Search Results For coronavirus

# Structural Analysis of Major Species Barriers between Humans and Palm Civets for Severe Acute Respiratory Syndrome

Coronavirus Infections,

2008

It is believed that a novel coronavirus, severe acute respiratory syndrome coronavirus (SARS-CoV), was passed from palm civets to humans and caused the epidemic of SARS in 2002 to 2003. The major species barriers between humans and civets for SARS-CoV infections are the specific interactions between a defined receptor-binding domain (RBD) on a viral spike protein and its host receptor, angiotensin-converting enzyme 2 (ACE2). In this study a chimeric ACE2 bearing the critical N-terminal helix from civet and the remaining peptidase domain from human was constructed, and it was shown that this construct has the same receptor activity as civet ACE2. In addition, crystal structures of the chimeric ACE2 complexed with RBDs from various human and civet SARS-CoV strains were determined. These structures, combined with a previously determined structure of human ACE2 complexed with the RBD from a human SARS-CoV strain, have revealed a structural basis for understanding the major species barriers between humans and civets for SARS-CoV infections. They show that the major species barriers are determined by interactions between four ACE2 residues (residues 31, 35, 38, and 353) and two RBD residues (residues 479 and 487), that early civet SARS-CoV isolates were prevented from infecting human cells due to imbalanced salt bridges at the hydrophobic virus/receptor interface, and that SARS-CoV has evolved to gain sustained infectivity for human cells by eliminating unfavorable free charges at the interface through stepwise mutations at positions 479 and 487. These result

# To the Editor: Porcine corona-

Coronavirus Hku,

2014

virus (PorCoV) HKU15 is a single-stranded, positive-sense, enveloped RNA virus belonging to the genus Deltacoronavirus (family Coronaviri-dae). PorCoV HKU15 was first iden-tified in 2012 in a surveillance study from China (1). Until February 2014, however, the role of this virus strain in clinical diseases of pigs had not been reported. We recently reported the de-tection of PorCoV strain HKU15-OH1987 in feces samples from sows and intestine samples from piglets in Ohio, United States; the infected ani-mals were from swine farms where outbreaks of diarrheal disease had oc-curred in late January and early Febru-ary 2014 (2). Genetic analysis showed that HKU15-OH1987 is closely re-lated to 2 deltacoronavirus strains that were detected in Hong Kong, China, in 2012: HKU15-155 and HKU15-44 (2). We also demonstrated the pres-ence of histopathologic lesions in the small intestines of PorCoV HKU15– infected piglets with diarrhea (L. Wang, unpub. data). In April 2014, a novel swine enteric coronavirus dis-ease caused by PorCoV HKU15 or porcine epidemic diarrhea virus was reported to the World Animal Health Organization by the US Department of Agricultur

# Neutralizing Antibodies in Patients with Severe Acute Respiratory Syndrome–Associated

Coronavirus Infection,

2016-10-04

Background. Severe acute respiratory syndrome (SARS)–associated coronavirus (SARS-CoV) is the principal etiologic agent of SARS. We analyzed serum samples obtained from 623 patients with SARS in Beijing, to determine whether infection with SARS-CoV can elicit neutralizing antibodies (NAbs). Methods. We developed a highly sensitive and safe neutralization assay using the SARS-CoV pseudotyped virus and used this assay to determine the titers of the NAbs in serum samples from patients with SARS. Results. We found that 85.9 % of serum samples contained NAbs against SARS-CoV and that most of the NAb activities could be attributed to immunoglobulin G. The NAbs became detectable first at 5–10 days after the onset of symptoms, and their levels peaked at 20–30 days and then were sustained for 1150 days. The serum samples could neutralize the pseudotype particles bearing the spike glycoproteins from different SARS-CoV strains, suggesting that the NAbs to SARS-CoV were broadly reactive. Conclusions. NAbs to SARS-CoV are broadly elicited in patients with SARS and, according to their kinetics, may correlate with viral load during the early stages of the disease. These results suggest that it is possible to develop effective vaccines against SARS and that NAbs provide a potential strategy for treating patients with SARS. Severe acute respiratory syndrome (SARS) is a life-threatening form of atypical pneumonia [1–3] that i

# Virus Discovery and Human

Author(s K. A. Pyrc, Human Coronavirus Nl, Krzysztof Pyrc, Coronavirus Nl, Coronavirus Nl,

2007

# Military Medical University, Xi’an, and 3Institute of Basic Medical Sciences, Academy of Military Medical Sciences, and 4Institute of Viral

