Joannopoulos and his MIT colleagues, however, were fortunate enough to have a setup at team member Henry Smith's lab that they could use.

Still, it took the MIT researchers 3 years to fabricate and test the design they worked out for trapping infrared light. The design is what Joannopoulos calls a hybrid. Part of it is traditional technology—in particular the silicon waveguide, down which light will travel in a straight line—and part is entirely new: a series of periodic holes that create the photonic crystal and the necessary band gap.

Using x-ray lithography, Jim Foresi, who was then a graduate student at MIT, drilled a series of four air holes, spaced 0.22 micrometer apart, into the waveguide, followed by a gap of 0.42 micrometer, and then another series of four holes. The regularly spaced holes provide periodicity in one dimension along the waveguide, while the gap in the middle is the defect that creates the microcavity, allowing only light at a wavelength of 1.5 micrometers to pass. "You send in a pulse of light with a whole bunch of frequencies," says Joannopoulos. "When it hits that configuration of four plus four plus the defect, it can only go through if it has the frequency of that defect state." The MIT team then tested the device by sending in a pulse from a semiconductor laser and analyzing the light that made it through to the other end. The result, says team member Henry Ippen, was "surprisingly right on the money."

To take the next step in the photonic revolution, however, researchers must solve another problem—creating microcavities that actively emit light, rather than just allowing a single wavelength to pass through. Although virtually everyone in the field is trying to accomplish this, they are all reluctant to talk about their progress until they have a publication in the works. The idea, though, is to put a classical semiconductor laser or an LED in the middle of the microcavity, which requires making considerably more complex structures on the submicrometer distance scales of the MIT device. These would be designed to emit a wavelength of light identical to the wavelength the microcavity allows to exist. If such devices can be achieved, given the degree of miniaturization required, they would be perfect for generating the light that would be used to transmit information on integrated circuits that combined both electronics and photonics.

Because only a single wavelength of light would be allowed to exist within the cavity, the efficiency of these "optoelectronic" devices would be much higher than today's strictly electronic devices, and they would need considerably less energy to run. Indeed, researchers predict they may someday make LEDs that will convert energy into light moving in a single direction with an efficiency of 90% compared to the 30% in present devices.

A microcavity laser would be what is known as a zero threshold laser. In traditional lasers, it takes time and energy to get to the point where the light is coherent and the laser is lasing. This would not be the case with a microcavity laser. "The very first photon that gets emitted goes straight into your laser beam," says MIT team member Pierre Villeneuve. With all other lasers, until that threshold is reached, "you're wasting energy."

Considering that LEDs are already ubiquitous, and someday photonic devices may be as densely packed on chips as present semiconductor devices, it becomes obvious why such savings in energy and heat loss make the photonic researchers believe they are onto something big. But they have some challenges to overcome first. One would be to devise ways of building photonic microcavities with technologies that are easier to come by than x-ray lithography. Villeneuve predicts, however, that that should be possible within a few years. If so, it should launch the photonic revolution out of the realm of fantasy.

Until then, however, Joannopoulos has one caveat. "We're still theorists," he says, "and so we have to learn how to play the game in the real world. It's fine to design something, but then someone has to make it and mass-produce and integrate what exists already. These are all difficult issues."

–Gary Taubes

BIOMEDICINE

Herpesvirus Linked to Multiple Sclerosis

A new study has yielded evidence linking a strain of herpesvirus to multiple sclerosis (MS). More than 70% of patients in the study with the most common form of MS showed signs of active infection with herpesvirus-6 (HHV-6). The finding, reported in the December issue of *Nature Medicine*, is not yet conclusive proof, however, and some researchers question whether the apparent association is a symptom rather than a possible cause of MS.

In multiple sclerosis, immune cells attack and inflame the myelin, fatlike sheaths surrounding neurons in the central nervous system. Symptoms can vary widely, but MS is generally characterized by muscle weakness and neurological impairments, and most patients see their condition wax and wane with new symptoms appearing or old ones becoming more severe, alternating with periods of remission. Eventually, however, it can lead to disability and paralysis. HHV-6—which infects young children, causing a condition called roseola marked by high fever and rashes—also inflames myelin. It is present in about 90% of the U.S. population and can become reactivated when the immune system is under stress from factors such as secondary infections or emotional strain. Virologist Steven Jacobson and his colleagues at the National Institute of Neurological Disorders and Stroke in Bethesda, Maryland, wondered whether there might be a link between HHV-6 infection and MS.

> **"Now, we think we're a little closer" to finding a viral link to multiple sclerosis.**
>
> **—Steven Jacobson**

The researchers looked for signatures of HHV-6 in the serum of 36 MS patients and 66 controls in a blind test. As expected, nearly all had long-term antibodies, known as IgGs, that react with HHV-6 antigens. But 73% of MS patients also had IgM, an early antibody response to HHV-6 antigens and a potential marker of active virus replication. Only 18% of the control group showed IgMs directed against HHV-6. DNA from the virus was also found in more than one-third of MS patients, but in none of the controls. Moreover, magnetic resonance imaging detected numerous lesions in the myelin in the brain of a recently deceased MS patient, and an autopsy revealed HHV-6 in the lesions, but not in the adjoining normal tissues.

Jacobson, who has been looking for a viral cause of MS for more than 20 years, says "Now, we think we're a little closer." But some other experts are not yet convinced. Patricia O'Looney, a biochemist with the National Multiple Sclerosis Society in New York City, points out that not all the MS patients showed indications of active HHV-6 infection. She also notes that "MS breaks down the blood-brain barrier," which "may allow the virus to migrate into the central nervous system." In that case, HHV-6 infection could simply be a symptom of MS.

Jacobson cautions that even if an association between HHV-6 and MS is strengthened with further studies, it will not lead directly to a treatment for MS because nothing is yet available that can kill the virus without aggravating the breakdown of the myelin sheath. But it could open up new avenues for research.

–Phil Berardelli

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