serves in the National Guard, says the case has already caused his name to be added to an immigration watch list: When his military unit reentered the United States after training in the Caribbean, he notes, he was delayed for hours while FBI officials checked out his story. "It's gotten Kafkaesque," he says.

University officials, meanwhile, have watched with concern as Foral's case has unfolded. Some schools, such as the Massachusetts Institute of Technology in Cambridge, had already hinted that the criminal sanctions and security requirements imposed by the Patriot Act and the more recent bioterrorism law (*Science*, 31 May, p. 1585) might force them to end research on regulated agents such as anthrax. "Many researchers are still unaware of these laws," says Atlas. "Deans are terrified," he adds, that one of their students could be next.

–DAVID MALAKOFF

PSYCHOLOGY

Violent Effects of Abuse Tied to Gene

Some children who suffer physical, sexual, or emotional abuse become violent adults. But many do not. Now a new study of both genetics and social surroundings points to the influence of a particular genotype on aggressive behavior in young adults from a troubled background.

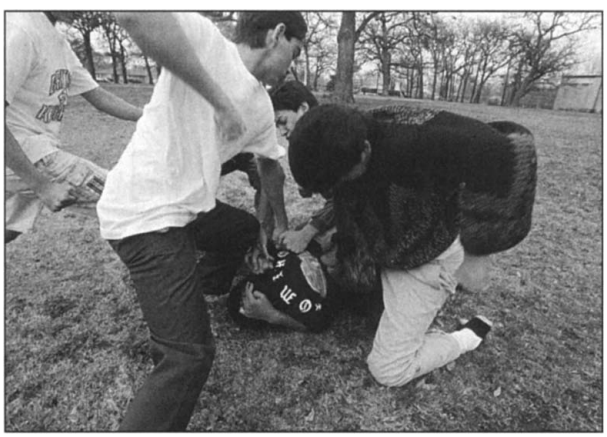
On page 851, a team led by clinical psychologists Terrie Moffitt and Avshalom Caspi, both of King's College London and the University of Wisconsin, Madison, reports that a certain form of a gene that breaks down neurotransmitters makes men more likely to be violent, but only if they were maltreated as children. "This is a very important piece of work," says geneticist Greg Carey of the University of Colorado, Boulder. "It's pretty convincing for just a single study."

The gene codes for an enzyme called monoamine oxidase A (MAOA), which metabolizes several kinds of neurotransmitters in the brain. By getting rid of excess neurotransmitters, MAOA helps keep communication between neurons functioning smoothly. Studies of lab animals show that knocking out the *MAOA* gene makes adult mice more aggressive. The first suggested evidence in humans came from a 1993 report of a Dutch family (*Science*, 18 June 1993, p. 1722). Several men in this family had a defective *MAOA* gene—none of the enzyme was found in their cerebrospinal fluid—and were prone to impulsive bouts of aggression. But because the mutation is extremely rare, no one has replicated the finding in other families.

To see whether the *MAOA* gene influences aggressive behavior in the broader population, Moffitt and Caspi's team turned to New Zealand's Dunedin Multidisciplinary Health and Development Study. The study, begun in 1972, has followed 1037 children since birth. Hoping to get as homogeneous a genetic background as possible, Moffitt and Caspi selected 442 subjects with four white grandparents. "It's about as refined as it can be," Moffitt says.

As expected, the team discovered that severely maltreated boys were more likely to exhibit so-called antisocial behavior than boys who had suffered little or no abuse. But the researchers also found that antisocial behavior was more likely in males with the genotype for low *MAOA* activity who had been mistreated. The 55 boys in this group were about twice as likely to have been diagnosed with conduct disorder in adolescence as the 99 mistreated boys with the high-activity genotype. And they were three times more likely to be convicted of a violent crime by age 26. Although the 55 males who had experienced moderate or severe maltreatment and also had the low-activity genotype made up only 12% of the study group, they committed 44% of the crimes. "They're doing four times their share of rape, robbery, and assault," Moffitt says.

But environmental influences were critical, Moffitt found. In the absence of abuse, having the low-activity genotype didn't make boys any more likely to be antisocial. Jon Beckwith of Harvard Medical School in Boston agrees, although he'd like to see the finding replicated: "I would use this as a wonderful class example of how social factors can play an enormous role in expression of behavioral traits." Moffitt views the results as an example of how accounting for environmental factors can help reveal a gene: "Finding the stressor can be a magic key."



