Mad Cow Disease, Human Illness Tied

Two groups of researchers have tightened the link between mad cow disease and a devastating brain disorder that has stricken 21 people in the United Kingdom in the past 3 years. Those people apparently ate infected beef.

The human illness, a variant of Creutzfeldt-Jakob disease (CJD), causes brain damage, dementia, tremors, and death. The mysterious agent behind this disease riddles the brain with tiny holes, a condition called spongiform encephalopathy (SN: 10/12/96, p. 238).

In the new studies, the researchers injected brain tissue from infected cows into a group of mice. Other mice received tissue from people who had died of variant CJD.

Less than a year after the injections, both groups of mice displayed the same bizarre symptoms, such as walking backward. The disease ultimately proved fatal, as it has in humans and cows.

The two studies, both published in the Oct. 2 NATURE, "confirm the notion that the variant CJD is very similar to BSE," or bovine spongiform encephalopathy, says Blas Frangione, a pathologist at New York University.

Classical CJD, first described in 1920, is a rare disease of unknown origin that strikes about 1 in 1 million elderly people. The recent variant, however, attacks people of any age.

One of the new studies, in which a team from Edinburgh examined the brains of mice given tissue from diseased cows or people with variant CJD, showed that the mice suffered brain degeneration. Brain material from infected domestic cats and two zoo antelopes—which ate food thought to have contained infected tissue from other animals—also caused the disease in mice. However, mice given tissue from older people who had died of classical CJD survived more than 600 days into the trial and showed no symptoms of disease.

The cause of mad cow disease and of the new human CJD variant has been difficult to pinpoint in living cows or people. At the center of the mystery is the prion, a malformed version of a protein that normally resides in mammalian brain tissue. The function of this protein is unknown, says Robert G. Will, a neurologist who coauthored the Scottish study and heads the National CJD Surveillance Unit at Western General Hospital in Edinburgh. Prions are thought to induce molecules of the normal protein to mimic their shape and participate in damaging the brain.

"I think the balance of evidence is very much in favor of the agent [being] a prion," Will says, "[but] my personal view is we still cannot be certain of that."

A competing theory, whose popularity has waned in recent years, holds that the prions derive from a virus or other infectious agent.

The second study, by John Collinge of Imperial College at St. Mary's Hospital in London and his colleagues, focused on prions. Using biomolecular analyses, the researchers found the same prions in mice regardless of whether they had been given tissue from humans with variant CJD or tissue from infected cows. The test provides "compelling evidence" that the two diseases are caused by the same prion strain, they report.

The human variant of the brain disease appears to have originated in sheep. The United Kingdom stopped farmers from adding sheep offal to cattle feed in 1989 after it appeared that the practice was infecting cows with scrapie, a communicable brain disease in sheep. Cattle continued to die in subsequent years as the disease took hold. The first reports of a person falling ill with the CJD variant surfaced in early 1996 (SN: 4/13/96, p. 228).

Jeffrey Almond of the University of Reading and John Pattison of University College London note in a NATURE commentary that the studies don't establish how many people are likely to get the disease or how long it takes the disease symptoms to appear in humans. They don't even prove that people contract it by eating beef. Still, contaminated beef remains "the most likely exposure," they concede. —*N. Seppa*

Satellite views Earth's living plumage

From their sunlit shallows to their pitch-black depths, the oceans provide an estimated 99 percent of Earth's living space, yet biologists have lacked a means of monitoring broad patterns of life in the seas. Now, a long-awaited color-sensing satellite fills that gap, according to NASA, which started releasing data from the instrument last month.

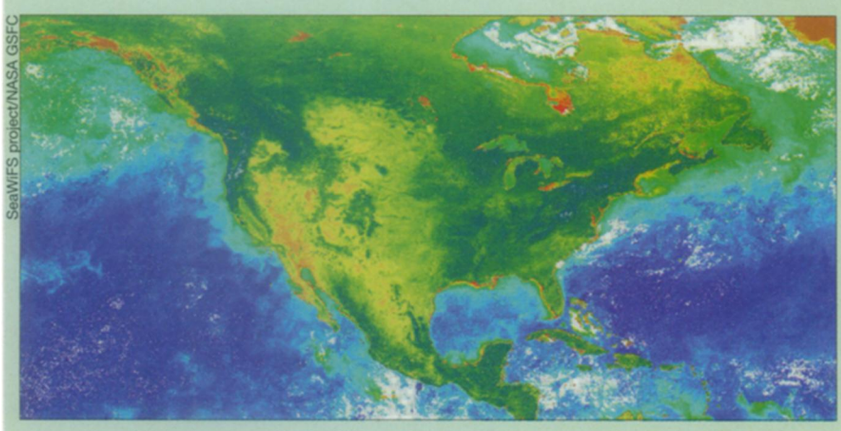
"We're going to see Earth the way we've never seen it before," says Gene C. Feldman of NASA's Goddard Space Flight Center in Greenbelt, Md.

The new satellite monitor, named the Sea-Viewing Wide Field-of-View Sensor (SeaWiFS), collects daily images of every point on Earth in eight separate wavelengths of light. By determining the color of the oceans, the instrument can indirectly measure the concentration of tiny marine plants called phytoplankton, which anchor the marine food chain.

SeaWiFS replaces a color-scanning satellite sensor that stopped working in 1986. Unlike the older version, however, SeaWiFS can measure the amount of plant life on land as well as in the oceans.

Marine biologists say the data from these satellites are invaluable for detecting the large-scale distribution of ocean life, which is impossible to see from the limited vantage point on board a ship. "You get a picture of what the world is really like, and the world is not exactly the way you thought it was," says Richard T. Barber of Duke University's Marine Laboratory in Beaufort, N.C.

—*R. Monastersky*



SeaWiFS project/NASA GSFC

A new satellite captures life in false color. In the oceans, reds denote the highest concentration of plant life, with lesser amounts shown in green and aqua. Dark blue indicates marine deserts. On land, vegetation appears dark green.