tiny samples, about 30 nanometers thick.

In those samples, the researchers tried varying numbers of copper-oxygen layers, and all showed signs of superconductivity at high temperatures, Laguës says. The most dramatic signs of superconductivity came in a sample made with eight copper-oxygen layers: Its electrical resistance dropped by a factor of 100,000 as it was cooled from 280 K to 250 K; it offered no resistance (within experimental error) to a small current at 235 K; and it seemed to show the Meissner effect—a tendency to prevent magnetic fields from penetrating the interior of a material, which is one of the hallmarks of a superconductor. Each of four other samples with from three to eight copper-oxygen layers also showed signs of superconductivity at temperatures from 130 K to 300 K.

This combination of evidence makes Laguës' case for superconductivity at 250 K much more compelling than earlier reports, says Rick Green of the University of Maryland's Center for Superconductivity Research. Still, other researchers familiar with Laguës' work caution that his data, like previous reports of very high-temperature superconductors, may have an explanation other than the existence of superconductivity near room temperature. "I have a funny feeling that a lot of this stuff is due to artifacts," says Venky Venkatesan, also of the Center for Superconductivity Research. In all of these cases, including Laguës,' the superconductivity appears to reside in only a small portion of the sample, Venkatesan notes, and researchers have discovered that there are many ways that structurally complex materials, such as the compounds Laguës' group has made, can fool researchers into thinking they've seen superconductivity. To separate the artifacts from the real thing, scientists want samples that reliably show the same behavior over and over again.

Laguës says he can provide just that kind of reliability, and scientists should be able to pronounce a verdict quickly on his claims. Laguës can make samples available to other labs for testing, and other researchers will almost certainly give Laguës' recipe a try now that they have reason to believe that near-room-temperature superconductivity may indeed exist. "It's like hunting for a needle in a haystack," Geballe says. "Before this work we didn't know there was a needle there. It's nice to know that there is a needle."

–Robert Pool

MICROBIOLOGY

New Bind for Ulcer Bacterium

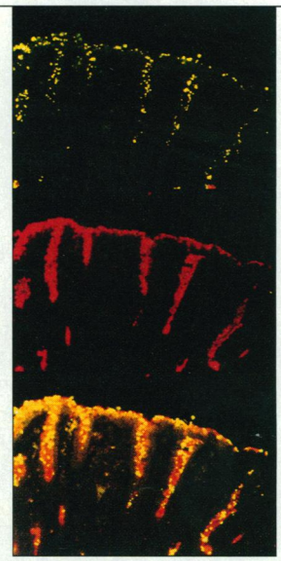
Ever since clinical studies published earlier this year firmly established in the minds of many researchers that the bacterium *Helicobacter pylori* is a common cause of stomach ulcers (*Science*, 9 April 1993, p. 159), researchers have been trying to pin down the unique mechanisms that enable this plucky bacterium to get a foothold in the hostile, highly acidic environment of the stomach.

Now researchers have met with success—not just once, but twice. On page 1892 of this issue, a group led by microbiologist Staffan Normark of the Washington University School of Medicine reports that *H. pylori* binds preferentially to Lewis^b (Le^b) antigens, located on the surface of gastric epithelial cells in the stomach, which are part of the blood group antigens that determine blood group O. Their report follows identification of another binding site by researchers at the Veterans Affairs Medical Center in Houston. Those scientists cloned an *H. pylori* gene that codes for a protein that binds specifically to the monosaccharide sialic acid, also found on glycoproteins on the surface of gastric epithelial cells. That protein presumably enables the bacterium to latch onto the cells (The discovery was reported by Dolores G. Evans and Doyle J. Evans Jr. in the February issue of the *Journal of Bacteriology*.)

Binding is one of the first steps in the process by which bacteria can bring on gastritis, gastric ulcers, even gastric carcinoma. After boring through the mucous layer that protects the gastric epithelium from stomach acid, the corkscrew shaped bacterium binds to the epithelium itself. What happens next isn't clear, but it appears that the immune system mounts an attack against the bacterium, which can inadvertently damage the epithelium, allowing *H. pylori* to strengthen its foothold. Over the years, this lesion may grow, resulting in an ulcer, or it may remain in check, resulting in gastritis. (Though the epidemiologic link between *H. pylori* infection and gastric carcinoma is strong, little is known about how infection leads to malignancy.)

The discovery of two distinct binding mechanisms might normally trigger a battle of competing theories, but not in this case. "We know that redundancy is the key to bacterial binding, so it's not surprising that first the Evans group and now Normark and his colleagues have found different targets to which *Helicobacter* binds," says microbiologist Martin Blaser of Vanderbilt University School of Medicine. Thomas Borén, a postdoc in Normark's lab, concurs: "It's likely that these are complementary mechanisms."

The discovery of the Le^b antigen binding has stirred particular excitement, because it may hold the key to a longstanding epidemiologic mystery. It's been known for some time that people with blood type O are between 1.5 and 2 times more likely to develop ulcers or stomach cancer than people with blood type A or B. According to Borén, *H. pylori* primarily binds to Le^b with the monosaccharide fucose at the end of its branched carbohydrate chains—the blood type O signature—and passes up Le^b with the antigenic determinants for blood groups A and B.



Matching up. *H. pylori*, stained green, binds to human stomach tissue *(top)*. That tissue also expresses specific blood group antigens, stained red *(middle)*. A double exposure shows the co-expression of bacterial receptors and blood group antigens *(bottom)*.

"This work is certainly consistent with the epidemiological data and provides a mechanism to account for these old observations," says Judah Folkman of Harvard Medical School, whose group there has also been studying *H. pylori* binding. But Blaser cautions against crediting Le^b binding with solely determining who gets infected with *H. pylori* and who does not. "This story has to be more complex because otherwise we would see more than just a 1.5 to 2-fold increase in ulcers and gastric cancer in people with blood type O," he explains. The second newly discovered binding site—sialic acid-terminated glycoproteins—could up the bacterial infection rate in non-O people; it might be one of those complexities.

With the discovery of the two binding sites, many researchers are now thinking about therapies aimed at loosening the bacterium's hold on the carbohydrate-containing glycoproteins on the stomach lining. "One would hope that it would be possible now to develop carbohydrate-based drugs that would compete with the natural binding sites and thus dislodge *Helicobacter* from the epithelium and allow it to be flushed from the stomach naturally," said Borén. But such drugs would need to get through the thick mucosal layer that protects the epithelium and *H. pylori* from stomach acid. Although the shot is difficult, researchers at least now have a target—or two—to aim at.

–Joseph Alper