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Opinion Article

General Introduction about Rhinoviruses

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DESCRIPTION

The Common Cold Research Unit was almost obscured by its pastoral surroundings, which were located in the rolling vividly green countryside of Wiltshire in Southwest England.The identification of the cold-causing agents now known as Rhinoviruses.

Work done in the UK and elsewhere resulted in two simple discoveries that allowed researchers to grow these fastidious viruses in the laboratory. First, the differentiated respiratory mucosa of human foetuses grown in organ culture proved to be uniquely susceptible to a wide range of pathogens. Second, their growth outside the human body was discovered to be temperature dependent on 33°C which maintains ceUs in the laboratory. Growing these viruses in the laboratory allowed researchers to examine their biological properties and conduct experiments to determine their pathogenicity.

Rhinoviruses are a subgenre of the picomavirus family. They are strikingly similar to entercniruses in molecular and structural terms, but differ in that they frequently have more fastidious growth requirements *in vitro,* and the virion is sensitive to add treatment in a laboratory setting. Over 100 distinct rhinovirus serotypes have been recovered from humans to date, but many more are likely to be discovered. These viruses are classified into two groups based on the cell surface receptors they use.

The majority of recognised strains attach to the ICAM-1 molecules of the respiratory epithelium. The second group consists of the remaining virus strains. They use a low-density lipoprotein cell surface receptor that has yet to be identified specifically. *In situ* hybridization studies have shown that rhinoviruses multiply in the mucosal cells of the nasal cavity and para nasal sinuses of humans and higher primates.Volunteers who have been experimentally infected appear to be susceptible to extremely small amounts of virus. Within 48 hours of infection,maximumvirus concentrations accumulate.

Concurrently, the all-too-familiar symptoms of the common cold appear suddenly. Respiratory complaints can last anywhere from 7 to 13 days. During the acute stages of infection, biopsies of the nasal mucosa show no cytological changes in the epithelial lining cells, but inflammatory cells are abundant in the mucosa and submucosa.

Furthermore, there are a large number of mucosal cells that are polymorphous. We can only speculate about their role in the signs and symptoms of the common cold. Only a few scattered cells of the nasal mucosa are infected, according to immunological labelling of the virus. These cells have an ill-defined topographical distribution, and not all regions of the nasal turbinate systems' mucosa are infected to the same extent. At this time, volunteer studies show a decrease in mucociliary function, as well as slower rates of transport of small (0.5 mm) foreign particles introduced experimentally onto the nasal mucosa.

In some cases, virus can be recovered from nasal secretions and cough specimens for up to three weeks. Finally, virus clearance is dependent on the production of type-specific secretory IgA. Although degenerative changes in the epithelium of infected organ cultures of the respiratory mucosa have been described, overt necrosis of mucosal cells does not occur in these models (as proves to be the case with influenza viruses). Mucus production in cultured nasal mucosa infected *in vitro* has been altered in studies conducted in the author's laboratory. The thick yellow-green nasal discharge of the common cold is caused by viscous mucus and an abundance of inflammatory cells.

In 30% to 50% of people infected with rhinoviruses, symptoms indicating tracheal and bronchial involvement occur. The most common complaint is a productive cough. While no consistent effects on pulmonary function have been observed.

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