal—enhancing the response to certain stimuli, diminishing responses to others. Alterations in the sensory maps of the cortex, often manifest as expansions or diminutions of specific representations, can be prevented by eliminating input from the basal forebrain. For example, after partial elimination of sensory input from the skin (such as a nerve lesion) in otherwise normal animals, the remaining intact regions of skin expand their representation in the contralateral somatosensory cortex. The expansion does not occur after a unilateral lesion of the basal forebrain, implicating this collection of nuclei in mediating the plastic changes (*4*).

Kilgard and Merzenich investigated how the projection from the basal forebrain might modulate the maps of the cortex. The authors electrically stimulated a nucleus in the basal forebrain [the nucleus basalis (NB)] of a rat while presenting a sound stimulus. They then compared the resulting map of the auditory cortex to maps from animals without NB stimulation. The map was dramatically altered by NB stimulation. An unexpected finding was that the changes were global, encompassing the entire cortical auditory map.



Basal forebrain input remakes maps. (**Left**) Without amplified input from the basal forebrain, neurons fire at a low rate and the representation of a theoretical sensory stimulus (blue) is small. (**Right**) With amplified basal forebrain input, neuronal firing is enhanced and more cortical sites are activated by the sensory stimulus (blue).

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They also found that modifications in the representation of different frequencies vary with the nature of the tone presented in conjunction with the basal forebrain stimulation. Presenting a temporally modulated tone or using two tones results in a different overall map than presenting a single-frequency tone.

Cortical maps can change quickly or slowly, but the mechanisms differ somewhat. Under the proper conditions, dramatic and large-scale alterations in the representation of a body part or visual image can shift within minutes or hours of changes in peripheral input (5). During these short-term changes, the balance of excitatory and inhibitory influences is presumably modified, and so-called "silent synapses" may be uncovered (6). Some changes are slower to develop. Several years after nerve damage, the corresponding cortical map of the body surface is different from the map 6 months or 1 year after the damage (7). The long-term changes probably require restructuring of axons and dendrites that specifically alters the density of cortical wiring (8), although modifications in the balance of excitation and inhibition probably also contribute.

The map changes reported by Kilgard and Merzenich were assessed after several weeks of stimulation and therefore could have resulted from restructuring and rewiring of the neocortex. Only 1 week of pairing the tone stimuli with NB stimulation also resulted in a substantial increase in the representation of the selected tone, but not of the magnitude found after 3 or 4 weeks of conditioning. Thus, map reordering likely occurs gradually, probably as a result of anatomical changes and changes in the balance of excitatory and inhibitory influences.

The input from the basal forebrain is mainly carried by neurons that use acetylcholine (ACh) as a neurotransmitter. The basal forebrain also allows the neocortex to emphasize behaviorally relevant stimuli (9, 10). One aspect of the basal forebrain's function in information processing has long been obvious in Alzheimer's disease, in which the basal forebrain degenerates, greatly diminishing its influence in older cortical regions, such as the hippocampus (a region of the brain involved with short-term memory formation and storage). Individuals with Alzheimer's disease have difficulty forming new memories, in part because of hippocampal damage.

As emphasized by Kilgard and Merzenich's results, presentation of behaviorally irrelevant stimuli do not result in changes in map representation. That is, tone stimuli presented without basal forebrain stimulation do not reorder auditory maps to favor the selected stimulus. Thus, when the basal forebrain functions poorly (as in Alzheimer's disease), the ability to emphasize behaviorally relevant stimuli is lost. The basal forebrain, therefore, is critical in determining which sensory representations are enhanced and strengthened.

Are the functional effects of basal forebrain stimulation mediated solely through the basal forebrain's cholinergic neurons? ACh certainly is a plausible candidate both to facilitate short-term attentional mechanisms and to promote long-term plasticity. After release, ACh can influence its target cells through two broad classes of receptors: muscarinic and nicotinic cholinergic receptors. It is likely that most of the cholinergic action in the cerebral cortex occurs through muscarinic receptors, although recent evidence suggests that nicotinic receptors may be involved as well, especially for inputs from the thalamus (11–13). The primary effect of ACh on cortical neurons seems to be facilitory in that it enhances the firing rate, although inhibition also occurs. At least some of the inhibitory effects may be due to a secondary effect through activation of γ-aminobutyric acid (GABA)–containing cells (11). ACh's action through specific muscarinic receptors, which are coupled to heterotrimeric guanosine triphosphate–binding proteins, leads to a voltage-dependent block of K^+ currents (11–14). These events result in a relatively long-lasting slow depolarization, which facilitates enhanced firing of pyramidal neurons (see figure). By modulating the train of action potentials after a stimulus in this way, ACh fine tunes the cortical responses so that the animal's appreciation of stimuli can be focused and enhanced (14–15). In addition, ACh sharpens stimulus processing in the neocortex by rapidly activating certain GABA-containing inhibitory interneurons (11, 16).

On a longer time scale, ACh can also facilitate neuronal activity. ACh action has been compared to the well-known mechanism of synaptic long-term potentiation, first described in the hippocampus, which causes enhancement of neuronal activity, usually at glutamatergic synapses (17). In addition, ACh's stimulation of muscarinic receptors can result in phosphoinositide (PI) turnover and subsequent protein kinase C activation. These events lead to phosphorylation of specific proteins implicated in development, growth, and plasticity (13) and possibly growth of new neuronal branches and connections. In addition, muscarinic receptors can regulate intracellular Ca^{2+} stores; mobilization of Ca^{2+} stores is crucial for various forms of plasticity (13).

A different line of evidence also suggests that the effects of projections from the basal forebrain are primarily cholinergic. Changes occurring in sensory regions of neocortex after either application of cholinergic or cholinomimetic drugs, or stimulation of the basal forebrain, can be pharmacologically blocked by cholinergic antagonists. Expansions of the representation of specific body parts can be prevented by delivery of cholinergic antagonists (9), and receptive-field plasticity induced by pairing basal forebrain stimulation with auditory tone stimuli can be blocked by the muscarinic antagonist atropine (18). Nevertheless, the NB contains cells that are not cholinergic but rather contain GABA or peptides. As a result, lesions or stimulation of the basal forebrain cannot unambiguously determine whether the cortical effects are cholinergic, and both the cholinergic and GABAergic neurons of the basal forebrain have been suggested to cooperate to sculpt the receptive-field properties of neurons in sensory cortex (17).

As elegantly demonstrated by Kilgard and Merzenich, the enhanced response to a behaviorally relevant sensory stimulus and resultant reordering of the cortical map can be accounted for by input from the basal forebrain. The singular properties of ACh make it a likely candidate to participate in this process. The precise mechanisms that remodel the cortical maps are not yet clear. The relative contributions of "silent synapses" and shifts in the balance of excitation and inhibition versus the remodeling and new growth of cortical structure remain to be determined. Both may occur and their individual contributions may evolve over time.

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