tracked isotopes of strontium and neodymium, which occur in different ratios in dust from different regions. They confirmed that during the dust pulse, the proportion of minerals with a composition similar to that of the soils of Mesopotamia and Arabia increased.

Given the uncertainties of carbon dating, the marine dust pulse and the abandonment of Tell Leilan could still have been several centuries apart. But Cullen and deMenocal found in the core another time marker that makes a somewhat tighter connection. Less than about 140 years before the dust pulse is a layer containing volcanic ash. And Weiss had already reported that a centimeter-thick ash layer lies just beneath the onset of aridity and abandonment at Tell Leilan. The strikingly similar elemental compositions of the two ashes imply that they stem from the same volcanic event. If so, then Tell Leilan was abandoned just after the start of a climatic change of considerable magnitude, geographical extent, and duration. "There's something going on, a shift of atmospheric circulation patterns over a fairly large region," says Cullen.

Some archaeologists agree that this climate shift did change history outside northern Mesopotamia. "Most people who work in this range of time don't pay much attention to climate," says archaeologist Frank Hole of Yale; "rather, it's political and social events [that matter]. ... But I think the evidence is overwhelming that we've got something going on here."

While conceding that climate and culture interact, a number of archaeologists still think that Weiss is pushing the connection too far. Drought may well have driven people from farmland dependent on rainfall, like that around Tell Leilan, says Lamberg-Karlovsky, but Weiss "generalizes from his northern Mesopotamia scenario to a global problem. That's utterly wrong. ... Archaeologists fall in love with their archaeological sites, and they generalize [unjustifiably] to a larger perspective."

Even in Mesopotamia, "you do not have by any means a universal collapse of cultural complexity," says Lamberg-Karlovsky. For example, at 2100 B.C., in the midst of the drying, the highly literate Ur III culture centered in far southern Mesopotamia was at its peak, he says, as was the Indus River civilization to the east, which thrived for another 200 years. Weiss counters that cuneiform records show that Ur III did in fact collapse 50 years later, apparently under the weight of a swelling immigrant population and crop failures. That timing still fails to impress Lamberg-Karlovsky, who concludes that Weiss is "getting little support for the global aspect of it."

Such support may yet come from climate records being retrieved from around the world. In an enticing look at the postglacial climate of North America, Walter Dean of the U.S. Geological Survey in Denver found three sharp peaks in the amount of dust that settled to the bottom of Elk Lake in Minnesota. Dust peaked at about 5800, 3800, and 2100 B.C., plus or minus 200 years, according to the counting of annual layers in the lake sediment. During the 2100 B.C. dust pulse, which lasted about a century, the lake received three times more dust each year than it did during the infamous Dust Bowl period in the U.S. in the 1930s. But the archaeological record doesn't reveal how this drought affected early North Americans, who at that time maintained no major population centers.

In another sign that the Mesopotamian drought was global, Lonnie G. Thompson of Ohio State University and his colleagues found a dust spike preserved in a Peruvian mountain glacier that marks "a major drought" in the Amazon Basin about 2200 B.C., give or take 200 years. It is by far the largest such event of the past 17,000 years. But it doesn't seem to have had entirely negative effects; indeed, it roughly coincides with a shift in population centers from coastal areas of Peru, where the ocean provided subsistence, to higher regions, where agriculture became important. As more such records accumulate in the rapidly accelerating study of recent climate, archaeologists will have a better idea of just how much history can be laid at the feet of climate change.

