known as the Zeeman effect. Each pair of levels corresponds to a particular direction of electron spin—up or down.

Once Zhao and his colleagues had split the energy levels, they applied a microwave field that had just the right energy to "couple" two of the lower energy levels by flipping the electrons' spins back and forth, forcing them to jump between the levels. By providing two competing paths for electrons to jump from a lower to a higher level, the coupling stymied the transition. "When you have light taking two paths and hitting a screen, at certain parts of the screen, the light cancels out," explains Scott Shepard, a physicist at Texas A&M University in College Station. "This is a very similar canceling out; the wave functions cancel." Because the electrons can't jump into the high-energy band, the ruby fails to absorb the wavelengths of light that would normally do the kicking.

The team was able to reduce absorption by about 20%. Other researchers have gotten much better results in gases. But "work in atomic vapors mainly demonstrates the principle," Zhao says: "Work in solid state may lead to real devices." Atac Imamoglu, a physicist at University of California, Santa Barbara, agrees. Even though the work is at a very early stage, he thinks that "several avenues [of possible application] are interesting." The ability to vary the transparency of an optical medium, he says, might be useful for storing bits in a quantum computer or creating switches for an optical computer. By cutting down on the amount of light absorbed in a laser's light-generating medium, he says, it could also lead to lower powered lasers.

–Charles Seife

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NEUROBIOLOGY

Schizophrenia Clues From Monkeys

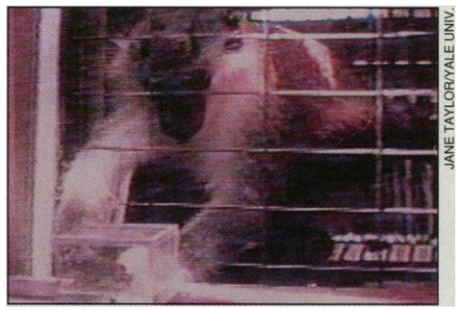
For decades researchers have tried, without much success, to stitch the patchwork of schizophrenia symptoms into a single picture. One in every hundred people suffers from this brain disorder, with manifestations that range from delusions and hallucinations, to lack of behavioral inhibition and cognitive problems—such as inability to make even simple decisions. Now, researchers from Yale University School of Medicine in New Haven, Connecticut, have taken a step toward understanding some of the brain changes involved in this disease.

On page 953, pharmacologist Robert Roth and his colleagues report that they can create cognitive problems in vervet monkeys by treating the animals with phencyclidine (PCP), a drug of abuse better known as "angel dust." Other researchers have studied how the drug affects behavior in animals, but they have not, for the most part, tested the animals on complex tasks such as those impaired in schizophrenia. What's more, the Roth team's results suggest that the more severe the cognitive deficits, the bigger the changes in dopamine—a neurotransmitter already known to be involved in schizophrenia—in the prefrontal cortex of the monkeys' brains. And they found the monkeys' problems can be partially reversed by a drug used to treat the condition in humans.

"I'm quite impressed," comments psychopharmacologist Klaus Miczek from Tufts University in Boston. "The combination of a complex behavioral test and the neurochemistry is very nice." Indeed, comments John Hsiao, a psychiatrist at the National Institute of Mental Health in Rockville, Maryland, "depending on how good a model [the PCP-treated monkey] is, this could be a tremendous advance." Studies of these monkeys may help clarify why cognitive problems arise in schizophrenia patients and could also help researchers evaluate therapies for improving cognitive function.

The Yale group decided to try PCP on the monkeys, because they knew the drug causes schizophrenic symptoms in people, particularly when used repeatedly. For the experiments, J. David Jentsch, a graduate student in Roth's lab, administered PCP twice daily to 15 monkeys for 2 weeks. A week later, he evaluated six of the animals—and an equal number of controls—with a behavioral test, in which each monkey was presented with a transparent cube that contained a slice of banana and was open on one side.

Instinctively, both PCP-treated and untreated monkeys grabbed for the banana by reaching straight for it. They all succeeded as long as the cube opening faced them. When the cube was rotated, untreated monkeys



Monkey do. PCP-treated monkeys can't figure out how to retrieve a treat from this cube.

quickly figured out that they needed to reach in from the side. But the PCP-treated monkeys, like animals whose prefrontal cortex is damaged, kept grabbing for the banana from the front, even though their hands kept banging into the cube wall. People with schizophrenia show a similar lack of behavioral inhibition. "They can't stop themselves, even though they know it is wrong," says Jentsch.

To look for chemical changes that might underlie this behavior, the Yale team sacrificed the other nine PCP-treated animals, as well as four controls. Then, to get an indication of dopamine usage, they measured the amounts of both dopamine and one of its breakdown products in various regions of the prefrontal cortex. The PCP treatment proved to be "very selective" in its effects, Roth notes, reducing dopamine usage in only two sections. One was the dorsal-lateral prefrontal cortex, which is responsible for working memory—essential if the monkey is to remember that it had already tried to grab the banana from the front. The other section was the prelimbic cortex, a section of the brain thought to control behavioral inhibition. "There's a direct and significant relationship in the degree of inhibition of dopamine [usage] and the degree of cognitive impairment," says Jentsch.

In a final test of the PCP monkey as a model for schizophrenia's cognitive symptoms, the Yale team evaluated the effects of clozapine, a drug used to treat the condition, on the surviving PCP monkeys. The drug improved their ability to figure out how to get the banana out of the cube, they report. This result highlights the power of using the PCP-treated monkey to study these deficits, says Roth. "There are other animal models of prefrontal cortical dysfunction, but they are not pharmacologically reversible," he points out.

The new work also fits with the growing view that dopamine's role in schizophrenia is more complicated than originally thought. Because many antipsychotic drugs block dopamine receptors, decreasing dopamine function, researchers once thought the symptoms were caused by an excess of this important nervous-system chemical. But this and other recent work suggest that while dopamine concentrations increase in some brain areas in schizophrenia, they decline in others—a result similar to what happened in the prefrontal cortex of the PCP-treated monkeys. "You can have both high and low dopamine at the same time," says Jentsch.

The Yale group hopes that continued work with the PCP-treated monkeys will lead to a better understanding of how dopamine is affected in the animals. And from those findings, says Jentsch, "we may be able to extrapolate what's going on in schizophrenia."

–Elizabeth Pennisi