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Mechanism for Multiplication of Atmospheric Ice Crystals: Apparent Charge Distribution on Laboratory Crystals

Abstract. *Replication of ice crystals with vapor of methyl 2-cyanoacrylate has produced evidence of whiskers on them; an additional mechanism for the necessary multiplication of ice crystals in the atmosphere is suggested. This replication technique also confirms crystal clustering in the atmosphere and appears to confirm the distribution of electrical charge on ice crystals.*

Photography of three-dimensional replicas of ice crystals produced by use of methyl 2-cyanoacrylate monomer (*1*), with a scanning electron microscope, revealed whiskers on the surfaces of growing crystals (*2*) and confirmed charge-induced clustering of ice crystals in the atmosphere (*3*). These results suggest a mechanism for multiplication of ice crystals and the existence of a charge distribution on the surface of a growing crystal.

The manner of multiplication of ice crystals in the atmosphere is potentially important in the ice-nucleus economy of clouds. Langham and Mason (*4*) suggested the importance of splintering and explosive rupture of droplets in the formation of ice nuclei. It was proposed (*5*) that ice whiskers serve as sources of new ice nuclei. We suggest that ice whiskers present on growing ice crystals, smaller by an order of magnitude than those reported (*5*), may serve this purpose.

The small diameter of these whiskers (of the order of 0.5 μ) and the large length-to-diameter ratio suggest that they may be easily broken. A collision between two whiskered ice crystals, or between a whiskered crystal and a water drop, could fracture many of these fragile whiskers, with the consequent production of new ice nuclei. The nature of the whiskers depends on the conditions of growth. Replicas of whiskered ice crystals, grown under different conditions, are shown in Fig. 1.

The electrical charge of ice crystals grown in a laboratory cloud chamber has been reported (*6*); its polarity appeared to depend on the temperature, and the sign-transition temperatures appeared to coincide with those of the reported (*7*) habit changes. Furthermore, modification of habit by impurities produced a corresponding modification of charge (*8*).

These results suggest that the crystal growth produces an excess of negative electrical charges on the basal faces and an excess of positive charges on the prism faces. Because ice is a poor conductor, while the crystal is growing, the negative charges appear to be concentrated near the center of the basal plane faces; the positive charges, on the edges between the prism faces.

The ice crystals agglomerate, fuse together (*9*), fall onto a metal pedestal, and are replicated. The metal substrate effectively prevents charge-induced rafting. An example is shown (see cover) of air clustering of two platelets grown at $-10°C$, each approximately 30 μ in diameter. Photographs of replicated clusters of ice crystals produced in a

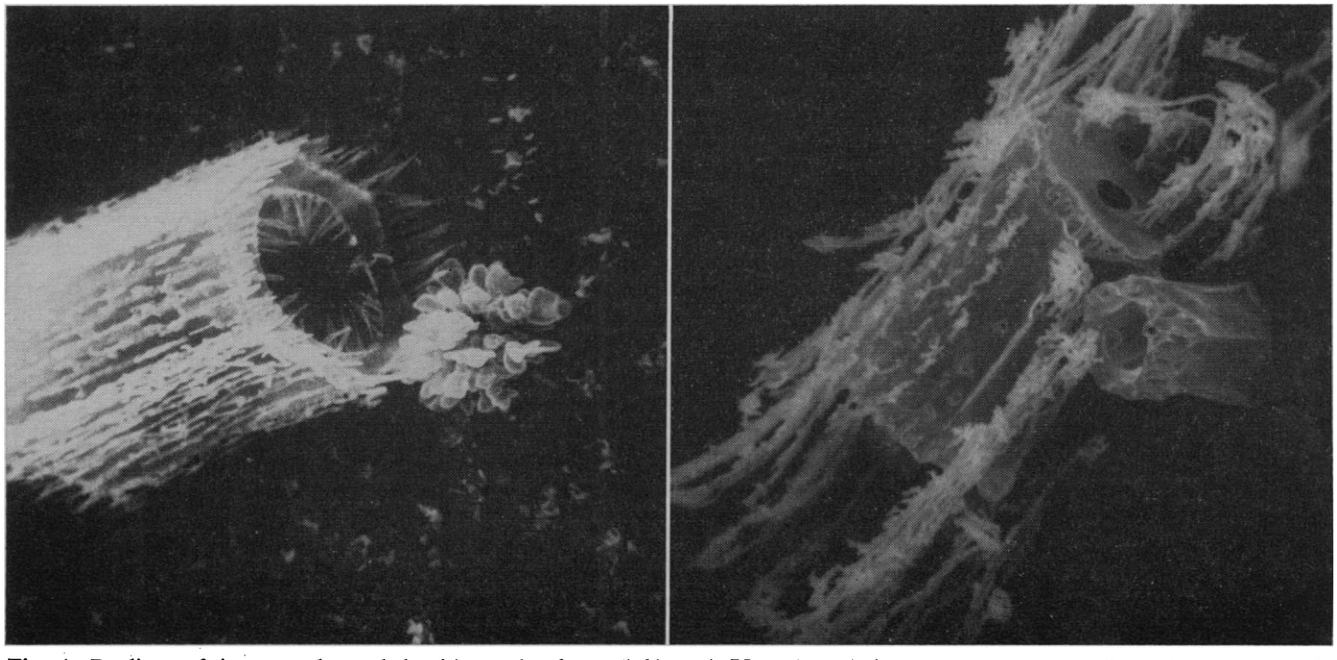

Fig. 1. Replicas of ice crystals seeded with smoke from (left) a $AgIO_3$ pyrotechnic and grown at $-6°C$ in the presence of NH_4OH contamination, and (right) from a $AgIO_3$–KIO_3 pyrotechnic and grown at $-7°C$ in air not deliberately contaminated; $\times$ 1000.

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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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