RECENT DEATHS

John Allison, 71; professor emeritus of psychology, Frostburg State College; 6 January.

Joseph C. Aub, 83; professor emeritus of medical research, Harvard University; 30 December.

Harry Bakwin, 79; retired professor of clinical pediatrics, New York University; 25 December.

Edmund deS. Brunner, 84; former professor of sociology, Columbia University; 21 December.

Richard S. Burington, 72; former professor of mathematics, Case Western Reserve University; 24 December.

Walter Cohen, 52; professor of psychology, State University of New York, Buffalo; 20 December.

George H. Estabrooks, 78; former chairman, psychology department, Colgate University; 30 December.

Griffith C. Evans, 86; professor emeritus of mathematics, University of California, Berkeley; 8 December.

Ralph H. Fox, 60; professor of mathematics, Princeton University; 23 December.

Joan P. Giles, 54; research associate professor of pediatrics, New York University; 28 November.

Vincent H. Gillson, 52; assistant professor of pathology, Fairleigh Dickinson College of Dentistry; 20 December.

Salvatore M. Marco, 65; professor emeritus of mechanical engineering, Ohio State University; 15 December.

Max Meenes, 73; professor emeritus of psychology, Howard University; 5 January.

Jacob van de Kamp, 69; retired head, Synthetic Organic Preparations Laboratory, Merck Sharp & Dohme; 22 November.

Wolf V. Vishniac, 51; professor of microbiology, University of Rochester; 10 December.

Joseph L. Walsh, 78; professor emeritus of mathematics, University of Maryland; 10 December.

Louis G. Welt, 60; chairman, internal medicine department, Yale School of Medicine; 13 January.

Gerald L. Wendt, 82; chemist and retired head, publication center, United Nations Education, Scientific and Cultural Organization; 22 December.

RESEARCH NEWS

RNA Viruses: The Age of Innocence Ends

Virologists have traditionally been among the most optimistic of cancer investigators, and for many of them the 1960's were an era of relative innocence. Secure in the knowledge that viruses cause tumors in animals, they were confident that these agents would provide an elegantly simple solution to the problem of human malignancies. If only a human cancer virus could be isolated, many virologists argued, a vaccine could be developed and control of cancer would be a reality.

That attitude engendered a tremendous outpouring of research results—a large number of little-recognized successes and a few more highly publicized failures. The investigators developed tissue culture systems for growing large numbers of virus particles, and thus learned a great deal about the biochemistry of oncogenic (tumor-forming) viruses. They discovered many animal tumor virus systems that served as models for what might occur in humans, and thus learned a great deal about the interaction of virus and host. They also isolated several putative human cancer viruses, and thus learned a great deal about humiliation and the loss of credibility as one after another of the ballyhooed candidates proved to be of nonhuman origin.

The age of innocence has slowly drawn to a close, however, as many virologists have begun to recognize that the problem is substantially more complex than they had originally anticipated. Although some still argue that a tangible oncogenic human virus will eventually be isolated, a growing number of investigators have concluded that this approach may be futile and have thus begun to reconsider the fundamental concepts of the nature of viruses and their role in animal biochemistry.

If viruses do play a causative role in human malignancies, these scientists suggest, it is most likely that the active agent is an incomplete or defective portion of one virus—or perhaps of several viruses—whose normal function is beneficial to the host. Research on oncogenic animal viruses, as a consequence, has been somewhat de-emphasized as investigators have pressed the search for virus fragments or information in human tumors. Nonetheless, there has been a continuing strong interest in ascertaining the normal role of oncogenic viruses, particularly those whose hereditary information is contained as RNA.

Oncogenic RNA viruses (also called oncornaviruses and RNA tumor viruses) are generally divided into three main classes, labeled A, B, and C. Type C RNA viruses, the most important class, have been shown to infect a large number of animal species. Most type C RNA viruses are oncogenic, causing mainly leukemias, lymphomas, and sarcomas—all tumors arising in tissues of mesodermal origin, such as bone, cartilage, connective tissue, and lymph nodes. Type B RNA viruses, which are fewer in number, have been associated primarily with certain tumors (carcinomas) of the breast. Type A RNA viruses, which are not infectious, are a very small group of viruslike particles that have not been found outside the confines of cells and that have not been shown to be oncogenic.

A principal difference between oncornaviruses and other animal RNA viruses lies in the size of the genome, the complete set of hereditary information contained in the chromosomes. Oncornavirus genomes have a mass of about 12×10^6 daltons, compared to about 6×10^6 daltons for the paramyxoviruses and about 2×10^6 daltons for poliomyelitis virus. Perhaps as a result of this large genome, oncornaviruses have a more complex internal structure with no clearly observable symmetry. They may also contain more types of proteins and more species of nucleic acids.