cloud chamber appear to confirm this result. Contacts at the edges between the prism faces, or at the center of basal faces, predominate.

Thus it appears that, in addition to a net charge on an ice crystal, a multipole charge-distribution exists while the crystal is growing.

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References

1. R. I. Smith-Johannsen, *Nature* **205**, 1204 (1965).
2. W. S. McEwan, F. K. Odencrantz, C. M. Drew, before American Meteorological Soc. 48th annual meeting, San Francisco, 1968.
3. W. G. Finnegan, L. A. Burkardt, F. K. Odencrantz, V. J. Schaefer, before American Meteorological Soc. 48th annual meeting, San Francisco, 1968.
4. E. J. Langham and B. J. Mason, *Proc. Roy. Soc. London Ser. A* **247**, 493 (1958).
5. S. M. De Micheli and A. R. Licenblat, *J. Atmos. Sci.* **24**, 312 (1967).
6. F. K. Odencrantz and R. W. Buecher, *Science* **158**, 256 (1967).
7. J. Hallett and B. J. Mason, *Proc. Roy. Soc. London Ser. A* **247**, 440 (1958).
8. F. K. Odencrantz, *J. Atmos. Sci.* **25**, 2, 337 (1968).
9. P. V. Hobbs, *ibid.* **22**, 296 (1965).

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Virus-Directed Protein Synthesis in Different Animal and Human Cells

Abstract. *The relative proportions of viral gene products (viral proteins) that are synthesized in different types of animal cells infected with the same RNA virus inoculum were compared. The relative rates of synthesis of the various virus proteins late in infection were remarkably constant regardless of cell type infected. This was true in cell lines that produced only small amounts of virus and virus proteins, as well as in those that gave large yields of viruses and virus proteins.*

Certain small RNA viruses adsorb to, replicate in, and kill host cells from a wide variety of animal sources (*1*). However, the efficiency of replication in different cells varied more than 1000-fold. The observed specificity of cell susceptibility and resistance to two different picornaviruses led us to suggest the possibility that different animal cells might translate the same viral RNA genetic message differently, or at different rates. Such a possibility has been indirectly reinforced by findings of chromatographic differences among transfer RNA's obtained from different animal cells (*2*).

If certain codons in a polycistronic viral RNA message were translated more slowly in some cells than in others, and if there were a number of initiator sites on the viral message, it might be possible to demonstrate differential rates of translation of various virus genes by comparing the ratios of gene products obtained when the same virus RNA acts as a messenger in two different types of cells. Maizel and his co-workers (*3*) have developed an excellent electrophoretic method for separating and identifying labeled proteins synthesized during the later stages of poliovirus infection. This method is used for comparison of the proteins synthesized in a variety of different animal cells after infection with poliovirus or mengovirus. It is shown below that the relative rate of synthesis of various picornavirus proteins is influenced little, if at all, by the host cell. Only the total amount of virus proteins synthesized varies from host cell to host cell. Their relative proportions remain constant.

The two viruses employed for this study were type 1 (Mahoney) poliovirus and mengovirus. Methods of cell culture and of infection have been described (*1*). Monolayer cultures containing about 10^7 cells were infected at an effective multiplicity of approximately 100.

Virus-coded proteins were labeled late in infection with a mixture of C^{14}- or H^3-labeled amino acids. The labeled amino acids in Eagle's minimum essential medium were added in small volume (0.6 ml) to thoroughly washed cells, which were then incubated for 1 hour at 37°C. The cells were washed and disrupted in 0.01*M* phosphate buffer (*p*H 7.2) containing 0.1 percent sodium dodecyl sulfate (SDS), 0.5*M* urea, and 0.1 percent mercaptoethanol (*3*). The cell proteins were dialyzed against this buffer for 24 hours. The H^3-labeled virus proteins were then mixed with an appropriate amount of C^{14}-labeled proteins with which they were to be compared, and were subjected to electrophoretic separation in polyacrylamide gels containing 0.1 percent SDS and 0.5*M* urea. The buffer contained 0.1*M* sodium phosphate (*p*H 7.2), 0.1 percent SDS, 0.1 percent ethylenediaminetetraacetate, and 0.1 percent mercaptoethanol (*3*). The gels were crushed on the Maizel linear gel-fractionator (*3*), and the radioactivity was measured with a Beckman scintillation counter.

