THE WALL STREET JOURNAL. ONLINE

December 31, 2003

BUSINESS WORLD

By HOLMAN W. JENKINS, JR.

Don't Let Mad Cow Make You Crazy

Scientists are still picking at the mystery of mad cow and related diseases, but we don't mind cheating a bit by anticipating the final chapter. When all the rocks have been turned over, such diseases will be seen as rare and spontaneous occurrences in many species after a certain protein, known as a prion, arranges itself in an abnormal shape, causing progressive brain damage.

Long before mad cow was discovered, humans were known to suffer Creutzfeldt-Jakob disease at a rate of one per million. The low incidence, evenly distributed among human populations, seems to indicate a random mutation rather than infection by diet or environmental contact. The same is likely to be true of other mammals. Not every mad cow, in other words, necessarily "catches" the disease from something it ate or touched.

It took the British disaster, with the attendant human illnesses and destruction of the country's cattle industry, to bring the possibility of an epidemic of prion disease into focus, though the risk had been noticed in earlier cases of human cannibalism, farm-raised minks and wild deer and elk. What did we learn from the British experience? That if you really work at it, you can create a system of mass feeding to ensure that prion disease, once it appears, will spread widely. And the British really worked at it: No other country has managed to produce a similar outbreak, though elements of British husbandry were adopted in other parts of the world.

Nearly 200,000 British cows were found to have been infected, and 4.5 million were slaughtered as a precaution -- because the disease incubates slowly and is hard to detect. It stands to reason, then, that millions of Britons consumed thousands of pounds of beef from infected cattle, and scientists once were willing to gamble their reputations by predicting thousands, even millions, of human cases over 50 or 60 years. Contrary to such

ABOUT THE AUTHOR

Holman W. Jenkins Jr. is a member of the editorial board of The Wall Street Journal and writes editorials and the weekly Business World column.

Mr. Jenkins joined the Journal in May 1992 as a writer for the editorial page in New York. In February 1994, he moved to Hong Kong as editor of The Asian Wall Street Journal's editorial page. He returned to the domestic Journal in December 1995 as a member of the paper's editorial board and was based in San Francisco. In April 1997, he returned to the Journal's New York office. Mr. Jenkins won a 1997 Gerald Loeb Award for distinguished business and financial coverage.

Born in Philadelphia, Mr. Jenkins received a bachelor's degree from Hobart and William Smith Colleges in Geneva, N.Y. He received a master's degree in journalism from Northwestern University in Evanston, Ill., and studied at the University of Michigan on a journalism fellowship.

Mr. Jenkins invites comments to holman.jenkins@wsj.com[1].

expectations, though, the number of people dying from presumed mad cow infection peaked at 28 in 2000 and has declined every year since. This year the number of deaths is just 16.

Could there be a nasty surprise? Yes, virtually all who've died display a genetic similarity, common to 40% of the population. It's just conceivable the rest of British humanity isn't genetically immune but simply takes much longer to show the disease. At the moment, most scientists are taking the data at face value and concluding there won't be a large epidemic of human illness, that in fact the total number of deaths may never break 200.

Even in Britain, four times as many people over the past decade died of normal one-in-a-million Creutzfeldt-Jakob disease as died of the mad cow-spawned version. For that matter, 5,000 Americans a year die from commonplace foodborne illnesses. Bottom line: The fluky cycling up of a mad cow epidemic in Britain through intensive reuse of animal brain matter as animal feed was and remains sui generis.

This should shed a poignant light on the tears of joy and huzzahs of triumph emanating from the U.S. Agriculture Department. Behind the celebration is the fact that the sole American case so far has been tentatively traced back to Canada, and even more delightful, the cow is believed to have been 6.5 years old. Interpretive mountains are being built on these molehills: If somebody's lax enforcement of feed rules was the culprit, please Lord, let it be Canada's. Even better if we can show -- in this case, by a mere four months -- that the animal had been born before suspect feeding practices were banned.

Uh huh. Trying to prove the cow got its disease from contaminated feed may be a fool's errand. Canada discovered its own first case in May but still hasn't fingered a source of infection. Never mind: In the future, the incidence of mad cow may well consist entirely of rare spontaneous cases. Japan now tests every cow slaughtered -- 1.2 million last year -- and has found nine cases in all, with the two latest being cattle born after feeding practices were cleaned up. Both also show an "atypical" pattern not seen in standard mad cow, though the atypical pattern has now been echoed in two recent French finds and two in Italy.

It's possible, therefore, that we are already returning to a world in which mad cow may be a rare and spontaneous disease -- and, importantly, not one spread by infection.

The hurdle Americans have to get over, as other nations have, is to realize the existence of mad cow in the world is not a reason to avoid beef. German beef consumption dropped 40% when the first mad cow was discovered but has bounced back strongly now that nearly 300 have been identified. Spain and Portugal are alone in still reporting rising numbers of British-legacy mad cow, with 274 cases this year.

Yet beef consumption has returned to normal there too.

For the time being, we will have to live with overkill as each instance of mad cow is treated as a harbinger of a British-style epidemic. Having taken steps to prevent brain tissue from entering the human and cattle food chain, though, we have probably done all that was necessary to stop the occasional spontaneous occurrence from threatening humans or cattle.

Other steps are probably more than enough, though we've taken those too. Indeed, nothing at this point will allay demands that "downer" cows (those that can't walk) be excluded from the nation's meat supply, however costly the gesture. A lame cow, in almost every instance, will not be one with mad cow. Likewise, an appearance of health is no proof that a cow hasn't come down with a case of prion disease.

Maybe, instead, it's not too early to begin noticing a silver lining in Britain's miserable experience, source of our current backward-looking panic. What we've been forced to learn about prion sickness may well point to cures for Alzheimer's, Parkinson's and other diseases where prions are increasingly suspected of playing a role.

URL for this article:
http://online.wsj.com/article/0,,SB107283716324279800,00.html

Hyperlinks in this Article:
(1) mailto:holman.jenkins@wsj.com

Updated December 31, 2003