# Preface

Early research in viral oncology focused primarily on agents that were artificially selected in the laboratory or agents that were oncogenic only when inoculated into foreign species. Either way, the viruses were generally not associated with naturally occurring tumors of outbred species of animals. During this early period, a considerable amount of information accumulated on the pathobiology of retrovirus-, papovavirus-, and adenovirus-induced tumors of laboratory rodents. During recent years, major developments have occurred. Improved methodology in biochemistry and genetics have provided new ways to study growth control and differentiation in normal and malignant cells. Because of their relatively small genomes, papovaviruses and retroviruses have been particularly appropriate subjects for this research. Another major development during this period was the recognition that horizontally transmitted retroviruses and herpesviruses cause tumors under natural conditions in such diverse species as chickens, cats, cattle, and gibbon apes.

Experimental evidence gained thus far from studies with human tumors indicates that most tumors contain neither viral particles nor complete copies of virus-specific DNA or RNA sequences that are closely related to currently recognized viral probes. Yet, for almost all types of human cancers, the etiology remains unknown. For many forms, especially those that occur at young ages, etiological hypotheses based solely on the accumulation of damage caused by chemical pollutants seem unlikely. Thus, the search continues for candidate viruses that might cause various forms of human cancers. Epidemiological studies have long supported an association between the Epstein-Barr virus and two distinct forms of human cancers, Burkitt's lymphoma and nasopharyngeal carcinoma. In the case of Burkitt's lymphoma, evidence continues to accumulate in support of the hypothesis that the Epstein-Barr virus represents an important component in a multifactorial process that results in the development of the disease. More recently, an impressive epidemiological association has been found between the development of primary hepatocellular carcinoma and exposure to the hepatitis B virus.

A major goal of this conference was to bring together researchers from different disciplines. The background and expertise of the participants ranged from molecular biology to epidemiology and clinical oncology. The common ground of interest was the association between viruses and naturally occurring cancers.

We are especially indebted to Jim Watson for both specific suggestions and general encouragement. We are grateful to the session chairpersons, Tom Benjamin, Peter Duesberg, Charlotte Friend, Ray Gilden, Gertrude Henle, Arthur Hurvitz, Rose Sheinin, Carl Olson, and David Yohn for stimulating discussion. We also wish to thank the National Cancer Institute; the Bureau of Biologics, Federal Drug Administration; and the Fogarty International Center for financial support.

The staff of the Cold Spring Harbor meetings office provided invaluable assistance, and we especially thank Gladys Kist and Winifred Modzeleski for their help in organizing the meeting. We also appreciate the efforts of the publications office, directed by Nancy Ford. In particular, we thank Dorothy Brown, Nadine Dumser, and Douglas Owen for their skillful editing, and Chris Nolan, Annette Kirk, and Kathleen Horan for helping to prepare the book for publication.

**M. Essex**
**G. Todaro**
**H. zur Hausen**