Syndrome Coronavirus,

2016-09-06

To identify the function of HAb18G/CD147 in invasion of host cells by severe acute respiratory syndrome (SARS) coronavirus (CoV), we analyzed the protein-protein interaction among HAb18G/CD147, cyclophilin A (CyPA), and SARS-CoV structural proteins by coimmunoprecipitation and surface plasmon resonance analy-sis. Although none of the SARS-CoV proteins was found to be directly bound to HAb18G/CD147, the nu-cleocapsid (N) protein of SARS-CoV was bound to CyPA, which interacted with HAb18G/CD147. Further research showed that HAb18G/CD147, a transmembrane molecule, was highly expressed on 293 cells and that CyPA was integrated with SARS-CoV. HAb18G/CD147–antagonistic peptide (AP)–9, an AP of HAb18G/CD147, had a high rate of binding to 293 cells and an inhibitory effect on SARS-CoV. These results show that HAb18G/ CD147, mediated by CyPA bound to SARS-CoV N protein, plays a functional role in facilitating invasion of host cells by SARS-CoV. Our finding provide some evidence for the cytologic mechanism of invasion by SARS-CoV and provide a molecular basis for screening anti-SARS drugs. Severe acute respiratory syndrome (SARS) coronavirus (CoV), the pathogen ascertained to be responsible for SARS, caused disastrous results around the world at the end of 2002 [1, 2]. How SARS-CoV infects host cell

# after Infection

Coronavirus Immunoglobulin,

2016-09-20

# RESEARCH ARTICLE Open Access

Human Coronavirus Oc,

2016-08-25

Full list of author information is available at the end of the articleHuman coronaviruses have a worldwide distribution and are frequently associated with self-limiting upper re-spiratory tract disease or “the common cold”. They can, however, also present with high morbidity outcomes of the lower respiratory tract including bronchiolitis, pneu-monia, [1-3], asthmatic exacerbations [4] and acute ex-acerbations of chronic obstructive pulmonary disease (COPD) [5], where human coronavirus OC43 has been Given the high burden of coronaviruses to human health, and their potential for genetic recombination to give rise to the emergence of completely novel strains, the presence of antiviral strategies is paramount. How-ever, there is currently no commercially available mo-lecular entity which is capable of inhibiting infection with human coronaviruses [7]. The 3CLpro has been vali

# CONTENT ALERTS

Coronavirus Nonstructural Protein,

2007

# A Mouse Model for Betacoronavirus Subgroup 2c Using a Bat

Coronavirus Strain, Hku Variant,

2016-10-04

ABSTRACT Cross-species transmission of zoonotic coronaviruses (CoVs) can result in pandemic disease outbreaks. Middle East respiratory syndrome CoV (MERS-CoV), identified in 2012, has caused 182 cases to date, with ~43%mortality, and no small ani-mal model has been reported. MERS-CoV and Pipistrellus bat coronavirus (BtCoV) strain HKU5 of Betacoronavirus (-CoV) subgroup 2c share&gt;65 % identity at the amino acid level in several regions, including nonstructural protein 5 (nsp5) and the nucleocapsid (N) protein, which are significant drug and vaccine targets. BtCoV HKU5 has been described in silico but has not been shown to replicate in culture, thus hampering drug and vaccine studies against subgroup 2c -CoVs. We report the syn-thetic reconstruction and testing of BtCoVHKU5 containing the severe acute respiratory syndrome (SARS)-CoV spike (S) glyco-protein ectodomain (BtCoVHKU5-SE). This virus replicates efficiently in cell culture and in young and agedmice, where the virus targets airway and alveolar epithelial cells. Unlike some subgroup 2b SARS-CoV vaccines that elicit a strong eosinophilia following challenge, we demonstrate that BtCoVHKU5 andMERS-CoVN-expressing Venezuelan equine encephalitis virus rep-licon particle (VRP) vaccines do not cause extensive eosinophilia following BtCoVHKU5-SE challenge. Passage of BtCoV HKU5-SE in youngmice resulted in enhanced virulence, causing 20%weight loss, diffuse alveolar damage, and hyaline mem

# Transcriptomic Analysis Reveals a Mechanism for a Prefibrotic Phenotype in STAT1 Knockout Mice during Severe Acute Respiratory Syndrome