Gel-electrophoresis techniques were essentially those described by Maizel *et al.* (*3*) except that a 5 percent gel, 23 cm long, was used. Samples were applied in 20 percent glycerol in volumes of 0.3 ml or less. Electrophoresis was carried out at constant voltage at 3 volt/cm for 16 hours.

The labeling amino acids in the H^3-labeling medium were (in microcuries per millimole) L-phenylalanine, 5000; L-tyrosine, 28,200; and L-valine, 2970; those in the C^{14}-labeling medium were L-phenylalanine, 504; L-tyrosine, 305; and L-valine, 267.

When a number of human cell lines were compared by dual label (H^3 and C^{14}) techniques, the electrophoretic positions and relative proportions of poliovirus-specified proteins produced in these cells were almost the same. Figure 1 shows nearly identical poliovirus-directed protein profiles in HeLa cells and in three other human cell lines. Similar results have been obtained with several monkey-kidney cell lines and with primary human-amnion cells.

Poliovirus can replicate only in primate cells because of a lack of receptors in nonprimate ones (*4*), but mengovirus allows comparison of cells from a wide variety of animals because it replicates in the cells of many unrelated animal species (*1*). However, it is clear that mengovirus-directed synthesis is very similar in cells of many different species. Figure 2 shows that mengovirus-specified proteins are nearly identical in electrophoretic position and relative proportions during late stages of infection of L cells, HeLa cells, monkey cells, primary mouse-embryo fibroblasts, and primary human-amnion cells. Note that the electrophoretic patterns of mengovirus proteins are quite different from those of poliovirus. We have found similar patterns with mengovirus-specified proteins produced during infection of rabbit cells, marsupial cells, hamster cells, and others. This is true in cells that give very poor, as well

as in those that support very large, virus yields (*1*, *5*).

Attempts are continuing in this laboratory to find a cell-virus system in which the cell alters the normal pattern of picornavirus protein synthesis, but it is already clear from the above results that the host cell usually has little influence on the relative proportions of viral proteins synthesized. However, it is equally clear that the host cell plays a profound role in determining the rate of synthesis and the total amount of synthesis of viral proteins and virions (*1*, *5*). It is still not known whether cells that replicate mengovirus poorly, and that synthesize viral proteins only at a very low rate, do so primarily because of inefficient translation, or only secondarily as a result of some other hindrance to rapid virus replication (that is, because of breakdown of viral RNA or poor synthesis of viral RNA, or both).

Shapiro *et al.* (*6*) have recently demonstrated that the electrophoretic migration of proteins in 5 percent gels with SDS is inversely proportional to molecular weight, with a nearly linear relation between the molecular weights of 15,000 and 90,000. Most of the virus proteins in our study are within this range, as determined by use of purified proteins for standards (*5*). Therefore, with a single initiator, 1:1 gene product relation, an electrophoretic pattern would occur in which each virus-specified protein peak would be somewhat larger when read from right (anode, proteins of low molecular weight) to left on the graphs. Since the patterns observed in this study, and in the work of Maizel and his co-workers (*3*) with 10 percent gels, deviate considerably from such a profile, it must be concluded that some regulatory mechanism is (or mechanisms are) involved. At least one intricate mechanism affecting these patterns is specific cleavage of very large, newly synthesized viral proteins into smaller functional proteins (*5*, *7*). Whether other regulatory effects are operative remains to be determined. Zinder and his co-workers (*8*) have shown with the small RNA phages that translation of virus proteins is strongly regulated and that in mutants of the coat protein gene this regulatory function breaks down. Sugiyama and Nakada have presented in vitro evidence for a role of the coat protein in repressing translation of the other two viral cistrons (*9*).

Further studies of late protein synthesis by the small RNA viruses of humans and animals should provide some insight into presently ill-defined aspects of replication of these viruses. It might then be possible to obtain an answer to the question of cellular susceptibility to viruses which these experiments were originally designed to examine: Are certain cistrons of picornavirus RNA translated with widely differing efficiencies in different animal and human cells?

