

size of which diminishes as a function of increasing pressure because of atomic compaction. This leads to the drastic change of water solubility in pyroxene at pressures between 3 and 5 GPa, corresponding to depths of 100 to 175 km. Depending on the tectonic environment and the temperature, the minimum in solubility is shallow in the case of the oceanic mantle but deepens in the case of the colder continental mantle.

Still, what are the physical and chemical changes that create the deceleration of seismic waves and the mechanical distinction between brittle lithosphere and ductile asthenosphere? Early models have invoked the presence of molten silicate to explain the reduced shear-wave velocity because these waves do not travel through liquids (1). This explanation was abandoned for two reasons: (i) To have an effect on wave propagation, the melt should form films lapping the boundaries between the solid grains, which is not the case; and (ii) the presence of excessive amounts of melts is required to effectively reduce the viscosity of the asthenosphere. The most accepted expla-

nation of mineral physicists has thus been that the asthenosphere is weak because it is hydrous and not because it is partially molten. Because the melt has an affinity for water that is 1000 times that of the minerals, melting does not occur in the asthenosphere as it would dehydrate the mantle (7, 8).

The consequence of this drying out would be that minerals become stiffer and deform less in response to convective flow. Such a model implies that the lithosphere-asthenosphere boundary limits the storage capacity of the mantle; this model would be in agreement with the interpretation of the electrical conductivity of the mantle. The oceanic mantle where the asthenosphere is shallower is more conductive than the adjacent continental lithosphere, which is deeper and less conductive (9).

Mierdel *et al.* conclude that melting must occur in the asthenosphere because their minimum in water storage capacity coincides with the depth of the low-velocity zone. This is an additional argument to constrain the water content in Earth's mantle, and yields a minimum of 0.07% weight occurring around 100 km in the

oceanic mantle. These conclusions agree with recent observations of the melting depth of a hydrous mantle (10) and further imply that melting starts deeper, in the garnet stability field. Thus, these findings show how mantle properties such as viscosity, melting, and differentiation are tied to its water storage capacity.

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NEUROSCIENCE

A Push-Me Pull-You Neural Design

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Networks of neurons producing behavioral outputs can be both excitatory and inhibitory—that is, they can activate or block the activity of their target neurons. The important interplay between these two opposing effects in the mammalian central nervous system has been recognized for at least 100 years, ever since Nobel laureate Sir Charles Sherrington investigated leg reflexes controlled by spinal cord motor neurons in cats (1). We now know that there is continual, intermixed chattering of inhibition and excitation in many parts of the mammalian brain, including the cerebral cortex (2, 3), that maintains a balance between responsiveness and stability (4–6). On page 390 of this issue (7), Berg *et al.* show the very surprising result that the rhythmic input onto motor neurons during scratching behavior in turtles excites and inhibits networks of motor neurons not alternately, as expected, but in phase. This synchronization of simultaneous excitation and inhibition—pushing and pulling at the same

time—appears to be counterproductive and wasteful. What possible function might it serve? To understand the possible importance requires a brief excursion into some other functions of neural inhibition.

Sir John Eccles and colleagues recorded the first inhibitory synaptic potential, in a cat spinal motor neuron, in 1952 (8). Since then, inhibition—a block to signal transmission through a neuron—has been found all over the central nervous system. For example, stimulating input from neurons to the mammalian cortex produces an initial direct excitation of target neurons followed quickly by strong indirect inhibition (9). The auditory systems of many animals have taken advantage of the relative timing difference of the excitation and inhibition elicited by neural signaling pathways from the two ears to determine the location of sound in space (10).

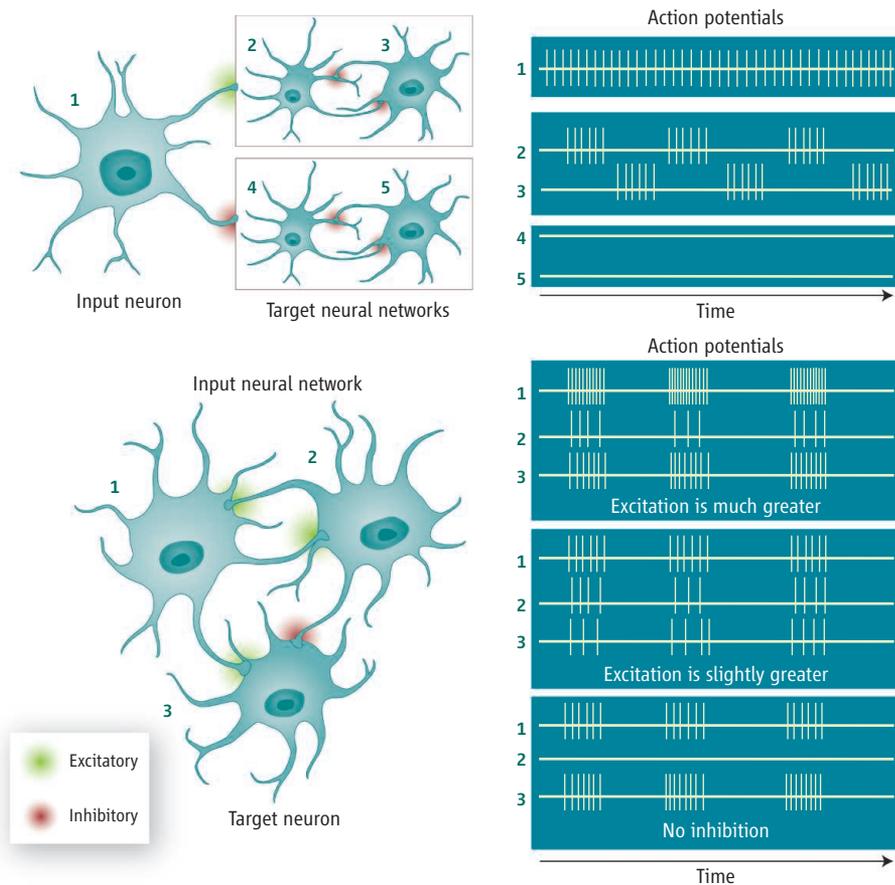
In a different network arrangement, a single source of neuronal activity can excite one population of neurons and inhibit another (see the figure) to activate one behavior and turn off others. The nature of the connection pattern found in the turtle scratching reflex circuit, which Berg *et al.* investigated, differs

