

saturated, oxygen consumption was normal, but as soon as NO exceeded the capacity of the sink, respiration was inhibited.

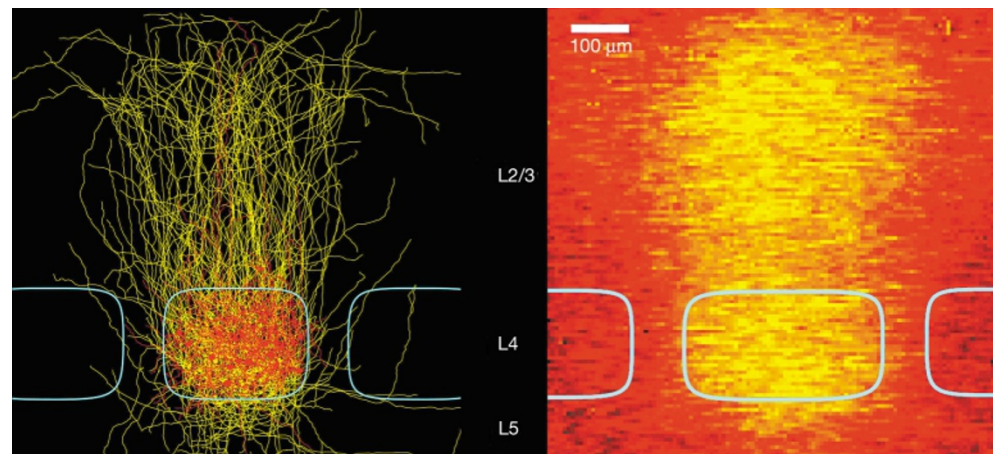
So cells can shape the levels of NO by means of a sink, the identity of which now needs to be established. Although this sink might have physiological and pathological relevance, the relationship between the NO concentrations measured in this study and those found *in vivo* is uncertain. It will therefore be necessary to embark on studies using more physiologically relevant systems to determine the actual role of this enigmatic sink in brain function.

Juan Carlos López

#### References and links

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**FURTHER READING** Thomas, D. D. *et al.* The biological lifetime of nitric oxide: implications for the perivascular dynamics of NO and O<sub>2</sub>. *Proc. Natl Acad. Sci. USA* **98**, 355–360 (2001)



Left: the anatomically defined barrel column, delineated by the normalized barrel boundaries (cyan), dendrites (red) and axons (yellow) of the excitatory neurons of layer 4. Right: the spatial extent of excitation at the peak of the functional response evoked by stimulating the barrel, imaged using a voltage-sensitive dye. Courtesy of Carl Petersen, Max-Planck-Institute for Medical Research, Germany; © 2001 Society for Neuroscience.

#### CORTICAL IMAGING

## Roll out the barrel

In rodents, each facial whisker is represented in layer 4 of the somatosensory cortex by a cylindrical array of neurons known as a barrel. The topographical organization of the barrels corresponds precisely to that of the whiskers, so the barrel cortex has proved to be extremely valuable for studying cortical responses to peripheral stimulation. Now, as reported in the *Journal of Neuroscience*, Petersen and Sakmann have devised an elegant *in vitro* system to investigate the functional anatomy of the barrel cortex.

Their experiments were carried out in slices of rat cortex, using a voltage-sensitive dye (vsd) to detect neuronal excitation. Following stimulation of a single barrel, the authors showed that the vsd response was initially confined to the stimulated barrel, but quickly spread vertically to an adjacent region of layer 2/3, of approximately the same width as the barrel. The signal decayed in the barrel and layer 2/3 simultaneously, and had disappeared entirely by 200 ms after stimulation.

Interestingly, *in vivo* studies have shown that stimulation of a single whisker activates an area of cortex much larger than the width of a barrel. What could account for this difference? A clue came from experiments in which Petersen and Sakmann treated their slices with bicuculline to inactivate GABA ( $\gamma$ -aminobutyric acid)-mediated inhibition. This treatment increased the lateral spreading of the vsd response in layer 2/3, whereas spreading in layer 4 was minimal and never extended into the adjacent barrel. So, GABA-mediated inhibition might limit the spread of activity in layer 2/3, but spread between barrels is more likely to be constrained by a lack of excitatory connections. The authors suggest that the reduced spread of activity in layer 2/3 in slices

might reflect differences in the balance between inhibition and excitation *in vivo* versus *in vitro*.

To replicate the type of barrel activity observed when a rat is actively exploring, the authors stimulated a single barrel continuously at the frequency of normal whisker movement (10 Hz), or stimulated two adjacent barrels simultaneously. Although the amplitude of the vsd response decreased with repeated stimulation, the spatial pattern of activity was unchanged. Simultaneous stimulation of adjacent barrels did not change the extent of the response in the individual barrels, and caused only a slight increase in the spread of activity in layer 2/3. This indicates that the barrels function largely independently of one another.

In a slightly more complex protocol, Petersen and Sakmann paired the stimulation of a barrel and a region of layer 2/3 adjoining an adjacent barrel. After repeated paired stimulation, the area of layer 2/3 that was excited by stimulating the barrel alone was shown to be increased. The authors suggest that such pairing protocols could provide models for cortical plasticity that results from alterations in sensory input.

Petersen and Sakmann's system certainly holds a great deal of promise for modelling cortical activity *in vitro*. By designing different stimulation protocols, it might be possible to replicate a wide variety of manipulations of sensory input that have already been observed *in vivo*.

Heather Wood

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**ORIGINAL RESEARCH PAPER** Petersen, C. C. H. & Sakmann, B. Functionally independent columns of rat somatosensory barrel cortex revealed with voltage-sensitive dye imaging. *J. Neurosci.* **21**, 8435–8446 (2001)

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