Brains show signs of two bilingual roads

Unlike people who become bilingual after childhood, those who learn a second language at an early age rely on the same critical patch of brain tissue when speaking either tongue, according to a new study.

Adult learners of language apparently recruit nearby groups of brain cells, suggest neuroscientist Joy Hirsch of Memorial Sloan-Kettering Cancer Center in New York and her colleagues.

"On the basis of our findings, the distinction between native and second languages may be less for [people who had] younger ages of exposure to a second language," Hirsch holds.

According to her study, bilingual individuals who acquired a second tongue during childhood display elevated activity in the same part of Broca's area—a frontal lobe structure considered crucial for language use—regardless of which language they use. In contrast, people employing a second language acquired later exhibit neuronal bustle in another segment of Broca's area, the researchers report in the July 12 NATURE.

Wernicke's area, located in the temporal lobe and also known to perform language functions, displayed comparable responses in both groups.

The researchers relied on a noninvasive technique known as functional magnetic resonance imaging (fMRI) to study changes in blood flow in the brains of 12 bilingual adults. Half of the group had learned a second language starting in infancy, while the remainder attained fluency as teenagers.

Together, the volunteers speak 10 native and second languages, including English, French, and Turkish. The two groups reported roughly equal fluency and frequency of use for their second tongues.

Researchers obtained brain scans as participants silently recited, first in one language and then the other, brief descriptions of an event from the previous day.

The findings may reflect either the sensitivity of part of Broca's area to language exposure during childhood or the existence of marked differences in the ways that children and adults learn languages, Hirsch says.

"These new results are interesting but inconclusive," comments neuroscientist Robert J. Zatorre of the Montreal Neurological Hospital. "It's devilishly difficult to study naturalistic types of language in a well-controlled way."

For instance, the short descriptions of personal events offered by volunteers in Hirsch's study allow for large individual differences in the amount of mental imagery generated during the task and the extent to which events sparked emotional reactions. Such differences may have influenced language-related brain activity, Zatorre contends.

Unpublished fMRI data obtained from bilingual speakers as they name various objects, a more restricted verbal task, yields the same disparity regarding age of learning a language, Hirsch responds. —*B. Bower*

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Brain images and expanded views of elevated activity in Broca's area (boxes) show a large area (mustard) shared by the two languages of an early bilingual person (top) and two adjacent areas (red and yellow) in a person who learned a second language as an adult.

Thieving bacteria use hot goods in hideout

Disease-causing microbes have achieved notoriety for their resourcefulness, often borrowing a molecule from the afflicted host organism to aid infection. The bacterium that produces Lyme disease takes this stratagem a step further. It steals a mammalian host's molecule and uses it to travel within an insect.

The Lyme disease bacterium, *Borrelia burgdorferi,* spends part of its life cycle in a tick, which then passes the microbe into a mammal. There, the invaders spread through the body, causing flulike symptoms and sometimes more serious illness.

In the past few years, several research groups have shown that *B. burgdorferi* binds to and activates a mammalian enzyme called plasminogen. In test-tube studies, this enzyme helped the bacterium cross a layer of human cells. These results suggested that *B. burgdorferi* uses plasminogen to spread within the mammalian host.

A team of microbiologists tested this idea using a genetically engineered mouse that does not make plasminogen. The researchers discovered that, rather than playing a major role in bacterial dissemination within the mouse, the enzyme helps the bacteria break down barriers inside the tick. Jorge L. Benach of the State University of New York at Stony Brook and his colleagues report their findings in the June 27 CELL.

"This is really a landmark paper," says Justin D. Radolf, an infectious disease physician at the University of Texas Southwestern Medical Center at Dallas. "Before this, several groups had shown that these bacteria can acquire plasminogen on their surface, but no one had come up with a way to examine how it fits into the bacterial life cycle."

To infect mammals, the bacteria must travel from a tick's gut, where they live most of the time, to the tick's salivary glands. This journey involves burrowing into the fluid-filled cavity that surrounds the gut, swimming through the fluid, and crossing another layer of tissue into the salivary glands. There the bacteria mix with saliva, which the tick spits into its mammalian host while feeding. Traversing these barriers requires an appropriate tool.

The bacteria acquire such a tool—plasminogen—when the tick gorges itself on the mammal's blood. Benach's team showed that if the tick feasts on plasminogen-deficient mice, most of the *B. burgdorferi* remain stuck in the insect's gut. Thirty times more bacteria can make it all the way to the salivary glands when the tick has fed on normal mice.

"Having plasminogen greatly facilitates the dissemination of *Borrelia* through the tick," says Benach. "All of the activity for migration is contributed by the mammalian host."

Somewhat surprisingly, the researchers found that the bacteria do not require mammalian plasminogen to spread within mice. When they inoculated mice with lab-raised *B. burgdorferi* not previously exposed to plasminogen, the bacteria invaded several organs, regardless of whether the mice made the enzyme. Moreover, when two plasminogen-defective mice were infected by ticks rather than needles, the bacteria spread to their internal organs.

Similar experiments, not yet published, by another research group yielded a different conclusion. When infected ticks fed on plasminogen-deficient mice, *B. burgdorferi* rarely reached the animals' internal organs, says Mark S. Klempner of Tufts University School of Medicine in Boston.

The Benach group's report is the first that "nails down" the role of a mammalian protein in an insect, says microbiologist Kathleen A. McDonough of the New York State Department of Health in Albany. "The bacterium has to deal with the tick as well as the mammal." The researchers used cutting-edge tools to dissect these relationships, which scientists must understand in order to figure out how to block infection, she adds. —*E. Strauss*

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