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Physiology

ANA 204(Histology)

 Corona virus infection is a severe acute respiratory syndrome of the Human Ciliated Airway Epithelia. First, there are two types of corona virus discovered lately, the SARS-CoV (severe acute respiratory syndrome-corona virus) and the HCoV (human corona virus).

 The human conducting airway epithelia of the nasal consist of a pseudostratified mucociliary epithelium with predominant ciliated cells interspersed with mucus-secreting goblet cells overlaying a basal cell layer. The predominant cells of the cuboidal epithelium of the human distal bronchiolar airways are also ciliated cells. The airway epithelium is often the first tissue encountered by intraluminal pathogens, and ciliated cells are major targets for common respiratory viruses, such as influenza etc . Vitro models of human airway epithelia derived from nasal were susceptible to SARS-CoV infection after luminal inoculation of virus, and the cell types targeted preferentially by SARS-CoV were ciliated epithelial cells.

HACE2(human Angiotesin-converting enzyme 2) which is an antigen expressed in the alveolar regions and the basal layer of the nonkeratinized squamous epithelium of the upper respiratory tract. The findings of hACE2 expression and productive SARS-CoV infection in ciliated cells derived from the nasal and tracheobronchial airways dispute this claim and support the hypothesis that SARS-CoV initiates a productive infection in human ciliated airway epithelium. It is interesting to consider the biological significance of hACE2 expression on the luminal surface of ciliated cells.

 The human coronavirus, HCoV, which primarily causes a mild upper respiratory tract infection in humans, has been shown with other models of HAE to principally target ciliated epithelia, causing cell morphology alterations, dyskinesia, and cilium dysfunction. However, shedding of infected ciliated cells, noted here during SARS-CoV infection, was not reported following HCoV infection. In contrast to other HCoV, SARS-CoV infection is associated with severe atypical pneumonia in adults. The major pathological findings upon autopsy suggest SARS-CoV involvement of type II pneumocytes located in the alveoli, as evidenced by detection of viral RNAs and EM detection of virus-like particles. Importantly, patients dying early following SARS-CoV exposure show marked bronchiolar disease with respiratory epithelial cell necrosis, loss of cilia, squamous metaplasia, and intrabronchiolar fibrin deposits. In fact, it has been suggested that early diffuse alveolar damage as a consequence of SARS-CoV infection may initiate in the respiratory bronchioles (13, 39).