Two strikes. Men who have a certain genotype for a brain enzyme—and were abused—tend to be more prone to violence.

CREDIT: JAY DICKMAN/CORBIS

There are caveats. The link between the *MAOA* alleles and the activity of the enzyme in these males is only inferred, Beckwith points out. Also potentially confounding the study is that antisocial behaviors might depend on social situations, not just genes, adds sociologist Troy Duster of New York University.

Replicating the results will be important, researchers say, although this might be easier than in previous studies because the sample was drawn from the general population. Confirmation could also lead to better intervention strategies. Social workers and therapists would benefit from knowing which abused kids are most at risk, notes criminologist Alfred Blumstein of Carnegie Mellon University in Pittsburgh.

Legal implications are less clear. Although some attorneys might argue that the *MAOA* genetic defect results in diminished capacity, Hal Edgar of Columbia Law School in New York City doesn't think judges will buy it. "This particular study in and of itself is not going to shape the [legal] culture," he says. And experts warn that it's much too early to discuss whether drugs might counter the effects of low *MAOA* activity.

Experts also say that it's important to remember that many genes probably influence violence and other antisocial behaviors. Or as Carey says, the strongest genetic marker for violence is still the presence of a Y (male) chromosome.

–ERIK STOKSTAD

NEUROSCIENCE

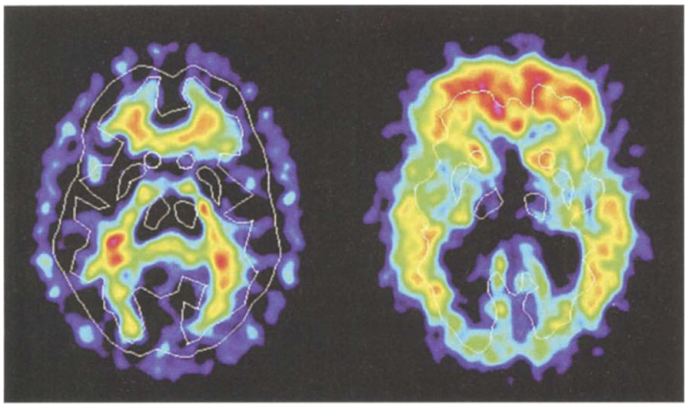
Long-Awaited Technique Spots Alzheimer's Toxin

STOCKHOLM—Alzheimer's disease is notoriously difficult to diagnose, particularly as it begins to take hold. Researchers suspect that therapies, when they become available, will work best if given early, however, raising the need for a test that spots the first signs of this dementia-causing disease. On 24 July at the International Conference of Alzheimer's Disease and Related Disorders here, a team revealed the first images from a positron emission tomography (PET) technique that picks up one of the defining—and first—features of Alzheimer's disease.

"People are going to point to this particular presentation and say, 'This is when we started making progress' " on visualizing Alzheimer's disease, says Mark Mintun of Washington University Medical Center in St. Louis, Missouri. This putative marker, as well as others reported at the meeting, could be invaluable not only for diagnosis but also in clinical research, conference attendees say.

Clinicians settle on a diagnosis of

Alzheimer's disease through a process of elimination. Pencil-and-paper tests of memory and problem solving, reports from family members, and standard brain scans suggest that Alzheimer's disease is the culprit, but researchers can't be entirely sure until an autopsy reveals brain tissue riddled with senile plaques and neurofibrillary tangles. The main ingredient in plaques is a protein called β amyloid, which congregates in the brain well before symptoms of dementia appear and is thought to initiate most of the damage.



Aglow with Alzheimer's. The first images of a β-amyloid tracer in humans show an ominous signal in the brains of patients with early symptoms of Alzheimer's disease (right).

CREDIT: W. KLUNK/UNIVERSITY OF PITTSBURGH

Several teams have been racing to find a nonintrusive way to visualize β amyloid in a living brain. William Klunk, Chester Mathis, and colleagues at the University of Pittsburgh spent about 10 years searching for a molecule that fits the bill. Their best candidate, dubbed PIB, crosses the blood-brain barrier, isn't toxic, and can be clipped to a radioactive tag to light up plaques, animal studies have shown.

Starting in February, a group led by Henry Engler of Uppsala University in Sweden injected the molecule into nine people with symptoms of "mild" Alzheimer's disease and five healthy controls and then used a PET scan to see where it went. At the conference, images of the results audibly took the audience's breath away: In healthy people, the marker sailed right through the brain and was well on its way to exiting via the central fluid-filled ventricles. But in people with early Alzheimer's disease, the marker stuck in the cortex, particularly in the frontal lobes and temporal-parietal areas, two of the brain regions most damaged in the disease.

