akin to the Defense Advanced Research Projects Agency—which prides itself on attracting daring ideas and funding them swiftly—and endowing it with $3 billion. "We need out-of-the-box thinking," says Barry Bloom, dean of the Harvard School of Public Health in Boston.

The meeting ended without a set of recommendations, but NIAID's Fauci says his institute will write a summary and pass it on to Health and Human Services Secretary Donna Shalala. Most participants, like Malegapuru Makgoba, president of the Medical Research Council of South Africa, were optimistic that tangible results are within reach: "I'm confident that we'll see an AIDS vaccine in the next 5 or 6 years."

But the lack of clear-cut plans on how to proceed left some a tad disappointed. "We shouldn't continue to have these very general meetings, where a shopping list is read out," says Richard Feachem, director of the Institute for Global Health in San Francisco and a veteran of similar gatherings, some with the same cast, over the past year. "This is a very special time," he asserts. "There's an energy now that we need to harness quickly, because it might be lost." **–MARTIN ENSERINK**

QUANTUM PHYSICS

Furtive Glances Trigger Radioactive Decay

Quantum physicists love to shatter conventional wisdom—even their own. Take radioactive decay. Common sense says you can't keep an atom's nucleus from decaying simply by looking at it. Quantum mechanics says you can. Now two Israeli physicists have come up with a way in which watching a nucleus might make it decay *faster*.

The decay-preventing process, known as the quantum Zeno effect, has fascinated physicists for 25 years. It takes its name from the paradox-mongering Greek philosopher who imagined himself repeatedly interrupting an arrow's flight to chop its trajectory into smaller and smaller bits—thus proving (he thought) that motion was impossible.

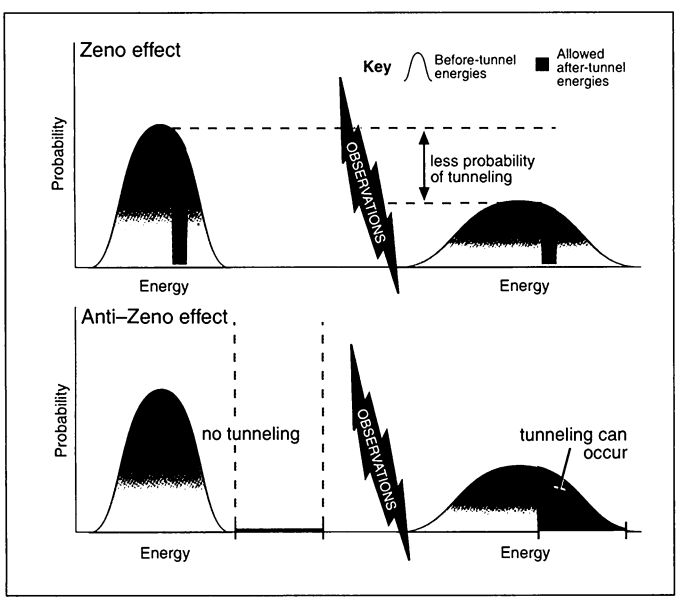
In the quantum case, what is ruined by interruption is not motion but processes such as nuclear decay. Imagine an alpha particle, two protons and two neutrons, lodged inside a much larger, radioactive nucleus. The particle is there because it can't hurdle the nuclear "energy barrier" that holds it in. Sooner or later, though, the particle probably *will* escape, causing the nucleus to decay. It can do that by tunneling through the barrier, in a strange quantum way. At first, the particle is firmly stuck on one side of the barrier, but as time goes on, it "spreads out" and starts to exist in a "superposition" of bound and free states that puts it, in effect, on both sides of the barrier at the same time. From this superposed state, the particle may decide that it is on the far side of the barrier, break free, and escape.

But there's a twist. If someone observes the particle, by, say, bouncing a photon off it, whatever superposition there is "collapses," and the particle must instantly decide which state it is in—inside or outside the barrier. "You make your measurement and, bingo! You're in one and only one state," explains Peter Milonni, a physicist at Los Alamos National Laboratory in New Mexico. By repeatedly measuring and prodding the particle, a scientist can keep destroying the superposition before it gets established, drastically reducing or even eliminating the possibility that the particle will tunnel through the barrier. "The exponential decay process could be slowed down or completely interrupted by the Zeno effect," says Gershon Kurizki of the Weizmann Institute of Science in Rehovot, Israel. In short, the watched pot never boils. Physicists think they've seen this quantum Zeno effect in experiments with photons and with trapped ions.