Coronavirus Infection, Marcus J,

2010

Severe acute respiratory syndrome coronavirus (SARS-CoV) infection can cause the development of severe end-stage lung disease characterized by acute respiratory distress syndrome (ARDS) and pulmonary fibrosis. The mechanisms by which pulmonary lesions and fibrosis are generated during SARS-CoV infection are not known. Using high-throughput mRNA profiling, we examined the transcriptional response of wild-type (WT), type I interferon receptor knockout (IFNAR1/), and STAT1 knockout (STAT1/) mice infected with a recombinant mouse-adapted SARS-CoV (rMA15) to better understand the contribution of specific gene expression changes to disease progression. Despite a deletion of the type I interferon receptor, strong expres-sion of interferon-stimulated genes was observed in the lungs of IFNAR1/ mice, contributing to clearance of the virus. In contrast, STAT1/ mice exhibited a defect in the expression of interferon-stimulated genes and were unable to clear the infection, resulting in a lethal outcome. STAT1/ mice exhibited dysregulation of T-cell and macrophage differentiation, leading to a TH2-biased immune response and the development of alternatively activated macrophages that mediate a profibrotic environment within the lung. We propose that a combination of impaired viral clearance and T-cell/macrophage dysregulation causes the formation of prefibrotic lesions in the lungs of rMA15-infected STAT1/ mice. The severe acute respiratory syndrome coronavirus (SARS

# A Mouse Model for Betacoronavirus Subgroup 2c Using a Bat

Coronavirus Strain, Hku Variant,

2016-08-21

ABSTRACT Cross-species transmission of zoonotic coronaviruses (CoVs) can result in pandemic disease outbreaks. Middle East respiratory syndrome CoV (MERS-CoV), identified in 2012, has caused 182 cases to date, with ~43%mortality, and no small ani-mal model has been reported. MERS-CoV and Pipistrellus bat coronavirus (BtCoV) strain HKU5 of Betacoronavirus (-CoV) subgroup 2c share&gt;65 % identity at the amino acid level in several regions, including nonstructural protein 5 (nsp5) and the nucleocapsid (N) protein, which are significant drug and vaccine targets. BtCoV HKU5 has been described in silico but has not been shown to replicate in culture, thus hampering drug and vaccine studies against subgroup 2c -CoVs. We report the syn-thetic reconstruction and testing of BtCoVHKU5 containing the severe acute respiratory syndrome (SARS)-CoV spike (S) glyco-protein ectodomain (BtCoVHKU5-SE). This virus replicates efficiently in cell culture and in young and agedmice, where the virus targets airway and alveolar epithelial cells. Unlike some subgroup 2b SARS-CoV vaccines that elicit a strong eosinophilia following challenge, we demonstrate that BtCoVHKU5 andMERS-CoVN-expressing Venezuelan equine encephalitis virus rep-licon particle (VRP) vaccines do not cause extensive eosinophilia following BtCoVHKU5-SE challenge. Passage of BtCoV HKU5-SE in youngmice resulted in enhanced virulence, causing 20%weight loss, diffuse alveolar damage, and hyaline mem

# RESEARCH Detection of new genetic

Coronavirus Chiroptera Pteropodidae Madagascar,

2016-09-02

is available at the end of the articleBackground Coronaviruses (CoVs) are enveloped viruses with single-stranded positive-sense RNA belonging to the subfamily Coronavirinae in the family Coronaviridae (order Nido-virales). Genomes of CoVs range from 25 to 32 kb and show high genetic diversity [1]. CoVs are classified into four genera: Alphacoronavirus, Betacoronavirus, Gam-macoronavirus, and Deltacoronavirus [2]. In mammals and birds, CoVs are associated with upper and lower respiratory illnesses or gastroenteritis. In humans, CoVs infections are commonly caused by HCoV-229E and HCoV-OC43 which generally cause mild respiratory illnesses [3]. A new CoV that causes se-vere acute respiratory syndrome (SARS-CoV) emerged in humans in 2002–2003 and infected more than 8,000 individuals with mortality rates estimated at around 10

