as an internal anticoincidence system. Difficulties with end effects and electronegative impurities should be overcome by this unique system.

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Biomacromolecules: Views and Models

A symposium entitled "Views and Models of Biomacromolecules" was held 15 May 1967 at the New York University Medical Center, New York City. A. K. Kleinschmidt (New York University School of Medicine) discussed the techniques used to determine the size and configuration of nucleic acid strands when removed from the core of different virus particles. The basic techniques consisted of extracting and letting the nucleic acids absorb from a solution to a protein monolayer, either by spreading a protein-nucleic acid mixture, or by utilizing the undisturbed diffusion of the filamentous macromolecules to a preformed absorptive stable film. Extraction of viral nucleic acids in various ways and originating the film can be performed in one step, so that the results show lengths of nucleic acids per virus particle. This was shown mainly with reovirus RNA extracted by urea. These nucleic acids have a tendency to fall apart in short pieces of trimodal size. DNA from many viruses was found in one filament that fit the lengths calculated from the known data of molecular weight. The shape predicted from models in solution was assumed to be transferable to electron micrographs. Measurements of length and end-to-end distances have been used to determine the spatial arrangement. Emma Shelton (National Cancer Institute) discussed the appearance of ribosomes in both monomeric and polymeric states. Using plasma cell ribosomes from neoplastic mice as the experimental material, the development of polyribosomes was traced. The differing degrees of coiling of free and membrane-bound polyribosomes were attributed to the intermolecular forces involved in attachment. Ribosomes polymerize; their smaller subunits bound to the extended messenger RNA give rise to a helical array of polyribosomes. The possibility of the pathway of messenger RNA between the large and small subunits as well as models for the possible method of transfer RNA and polypeptide formation were discussed. Morris J. Karnovsky (Harvard Medical School) demonstrated the use of peroxidases as tracers in the movement of macromolecules through cellular structures. By electron microscopy and measurements of the widths of cell unit membranes, as well as the tracer techniques, it was shown that the vascular channels in endothelial brain cells of rat and mouse were actually open when a small enough tracer was used. The peroxidase tracer and lanthanum nitrate were also used to demonstrate that, in mice, a barrier between blood and brain does exist. The cellular pores are closed between the chorioplexus and the nervous tissue; this may be demonstrated in both directions. Some further experiments to show the reactions of enzymes in muscle T-bands were illustrated with excellent micrographs.

Roderic Park (University of California, Berkeley) discussed the interpretation of cleaved membranes in chloroplasts. The use of freeze-etching and freeze-fracture techniques indicated that the fracture planes are most likely in the lipid layers between the grana. This interpretation is at variance with other workers in Zurich. The interpretation of electron micrographs of freeze-fractured surfaces has become a growing feature of electron microscopic research. It presents views of ultrastructure split at minimum interfacial tension, which varies considerably from previous methods of sample preparation. A very convincing model of chloroplast layers and structures was described and defended by Park. E. Kellenberger (Université de Genève) described experiments with mutants of bacteriophage T4 of *Escherichia coli* which were designed to reveal certain morphological features relating to their positions along the genetic map. The organized T4-phage head and its proteins of double-mutants were identified by electron microscopy and acrylamide gel techniques, combined with the localization of the genes responsible for the morphopoiesis of the phage heads. Some mutants had features which could be used to relate the specific genes responsible for the head formation of the T4 phage, either as a regular, or a prolate icosahedron. Considerable effort has gone into the examination of many mutant T4 strains, to determine precisely which morphological features are the results of the deletion, inclusion, or recombination of specific genes. S. S. Breese, Jr. (Plum Island Animal Disease Laboratory) presented material on the virus of foot-and-mouth disease as a macromolecule. Methods by which physical constants such as sedimentation and diffusion could be determined on impure but infectious samples were discussed. Examples were shown of the formation of crystalline arrays of virus in tissue culture cells and determination of the electron microscopic substructure of the virus. The difficulties of this virus as an experimental model as well as its advantages were pointed out.

The symposium was held under the auspices of the New York Society of Electron Microscopists and the New York University School of Medicine.

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Cellular Dynamics

The fifth conference on cellular dynamics was held at Princeton, New Jersey, 8–11 January 1967. Representatives of many disciplines—medical, biological, and biochemical—convened for a reevaluation, in the light of recent progress in those disciplines, of aging in cells and cell strains. On the assumption that such aging does take place, it was expected that the relation of these processes to more familiar manifestations of the syndrome in whole animals would be examined.

The sessions, arranged by M. D. Rosenberg, were broadly based in the several fields of clinical and biological research from which solutions to problems of aging must eventually flow. The argument that aging of animals is a reflection of aging in cells was reviewed by B. L. Strehler, who described the several classes of hypothesis currently held, and offered a speculative one of his own.

Some populations of fixed, postmitotic cells in the mammalian body show losses in number correlated with the other manifestations of aging. Even those populations for which a decrease in numbers cannot be demonstrated may show age-related loss in functional capacity. However, some cell populations and tissues showing no loss in basal function have, in age, a demonstrably reduced capacity to respond to stress.

