clearly shows the presence of a microbially derived organic-rich template for iron oxide formation. The clever use of carbon K-edge XANES analysis indicates a strong similarity between the spectra of synthetic acidic polysaccharides and the natural mineralized filaments.

The authors were also able to reproduce the formation of iron-rich filaments and elongated akaganeite crystals under laboratory conditions. Scanning transmission x-ray microscopy (STXM) and spectral analyses confirmed the presence of mineralized filaments rich in iron and carbon in an alginate solution mixed with ferric iron. Additional syntheses with a mixture of polysaccharides rich in carboxylic groups revealed the presence of elongated akaganeite crystals with smaller aspect ratios than those found in the natural sample. The combination of high-quality HRTEM images and XANES analysis provides compelling evidence that organic exopolymers secreted by bacterial cells can indeed template the crystallization of iron oxides. As stated by the authors, the use of high spatial resolution tools and powerful mineralogical analysis to characterize samples containing mineralized organic structures should improve our understanding of biomineralization mechanisms.

Chan *et al*. also propose a novel mechanism for the formation of crystals on bacterial exopolymers. They hypothesize that the oxidation of ferrous iron by iron-oxidizing bacteria increases the pH gradient across the cell membrane. The generation of protons near the cell wall is then thought to enhance the proton motive force and thus increase the energy-generating potential of the cell. This interesting hypothesis raises the possibility that bacteria do profit from encrusting themselves with various minerals (especially precipitation reactions leading to a pH gradient) because it allows them not only to survive, but also to gain useful energy in sometimes hostile or extreme environments.

Finally, a better understanding of the mechanisms leading to crystal nucleation on organic templates in natural environments and better characterization of such minerals might allow us to identify specific characteristics unique to biogenic minerals. Such characteristics could then become very helpful in the search for biosignatures in ancient environments on Earth and other planets. This is especially important for the NASA astrobiology program, which aims to learn how to recognize signatures of life on other worlds (*11*). The present and upcoming missions to Mars have already generated a lot of interest among the general public and in the scientific community. With more research such as that of Chan *et al*. (*10*), we may soon be able to ascertain whether life existed or still exists on other worlds. But with the lessons learned from the famous Martian meteorite (*12*), we must keep in mind that it is difficult to differentiate between biotic and abiotic mechanisms. With care, it should be possible to distinguish one from the other and apply our newly gained knowledge to samples returned from Mars or other planets.

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NEUROSCIENCE

Blocking Plasticity in the Visual Cortex

David Ferster

Few scientific questions strike as close to our hearts as the debate over nature versus nurture. Are human behavior and personality shaped by genes or by early experience? There is strong evidence that they are shaped by both. Single genetic mutations can profoundly affect complex behaviors such as sleep-wake cycles (*1*), parental care (*2*), and memory (*3*). Yet early experience can permanently alter adult behavior through its effect on development. In Harlow's classic experiments, for example, monkeys raised with inanimate objects as surrogate mothers suffered from social deficits later in life (*4*). One of the central pursuits of neuroscience, therefore, is to uncover the cellular and molecular mechanisms by which early experience alters brain development. Yet we have little idea where "mothering" and "social interaction" are generated in the brain, let alone how one might influence the other. To study the effects of early experience, neuroscientists have turned to model systems in which early experience is easy to manipulate and the resulting neural changes are easy to measure. The most intensely studied model of environmentally driven neural plasticity is that of ocular dominance columns in the developing mammalian visual cortex (*5*). In this issue, two studies—by Hensch and Stryker on page 1678 (*6*) and by Fagiolini *et al*. on page 1681 (*7*)—provide major insights into the activity-driven adaptation of the mammalian visual cortex to incoming visual stimuli during early postnatal life.

In the adult brain of many mammalian species, synaptic inputs from thalamic relay cells driven by one eye are clustered in the visual cortex into nearly parallel, stripelike regions or columns ~0.5 mm wide, which alternate with columns dominated by the other eye. At birth, however, thalamic inputs to the visual cortex from the two eyes start out completely overlapping with each other and segregate themselves into stripelike ocular dominance bands over the course of the next several weeks. This segregation process is exquisitely sensitive to visual experience: When vision in one eye is degraded during development (by surgically closing that eye), the ocular dominance bands for that eye narrow, and those for the normal eye broaden, so that the normal eye ends up with a much larger fraction of the cortical area. These changes become irreversible with the end of the early critical period (which, in kittens, is about 12 weeks after birth). Eye closure after the critical period has no effect on column width.