# Proteome Profile of Swine Testicular Cells Infected with

Porcine Transmissible, Gastroenteritis Coronavirus,

2016-08-21

The interactions occurring between a virus and a host cell during a viral infection are complex. The purpose of this paper was to analyze altered cellular protein levels in porcine transmissible gastroenteritis coronavirus (TGEV)-infected swine testicular (ST) cells in order to determine potential virus-host interactions. A proteomic approach using isobaric tags for relative and absolute quantitation (iTRAQ)-coupled two-dimensional liquid chromatography-tandem mass spectrometry identification was conducted on the TGEV-infected ST cells. The results showed that the 4-plex iTRAQ-based quantitative approach identified 4,112 proteins, 146 of which showed significant changes in expression 48 h after infection. At 64 h post infection, 219 of these proteins showed significant change, further indicating that a larger number of proteomic changes appear to occur during the later stages of infection. Gene ontology analysis of the altered proteins showed enrichment in multiple biological processes, including cell adhesion, response to stress, generation of precursor metabolites and energy, cell motility, protein complex assembly, growth, developmental maturation, immune system process, extracellular matrix organization, locomotion, cell-cell signaling, neurological system process, and cell junction organization. Changes in the expression levels of transforming growth factor beta 1 (TGF-b1), caspase-8, and heat shock protein 90 alpha (HSP90a) were also verified by western blot analysis. To our knowledge, this study is the first time the response profile of ST host cells following TGEV infection has been analyzed using iTRAQ technology, and our description of the late proteomic change

# Correspondence Questions Concerning

The New Haven Coronavirus,

2016-09-11

To the Editor—Esper et al. present the discovery of a novel human coronavirus (HCoV) in young children and infants with respiratory tract disease in New Haven: HCoV-NH [1]. However, they also men-tion that the virus is very similar to HCoV-NL63, a virus that was identified previously in Amsterdam, The Netherlands [2]. De-spite this, the 2 studies by Esper et al. [1, 3] and an Editorial Commentary by Mc-Intosh [4] avoid usage of the name “HCoV-NL63 ” while repeatedly claiming the dis-covery of a novel virus. To judge whether HCoV-NH is really a novel HCoV, a comparative analysis of HCoV-NH with a number of different fea-tures of established HCoVs should be per-formed. Examining the relatedness of ge-nome sequences is one facet of such an analysis. Unfortunately, limited data on the genome sequence for HCoV-NH are avail-able, but inspection of a 126-bp fragment clearly shows that all HCoV-NH isolates cluster together with the HCoV-NL63 Am-sterdam-1 strain (figure 3 in [1]). This re-sult strongly suggests that the viruses found by Esper et al. are New Haven isolates of HCoV-NL63. Moreover, the actual nucle-otide difference between the New Haven isolates (GenBank accession nos. AY870943– AY871008) and the HCoV-NL63 isolate Amsterdam-1 (GenBank accession no. NC \_005831) is 0%–6%. This degree of differ-ence falls well within the range of genetic variation observed among different HCoV-NL63 isolates from Amsterdam [2]. We re-ported the presence of distinct HCoV-NL63 variants that apparently are cocirculating, as has been confirmed recently by Arden et al. [5] and Bastien et al. [6]. Esper et al. do not seem to dispute that HCoV-NH is very similar to HCoV-NL63. What then made them decide to claim the identification of a novel virus? The only argument mentioned is that the research project was initiated before the first arti-cle on HCoV-NL63 was published. Is that how it works in science? No—only the first report can claim a novel scientific finding. In fact, for HCoV-NL63, Esper et al.’s is the third article that claims its dis-covery. The identification of HCoV-NL63 was first announced in an article in Nature Medicine (which was published electron-ically on 21 March 2004 [2]), and an ar-ticle by Fouchier et al. in the Proceeding

# Letter to the Editor Deubiquitination, a New Function of the Severe Acute Respiratory Syndrome

Coronavirus Papain-like Protease,

2016-10-11

A new coronavirus has been identified as the infectious agent of severe acute respiratory syndrome (SARS). Although SARS was successfully contained by quarantine measures, the reconstructed itinerary of the virus through 30 countries and its high mortality rate illustrate the global threat that this newly emerging disease represents (17). During the expression of the SARS coronavirus (SCoV) genome, two viral cysteine pro-teases, a papain-like protease (PLpro) and a chymotrypsin-like protease (3CLpro), process the encoded polyprotein precursor to release most of the proteins required for virus replication. PLpro refers to a domain of nonstructural protein 3, whose boundaries are defined by homology to the papain-like fold (7). The PLpro domain can be regarded as the catalytic core behind PLpro-mediated cleavages, even though processing by PLpros has been reported to be modulated by additiona

# Return to Work Guidelines

SHU Coronavirus Management Team,,

2020-07-01T07:00:00Z

# Vector Based on the Transmissible Gastroenteritis

Javier Ortego, David Escors, Hubert Laude, Coronavirus Genome, On The Transmissible Gastroenteritis, Coronavirus Genome,