–Richard A. Kerr

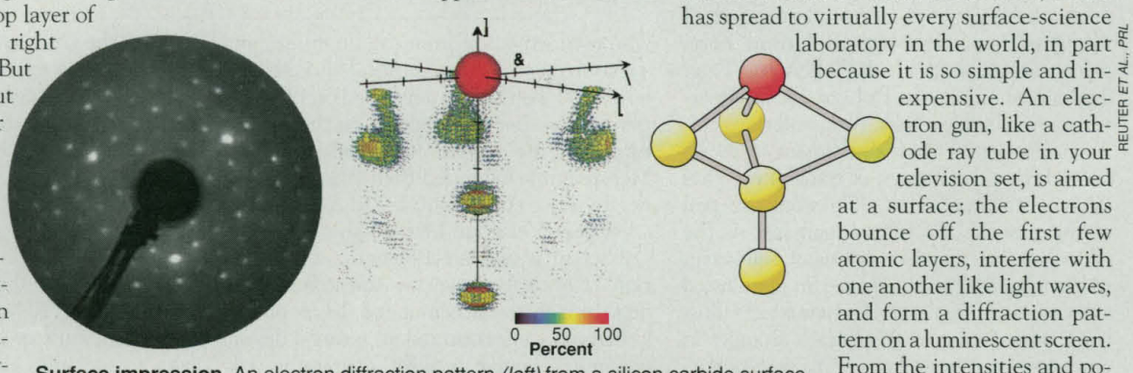
MATERIALS SCIENCE

Getting a 3D View of Surfaces

The true character of a material is often just skin deep. The arrangement of the first two or three layers of atoms can be enough to determine key properties such as friction, hardness, and chemical reactivity. Learning how those atoms are organized has been notoriously difficult, however. Scanning tunneling microscopes look at only the top layer of atoms, while x-rays pass right through to deeper layers. But imaging surfaces is about to get considerably easier and faster.

For years, researchers solved the atomic structure of a surface by sending in a beam of low-energy electrons, collecting the diffraction pattern generated when the electrons scatter off the surface, and comparing that to model calculations. But without an approximate picture of the surface structure to start with, low-energy electron diffraction, or LEED, was little help. Now, a team of surface physicists from the University of Erlangen-Nürnberg in Erlangen, Germany, has found a way to turn LEED data directly into three-dimensional images of the atomic arrangements.

"When we deal with complex materials, and we want to know more about the surface structure, or we want to tailor-make some structures for certain applications, then this will help us do it," says Ulrich Starke, a member of the Erlangen-Nürnberg team, which describes its work in the 15 December 1997 *Physical Review Letters* (*PRL*). "Anything for which a fundamental understanding of surface crystallography is important should benefit from this technology," adds Dilano Saldin, a solid-state theorist at the University of Wisconsin, Milwaukee.



REUTER *ET AL., PRL*

Surface impression. An electron diffraction pattern *(left)* from a silicon carbide surface yields the three-dimensional arrangement of atoms, which matches a model *(right)*.

The LEED technique dates back to the 1920s, although it wasn't until the late 1960s that researchers developed the algorithms needed to determine the atomic structure of a surface from the data. Since then, LEED has spread to virtually every surface-science laboratory in the world, in part because it is so simple and inexpensive. An electron gun, like a cathode ray tube in your television set, is aimed at a surface; the electrons bounce off the first few atomic layers, interfere with one another like light waves, and form a diffraction pattern on a luminescent screen. From the intensities and positions of the spots in the pattern, an algorithm recreates the arrangement of the surface atoms.

But it can only do so by iteration, starting with an approximate model of the surface in question. "You give this model to the computer and make the computer calculate the

intensities," says Klaus Heinz, head of the Erlangen-Nürnberg team. "If those calculated intensities coincide with those you measure, you're pretty sure you have the correct structure. But normally, you have to modify your model through trial and error. And in cases of complex structures, it may not ever be possible to find the correct structure."

In 1990, however, Saldin and Pedro de Andres of the Universidad Autonoma de Madrid suggested that the diffuse diffraction pattern generated when electrons scatter from a disordered surface might be interpreted as a hologram—a three-dimensional image captured in an interference pattern. Holograms are created when a beam scattered off an object interferes with a reference beam that has bypassed that object. Saldin and de Andres realized that they might be able to generate such a pattern if the surface has a single atom sticking out prominently, which can split the incoming electron beam. Half the beam bounces off the prominent atom and back to the detector, creating the reference beam, explains Heinz. The other half goes on to the other surface atoms and then scatters back to the detector, producing an object beam. The two interfere to produce the holographic image, which can be extracted from the LEED data.

Heinz's group collaborated with Saldin and his colleagues to produce atomic-resolution holograms of disordered oxygen and potassium atoms on a nickel surface. But measuring the intensity of a diffuse diffraction pattern accurately enough to extract a hologram was extremely difficult; only a few labs in the world could do it. Surface scientists also tend to be much more interested in ordered surfaces than in disordered ones because, says Starke, "those are the ones you can reproducibly prepare."

Heinz and his colleagues then realized that, with some modification, the same holographic reconstruction algorithms could work on the much brighter diffraction patterns that result from crystalline surfaces. The result is the pioneering image in the *PRL* paper: an electron hologram of a well-ordered atomic surface—in this case, silicon carbide.