How do spinal neurons control patterned motor output? A balance in simultaneous excitation and inhibition of neuronal circuits produces rhythmic activity that drives motor behavior.

from this behavioral choice connectivity pattern in that the excitation and inhibition onto a neuronal target overlap completely in both space and time (see the figure). In this configuration, the target neuronal network is effectively a comparator: It is active only when the input carried by the excitatory pathway is greater than that from the inhibitory one, and the magnitude, and even the qualitative nature, of its response will vary with the magnitude of the difference.

Berg *et al.* show that the input onto turtle motor neurons during the scratching reflex is oscillatory (from an input neural network that is a central pattern generator for scratching), with excitation being somewhat stronger than inhibition. In this mode, the target spinal motor neurons produce bursts of impulses (spikes) in phase with the synchronous excitatory and inhibitory bursts. At first glance, there would be no practical merit to having nearly equal input from excitatory and inhibitory inputs. The resultant activity of the target neuronal network would be the same if its total input were entirely from a much weaker excitatory input (see the figure). A little reflection shows that this is not accurate.

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Neural circuit designs. In simulated recordings of neuron activity, time runs left to right and the vertical blips represent action potentials (spikes) within the bursts of activity in the individual neurons. (**Top**) If a single input excites one neural network and inhibits another, it can turn one target network (pattern generator) on, resulting in alternate bursts of activity, and turn the other off. (**Bottom**) If there is excitatory and inhibitory input from a rhythmic pattern-generating circuit (shown is a circuit of two neurons) onto a single target neuron, such as a motor neuron, the magnitude of the target's response depends on the relative strengths of the inputs. When excitation is much stronger than inhibition, the same input from the neural circuit produces more spikes per burst. When the excitation is slightly stronger, the target's response is diminished. When there is no inhibition at all, the target's response increases—the same amount of excitatory input can produce a large output in the target neuron.

Both signaling pathways increase the target neuron's electrical conductance (i.e., it becomes leakier to electric current), and it is therefore in a very different state when excitatory and inhibitory inputs are both active than it would be if the input were exclusively a weaker excitation.

Because the conduction of action potentials along axons and the consequent chemical synaptic transmission are expensive energetically (11), there must be a strong benefit gained from simultaneous stimulation by inhibitory and excitatory sources. The reason proposed by Berg *et al.* is to randomize the timing of the spikes produced by different motor neurons to provide the muscles with a smoother excitation, and thus to produce smoother reflex responses. The authors provide several pieces of data in support of this hypothesis. For instance, they show that the

intervals between spikes become more regular when inhibition to the motor neurons is blocked pharmacologically.

Although convincing, this mechanism seems to be a long walk for a short drink. One can imagine a number of mechanisms with no involvement of inhibition that would do equally well at smoothing the motor response, including slow decay times in the motor neuronal membrane, asynchrony in the excitatory inputs to the motor neurons, or variability in synaptic transmission. In fact, nervous systems are usually faced with just the opposite problem: how to maintain timing accurately in the face of variability in all the components of a complicated neuronal circuit. Ultimately, the decoder of the motor neuron activity—the contraction of muscles—may not be affected very much by such subtleties as the degree of regularity in the motor neuronal spike bursts.

It may be that competing excitation and inhibition in a network are used for more interactive functions. Studies of seizures emphasize the need for inhibition to avoid the system's "blowing up," because there is a great tendency for neural systems with too much excitation to go into a maximal activity state (4, 6). In general, neuronal circuits with both excitation and inhibition are better able to control their level of activity because they have a broader dynamic range (5). They can thus change their gain more effectively than can purely excitatory networks. One consequence of this broader range of control may be that sensory input from a source that is external to the circuit, or parallel input from other behavioral circuits (onto either the excitatory or inhibitory pathways that impinge on the target motor neuron), can be integrated more smoothly into the ongoing activity of the primary circuit. These additional possibilities, however, do not detract from the one model proposed by Berg *et al.* In fact, a major strength of the turtle scratch reflex system is that the whole system—from sensory input through the central pattern generator to motor neuron output (12)—is much more tractable than is cortical brain activity, for instance. The turtle system, or other spinal cord locomotory networks, should provide a good test bed for approaching the many possible reasons for having conjoint inhibition and excitation.

The findings of Berg *et al.* are jarring because they provide clear evidence for an unlikely cellular system architecture in a completely unexpected place. This elicits both practical thoughts (Is there something like this in other neural systems?) and theoretical ones (What else can such a mechanism do?), sure signs of a particularly interesting piece of work.

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