Biomedicine

Mad cow disease diagnosed in humans

News that mad cow disease might have cropped up in 12 people this spring—perhaps as a result of eating meat from infected British cattle (SN: 4/13/96, p. 228)—sent researchers scrambling to their labs. There they focused on PrP, a protein suspected of causing that disease and some other fatal brain disorders in humans, including Creutzfeldt-Jakob disease (CJD).

Now, British researchers report they have direct evidence that mad cow disease, or bovine spongiform encephalopathy, was indeed transmitted from cattle to people. The evidence lies in PrP's shape, which appears to determine whether it coexists harmlessly with other proteins in brain cells or wreaks havoc.

Earlier studies indicated that normal PrP and its warped, disease-causing alter ego, known as a prion, are biochemically identical. This finding led researchers to conclude that changes in PrP structure result in functional differences, which would account for the different symptoms among infected species. They also theorized that a prion, which has no genetic material, replicates by twisting normal PrP into its own image.

John Collinge of the Prion Disease Group in the Imperial College School of Medicine at St. Mary's in London and his colleagues report in the Oct. 24 NATURE that they can exploit the different shapes to trace the transmission of prion strains within and between species.

The team used a standard laboratory technique to generate a band pattern for each protein. They found that most prions taken from different hosts formed distinctly different patterns. Since the proteins are biochemically alike, the variations probably signal differences in their shape.

The band pattern of prions taken from people who died of the human variant of mad cow disease matched the pattern from mice and monkeys infected in the lab with mad cow disease. It differed from the band pattern of prions from people with CJD. Such differences could form the basis of a new diagnostic test (SN: 10/12/96, p. 238), the researchers say.

Adriano Aguzzi and Charles Weissmann of Zurich University say in an accompanying editorial that the new work represents "an exciting new approach" to the study of prions and the role they play in these rare brain diseases.

Did rabies fell Edgar Allan Poe?

When literary figure Edgar Allan Poe collapsed in front of Ryan's Saloon in Baltimore on Oct. 3, 1849, everyone assumed the writer's boozy lifestyle had finally taken its toll.

Not so, says R. Michael Benitez of the University of Maryland Medical Center in Baltimore. Benitez' analysis of historical records shows that Poe probably died of rabies, a viral disease of the central nervous system.

Poe was taken to a hospital in Baltimore, where he suffered from delirium and tremors, both common in alcoholics who have not had a drink for 5 to 10 hours. After 3 days, he recovered briefly, then lapsed into delirium and confusion. The writer remained in this state until his death on Oct. 7, 1849.

The relapsing nature of Poe's illness doesn't match the symptoms of alcohol withdrawal, Benitez says. Furthermore, historical evidence suggests that Poe had abstained from alcohol for the 6 months prior to his collapse. He refused an alcoholic drink in the hospital.

The symptoms of Poe's illness mirror those of a rabies infection, Benitez notes in the September MARYLAND MEDICAL JOURNAL. Even more telling, Poe had great difficulty drinking water during his hospital stay. Rabies produces involuntary spasms of the throat that make swallowing difficult.

Poe was a well-known animal lover and was especially fond of cats, which can transmit the rabies virus. There was no record of an animal bite preceding Poe's ailment, but the illness can take more than a year to surface, Benitez says.

Nutrition

Iron pills improve kids' test scores . . .

Using dietary supplements to treat iron deficiency improves a girl's learning and memory, a new study finds.

Teenage girls face a particular risk of iron deficiency. "A lot of iron is used up laying down new muscle and expanding the blood volume" to meet the needs of a growing body, explains pediatrician Ann B. Bruner of Johns Hopkins Children's Center in Baltimore. However, this is also the time that girls begin to menstruate. "And it's probably the addition of this [iron loss in] menstrual blood," she says, "that leaves many girls in a negative iron balance during much of puberty."

Working with four local schools—two public, two private—Bruner's team recruited 716 girls age 14 to 18 for blood tests. Of the 112 girls who were iron-deficient but not anemic, 81 entered the study. After giving each girl four tests of attention and memory, Bruner's group randomly assigned half of the teens to receive daily pills containing 260 milligrams of iron and the other half to get identical pills containing no iron. Eight weeks later, all of the girls took the battery of tests again.

"Adults and teenagers with iron deficiency frequently report they have trouble concentrating," Bruner notes. In this study, however, iron had no effect on scores from the three tests measuring attention. It made a difference only on the Hopkins Verbal Learning Test. Here, each girl listened to 12 words and then tried to recall them. The researchers administered the exercise three times, using the same list, and added the correctly recalled words from all three trials.

Both groups of girls scored the same on the initial group of tests. Eight weeks later, the iron-supplemented girls remembered an average of one word more on each of the second and third trials, she reports in the Oct. 12 LANCET. The girls who received the ironfree pills scored the same on both batteries of tests.

Bruner would prefer that teens—especially the 15 percent of girls with iron deficiency—eat an extra serving of perhaps spinach, raisins, or wheat germ three times a week. But as a mother, she's learned that children don't always cooperate, making vitamin and mineral supplements a useful fallback. Indeed, she finds that none of the teenage girls she treats eats breakfast. "They're too busy doing their hair," she says. Then they skip those "nasty" school lunches, snack on chips and sodas after school, and pick up dinner at the fast-food franchise where they work. "Unfortunately," she laments, "this is real."

. . . as does breakfasting near test time

Children who eat breakfast at school score better on tests of verbal learning and memory than kids who either skip breakfast or eat at home, an Israeli study found. Its authors suggest that a meal's effect on learning may trace to how long it elevates concentrations of sugar in the blood.

Pediatrician Nachum Vaisman of Kaplan Hospital in Rehovot and his coworkers studied 569 children age 11 to 13. After giving the children a series of tests, the researchers assigned two-thirds of them to a breakfast of sugared cornflakes and milk at school for the next 2 weeks. At the end of the study, they retested the children, administering the tests just 30 minutes after the school-fed children had completed their meals.

On each of 10 different measures of memory and learning, children who ate at school performed better than those who had eaten at home and at least as well as—but usually better than—those who had skipped breakfast. Vaisman's team suspects that a transient increase in blood sugar explains why the children fed at school outperformed those who had eaten 2 hours earlier at home. They report their findings in the October ARCHIVES OF PEDIATRICS AND ADOLESCENT MEDICINE.