For some analysts, these decrements are signs of a more fundamental cellular process, possibly one that is programmed genetically (since there are values for the species, in aging and its consequences, that are not values for the individual). Suggestions about the identity of these fundamental processes, some old and some new ones, were reviewed. Argued with particular vigor was the proposal that aging may be a failure in maintenance in the differentiated state, that is, a failure in the capacity of certain cells to adhere to the correct pattern of macromolecule syntheses. Searching as was the discussion of the many reasonable mechanisms by which failures might arise, there were some participants who remained unconvinced that the syndrome called "aging" reflects any such fundamental cellular process.

Alternative possibilities were proposed and argued. For example, it was suggested that changes in the mechanical properties of extracellular tissue matrices, such as occur in connection with increased covalent cross-linking of collagen, are the basis of aging in whole animals. Presented by R. R. Kohn, this suggestion was nevertheless not more convincing than those involving intrinsic cellular alterations in protein synthesis, nucleic acid synthesis, cell cycle kinetics, mutation rate, differentiation, essential turnover processes, permeability, pigment accumulation, and lysosomal fragility. It is probably fair to say that while for some participants the decrease in adaptability to stress in age is an obvious consequence of decrements in processes taking place within cells (particularly in the fixed postmitotic populations) there is lacking in the argument the solid base of experimental fact.

Old animals and people do not, after all, die of aging. They die of cancer, stroke, heart failure, accident, infectious disease, fatigue, or boredom. They die of the inability to adjust to stress; adaptation potential declines in a predictable way for whole populations with time after maturity. Aging is in consequence an elusive entity—a syndrome, to be sure, but one diagnosed by its accidental sequelae, not by any demonstration of pathogen. "Old" and "aged" do not mean the same thing, not for men and not for their cells. The two words may sometimes refer to the same class of biological object, but even when this is clearly so we do not yet know why.

The data say that processes intrinsic to cells do change in time, both for cells in culture, outside the body, and for cells in somatic populations. Some of these changes, such as the accumulation of lipofuscin pigments, alterations in lysosomal function, loss of biosynthetic capacity, in normal surface properties, in contact geometry, variations in ploidy, in certain parts of the machinery that regulates transcription and translation, have at least a suspicious connection with clinical aging. Changes in extracellular proteins that are the undeniable concomitants of aging and of degenerative disease remain as likely to be consequences of one or more of the intrinsic changes as causes. For any suggested set of causes and consequences we must as yet agree upon no more committed a verdict than "not proven." But it is clear that no new information about the fundamental chemistry and biology of cells is irrelevant, and a great deal of this information will need to be exchanged by those concerned to understand, if not to control, the set of declining competences we recognize as aging.

This conference was arranged under the auspices of the Interdisciplinary Communications Program of the New York Academy of Sciences.

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Plant Physiology: Translocation in Plants

Translocation in plants is a topic which would seem to be easy to study in this age of tracers and chromatography. It would also seem to be fairly well understood if one glances at a text book or article on plant physiology written more than 6 years ago. That this is not the case was made abundantly clear at a recent symposium held in conjunction with a meeting of the Canadian Society of Plant Physiologists, held in Ottawa, Canada, 31 May–2 June 1967. The meeting was under the chairmanship of C. D. Nelson (Simon Fraser University, Burnaby, British Columbia), who, together with P. Gorham, is well known for his discovery of a small rapid component of translocation in phloem channels (more than 1000 centimeters per hour).

In the past a pressure flow mechanism has been thought to explain phloem transport. It is envisioned that sugars are loaded into the ends of phloem sieve tubes in the leaves; water flowing into the sugar solution under an osmotic gradient then produces pressure to force the solution down the plant. Sugar, of course, may be withdrawn along the length of the sieve tube. This theory was originally proposed by Münch. It is generally agreed that such a mechanism might operate by pressure to produce the observed flows of 50 to 100 cm/hr in the sieve tubes, if the tubes and sieve plates were free from obstruction by cytoplasm.

R. V. Evert (University of Wisconsin) pointed out that not only is there very good evidence now available that cytoplasmic strands pass through the sieve plates, but also he has found that sieve tubes contain a nucleus, mitochondria, plasmalemma, and tonoplast in the mature state. Moreover his study of the slime body and its subsequent development to produce slime strands, which penetrate the sieve plate, seem to make the older theory of mass flow highly improbable. More acceptable as a translocation mechanism would be the suggestion of Thaine that the slime strands themselves are the site of an active transport mechanism, related perhaps to protoplasmic streaming.

M. H. Zimmerman (Harvard University Cabot Foundation, Harvard Forest, Petersham, Massachusetts) discussed his findings of transport rates in tall trees 40 to 50 cm/hr. Such rates were measured by detecting a wave of atypical ratio of stachiose to raffinose in the sap which was induced by application of heat in an atmosphere of nitrogen. C. A. Swanson pointed out that in his work with tracers in *Phaseolus*, low temperatures (about 1°C) or high temperatures (about 50°C) both caused transport to cease. However, transport resumed after some time in the cold. Trip and Gorham (National Research Council, Ottawa) reported that adjacent sieve tubes carried sugar in different ways as indicated by micro-autoradiographs. J. A. Webb (Carleton University, Ottawa) reviewed the situation. He pointed out that while many facts about transport were known, the mechanism is unsolved. There remain objections both to this mass flow theory and to Thaine's protoplasmic theory. It was perhaps disappointing that the four or five other "active" transport hypotheses which have been proposed were not more seriously discussed.

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