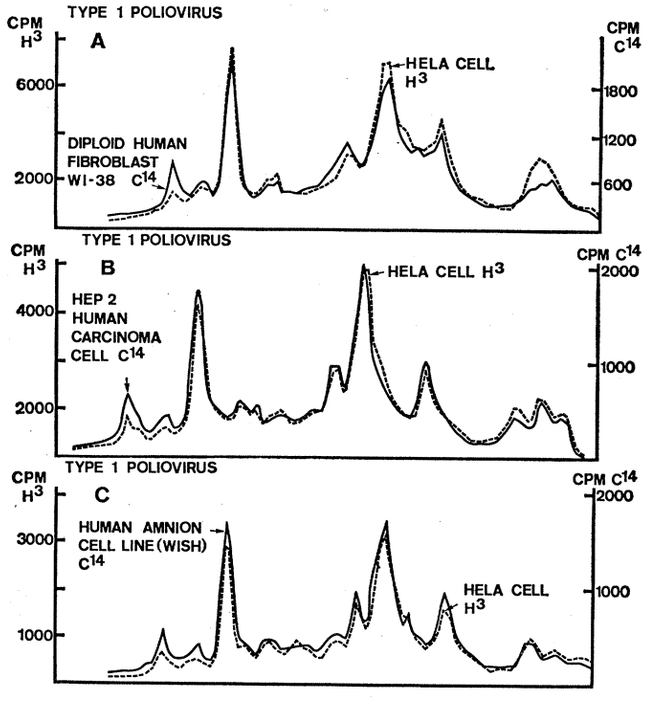

Fig. 1 (left). Acrylamide gel electrophorograms of poliovirus-specified proteins synthesized by different cells. Curves drawn from points representing 85 to 90 fractions. The anode is on the right. The origin (cathode end) is not shown since the last 20 fractions show insignificant labeling. HeLa cells in each case were labeled for 1 hour with H^3 amino acids between 4.5 and 5.5 hours after infection. (A) WI-38 cells were labeled with C^{14} amino acids between 6.0 and 7.0 hours after infection. (B and C) HEP 2 and WISH cells were labeled with C^{14} between 5.0 and 6.0 hours after infection. CPM, counts per minute.

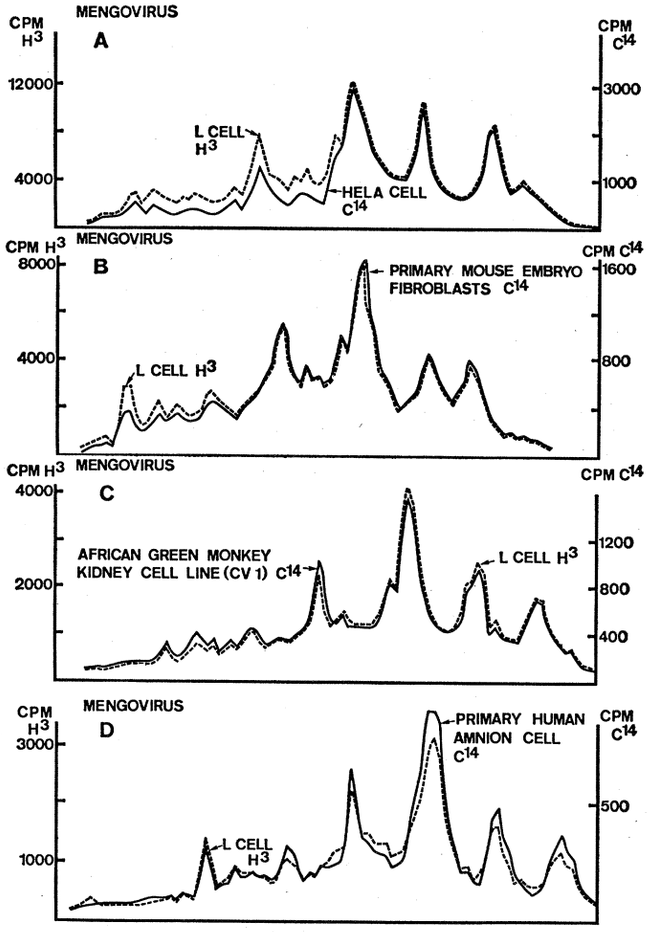

Fig. 2 (right). Acrylamide gel electrophorograms of mengovirus-specified proteins synthesized in different animal cells. In each case, L cells were labeled with H^3 amino acids for 1 hour between 5.5 and 6.5 hours after infection. HeLa cells (A) and CV 1 cells (C) were labeled with C^{14} amino acids for 1 hour between 5.5 and 6.5 hours after infection. Mouse-embryo fibroblasts (B) were labeled from 6.0 to 7.0 hours and human-amnion cells (D) from 6.5 to 7.5 hours after infection. CPM, counts per minute.

