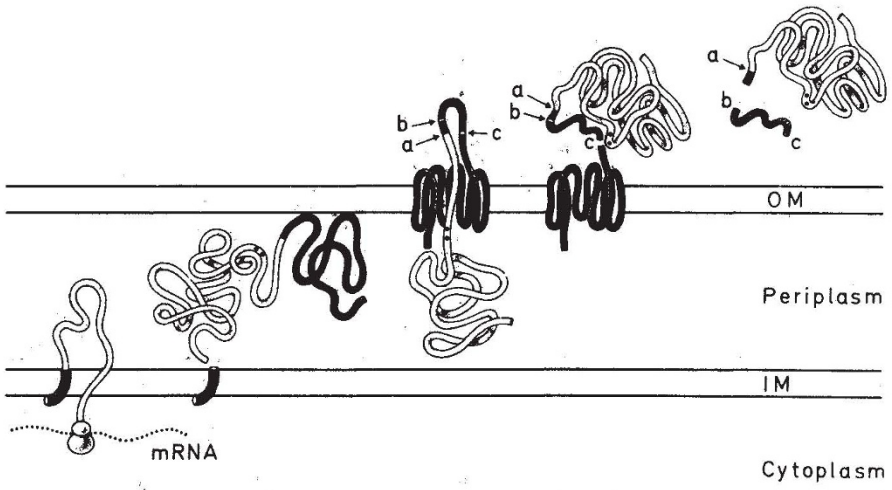

Fig. 5 Model for extracellular secretion of IgA protease. The leader peptide guides the IgA protease precursor into the periplasmic space via the regular co- or post-translational transport route taken by the majority of periplasmic and outer membrane proteins (ref. 31 for review). In a second step, the carboxy-terminal helper associates with the outer membrane to form a pore for excretion of the protease domain. This domain, still connected to the helper, gains its active conformation in the process of secretion. A proform of the IgA protease is released from the helper by autoproteolysis and further develops into α-protein and the mature enzyme.

brane fractions from gonococci and analysed them for the presence of the β-protein. Using a β-domain specific monoclonal antibody raised against fp42, we detected in the outer membrane of gonococci a 45 K protein (Fig. 4*c*), which precisely matches the β-domain in size.

Based on these results, we propose a model for the extracellular secretion of IgA protease (Fig. 5). We assume that the signal peptide allows the precursor to pass the inner membrane. Owing to its amphipathic features the helper becomes incorporated into the outer membrane to form a pore for the excretion of the protease domain and the α-protein. During excretion through the helper pore, IgA protease acquires an active conformation and is released from the helper by autoproteolysis. The extracellular form of IgA protease further develops by autoproteolysis to yield mature protease and the α-protein. This two-step mechanism is supported by the detection of residual IgA protease activity in the periplasm of cells harbouring either an intact *iga* gene or a helper mutant gene. A definitive distinction however is not possible between this model and a one-step secretion mechanism, using the inner membrane transport machinery, where the helper function is to direct the enzyme to a putative inner/outer membrane junction[13,14].

Few other examples of protein excretion in Gram-negative bacteria are known in detail[15–17]. The gonococcal IgA protease system is striking in that a single protein serves all specific functions of its own secretion, not only in the authentic host but also in the foreign environment of *E. coli*. It may prove a suitable system for manipulating and studying protein export in Gram-negative organisms. With respect to the virulence function of gonococcal IgA protease, our demonstration that IgA_1 is not an exclusive target of this enzyme suggests that other sensitive targets might exist in humans. Two other components, the membrane-associated helper and the soluble α-protein, appear to be intrinsically connected with IgA protease, it might be worthwhile to consider the significance of these factors in gonococcal pathogenesis.

We thank K. Neifer and B. Tries for technical assistance, K. Lamberti for artwork, R. Haas for computer assistance, G. Jähnig, J. R. McGhee and K. Muralidharan for critical suggestions, E. Beck and H. Schaller for releasing the pEX vector system before publication, S. Schweitzer and R. Sommer of Genbiotek GmbH for collaborating on the DNA analysis, and M. Ganss for hybridoma work. The project was supported through BMFT grant BCT 0364-1 and through generous commitments of H. Hoffmann-Berling and K. Rother.

Received 3 July; accepted 10 November 1986.

