cations resulting from research at universities and a national research center. Herrmann, who denies committing or knowing of any falsifications, has been barred from DFG advisory boards and temporarily suspended from the university while he fights the state science ministry's disciplinary proceedings. Brach, who had admitted to falsifying data in "two or three cases," lost her professorship at Lübeck.

Meanwhile last month, Germany's most prestigious scientific organization, the Munich-based Max Planck Society—which operates 73 basic research institutes—adopted its own new regulations on handling misconduct cases. Max Planck will set up a new standing committee, headed by an outsider, that will investigate any misconduct complaints and recommend sanctions, which will range from a warning, to dismissal. The final decision on sanctions will be made by the Max Planck president.

"I hope universities or other institutions will look closely at our new rules and perhaps use them as an example," says Max Planck President Hubert Markl. "Scientific misconduct is never wanted and never expected. But when it happens, you are apt to do things wrong if you haven't developed procedures on how to handle it." In addition to these new procedures, Markl has asked the Max Planck scientific council to develop a new educational program that will "help sharpen the awareness" of ethics issues at institutes.

The DFG also would like its panel's recommendations to reach a wider audience. It plans to send them to international science organizations, including the European Heads of Research Councils group, the European Science Foundation, and the scientific section of the G-8 organization of industrialized nations. "We hope each nation's scientific institutions [will] make use of these recommendations to examine their own rules," says Frühwald.

The only U.S.-based scientist on the DFG panel was Lennart Philipson, director of New York University's Skirball Institute of Biomolecular Medicine and a former director of Heidelberg's European Molecular Biology Laboratory. "It is healthy for the DFG, the Max Planck Society, and, ultimately, the German universities, to develop clear rules on how to deal with such issues," he said, adding that he particularly applauds the idea of an ombudsman. "Because the hierarchy at universities and at research institutes in Germany is so strong, it is extremely important to have neutral boards to examine such problems."

–Robert Koenig

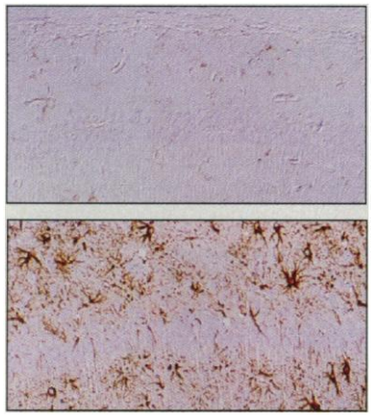
Robert Koenig is a writer in Bern, Switzerland.

NEURODEGENERATIVE DISEASE

B Cells May Propagate Prions

No one knows exactly what causes "mad cow disease" and related neurodegenerative conditions, such as Creutzfeldt-Jakob disease (CJD) in humans. But this uncertainty hasn't kept researchers from wondering how the agents that cause these diseases spread from the site of infection to the brain. New work by neuropathologist Adriano Aguzzi of the University of Zurich in Switzerland and his colleagues now suggests that B cells, a type of immune cell carried in the blood, play an important role in this propagation.

In this week's issue of *Nature*, the Aguzzi team reports that mice lacking B cells are resistant to infection with scrapie, a sheep condition similar to mad cow disease, when they are inoculated with infectious material in areas outside the brain. If B cells are necessary for the disease to propagate, the authors reason, they may also carry the infectious agent.



AGUZZI *ET AL./NATURE* **390**, P. 687 (1997)

Blocked. Brains of B cell–deficient mice *(top)* look healthy, but controls *(bottom)* show signs of scrapie.

In Britain, where 20 young people have already died from a new variant of CJD, possibly originating in meat and other products from cattle infected with mad cow disease, the finding has sparked fears about the safety of donated blood. Experts on the diseases say that no case of CJD in humans has ever been linked to blood transfusions. But the news—which Aguzzi presented at a closed meeting in November—has prompted calls for hemophiliacs in Britain to receive blood clotting factors made by recombinant DNA technology instead of prepared from the pooled blood of many donors. It has also led to suggestions that blood banks should remove white blood cells from donors' blood.

Nailing down how the agents that cause CJD and scrapie travel through the body has been difficult because researchers don't know exactly what to look for. Many believe that the agents are misfolded proteins called prions that propagate themselves, while others think an as yet unidentified slow-acting virus is to blame. But scientists showed as long ago as the 1970s that a wide variety of immune tissues can be infective, especially tonsils, thymus, lymph nodes, and spleen, when injected into animals' brains.

To try to pin down what particular component of the immune system carries the agent, the Aguzzi team tried to infect a number of mouse strains that had been engineered to lack specific immune cells or molecules. They found that mice lacking T cells or the immune-system protein interferon γ were infected as easily as normal mice, but mice that had no B cells were resistant. Out of 27 such mice, none developed symptoms of scrapie after more than a year (up to 534 days), although at least four of them did show evidence of scrapie in their brains. All other mice developed scrapie within 8 months.

Other researchers do not find the results particularly surprising. They note that the tissues previously shown to be infective are rich in B cells. More recent, unpublished work by neuroscientist Paul Brown of the National Institute of Neurological Disorders and Stroke in Bethesda, Maryland, and Robert Rohwer of the Molecular Neurovirology Unit at the Veteran's Administration Medical Center in Baltimore shows that both white blood cells and plasma from infected animals can transmit disease. And at a number of meetings Rohwer has reported that he and his colleagues have infected at least one hamster—out of 22 tested—with scrapie through a transfusion.

The researchers caution, though, that the results only show that blood-borne transmission is possible in the laboratory, and say nothing about the likelihood of it in humans or animals. They point out that no human case of CJD has been traced to a blood transfusion. There is a "tremendous amount of epidemiology that all speaks against the possibility of blood-borne transmission of the agent," Aguzzi says. Rohwer points out, however, that while those results apply to classic CJD, with which "we have been living since the very first transfusion," the situation may be different for the U.K.'s new CJD variant.

There's at least one indication that the immune system could play a bigger role in transmitting the new variant. While doctors have never spotted abnormal prion proteins in the tonsils of patients with classic CJD, in new variant patients, tonsils are "full of abnormal proteins," Aguzzi says.

Aguzzi suspects that the B cells in tonsils carry the prions. But it is not clear whether all B cells harbor the infectious agent, or if only a subset do so. Says Aguzzi, "There is still a tremendous amount of work to be done." That's surely one of the only things on which all researchers in the field can agree.

–Gretchen Vogel