Note added in proof: We have recently found evidence with Coxsackie B_1 virus and mengovirus that all or nearly all viral proteins are cleaved from precursor protein molecules of high molecular weight (*10*). This probably explains the constant ratio of viral gene products observed here.

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References and Notes

1. C. A. Buck, G. A. Granger, M. W. Taylor, J. J. Holland, *Virology* **33**, 36 (1967).
2. J. C. Lee and V. M. Ingram, *Science* **158**, 1330 (1967); J. J. Holland, M. W. Taylor, C. A. Buck, *Proc. Nat. Acad. Sci. U.S.* **58**, 2437 (1967); B. L. Strehler, D. D. Hendley, G. P. Hirsch, *ibid.* **57**, 1751 (1967); T. Kano Sueoka and N. Sueoka, *J. Mol. Biol.* **20**, 183 (1966); H. Subak-Sharpe, W. M. Shepherd, J. Hay, in *Cold Spring Harbor Symp. Quant. Biol.* **31**, 583 (1966); M. W. Taylor, G. A. Granger, C. A. Buck, J. J. Holland, *Proc. Nat. Acad. Sci. U.S.* **57**, 1712 (1967); S. Yang and D. Comb, *J. Mol. Biol.* **31**, 139 (1968).
3. J. V. Maizel, *Science* **151**, 988 (1966); D. F. Summers, J. V. Maizel, J. E. Darnell, *Proc. Nat. Acad. Sci. U.S.* **54**, 505 (196[illegible]), *Virology* **31**, 427 (1967); J. V. Maizel, B. A. Philips, D. F. Summers, *ibid.* **32**, 692 (1967).
4. L. C. McLaren and J. J. Holland, *J. Exp. Med.* **109**, 475 (1959); J. J. Holland and B. H. Hoyer, *Cold Spring Harbor Symp. Quant. Biol.* **27**, 101 (1962).
5. J. J. Holland, in preparation.
6. A. L. Shapiro, E. Vinuela, J. V. Maizel, *Biochem. Biophys. Res. Commun.* **28**, 815 (1967); J. Zivan, *Anal. Biochem.* **21**, 155 (1967).
7. D. F. Summers and J. V. Maizel, *Proc. Nat. Acad. Sci. U.S.* **59**, 966 (1968).
8. H. F. Lodish and N. D. Zinder, *J. Mol. Biol.* **19**, 333 (1966); N. D. Zinder and L. B. Lyons, *Science* **159**, 84 (1967).
9. T. Sugiyama and D. Nakada, *Proc. Nat. Acad. Sci. U.S.* **57**, 1744 (1967).
10. J. J. Holland and E. D. Kiehn, *ibid.*, in press.
11. I thank James B. Massey for technical assistance. Supported by grant number GM 14124-02 from NIH.

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Aluminum-26 and Beryllium-10 in Marine Sediment

Abstract. *Activities of aluminum-26 and beryllium-10 in marine sediment were measured at 0.01 ± 0.13 and 4.4 ± 0.9 disintegrations per minute, per kilogram dry weight, respectively. Only an upper limit of 0.03 could be determined for the ratio of aluminum-26 to beryllium-10 in the sediment. The ratio is probably explained by production by cosmic rays in the atmosphere.*

Pelagic sediment is one of the best sources of information on radioactivities produced by cosmic rays. Generally two types of processes are expected to cause accumulation of long-lived radioactive products in marine sediment: (i) precipitation of spallation products resulting from nuclear interactions of high-energy galactic cosmic rays with atmospheric substances; and (ii) sedimentation of cosmic dust, when radioactivities induced by low-energy solar protons may be expected. It has been proposed that simultaneous pairs of measurements of Al^{26} and Be^{10} would be an effective way to investigate these processes (*1*).

