dinosaurs offer clues to another paleontological mystery—how the first birds got off the ground. Perhaps, says Chiappe, what started as predatory movements of the arms of a dinosaur like the one in Patagonia eventually evolved into flapping that was rapidly refined into modern flight.

Yet another fossil described at the meeting, a fledgling bird the size of a sparrow that lived briefly in Sierra del Montsec, Spain, about 130 million years ago, is offering a glimpse of how flight was perfected. Like *Archaeopteryx*, this sparrow-sized creature had a primitive, dinosaurian skull, with a depression between its eye socket and brain case, reported Chiappe, who has studied the bird with a team from the University Autonoma of Madrid. But unlike *Archaeopteryx*, which had a short, primitive wing, this small, Spanish bird and its closest cousins probably had a longer, more sophisticated wing with the beginnings of a "slot"—the set of quill-like feathers that allows modern birds to maneuver better at slower speeds (*Nature*, 1 August, p. 442). "This is exciting because you see that in the early evolution of birds, the flight apparatus was a priority," says Chiappe.

The new findings haven't swayed Feduccia or University of Kansas paleontologist Larry Martin, another skeptic of the bird-dinosaur link. Says Feduccia: "It's biophysically impossible to evolve flight from such large bipeds with foreshortened forelimbs and heavy, balancing tails"—exactly the wrong anatomy for flight. And as for the suite of other strange-looking characters that link dinosaurs and birds, Martin says that they could have been inherited from an ancient reptilian ancestor that gave rise to both dinosaurs and birds. "In my opinion, the theropod origin of birds will be the greatest embarrassment of paleontology in the 20th century," says Feduccia.

But to Ostrom, who 23 years ago breathed new life into the theory that dinosaurs gave rise to birds, the wealth of new fossils speaks for itself: "I'm satisfied I know where birds have come from."

–Ann Gibbons

MAD COW DISEASE

Protein Test Favors BSE-CJD Link

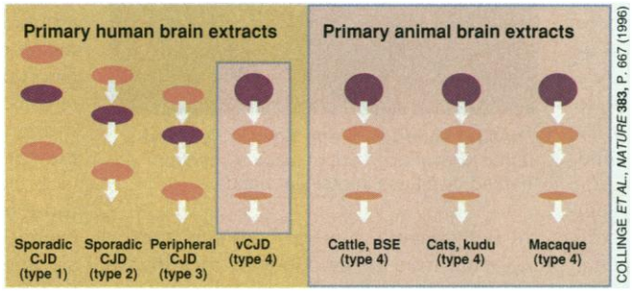
LONDON—"Beef blamed for CJD deaths" ran the front-page headline in the London *Times* on 24 October, after British scientists published the first evidence of biochemical similarities between recent anomalous cases of Creutzfeldt-Jakob disease (CJD) in humans and bovine spongiform encephalopathy (BSE) in cows, mice, and other animals. By analyzing the proteins thought to transmit these degenerative diseases of the brain, researchers had shown that the new variant of the human disease, known as vCJD—which has now struck 14 young people since 1994—resembles BSE more closely than classical CJD.

The new work, which appeared in last week's *Nature*, strengthens the case that beef infected with BSE, or "mad cow disease," is the cause of vCJD, but scientists do not think the link has yet been proved. They see the British team's success at chemically characterizing BSE and four strains of CJD as a milestone of a different kind. "The important result is that [vCJD] can be diagnosed" by applying this technique, says neurovirologist Corinne Lasmézas of France's Atomic Energy Commission (CEA) in Fontenay-aux-Roses.

Suspicions of a BSE-CJD link first surfaced last March, when British government scientists who monitor CJD spotted 10 cases of the disease in people under the age of 45. CJD normally afflicts only the elderly, and in all 10 of the new cases the pattern of brain damage, or neuropathology, was different from the usual CJD pattern (*Science*, 29 March 1996, p. 1798). The search was on for further evidence that the new vCJD might in fact be a human BSE infection.

The test, developed by John Collinge's team at Imperial College Medical School at St. Mary's Hospital, London, provides what virologist Clarence Gibbs of the U.S. National Institutes of Health (NIH) in Bethesda calls "the second piece of evidence suggesting a link." It does so by giving biochemists a way to differentiate chemically between different strains of CJD, BSE, scrapie in sheep, and related diseases—something that has been impossible until now.

The infectious particle responsible for these diseases seems to be an abnormal form of a naturally occurring protein, dubbed a prion (*Science*, 12 July 1996, p. 184). Prions apparently lack DNA or RNA, which can be decoded to indicate the strain of infection if the agent is a virus or a bacterium. What's more, the basic chemical makeup of prions causing different strains of the same disease is identical. Prion researchers have proposed instead that the different strains reflect differences in the protein's shape, which current techniques cannot distinguish.



COLLINGE *ET AL.*, *NATURE* **383**, P. 667 (1996)

Spot check. Collinge's test shows clearly that the pattern of prion masses in new variant CJD mirrors that of BSE.

Collinge's team, extending a method that had previously shown a distinction between two "normal" CJD strains, examined how many sugar groups are attached to the prion proteins. They extracted prion proteins from infected brains and separated them by mass on a gel. Classical CJD material produces a characteristic pattern of three bands, corresponding to prions with no attached sugar groups, with one, or with two. The researchers identified three variations on this three-band pattern, but in each case prions with one attached sugar group predominated.

The team then analyzed samples from the first 10 vCJD cases and found a fourth pattern in which prions with two sugars were the most common. When the scientists later ran samples of brain from BSE-infected cattle, mouse, cat, and monkey, they displayed an almost identical two-sugar pattern. "It's the most suggestive evidence so far" of a link between the animal and human diseases, says biochemist Byron Caughey of the NIH Rocky Mountain Laboratory in Montana.

But the welcome for the new test has not been unanimous. "It's a very interesting suggestion that the [sugar] pattern is common, but I don't know how strong that evidence is," says Pierluigi Gambetti at Case Western Reserve University in Cleveland, Ohio. Vindication of Collinge's results will have to wait until the only other method of identifying strains yields results. It involves injecting infected brain material into mice, then comparing the incubation time and neuropathology when the animals come down with the disease. This experiment, now under way at the Neuropathogenesis Unit of the Institute of Animal Health in Edinburgh, "will furnish a much more persuasive view" of BSE-vCJD similarities, says Paul Brown of the NIH Laboratory of Central Nervous System Studies in Bethesda. Gibbs agrees: "We're all awaiting the outcome."

Collinge is now looking at whether the test could be applied to tissues other than brain, such as the tonsils and lymph nodes, making it easier to acquire samples while the patient is alive. If the work leads to a test able to detect the disease early on, he says, "we should think seriously about developing therapeutics."

–Claire O'Brien

Claire O'Brien is a writer in Cambridge, U.K.