of the phospholipid fraction of the virus which is grown on these cells without a concomitant increase in the specific labelling of the virus ribonucleic acid. Similar observations have been made during the P^{32} labelling of Newcastle disease virus[31]. This suggests that the virus lipid is obtained from the host cells as intact lipid material whereas the virus nucleic acid is newly synthesized from smaller components. The fact that the composition of the phospholipid fraction of several strains of influenza virus is the same as that of the normal chorioallantoic membrane[32] further supports this idea.

These facts lead to the following hypothesis: *viruses containing lipids as essential components are assembled from viral subunits or from vegetative forms at or near the cell surface and are completed by obtaining a protective external lipid coat from the lipid structures at the cell periphery as the particles pass to the external cell surface.* As a corollary to this, viruses which do not contain essential lipid components do not form a close union with the periphery of the cell but are completed within the cell and may be released in large amounts by a burst process.

It may be that each type of virus which is released continuously receives an equivalent outer shell of lipid material as it is being completed. It is striking that this hypothetical lipid shell has the same thickness of 40 Å for two unrelated viruses, fowl plague and equine encephalitis, as estimated assuming an average virus density of 1·1 and an average lipid density of 0·9.

Since cells infected with tumor viruses continue to divide, one would expect that these viruses are all released by a continuous process and hence contain essential lipid components. Except for the Rous sarcoma virus, little is known concerning the growth of tumor viruses. Infectious virus particles are continuously released from Rous sarcoma cells in tissue culture[33] and this virus is ether sensitive[29].

Of the other tumor viruses which have been studied, the following are ether sensitive: myxoma[29], fibroma[29], and the agent of mouse leukemia[34]; and the following are ether resistant: Shope papilloma[29], mouse parotid tumor agent[35], and mouse sarcoma agent[35]. The ether resistant Shope papilloma virus, identified by specific fluorescent antibodies, is located for the most part in the differentiating keratohyaline layer rather than in the actively multiplying tissue[36].

The fact that some tumor viruses are ether resistant suggests that they may behave like lysogenic phage, being released by a rare burst event. The ether sensitive tumor viruses may all multiply like the Roux sarcoma virus.

Although this survey is concerned with animal viruses, it should be pointed out that bacterial viruses are released by a burst mechanism and do not contain lipids. Plant viruses are also free of lipids but the release mechanism is a highly specialized process, the virus passing from cell to cell by means of plasmodesmata[37].

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Max-Planck-Institut für Virusforschung, Tübingen, 24. April 1958.

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Zusammenfassung

Eine Hypothese über die Synthese und die Freisetzung von tierpathogenen Viren wird entwickelt. Diejenigen Viren, die durch einen kontinuierlichen Prozess freigesetzt werden, werden an der Zellperipherie zusammengefügt. Beim Durchtritt durch die Zellmembran erhalten sie eine Schutzhülle aus Lipiden, die aus den in der Zellmembran lokalisierten Lipiden der Wirtszelle stammen. Alle derartigen Viren sind gegen Äther empfindlich und enthalten Lipide als essentiellen Bestandteil. Diejenigen Viren, die stossartig freigesetzt werden, werden dagegen im Innern der Zelle zusammengefügt und enthalten keine essentielle Lipid-Komponente. Diese Hypothese wird auch auf die Tumor-Viren angewandt.

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Corrigenda

B. L. VAN DER WAERDEN, *Erwachende Wissenschaft* (Ref. N. STULOFF). Exper. *14*, Fasc. 6, 228 (1958).

Die letzte Zeile des vorletzten Absatzes der Buchbesprechung muss richtig lauten: «Ursachen des Niederganges der griechischen Mathematik».

H. LINDE und K. MEYER, *Zur Konstitution des Resibufogenins und Artebufogenins*. Exper. *14*, fasc. 7, 238 (1958).

Auf Seite 239 ist in Formel VIII das H-Atom an C-14 *α-ständig* zu formulieren.