1. Plaut, A. G., Gilbert, J. V., Artenstein, M. S. & Capra, J. D. *Science* **1290**, 1103–1105 (1975).
2. Kilian, M., Mestecky, J. & Schrohenloher, R. E. *Infect. Immun.* **26**, 143–149 (1979).
3. Koomey, J. M., Gill, R. E. & Falkow, S. *Proc. natn. Acad. Sci. U.S.A.* **79**, 7881–7885 (1982).
4. Halter, R., Pohlner, J. & Meyer, T. F. *EMBO J.* **3**, 1595–1601 (1984).
5. Bricker, J., Mulks, M. H., Plaut, A. G., Moxon, E. R. & Wright, A. *Proc. natn. Acad. Sci. U.S.A.* **80**, 2681–2685 (1983).
6. Fishman, Y., Bricker, J., Gilbert, J. V., Plaut, A. G. & Wright, A. in *Pathogenic Neisseriae Proc. Fourth Int. Microbiol.* (eds Schoolnik, G. K. *et al.*) 164–168 (1985).
7. Rahr, S., Halter, R., Müller, H., Pohlner, J. & Meyer, T. F. in *Pathogenic Neisseriae Proc. Fourth Int. Symp. Soc. Microbiol.* (eds Schoolnik, G. K. *et al.*) 157–163 (1985).
8. Cooper, M. D., McGee, Z. A., Mulks, M. H., Koomey, J. M. & Hindman, T. L. *J. inf. Dis.* **150**, 737–744 (1984).
9. Strebel, K., Beck, E., Strohmaier, K. & Schaller, H. *Virology* **57**, 983–991 (1986).
10. Watson, M. E. E. *Nucleic Acids Res.* **12**, 5145–5164 (1984).
11. Pollock, M. R. & Richmond, M. H. *Nature* **194**, 446–449 (1962).
12. Mulks, M. H., Plaut, A. G., Feldman, H. A. & Frangione, B. *J. exp. Med.* **152**, 1442–1447 (1980).
13. Lory, S., Tai, P. C. & Dabis, B. D. *J. Bact.* **156**, 695–702 (1983).
14. Bayer, M. E. in *Bacterial outer membranes: biogenesis and function* (ed. Inouye, M.) 167–202 (Wiley, New York, 1979).
15. Wagner, W., Vogel, M. & Goebel, W. *J. Bact.* **154**, 200–210 (1983).
16. Mackman, N., Nicaud, J.-M., Gray, L. & Holland, I. B. *Molec. gen. Genet.* **201**, 529–536 (1985).
17. Hawley, D. K. & McClure, W. R. *Nucleic Acids Res.* **11**, 2237–2255 (1983).
18. Schaller, H., Grey, C. & Herrmann, K. *Proc. natn. Acad. Sci. U.S.A.* **72**, 737–456 (1975).
19. Gentz, R. Bujard, H. *J. Bact.* **164**, 70–77 (1985).
20. Rosenberg, M. & Court, D. *A. Rev. Genet.* **13**, 319–353 (1979).
21. Shine, J. & Dalgarno, L. *Proc. natn. Acad. Sci. U.S.A.* **71**, 1342–1346 (1974).
22. Maxam, A. M. & Gilbert, W. *Meth. Enzym.* **65**, 499–560 (1980).
23. Sanger, F., Nicklen, S. & Coulson, A. R. *Proc. natn. Acad. Sci. U.S.A.* **74**, 5463–5467 (1977).
24. Ghou, P. Y. & Fasman, G. D. *A. Rev. Biochem.* **47**, 251–276 (1978).
25. Hopp, Th. P. & Woods, K. R. *Proc. natn. Acad. Sci. U.S.A.* **78**, 3824–3828 (1981).
26. Neu, H. D. & Heppel, L. A. *J. biol. Chem.* **240**, 3685–3692 (1965).
27. Craven, G. R., Steers, E., Jr & Anfinsen, C. B. *J. biol. Chem.* **240**, 2468–2473 (1965).
28. Howard, S. P. & Buckley, J. T. *J. Bact.* **161**, 1118–1124 (1985).
29. Stern, A., Brown, M., Nickel, P. & Meyer, T. F. *Cell* **47**, 61–71 (1986).
30. Hunkapiller, M. W., Lujan, E., Ostran der, F. & Hood, L. E. *Meth. Enzym.* **91**, 227–236 (1983).
31. Randall, L. L. & Hardy, S. J. S. *Microbiol. Rev.* **48**, 290–298 (1984).

Erratum

Energy conversion catalysis using semiconducting transition metal cluster compounds

N. Alonso Vante & H. Tributsch
Nature **323**, 431–432 (1986).

IN this letter, the metal atoms in the Fig. 1 legend were incorrectly defined. They should read: ●, Mo; ⊜, Ru; ○, Se.

US Supreme Court to review Louisiana appeal

Thomas H. Jukes
Nature **324**, 423–424 (1986).

IN this commentary a sentence in the seventh paragraph contained an incorrect age for the Earth and the Universe. It is correct as printed here.

"This places the date of creation as about 10,000 years ago, which accounts for opposition by creationists towards any of the natural sciences that state that the Earth and the Universe are thousands of millions of years old."