What drives apart the initially overlapping thalamic inputs from the two eyes, and what determines the pattern of segregation? Why stripes and not checkerboards? Why 0.5 mm? One possibility is that the inputs are guided by unknown molecular cues that differentially attract inputs from the two eyes. These cues could be arranged in preexisting stripelike patterns within the visual cortex (*8*), and

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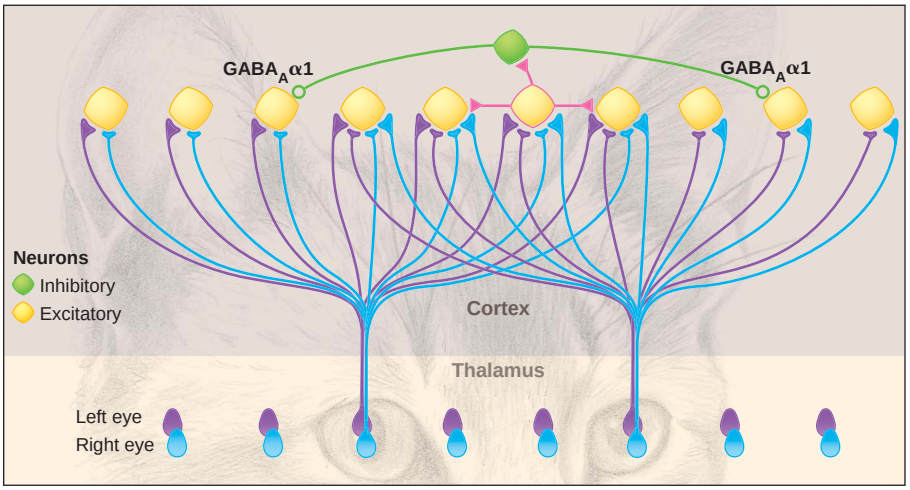
could explain how inputs from the two eyes start to segregate prior to the period when manipulations of visual experience alter the segregation process (*8*). Alternative models of segregation propose local interactions between the individual afferent axons of thalamic relay cells and their neuronal targets in the visual cortex. One model, for example, relies on Hebbian rules, a winner-take-all competition in which synaptic inputs that most strongly activate a cortical neuron are strengthened at the expense of weaker ones (*9*) (see the figure). The strong get stronger and the weak get weaker. Through this process, even small, random imbalances in the overlapping input from the two eyes get amplified to the point that the weaker eye's input fades entirely and each cortical cell's input becomes predominantly monocular. The effects of eye closure are now easily explained: The closed eye's inputs are reduced in activity and can no longer compete effectively with their counterparts from the other eye.



Hebb rules in the visual cortex. Model of neuronal connections in the developing kitten visual cortex (*9*). At birth, thalamic neurons from the two eyes project their afferent axons (blue, purple) to completely overlapping regions of the visual cortex. Activity in thalamic neurons driven by the same eye is correlated; activity in neurons driven by opposite eyes is not correlated. Short-range excitatory connections within the visual cortex (pink triangular synapses) increase correlation in activity among neighboring cortical neurons (yellow). Long-range inhibitory connections (small green circular synapses) with GABAergic interneurons (green) decrease correlations among cortical neurons that are farther apart from one another. The scale of these inhibitory connections is critical for determining the width of fully developed ocular dominance columns.

