same procedures. Both human and animal bones have many cut marks in places similar to those seen in bones butchered by modern techniques[2,4]. All the long bones were broken, to extract marrow[2–4], and the patterns of discard are identical, showing identical treatment of human and animal remains.

If, as Bahn suggests, human bones indicate secondary burial, it logically follows that the Fontbrégoua people hunted, herded and butchered, but did not eat, food animals, and that they gave secondary burial to boars, deer, sheep, roe deer, badgers and marten. At Fontbrégoua the burial hypothesis is not a reasonable alternative and is, in fact, implausible; cannibalism is the only satisfactory explanation.

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BAHN REPLIES — I stated, on the basis of ethnographic data from Australia which had been specifically compared with Villa and Courtin's evidence[5], that mortuary rituals among some Aborigines can produce the same results as alleged cannibalism.

I would like to make three brief points. First, the Fontbrégoua human remains are not mixed with animal bones, but treated separately. Second, the limitation of the remains to a brief episode could equally be argued to indicate a mortuary ritual rather than a sudden and short-lived craving for human flesh. Third, one could apply the same logic to both animal and human remains, and assume identical butchery and discard equals consumption in both cases.

At first glance, the last possibility appears to be correct; but whereas there is much evidence throughout history for the consumption of animals by humans, there is virtually no solid, reliable evidence from any period for human cannibalism. Although Fontbrégoua is the best documented case yet published for the existence of prehistoric cannibalism, its separate treatment of human remains, together with the Australian ethnography which can account for every feature in its data, weaken that case considerably.

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1. Bahn, P. G. *Nature* **348**, 395 (1990).
2. Villa, P. *et al. Bull. Soc. Préhist. Française* **82**, 389–421 (1985).
3. Villa, P. *et al. Science* **233**, 431–437 (1986).
4. Villa, P. & Mahieu, E. *J. hum. Evol.* (in the press).
5. Pickering, M. P. *Aust. Archaeol.* **28**, 35–39 (1989).

Analysis of choroideraemia gene

SIR — Choroideraemia is a heritable X-linked disease that leads to progressive retinal degeneration and blindness. Although this disease is so rare that no accurate data on prevalence are obtainable, choroideraemia is important because it and gyrate atrophy of the retina and choroid are the two hereditary retinal degenerations in the general category of retinitis pigmentosa that can be specifically recognized by the appearance of the retina. The choroideraemia locus has been mapped[1,2] to the q21 band on the X chromosome and DNA clones that span Xq21 deletions in patients with the disease have been isolated. Sequence analysis reveals that these deletions interrupt an open-reading frame of 948 base pairs that could encode a protein of 316 amino acids. RNA transcripts from this open reading frame are absent or structurally altered in patients with choroideraemia, supporting the presumption that it represents the choroideraemia gene. To understand how alterations or the absence of the choroideraemia gene contribute to retinal degeneration its function in eye physiology must be identified. But as Cremers *et al.* point out[1], neither the nucleotide nor the predicted amino-acid sequence of the choroideraemia gene have revealed significant homology to gene or protein sequences in the nucleic acid or protein databases (up to August, 1990). Moreover, no topogenic sequences or domains with biological function have so far been identified. We now report the identification of a significant similarity in the amino-terminal region of the putative choroideraemia gene product to a recently described p25A GDP dissociation inhibitor (smg p25A-GDI) (ref. 3), following a search of the updated NBRF protein database.

In vitro, p25A-GDI inhibits the rate of GDP release from a ras-like protein, p25A (ref. 3). It has been proposed, as in the case of GAP, IRA1, IRA2 and NF1[4,5], that smg p25A-GDI could negatively regulate the signalling pathway of p25A by decreasing the intracellular concentration of the active GTP-bound form[6]. But neither the effector function nor the physiological role of smg p25A have been clarified.

An alignment of the regions of smg p25A-GDI and choroideraemia that are similar is shown in the figure. Choroideraemia and smg p25A-GDI are 76 per cent similar over a 46 amino-acid sequence (amino acids 22–67 of choroideraemia and 210–255 of p25A-GDI) with 23 identities and 12 conservative amino-acid residue replacements with no gaps. Particularly striking is the conservation of three prolines at positions 35/223, 39/227, and 47/235 (choroideraemia/p25A-GDI), suggesting a similar secondary structure.

Signal transmission in the retina is mediated by transducin, a trimeric G protein that links photoactivation of rhodopsin to activation of retinal cyclic GMP-phosphodiesterase[7]. Analysis of rod transducin α-subunit reveals several regions that are similar to the *ras* gene products which correspond to domains involved in guanine nucleotide binding and GTPase activity[8].

It is, therefore, possible that transducin and the ras-like proteins share related control mechanisms. It is not known whether the similar domain between choroideraemia and p25A-GDI is involved directly in guanine nucleotide metabolism or if it represents a recognition motif for another function. Furthermore, it is noteworthy that Bowes *et al.*[9] have demonstrated that retinal degeneration in the rd mouse model is caused by a defect in the β-subunit of the rod cGMP-phosphodiesterase. So we feel it is likely that other lesions in the visual signalling pathway involving GTP could precipitate retinal degeneration. The homology we present here should help in investigating the function of the choroideraemia gene in retinal disorder.

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                                                 *           *                             *
Chor. 22  K  N  F  L  H  C  L  G  R  Y  G  N  T  P  F  L  F  P  L  Y  G  Q  G  Q  G  E  L  P  Q  C  F  C  R  M  C  A  V  F  G  G  I  Y  C  L  R  H  S  V
          ||    |           || |  || || ||    |  || |  || |  || || || ||    ||    || || || || ||    ||    || |     || |  |  || ||    ||    ||    |  |  ||
GDI 210   K  L  Y  S  E  S  L  A  R  Y  G  K  S  P  Y  L  Y  P  L  Y  G  L  G  L  G  E  L  P  Q  G  F  A  R  L  S  A  I  Y  G  G  T  Y  M  L  N  K  P  V
                                                 *           *                             *
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Alignment of the choroideraemia product and smg p25AGDI. ‖, similar amino acids; |, conservative replacements; *, proline residues. A Monte-Carlo statistical analysis of this homology was carried out using the EUGENE sequence analysis program (Baylor University) yielding a score of 12, indication a high probability of relatedness.

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1. Cremers, F. P. N., van der Pol, D. J. R., van Kerkhoff, L. P. M., Wieringenm, B. & Ropers H. -H. *Nature* **347**, 674–677 (1990).
2. Nussbaum, R. L., Lewis, R. A., Leskom, J. G. & Ferrell, R. *Am. J. hum. Genet.* **37**, 473–481 (1985).
3. Matsui, Y. *et al. Molec. cell. Biol.* **10**(8), 4116–4122 (1990).
4. Martin, G. *et al. Cell* **63**, 843–849 (1990).
5. Ballester, R., Sanders, D. A. & McCormick, F. *Cell* **63**, 851–859 (1990).
6. Bourne, H., *et al. Nature* **348**, 125–132 (1990).
7. Yau, K. & Baylor, D. A. *A. Rev. Neurosci.* **12**, 289–327 (1989).
8. Tanabe, T., *et al. Nature* **315**, 242–245 (1985).
9. Bowes, C. *et al. Nature* **347**, 677–680 (1990).

Scientific Correspondence

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