Lal and Peters (*2*) have estimated the global rates of production of Al^{26} and Be^{10} by spallation of atmospheric constituents at 1.4×10^{-4} and 4.5×10^{-2} atom/cm^2, respectively; these values correspond to a ratio of about 0.01 for the specific activities of Al^{26} and Be^{10} in marine sediment. The estimated production rate for Be^{10} generally accords with the observed occurrence of 1 to 10 dpm/kg (dry weight) (*3*) (dpm, disintegrations per minute). Wasson's estimate (*4*) of the Al^{26} introduced to marine sediment by process (ii) suggests that this contribution to the content exceeds the contribution by process (i) by more than an order of magnitude; a higher Al^{26}:Be^{10} ratio is expected by virtue of the fact that little Be^{10} can be produced by process (ii). However, there are still large ambiguities in his estimate because of the lack of accurate knowledge of both the rate of accretion to Earth of cosmic dust and the flux of solar protons.

Amin *et al.* (*5*) have reported the mean specific activities of Al^{26} and Be^{10} in two cores from the Pacific basin at 0.5 and 4.0 dpm/kg (dry weight), which correspond to an average Al^{26}:Be^{10} ratio of 0.12. Wasson *et al.* (*6*) have reported an Al^{26} content of 0.8 dpm/kg (dry weight). Both results imply that the Al^{26} content is higher by about an order of magnitude than that expected from the spallation of atmospheric argon. The high Al^{26} content was taken to originate from cosmic dust accreted by Earth, and the necessary flux of solar protons and the accretion rate of cosmic dust were discussed (*7*). On the other hand, Yokoyama (*8*) says that the Al^{26}:Be^{10} ratio of around 0.1 can be explained as resulting only from spallation of atmospheric nuclei, in the light of new cross-sectional data from Bernas *et al.*

However, the important point is that there seem to be uncertainties in the earlier Al^{26} measurements, resulting mainly from difficulties in obtaining a counting sample completely free from radioactive impurities. While investigating Al^{26} and Be^{10} in marine sediment, we have obtained a value for Al^{26} lower than the earlier results.

Our core sample of red clay was 7.8 cm in diameter and 1 m long; it came from a depth of 5439 m at 23°07′N, 135°45′E. The core was halved vertically, one half being available for our experiments. The sample was cut into four sections, each about 25 cm long. The Al_2O_3 content was vertically uniform throughout the core: 16.38 ± 0.32 percent (dry weight).

Dried clay was completely dissolved by treatment with HF and subsequent fusion of the residue with sodium carbonate. From the solution, Be and Al were separated and purified (*9*). The purified Be was deposited on a platinum disk, and Be^{10} was counted with a small flow-type Geiger counter. Because of the paucity of the sample and the rather high background of the counter, the statistical errors in the Be^{10} measurements (Table 1) are relatively high. However, the observed count rates accord with previously reported values. Sample 1A (Table 1) was subjected to absorption measurement with an aluminum absorber, which gave a half-thickness of 20 mg in fair agreement with the maximum energy of Be^{10}.

The purified Al was finally converted to Al_2O_3, and Al^{26} was counted with a low-level gamma-gamma coincidence spectrometer developed by us (*10*). The spectrometer comprises two quartz-faced NaI crystals, each 7.5 by 7.5 cm, positioned face to face. The coincidence spectrometry was performed with gating at 465 to 550 kev, and the counting efficiency for positrons was determined to be 2.8 percent.

Figure 1 illustrates the gamma-ray coincidence spectra of samples of Al_2O_3 after subtraction of background.

Table 1. Measurements of Be^{10} in four samples of sediment; the chemical yield of Be was determined to be 40 ± 5 percent by separate tracer experiments.

Sample			Be^{10} [dpm/kg (dry wt)]
No.	Depth in core (cm)	Dry clay(g)	
1A	0–23	156	6.1 ± 1.6
1B	23–47	182	3.7 ± 1.3
2A	47–73	132	4.4 ± 1.8
2B	73–100	144	3.5 ± 1.7