Slender science feeds beef panic

On 20 March, Stephen Dorrell, the UK's Secretary of State for Health, told the House of Commons that a distinctly new form of the neurodegenerative disorder Creutzfeldt-Jakob Disease (CJD) had been identified in ten people and the most likely cause was eating beef from cattle with bovine spongiform encephalopathy (BSE). Panic ensued, sweeping through Britain and beyond (including a European Union ban on importation of British beef, and an announcement by the New York State Department of Agriculture that 13 cows imported from Britain before 1989 would be destroyed). But the evidence for a link between English beef and CJD is minimal at best, and government mishandling of the slim scientific information bears, in large part, the responsibility for the collapse of the historic British beef market.

Dorrell made his calamitous statement on the recommendation of the government's Spongiform Encephalopathy Advisory Committee. Unlike comparable groups in the United States, the committee's deliberation was behind closed doors, and the scientific findings that prompted Dorrell's announcement were not published until 8 April in the *Lancet*, too late to head off the ensuing panic.

Although Dorrell did say that the risk associated with eating beef was "small," many people recalled a former minister saying there was no link between BSE and CJD, and thus ignored the caveat. Other government ministers compounded the problem by distancing themselves from the controversy, saying that it was the scientists, not the ministers, who said there was a risk in eating beef, and people should make their judgments on the basis of the science — a disingenuous suggestion, as the data were not yet published.

The "science" of CJD and BSE is mostly unanswered questions, including; What is the agent or agents that causes BSE and CJD (currently thought to be mutant prion proteins)? Where does BSE come from in the first place, and can it cause CJD? There are few solid answers. However, these questions of epidemiology and pathogenesis must be addressed before determining what risk, if any, comes with eating beef.

CJD is rare, occurring in about one in a million people. In 1990, the CJD Surveillance Unit was set up in Edinburgh to see whether the BSE epidemic in cattle affected the incidence of CJD in humans. Of the 207 cases examined since 1990, the CJD unit found that 10 were atypical. It is these investigations that were published in the *Lancet*.

However, atypical cases of CJD may be even more common than these results suggest, and may have occurred well before BSE was identified in cattle. Gareth Roberts, a neuropathologist at Smith-Kline Beecham Pharmaceuticals, and his colleagues examined brain tissues of the more than 1,000 cases of dementia collected between 1964 and 1990. They found 19 cases of spongiform encephalopathy, eleven with typical symptoms of CJD and six with atypical symptoms. Roberts argues that a reinvestigation of all archival material from people who died with neurological disorders before the age of forty-five, especially those prior to 1986 (when BSE was discovered), should be undertaken to see if there is a link between BSE and CJD.

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A "Beefeater," reminder of the historic importance of UK beef.

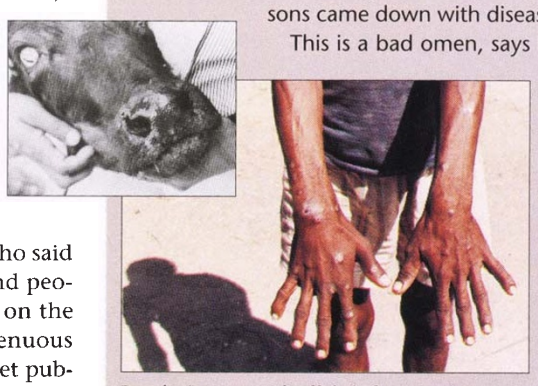
Is buffalo pox now a human disease?

Human-to-human transmission of buffalo pox virus (BPV) infection in recent outbreaks in India is causing concern among medical researchers. Although they publicly say there is nothing alarming about it, the researchers caution that the virus should be monitored, as it could become a public health problem.

Pox disease in buffalo and cattle has been known in the Indo-Pakistani subcontinent since 1934. The disease has also been found in Indonesia, Russia and Egypt. The virus was known to spread to humans coming in close contact with infected animals, causing mild fever, pox lesions, and occasionally, swelling of the lymph nodes.

But there is a new threat: "In this country we are now seeing BPV infection spreading from one person to another," says Kalyan Banerjee, director of the National Institute of Virology (NIV) in Pune. Banerjee and his colleagues, who investigated a recent outbreak in Beed district of Maharashtra, found five human cases — all infants who had never been near buffalo. During the same period, in another district of Maharashtra, 58 persons came down with disease, several of them unexposed to buffalo.

This is a bad omen, says N.P. Gupta, former director of NIV, because the animal virus can change its behavior once it starts circulating in humans. "However, what changes *will* take place in BPV is difficult to tell," he says.



Pox lesions on a buffalo's face and on an infected person's arms and hands.

"We suspect that the animal virus has already acquired some virulence," says Krishna Kumar Datta, director of the National Institute of Communicable Diseases in New Delhi. In 1977, Institute researchers investigating an outbreak in Dhulia district of Maharashtra concluded that the human illness "was mild and uneventful, lasting for one to two weeks." In contrast, Datta says the illness during this year's Maharashtra outbreak "was of a longer duration and more severe." The situation is worsened by the fact that pox outbreaks in buffaloes have become frequent, and that rodents are now suspected to be reservoirs of BPV.

Recent studies, according to Banerjee, indicate that the BPV genome circulating in Maharashtra is different from the original strain found in Hissar, north of Delhi, 35 years ago. Gupta says it is now imperative for health authorities to isolate the virus strain from each human case and preserve it for future studies.

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