Now Kurizki and his colleague Abraham Kofman argue in this week's issue of *Nature* that the reverse can happen: Under certain conditions, the watched pot *always* boils. Kurizki and Kofman think of the Zeno effect as an interaction of overlapping energy states. Before tunneling, a particle can take on a certain range of energies; after tunneling, it has another range. A particle can tunnel only if those energy ranges overlap. Energy ranges, however, can change. If you knock a particle by measuring it, for example, the jolt from the photon broadens the range of energies the particle can take on. The faster you repeatedly measure the particle, the broader the range gets. With more energy options to choose from, the particle spends less time in any particular part of its range. Thus, by repeatedly observing a before-tunneling particle, physicists can ensure that it spends almost all its time at energies that don't overlap with after-tunneling energies. The result: no tunneling, and no nuclear decay.

Kurizki and Kofman realized that the exact opposite can happen. Suppose, they said, your before-tunneling energies and after-tunneling energies don't overlap to begin with. In that case, the particle can't escape. But repeated measurements might broaden the range of before-tunneling energies so that it creeps into the after-tunneling zone, allowing the nucleus to decay. "If you do it sufficiently fast, you would see an increase of the decay rate," Kurizki says. "The same procedure leads to the opposite of what is expected."

Although nobody has yet seen the anti–Zeno effect in action, Kurizki believes experiments will verify it within a few years. In fact, he thinks the anti–Zeno effect ought to be much more common than the Zeno effect—"the rule rather than the exception." If so, that could be bad news for scientists trying to develop quantum computers. Some physicists have proposed using the Zeno effect to keep quantum bits from losing the information they contain. But a repeated measurement might induce an anti–Zeno effect instead, Kurizki says. "It might have the opposite effect." **–CHARLES SEIFE**



Zeno phobia. By spreading out a particle's energy curve, observations can make it either less (top) or more likely to escape by quantum tunneling.

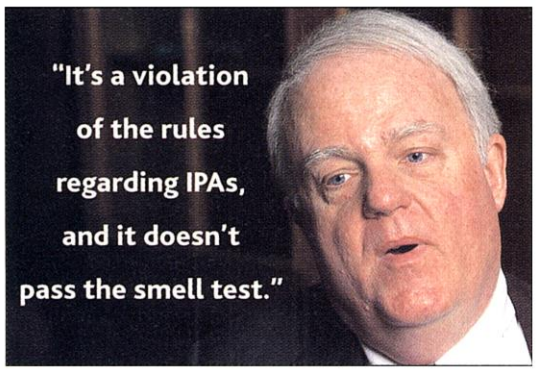
NSF REAUTHORIZATION

Closed Ethics Case Sparks Dueling Bills

A 2-year-old case of financial impropriety by a former National Science Foundation (NSF) senior staffer has exploded like a time bomb on Capitol Hill, sending the agency running for cover. The surprise battleground is new legislation to reauthorize NSF's programs, a process normally carried out with little fanfare. The dispute pits the chair of the House Science Committee, James Sensenbrenner (R–WI), against Nick Smith (R–MI), chair of the panel's basic research subcommittee. Caught in the crossfire is NSF Director Rita

Colwell, who needs both men as allies in the annual fight for federal dollars.

On 17 May Sensenbrenner proposed cutting the director's travel budget, staff levels, and her ability to temporarily transfer employees to outside organizations as part of legislation, H.R. 4485, to authorize NSF's programs for the next 4 years. He says it's a response to the "fiasco" stemming from NSF's reaction to rule-breaking by Luther Williams, former head of the education directorate. Williams was fined $24,900 in June 1998 for improperly accepting outside honoraria (*Science*, 26 June 1998, p. 2053). "They completely mishandled the case," Sensenbrenner says about NSF's decision to reprimand Williams but allow him to keep his job for more than a year. Then, last summer, the agency reassigned him under the Intergovernmental Personnel Act (IPA) to a new program run by Louisiana's Tulane University (*Science*, 13 August 1999, p. 997).



In particular, Sensenbrenner says he's incensed because Williams was allowed to retire 5 months after the reassignment, rather than return to NSF, and because the Tulane program's founder, president emeritus Eamon Kelley, also heads the National Science Board. "[The first] is a violation of the rules regarding IPAs," he says, "and [the second] doesn't pass the smell test." An NSF spokesperson said that Colwell did not wish to comment on the matter.

