Phillips, IBM attorney Michael Templeton wrote that publication of the study would represent "a misappropriation of data that Dr. Clapp has no right to use for such purposes," and that "IBM expressly reserves all of its rights to take any appropriate action." Clapp's attorney, Indira Talwani, calls that "a threat." Clapp told me that if he hadn't received the letter from IBM's lawyers, he would have gone ahead and published the study in COEM. Brooks maintains that publishing the study would violate four court orders; Talwani, in contrast, maintains that the study is in the public domain.

I did not claim that "IBM has 'blocked' publication of legitimate scientific research into workplace health issues." In fact, the story describes the University of Alabama study led by Elizabeth Delzell and clearly states that Delzell intends to publish that study in a peer-reviewed journal.

Finally, the fact that a judge ruled a scientific analysis irrelevant to a particular lawsuit on legal grounds does not necessarily reflect on the quality of the underlying science. I asked three independent epidemiologists unconnected with the litigation to look at the study. All three agreed that Clapp and his co-author Rebecca Johnson's conclusions, while preliminary, were scientifically valid and deserving of further study.

DAN FERBER

Prion Diseases and a Penchant for Brains

IN THEIR REPORT "BALANCING SELECTION AT THE prion protein gene consistent with prehistoric kurulike epidemics," S. Mead *et al.* provide an interesting and persuasive argument on how kuru transmitted by endocannibalism resulted in balancing selection at codon 129 of human prion protein gene in the Fore linguistic group of Papua New Guinea (25 Apr. 2003, p. 640). I am not convinced, however, that acquired prion disease causing the selective pressure results mainly from cannibalism in other populations around the world. Although not commented on in the Report, it is intriguing that the sampled Turkish population has the second highest polymorphic frequency of 129V (0.48), which is close to the that of the Fore population (0.55), and even closer to the expected equilibrium frequency of 0.45 (*1*).

Given that there are no records of cannibalism in Turkish history, and the impossibility of the existence of undetected cannibalism of the scale observed in the Fore, there must be a different explanation for this high frequency. Sheep and cow brains are delicacies in Turkey, and certain popular restaurants (kelle paça) specialize in sheep brains and heads. The brains of sheep, cattle, pigs, primates, and other mammals are esteemed dietary items by

people in many parts of the world, including the Middle East; north and central Africa; some Caribbean islands; central, south, and southeast Asia; Russia; Iceland; southern Europe; and North and Latin America. In Mead *et al.*'s Table 1, the samples from many of these groups also show relatively high M129V frequencies.

In cases of bovine spongiform encephalopathy (BSE) and scrapie, prion diseases found in cattle and sheep, the prions are concentrated in the brain and other nervous tissues (*2*), and the high frequency of M129V in many groups can also be explained by regular exposure to prion diseases as a result of frequent consumption of animal brains. Even though Mead *et al*. mention the possibility of animal prion disease as an explanation for the observed pattern, they clearly favor cannibalism as the main cause, citing the evidence for prehistoric cannibalism in certain human populations. However, variant Creutzfeldt-Jakob disease (vCJD) has been transmitted from BSE-infected cattle to humans (*3*); the BSE agent can infect various animals (such as cattle, goats, pigs, and sheep) that are often consumed by people (*4*); the significant increase in vCJD incidence in Leicestershire, England, was a result of the contamination of cow meat with central nervous system tissue (*2*); the high incidence of CJD in Libyan Jews (*5*) and Slovakian herdsmen (*6*) correlates with a high preference for sheep brains; and in rural Kentucky, a fondness for squirrel brains suggests a similar connection (*7*). An important reason for the dietary preference for brain tissue is its high fat content. This can also be seen in wild chimpanzees feeding on monkeys (*8*), and it is likely to have been present in prehistoric human populations feeding on a diversity of animals.

That 85% of human transmissible spongiform encephalopathies are sporadic and have no known etiologies (*2*); that many people regularly consume ruminant intestinal tissue, which also has a high concentration of BSE prions (*9*); and that other possible vectors of human prion diseases include rodents (*10*) and flies (*11*) necessitate further caution in assigning cannibalism as the predominant cause of balancing selection at the prion protein gene in human populations worldwide.

Prion diseases can have very long incubation periods (*12*, *13*), and carriers may not show clinical symptoms (*14*), which meant that solving the riddle of kuru took many years. Therefore, prion diseases in people, especially outside the developed world, are likely to be misdiagnosed and underrecorded. These factors make studying the relationship between dietary habits and prion diseases even more difficult. However, given the public health implications, research in this field is crucial. To the extent possible, consumption of mammal brains, intestines, and other highly infective tissue should be evaluated as a potential causal factor in any study of human prion diseases.

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References

1. P. W. Hedrick, *Science* **302**, 57 (2003).
2. C. R. Trevitt, P. N. Singh, *Am. J. Clin. Nutr.* **78**, 651S (2003).
3. A. F. Hill *et al.*, *Nature* **389**, 448 (1997).
4. D. M. Taylor, *Clinical Microbiol. Infect.* **8**, 332 (2002).
5. M. Alter, *Science* **186**, 848 (1974).
6. E. Mitrova, in *Search for the Causes of Multiple Sclerosis and Other Chronic Diseases of the Central Nervous System*, A. Boese, Ed. (Verlag Chemie, Weinheim, Germany, 1980), pp. 356–366.
7. J. R. Berger, E. Weisman, B. Weisman, *Lancet* **350**, 642 (1997).
8. B. Bower, *Science News* **147**, 316 (1995).
9. H. W. Moon, *J. Food Protect.* **59**, 1106 (1996).
10. G. P. Concepcion, E. A. Padlan, *Med. Hypotheses* **60**, 699 (2003).
11. O. Lupi, *Int. J. Epidemiol.* **32**, 425 (2003).
12. A. G. Dickinson, H. Fraser, G. W. Outram, *Nature* **256**, 732 (1975).
13. G. Stoltenburg-Didinger, *Fleischwirtschaft* **82**, 107 (2002).
14. A. F. Hill *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **97**, 10248 (2000).