Another neglected area consists of so-called process studies, which are focused research efforts seeking to understand individual elements of the climate puzzle. Strikingly, the committees also report that the United States has lost the leadership position it once held in constructing computer models of the climate.

Some authors of the report make more pointed comments. "[Terra] is old technology. It gathers massive amounts of data without cutting the problem acutely with respect to diagnosing what is going on," says Harvard University atmospheric chemist James G. Anderson. "Scaling back and reducing EOS in scope isn't really the answer because you have to go after this from an entirely different point of view to solve the problem."

Instead of simply harvesting data, scientists need to pinpoint the most important questions and test individual hypotheses, Anderson says. As positive examples of focused federal research programs, he cites AIDS studies, the human genome project, efforts to understand stratospheric ozone loss, and investigations concerning El Niño.

"We cannot as a nation afford to run programs other than [in this] way," he says.

NASA has gotten the message from criticisms over the years and has responded to some by restructuring the EOS program. The agency will send up a series of small spacecraft designed to address particular problems, such as changes in solar radiation, ocean circulation, and polar ice sheets.

By all accounts, EOS as currently structured will fail to deliver in one important respect: It will not measure how the planet's climate is changing over the long term.

Although NASA originally regarded EOS as a monitoring mission, the redesigns of the early 1990s shifted the emphasis toward understanding how the many different facets of atmosphere, land, and ocean interact. Lost by the wayside were plans to track land temperatures, sea temperatures, cloud cover, and other critical indicators of the planet's health. Although debate concerning the planet's temperature has simmered since the 1980s (SN: 3/15/97, p. 156), the USGCRP lacks a coherent plan for determining how quickly the globe is warming.

"We as a country do not have a strategy for making long-term climate-relevant measurements," says Berrien Moore III, an EOS investigator from the University of New Hampshire in Durham. "And if we don't begin to put that strategy into place, then EOS really will not achieve what it should. . . . That would be a very grave error." Chairperson of the National Academy of Science's committee on global-change research, Moore played a substantial role in devising the original EOS plans that he now admits were "fundamentally flawed."

Despite the decade of criticisms leading up to this summer's launch and the lingering questions concerning the future, Moore and others agree that Terra will provide scientists with completely new ways of looking at Earth. By observing the same point simultaneously with so many instruments, it will enable researchers to examine the ties between different facets of climate, exploring such topics as how temperature affects tree growth and vice versa.

"It's a wonderful opportunity," says Inez Y. Fung, director of the Center for Atmospheric Sciences at the University of California, Berkeley, who formerly criticized the U.S. program.

Fung, now part of the EOS team, likens the Terra launch to sending a patient to the hospital for a full, and costly, examination. "Maybe this is our only chance to check Earth into the Mayo Clinic to get a thorough scan of everything we can," she says. □

Biomedicine

Genetic variation helps ward off AIDS

Diversity is good. That's not a politically correct conclusion but a medical one. Scientists have begun to study how people's genetic variation may slow the speed at which an HIV infection leads to AIDS (SN: 8/16/97, p. 103).

"A few years ago, we really didn't have any genes that we knew influenced the outcome of an infectious disease. Now, we have a handful," says Stephen J. O'Brien of the National Cancer Institute in Frederick, Md., a leader in the effort.

In the March 12 SCIENCE, O'Brien and his colleagues report that HIV-infected people with a limited repertoire of so-called HLA genes are likely to develop AIDS within 3 to 5 years rather than the usual 10 or so. The researchers also found a quicker progression to AIDS in people having either of two particular HLA genes, even if they have diversity within their HLA genes.

HLA genes encode proteins that help cells present pieces of invading viruses or bacteria as targets for the immune system to attack. Each of the dozen or so HLA genes that people carry comes in many slightly different forms, or alleles. Some of these genes have more than 100 alleles. People typically inherit different HLA alleles from each parent, but a mother and father occasionally pass on the same HLA allele. Those cases, known as HLA homozygosity, seem to pose a threat.

Scientists have long suspected that having a diverse set of HLA genes allows people to present a wider range of targets to their immune system, but finding proof was tough. O'Brien's group now has examined the variation among three HLA genes (*A*, *B*, and *C*) in nearly 500 HIV-infected people. Those with different forms of all three genes—adding up to six different HLA alleles—avoided AIDS on average for 10 to 12 years, and many stayed healthy even longer. "If you're optimally represented with HLA types, you'll have a better defense against a virus that changes a lot, like HIV," concludes O'Brien.

He's now studying why having at least one of the HLA alleles called *B*35* and *Cw*04*, even if the person isn't homozygous for it, makes one vulnerable to rapid AIDS progression. Since almost half of the population is homozygous at one of the three HLA genes or has a *B*35* or *Cw*04* allele, O'Brien says it's crucial to know how these genes influence HIV infections.

Identifying such genetic factors, he adds, should help in the evaluation of new AIDS drugs and vaccines. People who don't respond may have a vulnerable genome. —*J.T.*

Misplaced DNA generates problems

Autoimmune diseases largely remain a mystery. Infections or injuries often precede autoimmune attacks, but how can those events make the body attack its own tissues?

Misplaced DNA inside cells may lead them to rally the immune system to destroy normal tissues, suggests Leonard D. Kohn of the National Institute of Diabetes and Digestive and Kidney Diseases in Bethesda, Md. In the March 2 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, he and his colleagues show that introducing DNA into a cell's cytoplasm—rather than the nucleus, where most genetic material resides—triggers changes that scientists had overlooked.

In response to the added DNA, the cell turns on genes usually employed only by specialty cells that display targets for the immune system to attack. Since viruses often introduce their DNA into a cell's cytoplasm, and damaged cells may have their DNA leak out of the nucleus, Kohn's finding may explain why infections and tissue injuries are associated with autoimmunity.

The results may also indicate why gene therapy has had little success. Viruses are often used to deliver the genes to the cytoplasm, and frequently the immune system later destroys the cells that have received the DNA. Scientists are studying compounds that may suppress the changes brought about by DNA in the cytoplasm, says Kohn. —*J.T.*