ENTRY OF RETROVIRUS INTO MOUSE FIBROBLASTS. Klaus B. Andersen. The Fibiger Laboratory, Nordre Frihavnsgade 70, DK-2100 Copenhagen Ø, Denmark.

On the basis of experiments with radioactively labelled virus particles and studies of the inhibitory effect of lysosomotropic bases on retrovirus infection, we have proposed the following route for the entry of retrovirus into the host cells (Andersen and Nexø, Virology, 125, 85, 1983): The virus particles bind to receptors on the cell surface, they are internalized by endocytosis, and they are collected in the lysosomes, where they are degraded. However, a part of the particles enters the cytoplasm, presumably by a membrane fusion between the virus membrane and the vesicle membrane. This is the infectious route.

The lysosomotropic bases both inhibit infection and virus degradation. Is degradation then necessary for infection? Leupeptin is an inhibitor of various lysosomal proteases. Leupeptin was shown to inhibit infection at a step shortly after internalization. A limited degradation, therefore, appears to be necessary for infection. Besides the particles which are fully degraded, the membranes of the virus particles entering the cytoplasm remain in the vesicles according to the model. Are the surface proteins then degraded to a larger extent than the core proteins? The proteins of radioactive virus particles were followed during entry, and the surface protein gp70 was shown to be degraded faster and to a larger extent than the core proteins.

IMMUNE STUDIES ON URANIUM MINERS IN SEVERAL LOCALITIES.
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The uranium miners represent one of the "high risk groups" with respect to lung cancer. In this case the immunological aspects of ionizing radiation must be taken into consideration. There are, however, localities with chemical carcinogens in some mines. The purpose of our study is to analyse the interferrence between these two influences in modifying the immune status of uranium miners. We have investigated serum immunoglobulin, alfa-2-macroglobulin and alfa-1-antitrypsin levels in miners, who underwent periodical preventive medical investigation. We also investigated cellular immunity in selected persons. Further, the number of cigarettes per day, age at which smoking began and total of years were assessed.

EFFECTS OF TUMOUR PROMOTER ON CULTURED SQUAMOUS EPITHELIAL CELLS DERIVED FROM RAT TONGUE EPITHELIUM. D.Arenholt, A.Jepsen, L.Andersen, A.V.Fisker and H.P.Philipsen.
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Data are presented indicating that not only TPA but also chemically unrelated tumour promoters such as teleocidin, anthralin and meserein induce characteristic morphological changes of epithelial cells in the form of elongation, formation of cytoplasmic extensions and widening of the intercellular spaces. Non-promoting chemicals (ethylphenylpropiolat, phorbol, acetic acid, acetone) did not cause the same changes. The changed morphology resembles that of single cells in low density cultures before they reach contact with neighbouring cells. We suggest that there is a link between the morphological reactions and the known tumour promoter ability to block intercellular communication. The morphological changes were examined in phase contrast and scanning electron microscopy.

A cell kinetic study has shown that incubation with concentrations higher than 5 ng TPA/ml medium caused inhibition, below 5 ng TPA/ml stimulation of the mitotic activity during a 24 hr incubation period, reaching a maximum 4-5 hr after change to medium without promoter. Preliminary results indicate that the promoter induced stimulation is modified by addition of atoxic doses of retinoic acid (3.3 μM) or selenium (10^{-6} M).