Not every surface will give up its secrets to electron diffraction holography. It only works on materials with the occasional prominent atom, although such atoms can be chemically attached to a surface of interest before it is imaged. What's more, the technique can achieve a resolution of only 0.5 angstrom, compared to 0.1 angstrom for conventional LEED. For now, says Saldin, the technique is best suited to providing a quick first guess to plug into the LEED algorithm, which can then generate the correct, well-resolved structure. "It is a direct method of very quickly getting an approximate view of the structure," he says—a quick, surface impression of a material.

–Gary Taubes

AIDS RESEARCH

Chemokine Mutation Slows Progression

One of the most insidious features of the AIDS virus, HIV, is its habit of lurking in the body for years before causing overt disease. Why HIV takes so long to destroy the immune system of most infected patients is a central question in AIDS research. Now, findings presented in this issue may provide fresh clues to the mystery—as well as suggest new therapies that could slow or stop progression of the disease.

On page 389, geneticists Stephen O'Brien, Cheryl Winkler, and their colleagues at the National Cancer Institute in Frederick, Maryland, together with collaborators from other institutions in the United States and Japan, report that HIV-infected patients who have a mutant gene for a molecule called SDF-1 progress much more slowly to full-blown AIDS or death than do people with a normal version of the gene.

SDF-1 is a member of a class of molecules called chemokines, which help recruit immune cells to sites of inflammation. It normally binds to a receptor molecule on T lymphocytes—the cells targeted by HIV—called CXCR4, which the virus also uses to enter T cells during later stages of the disease. The results suggest that the mutant gene, called *SDF1-3'A*, helps protect infected people from the ravages of these late-stage viruses. These findings mark the first time that a mutation in a gene coding for a chemokine, rather than a chemokine receptor, has been shown to affect the course of HIV infection.

M. CRUMP/PENCE

Protector? SDF-1 chemokine may tie up T cell receptors.

O'Brien's team found the mutation during a genetic screen of blood samples taken from 2419 HIV-infected patients in study cohorts across the United States, as well as 435 people who had been exposed to HIV but remained uninfected. In earlier work on these patients (*Science*, 27 September 1996, p. 1856), the team found that people with two mutant copies of the gene coding for CCR5—a chemokine receptor targeted by HIV in the earlier stages of infection—are highly resistant to infection. The new study indicates that subjects who are homozygous for the SDF1-3'A mutation—meaning they carry two copies of it—are also protected, but primarily against progression of the disease after infection. Disease progression in homozygous Caucasians, for example, takes three times longer than in similar individuals who possess only one mutant copy or none at all.

The SDF1-3'A variation, which occurred in homozygous form in less than 5% of the patients studied, is located in a part of the gene that is not "translated" into the building blocks of SDF-1. Instead, it is in an adjacent, untranslated portion whose sequence is conserved among mammalian species, indicating that it may have an important regulatory function. O'Brien and his colleagues suggest that this segment may control the production or transport of the chemokine. If so, the mutation may protect infected individuals by increasing the production or availability of SDF-1, which would bind to CXCR4 and block the virus from entering the T cells.

The study provides no direct evidence for this idea, but other researchers told *Science* that it is a reasonable—and attractive—interpretation of the results. Viral immunologist Jean-Louis Virelizier of the Pasteur Institute in Paris says that an increased level of the chemokine is the "simplest explanation" for the findings. Although SDF-1 has previously been shown to block CXCR4-using viruses in the test tube, Virelizier says the new results "provide the first evidence" that the chemokine "may participate in vivo in the host's defense against HIV infection." And Dan Littman, an AIDS researcher at the New York University Medical Center, says that if the mutation does affect SDF-1 levels, "it would be very exciting indeed, because it would suggest that progression to disease is in large part dependent on HIV interacting with CXCR4." That is a popular, but not yet proven, hypothesis for how HIV becomes increasingly lethal to its target cells.

The next step, researchers say, will be to prove that the mutation really does increase SDF-1 levels. If O'Brien's hypothesis is right, the findings could point the way to development of new anti-HIV drugs: SDF-1 or a laboratory-modified version of the molecule "may have antiviral effects even at late stages of HIV infection," says Virelizier. Indeed, O'Brien argues that the protective effects of the SDF1-3'A mutation could be considered a first clinical test of that appealing possibility. "This was not done in the test tube, but with patients infected with HIV," he says. "It has parallels with a clinical trial. The results are very provocative."

–Michael Balter