as a lower priority because it is "a sort of stand-alone thing," says Thompson, that doesn't fit in the usual mission categories, such as astronomy, observing platforms, or life sciences. Furthermore, Thompson says, it "doesn't have the broad constituency that the astronomy missions do"—meaning the experiment will be built and used by only one university, not many, as some projects are.

Despite these hammer blows, Everitt always revived his project, partly with the help of NASA insiders such as Charles Pellerin (head of astrophysics and physics) and researchers at NASA's Marshall Space Flight Center. "I did what people usually do after they've been zeroed. You learn to lobby," he says. Two congressional aides gave Everitt and Fairbank a crash course, and they took to the halls of Congress—learning to peddle the romance of Einstein, the glamour of high tech, and the value of educating students (the project has produced 33 Ph.D.s). Stanford even published a glossy 28-page brochure on the experiment.

The work paid off each time when Congress restored Gravity Probe B to NASA's budget. By now the project "has significant congressional support," says a staff member of the Senate Appropriations Committee. "It's good science, it's affordably priced, and it's not the kind of science that NASA usually supports. We think it would be a real tragedy to cut it." Obviously, so would the researchers at Stanford, more than one of whom has made it his life's work. "If I had known how long it would take when I started this at age 28, I would have thought I was a fool to have gotten into it," admits Everitt. But as Gravity Probe B stands now—with NASA funding, a launch date, and an enviable technological track record—Everitt can still add, "I'm delighted that I did." ■ ANN GIBBONS

AIDS: The Evolution of an Infection

AIDS researchers have long been puzzled by the prolonged clinical course of the disease: A person infected with HIV can apparently combat the virus for more than a decade, and then his or her immune defenses give out, opening the door to an onslaught of opportunistic infections, and, in most cases, death. Some investigators have insisted that a cofactor—possibly another infectious agent such as a mycoplasm—must be involved. Others have speculated that the AIDS virus may become more pathogenic as it replicates inside the infected host.

But now a team of researchers in England and the Netherlands has come up with a radically different explanation of what happens during the years that the immune system is under assault by HIV. They suggest, in an article on p. 963 of this issue of *Science*, that the progression to disease can be viewed as an evolutionary process with a timescale measured in years rather than millennia. And they have developed a mathematical model that not only describes the clinical course of the disease but also raises doubts about some of the strategies being used to develop vaccines or drug therapies against HIV. While virologists who have seen the model are intrigued, they are generally skeptical because so far it is supported by scant experimental evidence: Only two patients' infections are chronicled in the *Science* paper.

Martin A. Nowak, a mathematical biologist working with population biologists Roy M. Anderson and Robert M. May at the University of Oxford department of zoology, first sketched out the hypothesis last year in the journal *AIDS* (vol. 4, p. 995). It is based on a biological property that HIV shares with all retroviruses: It lacks any mechanisms to correct errors that occur when its genetic material is being duplicated. This means that every time the virus makes a copy of itself there will be, on average, at least one genetic "mistake" incorporated in the new virus. So a few days or weeks after initial infection, there may be a large population of closely related, but not identical, viruses replicating in an infected individual. While the immune system will recognize most members of this population of viruses, some mutants will evade the immune response for a time. Until they are brought under immune control, these so-called escape mutants will attack a class of T cells that express a receptor called CD4. It is these CD4 cells that are key to orchestrating the overall immune response, and once they are gone the immune system collapses.

As the virus grows and continues to produce mutant forms, the immune system and responds to these new forms. But ultimately, Nowak and his colleagues conclude, the sheer number of different viruses to which the immune system must respond becomes overwhelming. It's a bit like the juggler who tries to keep one too many balls in the air: The result is disastrous. Once the immune system is overwhelmed, the latest escape mutant—which may not necessarily be the most pathogenic one to come along—will predominate.

Once they had worked out their model on paper the Oxford group, along with Tom F. W. Wolfs and Jaap Goudsmit of the Human Retrovirus Laboratory in Amsterdam, looked at the pattern of viral diversity in two HIV-infected patients to see whether they had accurately predicted the course of the disease. Both patients first developed antibodies to HIV in 1985. One man, who developed AIDS, did show a rapid decline of the number of different HIV quasispecies after AIDS symptoms began to appear. For the other, who remained asymptomatic over the duration of the study, the diversity continued to grow.

More studies of this kind will be needed to convince other AIDS researchers that the model is valid. "This is a reasonable hypothesis," says virologist Harold Burger of the New York State Health Department's Wadsworth Center for Research, "but it is important to get an adequate amount of data to confirm it." Gerald Myers, who studies viral diversity at Los Alamos National Laboratory, is more skeptical. He thinks that the decline in viral diversity in the one patient presented in Nowak's paper is an artifact that is explained by that patient's use of the antiviral drugs DDI and AZT. But Myers agrees with Nowak and his colleagues that the model raises some interesting questions for future research.

For example, if a vaccine is targeted against a particular strain of the virus—say one that has stabilized after years of growing in culture—will it be effective against a constantly diversifying virus population? Nowak's model suggests it won't. The same goes for therapies that try to enhance the ability of the immune system to respond to the virus. If the therapy is begun after there is already a lot of viral diversity, these therapies will not be effective. On the other hand, a therapy that will slow viral diversity early in the course of infection—such as DDI and AZT appear capable of doing—could delay the onset of symptoms by years.

For now, the search is on for more data to validate—or invalidate—the model. Myers thinks these data may already exist in labs that have been looking into the change in the virus during an infection. If so, Nowak and his colleagues won't have long to wait to see whether their novel mathematical model accurately reflects the real world. ■ JOSEPH PALCA