These rules might explain why single neurons in the adult visual cortex receive strong monocular inputs, but not why segregation leads to stripes instead of random intermixing of left and right eye–dominated neurons. For stripes to form, three additional rules seem to be required: (i) Activity in nearby inputs from the same eye must be correlated with each other (but not with those from the other eye) so that they can cooperate with one another in their bid to take over cortical territory from the other eye. This correlation can occur even in the absence of vision and may be intrinsic to the thalamic relay nucleus itself. (ii) Activity in nearby cortical cells must be correlated, presumably through mutual excitatory connections. Together, the correlation in nearby inputs and in neighboring cortical cells implies that clustered groups of cells get taken over by groups of nearby afferent axons. (iii) Activity in more distant cells separated by 0.5 mm or so must be anticorrelated with each other, so that when a set of inputs from one eye take over one group of cortical cells, they will be discouraged from simultaneously claiming the cells 0.5 mm away. In this model, anticorrelations are introduced by lateral inhibitory connections formed by GABAergic interneurons. With the right parameters, the model predicts ocular dominance columns of just the right size and shape, and demonstrates the correct response to simulated eye closure. So global patterns of ocular dominance can self-organize based on simple, local rules: Strong inputs get stronger; nearby inputs are correlated; nearby cells excite one another; and more distant cells inhibit one another.

As elegant as the model is, testing it has proven difficult. Disrupting the inhibitory activity of the neurotransmitter GABA$_A$ during development disrupts the segregation process (*10*). But such treatment also has profound effects on cortical activity, which confounds interpretation of the results. In their new study, Hensch and Stryker (*6*) have accomplished a far more subtle and compelling manipulation of the process. By modulating, rather than completely supplanting, the intrinsic GABA-mediated inhibitory activity in the developing kitten visual cortex, they have induced changes in the width of the ocular dominance bands without dramatically altering cortical function. Potentiating inhibition with diazepam broadened cortical columns, whereas reducing inhibition with the benzodiazepine inverse agonist, DMCM, narrowed them. These systematic changes in width—nearly 30% in the case of diazepam—far exceeded the natural variability in column widths, and yet the drugs induced few changes in the physiology of cortical neurons. Orientation selectivity, orientation maps, and spontaneous activity of the visual cortex all remained normal. Potentiating inhibition with diazepam also sharpened the segregation process in that many more cortical neurons than normal were completely dominated by one eye or the other. Fewer neurons than normal received mixed input. This enhanced segregation is in complete agreement with the model, which predicts that enhancing inhibition would further decrease correlations in activity among more distant neurons, which in turn would facilitate the segregation process.

As specific as the effects of benzodiazepines are, these drugs are still blunt instruments when applied to the complex inhibitory circuitry of the visual cortex. There are dozens of different kinds of inhibitory interneurons, and there are many different subtypes of GABA receptors present in the visual cortex. To identify which interneurons and which GABA receptors were responsible for the diazepam effects on eye-specific segregation, Fagiolini *et al.* (*7*) turned to the mouse visual cortex. As in cats, monkeys, and humans, there is a critical period in mouse development during which eye closure can bias the outcome in favor of the open eye. Although sensitivity to eye closure normally begins at 3 weeks after birth in the mouse, it can be accelerated to start at a much earlier stage, again by enhancing inhibition through the application of diazepam. By showing that the selective benzodiazepine agonist, zolpidem, also accelerates the critical period, the authors narrowed the list of possible GABA$_A$ receptor subtypes responsible for the effects to those containing the α1, α2, or α3 subunits (the α4 and α6 subunits are generally benzodiazepine insensitive, and α5 is zolpidem insensitive). To further narrow the possibilities, the authors tested knockin mice in which the α1, α2, or α3 subunits had been selectively rendered insensitive to benzodi-

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azepines (but still functioned normally). Only in mice with the α1 mutation did diazepam fail to accelerate ocular dominance plasticity. In addition, these mice showed proper segregation and normal sensitivity to eye closure during the natural critical period starting at 3 weeks. Nor did the mutation have noticeable effects on neuronal responses to visual stimuli, or on the normal distribution of α1-labeled inhibitory synapses.

Somatic GABA receptors containing the α1 subunit are found preferentially at synapses formed by large basket cells, one particular subtype of inhibitory interneuron. Basket cells stain positively for parvalbumin and their axonal arbors spread widely. These cells are electrically coupled with one another (*11*) in a way that could further increase their ability to decorrelate activity over wide areas of the cortex, allowing them to provide the spatially patterned inhibition required by the developmental model. Finally, disrupting the activity of the parvalbumin-positive interneurons (by knocking out a potassium channel specific to these neurons) attenuates the plasticity that occurs during the normal critical period (*12*).

