

VIRAL INFECTIONS IN ORAL MEDICINE

Proceedings of the International Symposium on Viruses and Oral Diseases held 22-23
September 1980 at the National Institutes of Health, Bethesda, Maryland, U.S.A.

Editors:

JOHN J. HOOKS, Ph.D.

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GEORGE W. JORDAN, M.D.



1985年1月19日

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Preface

It is well known that a majority of viruses either induce oral pathology or are transmitted via the oral mucosa. Nevertheless, there is no authoritative description of viruses and viral diseases focusing on the oral cavity. This book is designed to fill that gap. Authorities in basic virology, immunology, laboratory diagnosis, epidemiology, prevention, and therapy cooperate in examining viruses and oral diseases.

This book is designed as up-to-date, authoritative reference both for dental and medical students and also for specialists in dentistry, dermatology, and infectious diseases. The introductory section deals with basic biological mechanisms involved in the pathogenesis of viral diseases with emphasis on the immune response and the interferon system. An indepth analysis of viruses follows. It includes descriptions of virus-induced oral pathology and also diagnostic laboratory procedures. The next sections cover prevention, control of spread of infection, and epidemiology. In these sections, risks to dental and medical personnel and patients are examined. Moreover, the authors consider biological safety in hepatitis, infectious mononucleosis, and other viral infections. The book concludes with a discussion of diseases of the oral cavity with possible viral and immunologic etiology.

The National Institute of Dental Research (NIDR) sponsors workshops to discuss fundamental research problems, clinical trials, and treatment of certain diseases of the oral mucosa. The workshop on "Viruses and Oral Diseases" was convened by NIDR on September 22-23, 1980. We wish to thank Mrs. Eloise Mange for her skillful assistance in technical arrangements for this workshop. We also thank Dr. Abner L. Notkins for his advice and support.

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SECTION I

Biology of Viral Diseases

SECTION I

Biology of Viral Diseases

Pathogenesis of Viral Infections

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Viruses have given us insights into life as well as disease at a fundamental level. It has been argued that viruses are not alive, since they contain only a single nucleic acid, lack organelles or nuclei, fail to grow or undergo binary fission and are incapable of energy production. However, they transmit information between living cells and, therefore, their vitality can be defended on the premise that genetic perpetuation is the very essence of life (Luria et al., 1978).

Viruses contain single or double strands of a single nucleic acid, either DNA or RNA, which retain the information and direct the cell to construct a series of polypeptides and enzymes. The cell, under virus direction, then assists in replication of the nucleic acid and assembles viral proteins into a protective coat, the capsid. The mature particles, called virions, consist of the naked nucleocapsid in some virus families, whereas in other families the virion has an outer envelope. This envelope is added when the nucleocapsid buds through modified cell membranes in which viral proteins have replaced cell proteins. The virion structure facilitates transmission of the informational viral nucleic acids to other cells.

Cellular Infection

Viral infection can be viewed at three levels: the level of the cell, the level of the host animal, and the level of host populations. The pathogenesis at the single cell level depends on virus attachment to the cell, penetration of the virus, uncoating of the nucleic acid, transcription of messages to direct the cell to synthesize protein, replication of the nucleic acid, and assembly and release of the mature virions. These processes in turn may have varied effects upon the

host cell, including lysis, transformation, or moderate effects with subtler modification of cell function (Figure 1).

Susceptibility of the individual cell to infection is often determined by the presence or absence of specific receptor sites on the cell surface. Viruses have no inherent mobility and contact cells by Brownian movement or by passive transport in host cells or fluids. Random contact usually does not result in attachment, since surface polypeptides of the virus must attach to specific receptors on the cytoplasmic membrane of the cells. Attachment does not necessarily lead to penetration, but attachment alone, in some cases, can lead to alteration of the host cell. Virus may penetrate the cell by endopinocytosis or by fusion of the virus envelope with cytoplasmic membranes releasing the nucleocapsid directly into the cytoplasm. Uncoating is the functional release of nucleic acid from the protein and lipid coverings sufficient to allow transcription of messenger RNA. Complete release of the nucleic acids is not always required; for example, paramyxovirus messenger RNA appears to be transcribed while the viral RNA is still encapsidated with capsid protein. For permissive infection in which progeny virus are produced, the host cell must be

Figure 1. Schematic diagram of the infectious cycle and the effects on the host cell. The steps of infection (left) represent an enveloped virus. The host cell nucleus and cytoplasm are not indicated since different viruses are replicated and assembled in different locations. Some viruses lose envelopes at the time of penetration, and the envelope of progeny virions may be acquired from nuclear, cytoplasmic, or plasma membranes. For simplification, the transcription of messenger RNA and translation of nonstructural proteins have not been included. The alterations in the host cells (right) do not necessarily require completion of the infectious cycle, since nonproductive infection can frequently lead to cytolysis or transformation.

SOURCE: Reproduced from Johnson (1974) with permission of the American Association of Pathologists and Bacteriologists.

