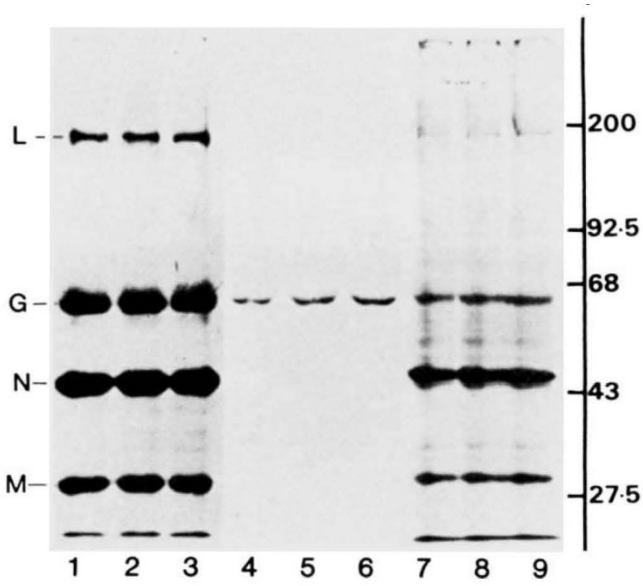

Fig. 3 Effect of IFN on protein G. L-cells were treated with IFN and metabolically labelled as described in Fig. 1 legend. To prepare purified viruses, the culture media were collected and centrifuged at 10,000*g* for 10 min to remove large cellular debris. The supernatants were centrifuged at 48,000*g* for 2 h. The pellets containing the virus were further purified by the sucrose density gradient procedure of Leavitt *et al.*[13]. The viruses were resuspended in MEM and centrifuged for 1 h at 100,000*g* to remove the sucrose. The viruses were then resuspended in MEM (without supplements) containing 100 μg ml^{-1} antifibronectin coupled to Sepharose 4B gel beads and incubated for 1 h at 5 °C on a rotatory mixer. The gel beads with the fibronectin containing membrane particles were removed by centrifugation at 10,000*g* for 10 min. The purified virus particles were stored in MEM at −70 °C, or immediately analysed by SDS-PAGE. Lanes 1, 2 and 3 represent the highly purified viral particles from cells treated with 0, 30 and 250 U ml^{-1} IFN. Glycoprotein G (lanes 4–6) was immunoprecipitated from 1 ml of a 1% Triton X-100 extract of viral particles (100 μg protein) by the addition of 50 μg of affinity-purified anti-G, and processed as described in Fig. 2 legend. Lanes 7–9 represent the crude viral particles from cells treated with 0, 30 and 250 U ml^{-1} IFN.

Maheshwari *et al.* (ref. 1, Fig. 1) shows that one likewise cannot make a strong case for a significant and specific decrease in glycoprotein G, contrary to their previous reports[6,7].

The results shown in lanes 1–3 of Fig. 3 are with viral particles that have undergone extensive purification, hence it is possible that virus with altered G protein was preferentially lost during purification. This possibility was evaluated by two different approaches. First, we determined the rate of G protein synthesis and glycosylation by pulse labelling with ^{14}C-leucine, ^{3}H-mannose or ^{3}H-glucosamine for 1 h at 3, 6 and 9 h post-infection of L-cells. Cells containing the newly synthesized viral protein were homogenized and analysed by SDS-PAGE. The polypeptide band corresponding to the G protein was cut from the gel, dissolved in 30% hydrogen peroxide and the radioactivity determined by liquid scintillation spectrometry. There was no apparent difference in the specific activity (^{14}C c.p.m./^{3}H c.p.m.) of carbohydrate precursor incorporation in the presence or absence of IFN (30–500 U ml^{-1}). The second approach involved the rapid isolation of viral particles without extensive purification. Media from VSV-infected cells, cultured in the presence or absence of IFN (30–500 U ml^{-1}), was centrifuged at 10,000*g* for 10 min, and the resulting supernatant at 100,000*g* for 30 min. The pellets containing the virus were homogenized and equal amounts of protein were electrophoresed. The fluorograms are shown in lanes 7–9 of Fig. 3. It is apparent that similar results are obtained with crude and highly purified viral preparations.

The results presented here show that IFN does not inhibit the glycosylation of asparagine-linked glycoproteins as previously reported[1]. When [2-^{3}H]mannose was used as an alternative labelling substrate for glycoproteins, similar results were obtained. Therefore, the results obtained with ^{14}C-D-glucosamine are not experimental artifacts. We conclude that the antiviral, antitumour and immunomodulatory actions of IFN are probably unrelated to glycosylation.