Together, the studies by Hench and Stryker (*6*) and Fagiolini *et al*. (*7*) give a remarkably detailed picture of the events that occur during the segregation of eye-specific inputs to the visual cortex. The cortex itself, through one specific subtype of GABAergic inhibitory interneuron, plays a central role in organizing its activity into patterns that guide the incoming thalamic afferent axons so that they become segregated into their allotted areas. Whether and to what extent these mechanisms operate in other parts of the brain, and how they interact with developmental processes that are less susceptible to environmental manipulation, remain to be explored. But they will surely be the foundation for much new work. Ultimately, we may indeed learn not only how genes shape behavior directly, but also how the environment shapes behavior through its effect on gene expression.

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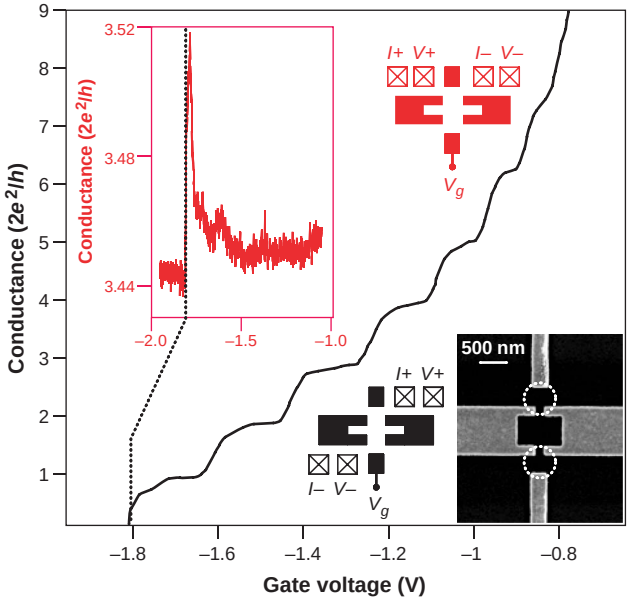
PHYSICS

Electron Spin Polarization in Nanoscale Constrictions

Jonathan P. Bird and Yuichi Ochiai

Do electrons in a nonmagnetic semiconductor spontaneously arrange themselves so that their spins all point in the same direction, forming a magnetic state, when confined within a nanoscale constriction? This question has been attracting increasing interest in recent years, and may have important implications for the development of future devices that depend on electron spin. A growing body of experimental data suggests that spontaneous spin polarization is indeed occurring.

The origins of this question may be traced to the discovery (*1*, *2*) that the conductance of quantum point contacts is quantized at low temperatures, in integer multiples of $2e^2/h$ (where e is the electron charge and h is Planck's constant). A quantum point contact is a constriction, whose dimensions are comparable to the electron wavelength, fabricated from an extremely pure semiconductor. Current through such a constriction is carried by means of one-dimensional modes, which are analogous to the quantized energy levels of a microscopic particle confined inside a one-dimensional box. The constriction is created by applying a depletion bias voltage to lithographically defined metal gates, formed on the surface of the semiconductor. An increase in this bias steadily reduces the width of the constriction and, thus, the number of modes that contribute to current flow. A simple analysis (based on a single-particle picture of electron transport) shows that each mode has a conductance $2e^2/h$, so that, as the number of modes is reduced one at a time, the conductance of the constriction exhibits a set of plateaus at decreasing integer multiples of this fundamental unit (see the figure).



Detection of the spin-polarized state? In studies of spin polarization, quantum point contacts (circled with dotted lines in the lower right inset) are connected by a quantum dot. The measured conductance through the upper contact (red curve) shows a resonant peak that is correlated to the lower contact becoming nonconducting (black curve) (*9*).

According to this simple picture, the last plateau in the conductance should occur at $2e^2/h$, after which it should decrease quickly to zero as the constriction narrows and closes off the current. In many experiments, however, an additional plateau is observed near $0.7 \times 2e^2/h$ (*3*, *4*), and much interest has focused on whether this represents the signature of a spin-polarized electron state. Conductance quantization in units of $2e^2/h$ arises because, at zero magnetic field, each one-dimensional mode is typically spin degenerate, meaning that it actually consists of two "parallel" modes. By

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