atomic trampoline.

The ultimate goal of all these shenanigans is to better understand how atoms interact—with each other, with light, and with the rest of the world. One area in which researchers have already seen some new physics is in slow collisions.

At room temperature, atoms in a gas move at several hundred meters per second. When two atoms collide at these speeds, they bounce off one another like billiard balls. But near absolute zero, the atoms are moving at about 10 centimeters per second and collisions proceed much more slowly, which allows quantum effects to appear. For example, Wieman and colleagues at JILA have studied collisions between cesium atoms held in a laser trap at around 250 μK. At this temperature, a collision between two cesium atoms stretches over about 30 nanoseconds, which is approximately the same amount of time it takes a cesium atom to reemit a photon that it has absorbed.

"The collisions that we've observed are quite novel," says Walker. They are slow enough that if one of the atoms is in an excited state, it can emit a photon in the middle of bouncing off the other atom. These quantum effects not only modify the way two atoms collide but actually affect the probability of a collision occurring. Theorists Alan Gallagher of JILA and David Pritchard of the Massachusetts Institute of Technology have a rough model that is "probably close to correct," Walker says, but the fine points of the collisions are still not understood.

One reason more labs aren't doing laser trapping and cooling experiments is their traditional dependence on bulky and expensive lasers and atomic beams, but work at Wieman's lab seems likely to change that. Wieman says he has cooled atoms to much less than 1 mK with a system that costs about $1,000, as compared to about $250,000 for a standard setup.

Instead of using an atomic beam to supply the atoms, the researchers found they could use a small glass bulb that contains a dilute vapor of cesium atoms. And in place of the normal laboratory lasers, the Colorado lab's system uses two tiny and inexpensive diode lasers that are each split into three beams, with all six focused on a spot in the center of the bulb. As the atoms move around the bulb, they are caught in the focus of the laser trap and held there. Although the whole trap takes up only "half a desktop," it is powerful enough to hold tens of millions of cesium atoms at a time. It is cheap enough and simple enough to be used in an undergraduate physics laboratory, Wieman says, and it's way ahead of the state-of-the-art equipment of just 3½ years ago. It could introduce a whole new generation of researchers to the joys of making atoms jump through hoops. ■ ROBERT POOL

A Reliable Animal Model for AIDS

Much of what is known about how human immunodeficiency virus (HIV) causes AIDS has been inferred from studying its effects on cells growing in the laboratory. Researchers have had little alternative: HIV only infects humans and chimpanzees and it doesn't make chimpanzees sick, so there have been no good models to work with. Now, however, that is changing.

On page 1109 of this issue, Ronald C. Desrosier and his colleagues at the New England Regional Primate Center in Southborough, Massachusetts, report that they have identified and cloned a simian immunodeficiency virus (SIV) that will reliably cause AIDS-like symptoms—and ultimately death—in rhesus monkeys. SIV has already been shown to cause a simian form of AIDS, but the infectivity and pathogenicity of wild strains of the virus is variable. The significance of Desrosier's work is that it starts with a thoroughly characterized virus—not a wild virus grown in culture but a clone with a known sequence that consistently causes disease.

HIV and SIV are closely related, both genetically and biologically, and simian AIDS closely parallels the human disease. By using this new cloned virus, scientists can design experiments that will help reveal just how this retrovirus causes disease.

"It's some of the most exciting stuff that I've heard," enthuses Dani Bolognesi, an AIDS researcher from Duke University School of Madison. "With this clone and mutants of it he has a handle on resolving issues of pathogenesis."

And that's just for starters.

In studies conducted both at the New England Regional Primate Center and the California Regional Primate Center, Davis all 11 monkeys inoculated with the SIV clone became infected and half died within 1 year. Murray Gardner, an AIDS researcher at the University of California in Davis, says Desrosier's animal model is "the gold standard." "Using this model you can do the systematic, grunt science—the step by step things that have to be done to work out the best vaccine and the best treatment," says Gardner.

Desrosier has already launched on three separate lines of research. First, he is studying how the virus changes in its host over time and how those changes correlate with the progression of disease. "We have an experimental system derived from a single DNA molecule, whose sequence we know, and we've been able to precisely measure the rate at which the envelope gene mutates as a function of infection of the animal," says Desrosier. "This is really the first time that anyone has ever been able to quantitate the rate of genetic change in an infection of animals."

A second direction is to study the so-called nonessential genes in the SIV genome. Like HIV, SIV has several genes—including *rev*, *vip*, *vpr*, and *nef*—that are thought to regulate the virus's growth, but they are called nonessential because the virus will still grow in tissue culture even after they have been removed. Desrosier believes it may be a different story in vivo. "Some of [the nonessential genes] might play a role in getting the virus into certain secretions, like vaginal fluid or semen," he says. "Now we have the capability to look at each of these 'nonessential' genes and ask if they're essential for the pathogenic potential of the virus. If so, they would become targets for drug development."

Finally, Desrosier is studying how the virus's affinity for different types of cells changes during the course of an infection. For example, Desrosier's SIV clone does not grow in macrophages in the laboratory. But virus recovered from one monkey just before it died did grow in those cells. Intriguingly, this was the only monkey that exhibited granulomatous encephalitis, rash, and giant cell pneumonia. Could these particular symptoms be related to a change in the virus that makes it target macrophages?

Gardner points to one other crucial issue that is resolved by Desrosier's work. All by itself, the SIV clone causes disease in otherwise healthy animals. "This is what virologists and others want to have as the ultimate proof that a virus is the etiologic agent," says Gardner. "This is the sine qua non. This nails it down." And if SIV infection is all that is needed to cause simian AIDS, that's one more indication that HIV is all that is needed to cause human AIDS. ■ JOSEPH PALCA