The present study does not agree with earlier reports by Maheshwari *et al.* claiming that IFN is an inhibitor of glycosylation[1] and that VSV released from IFN treated cells is deficient in glycoprotein G[6,7] and, as a consequence, is less infectious than virions released by untreated cells[1,5–8]. The basis for the apparent discrepancies in results are not immediately obvious as we used what we believe to be the same cells, virus and IFN as the previous investigators, and as we also used the same general procedures. Also, we were able to repeat the above results with a similar preparation of IFN obtained from Lee Biomolecular Research Laboratories. There is a major difference in experimental approach relative to viral purification; however, the apparent contradictions cannot be due to the isolation procedure as we were unable to repeat their findings even when we used less pure viral preparations. It is also important to point out that Maheshwari and Friedman[1,5–8] never studied the effect of IFN on the synthesis of host cell glycoprotein. Also, we have no information relevant to their finding that IFN inhibits the transfer of *N*-acetylglucosamine from UDP-*N*-acetylglucosamine into glycolipids[1].

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1. Maheshwari, R. K., Banerjee, D. K., Waechter, C. J., Olden, K. & Friedman, R. M. *Nature* **287**, 454–456 (1980).
2. Yamada, K. M. & Olden, K. in *Tunicamycin* (ed. Tamura, G.) 119–139 (Japan Scientific Societies Press, Tokyo, 1982).
3. Pouyssegur, J., Shiu, R. P. C. & Pastan, I. *Cell* **11**, 941–947 (1977).
4. Olden, K., Pratt, R. M. & Yamada, K. M. *Cell* **13**, 461–473 (1978).
5. Maheshwari, R. K. & Friedman, R. M. *J. gen. Virol.* **44**, 261–264 (1979).
6. Maheshwari, R. K., Demsey, A. E., Mohanty, S. B. & Friedman, R. M. *Proc. natn. Acad. Sci. U.S.A.* **77**, 2284–2287 (1980).
7. Maheshwari, R. K., Jay, F. T. & Friedman, R. M. *Science* **207**, 540–541 (1980).
8. Maheshwari, R. K. & Friedman, R. M. *Virology* **101**, 399–407 (1980).
9. Vogt, P. I. in *Fundamental Techniques in Virology* (eds Habel K. & Salzman, N.) 198–211 (Academic, New York, 1969).
10. Dulbecco, R. in *Microbiology* (eds Davis, B. A., Dulbecco, R. Eisen, H. N. & Ginsberg, H. S.) 874–877 (Harper & Row, Cambridge, Massachusetts, 1980).
11. Olden, K., Pratt, R. M. & Yamada, K. M. *Proc. natn. Acad. Sci. U.S.A.* **76**, 3343–3347 (1979).
12. Schubert, D. J. *Molec. Biol.* **51**, 287–301 (1970).
13. Leavitt, R., Schlesinger, S. & Kornfeld, S. *J. Virol.* **21**, 375–385 (1977).
14. Leavitt, R., Schlesinger, S. & Kornfeld, S. *J. biol. Chem.* **252**, 9018–9023 (1977).
15. Gibson, R., Leavitt, R., Kornfeld, S. & Schlesinger, S. *cell* **13**, 671–679 (1978).
16. Schulze, I. *Virology*. **42**, 890–904 (1970).
17. Compans, R. W., Klenk, H. D., Caliguri, L. A. & Choppin, P. W. *Virology* **42**, 880–889 (1970).
18. Klenk, H. D., Rott, R. & Becht, H. *Virology* **47**, 579–591 (1972).
19. Compans, R. W. *Nature new Biol.* **229**, 114–116 (1971).
20. Cartwright, B. & Brown, F. *J. gen. Virol.* **35**, 197–199 (1977).
21. Bishop, D. H. L., Repik, P. Obijeski, J. F., Moore, N. F. & Wagner, R. R. *J. Virol.* **16**, 75–84 (1975).
22. Yabrov, A. A. *Interferon and Nonspecific Resistance* (Human Science Press, London, 1980).
23. Olden, K. & Yamada, K. M. *Cell* **11**, 957–969 (1977).
24. Laemmli, U. K. *Nature* **227**, 680–685 (1970).
25. Lowry, O. H., Rosenbrough, N. J., Farr, A. L. & Randall, R. J. *J. biol. Chem.* **193**, 265–275 (1951).

Corrigenda

In the letter 'Crystal structure of a microbial ribonuclease, RNase St' by K. T. Nakamura *et al.*, *Nature* **299**, 564–566 (1982), four residues are shown incorrectly in Table 1. In RNase St, residue 212 should be 'Q', in RNase Ms residue 32 should be 'D', in RNase Ba residue 49 should be 'K' and in RNase U_2 residue 75 should be 'D'.

In the article 'Fossil mammals and artefacts from the Middle Awash Valley, Ethiopia' by J. E. Kalb *et al.*, *Nature* **298**, 25–29 (1982), the acknowledgements section should include thanks to the Boise Fund (Oxford University) for supporting one of the authors (Dr P. Whitehead).