The reauthorization bill, which sets funding levels for individual programs, would prohibit NSF from detailing staff to any outside organization as an IPA, mandate ethics training for all NSF staff, and require Colwell to update Congress twice a year on the training and related matters. "It's an attempt to make sure that this stuff never happens again," says Sensenbrenner. He calls the language "a preemptive strike" to protect NSF against legislators who might use the controversy as an excuse to trim the agency's budget. The bill matches the president's 17% requested increase for NSF in the fiscal year that begins on 1 October and includes much more modest annual increases—from 2.5% to 3.3%—for the next 3 years. "I'm a fan of NSF, but sometimes basic research is a hard sell," he explains.

Two days after Sensenbrenner introduced his bill, Smith introduced his own version, H.R. 4500. It hews closely to Sensenbrenner's on most counts—but omits the prohibition on IPA transfers as well as the travel cuts and the ethics program. Legislative aides say it is highly unusual for a subcommittee chair to introduce a bill that competes with one by his boss. In another bizarre turn, H.R. 4500 was actually Smith's second NSF authorization bill. The day before, he introduced H.R. 4491, which retains all of Sensenbrenner's punitive language and differs only in proposing two, rather than four, years of future budgets. Smith declined to comment on either of his bills, but Capitol Hill sources speculate that the earlier version had been filed in haste.

Sensenbrenner says that he hopes the full science committee can take up his bill sometime this month. Such a move would bypass Smith's basic science subcommittee, which oversees NSF, but Sensenbrenner says it is standard practice. The move would also make it harder for Smith to hold a hearing on his legislation.

Even if approved, however, Sensenbrenner's bill stands only an outside chance of being considered by the full House before Congress adjourns in the fall. It also lacks a counterpart in the Senate. The bill's demise certainly wouldn't upset NSF officials, who hope that the flap doesn't affect their long-term relationship with their closest congressional overseers. **–JEFFREY MERVIS**

BIOMEDICAL RESEARCH

Patients Help Track Down Disease Gene

In the Terry household, patient advocacy is a family affair. At age 11, Elizabeth Terry testified before Congress to promote biomedical research. Her mother travels around the country seeking out patients with pseudoxanthoma elasticum (PXE), the disease Elizabeth and her younger brother, Ian, were diagnosed with some 5 years ago. Their father makes regular trips to the local medical library to follow up leads.

Together, they established and run a patient advocacy group from their house, one that is promoting "a new breed of advocacy," says Francis Collins, director of the National Human Genome Research Institute in Bethesda, Maryland. Like the Hereditary Disease Foundation, which played a pivotal role in coordinating research and tracking down the gene for Huntington's disease, the Terrys' organization has become "aggressively involved in the science," says Collins, as well as in securing funding and working with PXE patients. In the past few weeks, their relentless work has paid off in a big way.

Their organization, PXE International, has helped three research teams pinpoint the identity of the gene for this rare inherited disorder. Two labs, led by Charles Boyd at the University of Hawaii, Honolulu, and Arthur Bergen at the Netherlands Ophthalmic Research Institute in Amsterdam, report their results in the June issue of *Nature Genetics*; the third group, led by Jouni Uitto at Thomas Jefferson University in Philadelphia, published its findings in the 23 May *Proceedings of the National Academy of Sciences.*

But PXE International isn't the only patient group to play a key role in unmasking the identity of the PXE gene. An older, albeit less media-savvy, organization called the National Association for PXE (NAPE) teamed up with the March of Dimes, a Texas dermatologist, and a Harvard genetics lab to help place the gene for this disorder on the genome map—literally—in 1997. Now, the collaboration, led by Klaus Lindpaintner, a physician-geneticist at Harvard's Brigham and Women's Hospital, has also identified the gene itself. It reported its results online in the 26 May *Journal of Molecular Medicine*.

All the researchers say that having a group of families willing to work with them was critical to their success. "If the patient community had not been involved, there would have been no studies," agrees Sherri Bale, a geneticist with GeneDX, a start-up in Rockville, Maryland, that makes genetic diagnostic tests.

Young advocate. Elizabeth Terry urges Congress to spend more money on biomedical research.

The four teams converged on a little-studied gene, called *ABCC6* or *MRP6*, on chromosome 16 as the culprit in PXE. Traditionally considered a skin disease, PXE causes calcium to accumulate in connective tissue of the eye, gut, heart, and skin, sometimes causing vision loss, gastrointestinal

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