"This is something we've all been waiting for," says Michael Pontecorvo of Mitsubishi Pharma America in Warren, New Jersey. Adds Randy Buckner of Washington University, "this opens a new window on what we think to be the primary marker associated with disease."

β amyloid can be detected in a far more unexpected place, another team reported. Lee Goldstein of Brigham and Women's Hospital in Boston and colleagues have found that the protein also collects in the lens of the eye. What's more, in a postmortem study of eyes from 16 elderly donors, half of whom suffered from Alzheimer's disease, all of the patients had a rare type of cataract on the edges of the lens—apparently caused by β-amyloid deposits. But this doesn't mean that an eye exam for Alzheimer's disease is around the corner, cautions Goldstein, in part because it's not yet clear how early in the disease's progression the protein might become visible in the lens.

Standard brain scans, if analyzed carefully, can also show signs of impending Alzheimer's disease, reported Nick Fox of the National Hospital for Neurology and Neurosurgery in London. By comparing magnetic resonance images taken 1 year apart, Fox and his colleagues found that healthy elderly people lose about 0.2% of their brain volume each year. People who start out with minor memory complaints and progress to Alzheimer's disease, on the other hand, lose about 2.8% a year. This dramatic shrinking is evident in the hippocampus, which helps store memories, and the frontal and temporal lobes.

The technique doesn't provide a quick diagnosis: It takes time to get a clear picture of Alzheimer-like decline. But it could help monitor patients' progress in clinical trials, says Fox. Their performance on tests of memory and problem solving varies a lot from day to day, making it tough to tease out any potential improvements. By providing a more accurate endpoint, he suggests, brain scans might enable researchers to make clinical trials "smaller and faster." –LAURA HELMUTH

ASTROPHYSICS

'Winged' Galaxies Point To Black Hole Mergers

X literally may mark the spot as astrophysicists hunt for colliding black holes. Results of a new mathematical model, published online this week by *Science* (www.sciencemag.org/cgi/content/abstract/1074688), maintain that cross-shaped radio galaxies harbor massive black holes that suddenly flipped their spins, probably by absorbing black holes from other galaxies. When combined with a census of these distinctive galaxies, the model suggests that such titanic encounters happen about once a year in the cosmos.

Observations of galactic cores, including

ScienceScope

Trickle-Down The global economic slump could delay construction of the world's most powerful particle accelerator now that a key parts supplier has gone bust. Babcock Noell Nuclear in Würzburg, Germany, is supposed to supply one-third of the 1236 superconducting magnets needed for the Large Hadron Collider (LHC), being built at the European particle physics laboratory CERN near Geneva. But on 4 July, Noell's parent company, the industrial giant Babcock Borsig AG, declared bankruptcy. Noell is now in the hands of receivers—and physicists are wondering if they'll get their magnets on time.

The bankruptcy "came as a real surprise," says LHC director Lyndon Evans, and it threatens to add to LHC's woes. The project is already $300 million over its original $1.6 billion budget and 2 years behind schedule, with completion now slated for 2007 (*Science*, 28 June, p. 2317).

Noell executives say the company remains profitable despite the parent company's troubles, and they plan on finishing CERN's magnets. If they can't, two other magnet suppliers—Italy's Ansaldo and France's Alstom-Jeumont—will scramble to meet LHC's needs.

Dependence Day Greater autonomy and a $3.5 million annual bank account might sound like a pretty good deal to most people—but not to the National Science Board, the presidentially appointed oversight body for the National Science Foundation (NSF).

Last week the Senate Appropriations Committee created a separate account for the 24-member board in its proposed 2003 budget for NSF (see p. 755). It argues that the board—which oversees the $5-billion-plus agency and advises the nation on scientific issues—should be financially and operationally independent of NSF. But board chair Warren Washington says he prefers the status quo, under which the board's staff members are NSF employees and the board gets its resources from the agency. Washington also says that borrowing NSF employees as needed gives the board greater flexibility than hiring a permanent staff.

The board is expected to discuss the idea at its meeting 14 to 15 August. Legislators will also get another crack at it as the spending bill moves through Congress.

Contributors: Adam Bostanci, Charles Seife, Adrian Cho, Jeffrey Mervis