

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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