

Devouring a cannibal myth

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In 2003, a study of prion disease associated with cannibalism was published in *Science*,¹ with significant implications for anthropology. Now an equally significant rebuttal has appeared in *Genome Research*.²

Prion diseases³ are classed in three ways as to their occurrence: sporadic (particularly puzzling, with no identifiable antecedent event); familial (by genetic inheritance); or acquired (by contact with an infectious agent). Kuru is classed in the last category. In the 1950s, an epidemic of the prion disease Kuru was discovered among the Fore linguistic group of tribes in Papua New Guinea. The general consensus was that it was transmitted in the practice of ritual cannibalism. Endocannibalism, the ritual consumption of deceased ancestors as an act of respect, is thought to have been part of Fore culture until the practice was abruptly ceased in the late 1950s with the establishment of government patrols into the highlands.⁴

Heterozygosity selected?

In the 2003 *Science* study, Simon Mead of University College London and a team of researchers presented genetic evidence ‘consistent with prehistoric Kurulike epidemics’.¹ Mead’s study centred on an analysis of the human prion protein gene *PRNP*. It has been established elsewhere that homozygosity at codon 129 contributes significantly to greater susceptibility to Kuru. In light of this, Mead *et al.* first reported that ‘Fore women who participated in cannibalistic feasts were significantly more likely to be heterozygous at codon 129 than expected by chance.’⁵ Second, Mead *et al.* did an international comparison of the *PRNP* gene, and announced that there is an international higher rate of occurrence of heterozygosity than would be expected

from chance. In conclusion, Mead *et al.* noted that this finding indicates that natural selection favoured heterozygous genes worldwide in response to worldwide occurrences of Kuru or a similar disease, which was spread by cannibalism:

‘... available evidence appears consistent with the explanation that repeated episodes of endocannibalism-related prion disease epidemics in ancient human populations made coding heterozygosity at *PRNP* a significant selective advantage leading to the signature of balancing selection observed today.’¹

Problems for the cannibalism hypothesis

Mead’s article came in for some criticism in 2004 when Martin Kreitman and Anna Di Rienzo, both of the University of Chicago, published a paper suggesting the presence of ascertainment bias in Mead’s study.⁶ As Kreitman and Di Rienzo point out, ‘Under neutrality, the majority of variants are expected to be rare but intermediate-frequency alleles are also expected.’⁷ But in Mead *et al.*’s study, only common *PRNP* polymorphisms were scored.

‘Without resequencing every individual in the final study, the authors likely biased their results toward common polymorphisms, the signature of balancing selection.’⁸

This is a classic case of ascertainment bias, where the test statistic is thrown off because the data had

‘prior restrictions about whether any particular SNP [single nucleotide polymorphism] will be “discovered” (i.e. included) in a sample.’⁹

Soldevila *et al.*’s definitive study² confirms Kreitman and Di Rienzo’s opinion. The new study in *Genome Research* criticizes Mead *et al.*’s international findings after a thorough resequencing of the prion-coding region of 174 individuals from around

the world. Soldevila *et al.*’s thorough study confirmed the suspicions of Kreitman and Di Rienzo:

‘... we can reject the existence of an ancient, stable, balanced polymorphism of the kind that skews the frequency spectrum to an excess of intermediate frequency variants described by Mead *et al.* (2003). Thus, a general pattern of balancing selection, presumably related to prion diseases and cannibalism, can be rejected in human history and is shown to be due to ascertainment bias.’¹⁰

Cannibalism and worldviews

The significance of this research for anthropology should be obvious. The initial Mead paper suggested (1) worldwide endocannibalism creating (2) worldwide prion disease epidemics. Such a finding fits well with the basic evolutionary view of primitive man. The anthropologist Raymond Dart wrote that the ‘blood-spattered, slaughter-gutted archives of human history ... accord with early universal cannibalism’,¹¹ but others have challenged this view.¹² Worldwide cannibalism logically follows from the presupposition that man is an animal and, if animals prey on each other, humans will do likewise. Mead used his study to promote this idea but his result was only ‘consistent with’ such an explanation. Why did he not seek any other explanation? And why did he not see the ‘ascertainment bias’ problem? Was it because the result was consistent with his worldview?

Biblical historical anthropology would not predict worldwide cannibalism.¹³ The key difference between biblical anthropology and secular anthropology is the presence of a written history against which individual facts can be checked and interpreted. The biblical historical record leaves a place for *instances* (tribal or regional¹⁴) of cannibalism in that it provides very clear descriptions of sinful man; however, cannibalism



Was there widespread cannibalism among our ancestors? The latest research does not support this hypothesis.

is not a *normative* human practice. In biblical chronology, it would be difficult to find a period of time in which to place a virtually worldwide practice of cannibalism, and extremely difficult where it is accompanied by worldwide Kuru-like epidemics.

Mead's genetic argument for what one writer called a 'cannibalism signature'⁵ on the human genome was in effect an argument contra the biblical account. The more thorough study of Soldevila *et al.* refutes the statistics underlying Mead's argument, and accords with the predictions of biblical anthropology.

References

1. Mead, S. *et al.*, Balancing selection at the prion protein gene consistent with prehistoric Kuru-like epidemics, *Science* **300**:640–643, 2003.
2. Soldevila, M. *et al.*, The prion protein gene in humans revisited: lessons from a worldwide resequencing study, *Genome Research*, Epub, 20 December 2005.

3. Probably the best-known prion disease was the much-publicized 'mad cow' disease, bovine spongiform encephalopathy (BSE). For an introduction to the prion diseases, see Ridley, R.M. and Baker, H.F., *Fatal Protein: The Story of CJD, BSE, and Other Prion Diseases*, Oxford University Press, New York, 1998.

4. See Ridley and Baker, ref. 3, ch. 3.

5. Phillips, M.L., No cannibalism signature in human gene, <www.the-scientist.com/news/display/22927/>, 7 April 2006.

6. Kreitman, M. and Di Rienzo, A., Balancing claims for balancing selection, *Trends in Genetics* **20**(7):300–304, 2004.

7. Kreitman and Di Rienzo, ref. 6, p. 301.

8. Phillips, ref. 5, summarizing Kreitman and Di Rienzo.

9. Kreitman and Di Rienzo, ref. 6, p. 302.

10. Soldevila *et al.*, ref. 2, p. 7.

11. Quoted in Leakey, R.E. and Lewin, R., *Origins*, E.P. Dutton, New York, p. 208, 1977.

12. For example, Richard Leakey believes that cannibalism has always been rare (see Leakey and Lewin, ref. 11, pp. 217–221). William Arens has gone much further, arguing that cannibalism is basically a myth. See Kolata, G., Anthropologists suggest cannibalism is a myth, *Science* **232**:1497–1500, 1986. Arens' radical argument provoked very strong words in response: Riley, T.J., Lidz, T. and Schryer, D.R., The existence of cannibalism, *Science* **233**:926, 1986. For a more recent summary of research on cannibalism and early man, see Gibbons, A., Archaeologists rediscover cannibals, *Science* **277**:635–637, 1997.

13. For one general creationist analysis of cannibalism, see Morris, H.M., *The Biblical Basis of Modern Science*, Baker, Grand Rapids, MI, pp. 402–405, 1984.

14. Because of this, the regional cannibalism for which there is a very strong archaeological argument (as in Gibbons, ref. 14), does not pose a problem for biblical anthropology.