of AD, there is plenty left to do. Besides *S182*, genes that cause other autosomal dominant forms of AD await identification. How each of these (as well as *ε4* of apolipoprotein E) triggers or modifies the relatively stereotyped cascade of AD pathology will need to be determined. Environmental factors that influence the course of the disease also remain to be clarified. Most importantly, small molecules that block one or another step in AD pathogenesis must be developed. Our patients, and science, will be the beneficiaries. □

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NEUROBIOLOGY

How cortex reorganizes

Jon H. Kaas

MOST of us used to be convinced that sensory areas of mature cortex in the brains of adult mammals are highly stable in their organization. This idea was consistent with evidence that visual cortex is altered by certain experiences only during a limited period of development (see ref. 1, for example). Moreover, it was presumed that processing circuits must be stable in order to perform reliably. But this assumption has been overturned by a host of experiments showing that cortical maps of receptor surfaces can reorganize after partial deactivation and after altered patterns of activation[2,3]. Deactivation may involve damage to sensory nerves from the skin, small retinal lesions, or selective loss of hair cells of the cochlea. Regions of cortex thereby deprived of their usual source of activation typically become responsive to the remaining sensory inputs. Changes in sensory use also alter cortex, but usually less dramatically. Now on page 780 of this issue, Das and Gilbert[4] look into just how cortex reorganizes after restricted retinal lesions in cats.

Cortical reorganization could depend on the potentiation of any of several types of existing but normally ineffective neural connections, the growth of new connections, or both. Das and Gilbert[4] propose that cortical reorganization after retinal lesion is mediated by the potentiation of a highly organized system of long-range horizontal connections in visual cortex which seem to have significant, but largely subthreshold, neuronal effects in normal animals[5]. After retinal lesions remove a competing source of activation, these subthreshold influences gradually become effective in activating neurons to suprathreshold levels. This proposal is supported by a detailed comparison of the horizontal or surface-view extent of cortex activated at suprathreshold or at suprathreshold plus subthreshold levels by a minimal visual stimulus, the restricted movement of a very small bar in the visual field.

As expected, the extent of suprathreshold activity, the spiking of neurons, measured conventionally with an array of microelectrode penetrations, was quite limited. A much larger surface area of cortex showed enhanced metabolic activity in response to the same stimulus when measured with optical imaging. As all neural events, both threshold and subthreshold, require energy, the larger region of responsive cortex measured with optical imaging includes a core region of spiking neurons and a surrounding fringe of neurons with subthreshold activity. The extent and topographic features of the fringe suggest that it depends on horizontal cortical connections. Months after binocular retinal lesions, measurements by both procedures showed that the subthreshold fringe can be driven to suprathreshold levels. Thus, horizontal connections may provide a large zone of subthreshold activity that can become suprathreshold after the deactivation of competing inputs.

Interestingly, an early and now largely abandoned procedure, that of recording evoked slow potentials with electrodes on the brain surface, may have provided comparable evidence. Surface recordings were used to reveal cortical organization during the 1940s to 1960s, but were re-

New look for old reptiles

Two historic collections of fossil reptiles return to public view this month after three-year absences. This specimen of *Triceratops* is a star turn in the revamped dinosaur galleries at the American Museum of Natural History in New York. Many familiar specimens, such as *Apatosaurus* and *Tyrannosaurus*, are displayed in new-look, dynamic poses, and the overall effect is stunning.

The renovation is part of the AMNH's continuing redesign of its fossil vertebrate galleries that started last May with 'Mammals and their Extinct Relatives', and will go on until 1996, when the hall of vertebrate origins opens. A visitor with enough time and stamina will then be able to do a circuit of the museum's fourth floor, covering the scope of vertebrate evolution. Like the mammals gallery, the dinosaur displays are arranged along systematic rather than chronological lines, reflecting the museum's research strengths. This is far less daunting than it sounds: technological aids such as interactive CD-ROMs help visitors pick their way through the taxonomic jungle (and children, of course, take to both technology and technicality like *Anatotitan* to water).

IMAGE UNAVAILABLE FOR COPYRIGHT REASONS

American Museum of Natural History

Another lengthy conservation effort, this time at the Natural History Museum in London, has also restored a spectacular display to public view. The NHM's unrivalled collection of Lower Jurassic marine reptiles is now back on show, the 112 mounted fossil skeletons lining the walls of a 60-metre gallery. Some of the specimens were found at Lyme Regis, Dorset, by the famous collector Mary Anning in the 1830s. Time and heavy-handed Victorian museum techniques had taken their toll, but after extensive cleaning and restoration, the NHM's ichthyosaurs and plesiosaurs have been returned to that pristine, just-excavated state. Henry Gee