2002

# Research Involving Human Subjects

Coronavirus Hku, The United States,

2013-07-31

# RESEARCH ARTICLE Critical Assessment of the Important Residues Involved in the Dimerization and

Catalysis Mers, Coronavirus Main Protease,

1371

Background A highly pathogenic human coronavirus (CoV), Middle East respiratory syndrome coronavirus (MERS-CoV), has emerged in Jeddah and other places in Saudi Arabia, and has quickly spread to European and Asian countries since September 2012. Up to the 1st October 2015 it has infected at least 1593 people with a global fatality rate of about 35%. Studies to understand the virus are necessary and urgent. In the present study, MERS-CoVmain protease (Mpro) is expressed; the dimerization of the protein and its relationship to catalysis are investigated. Methods and Results The crystal structure of MERS-CoV Mpro indicates that it shares a similar scaffold to that of other coronaviral Mpro and consists of chymotrypsin-like domains I and II and a helical domain III of five helices. Analytical ultracentrifugation analysis demonstrated that MERS-CoV Mpro undergoes a monomer to dimer conversion in the presence of a peptide substrate. Glu169 is a key residue and plays a dual role in both dimerization and catalysis. The muta-genesis of other residues found on the dimerization interface indicate that dimerization of MERS-CoV Mpro is required for its catalytic activity. One mutation, M298R, resulted in a sta-ble dimer with a higher level of proteolytic activity than the wild-type enzyme. Conclusions MERS-CoV Mpro shows substrate-induced dimerization and potent proteolytic activity. A critical assessment of the residues important to these processes provides insights into the correlation between dimerization and catalysis within the coronaviral Mpro family

# Key words: coronavirus MHV-JHM/nucleotide sequence/surfi~ce projection glycoprotein gene Nucleotide Sequence of the Gene Encoding the Surface Projection

Glycoprotein Of Coronavirus Mhv-jhm,

1986

Sequences encoding the surface projection glycoprotein of the coronavirus, murine hepatitis virus (MHV), strain JHM, have been cloned into pAT153 using cDNA produced by priming with specific oligonucleotides on infected cell RNA. The regions of three clones pJMS1010, pJS112 and pJS92, which together encompass the surface protein gene have been sequenced by the chain termination method. The sequence of the primary translation product, deduced from the DNA sequence, predicts a polypeptide of 1235 amino acids with a molecular weight of 136600. This polypeptide displays the features characteristic of a group 1 membrane protein; an amino-terminal signal sequence and carboxy-terminal membrane and cytoplasmic domains. There are 21 potential glycosylation sites in the polypeptide and a cysteine-rich region in the vicinity of the transmembrane domain. During maturation proteolytic processing of the polypeptide occurs and at positions 624 to 628 the sequence Arg Arg-Ala-Arg-Arg is found, which is similar to a number of basic sequences involved in the cleavage of enveloped RNA virus glycoproteins. The fusogenic properties of the MHV surface protein do not appear to correlate with a strongly hydrophobic region at the putative amino terminus of the carboxy-terminat cleavage product

# doi:10.1155/2011/734690 Research Article The Effects of Temperature and Relative Humidity on the

Viability Of The Sars Coronavirus,

2013-02-14

Copyright © 2011 K. H. Chan et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. The main route of transmission of SARS CoV infection is presumed to be respiratory droplets. However the virus is also detectable in other body fluids and excreta. The stability of the virus at different temperatures and relative humidity on smooth surfaces were studied. The dried virus on smooth surfaces retained its viability for over 5 days at temperatures of 22–25 ◦ Candrelative humidity of 40–50%, that is, typical air-conditioned environments. However, virus viability was rapidly lost (&gt;3 log10) at higher temperatures and higher relative humidity (e.g., 38 ◦ C, and relative humidity of&gt;95%). The better stability of SARS coronavirus at low temperature and low humidity environment may facilitate its transmission in community in subtropical area (such as Hong Kong) during the spring and in air-conditioned environments. It may also explain why some Asian countries in tropical area (such as Malaysia, Indonesia or Thailand) with high temperature and high relative humidity environment did not have major community outbreaks of SARS. 1

# Identification and Characterization of the Putative Fusion Peptide of the Severe Acute Respiratory Syndrome-Associated

Coronavirus Spike Protein, William C. Wimley,

2004

Severe acute respiratory syndrome-associated coronavirus (SARS-CoV) is a newly identified member of the family Coronaviridae and poses a serious public health threat. Recent studies indicated that the SARS-CoV viral spike glycoprotein is a class I viral fusion protein. A fusion peptide present at the N-terminal region of class I viral fusion proteins is believed to initiate viral and cell membrane interactions and subsequent fusion. Although the SARS-CoV fusion protein heptad repeats have been well characterized, the fusion peptide has yet to be identified. Based on the conserved features of known viral fusion peptides and using Wimley and White interfacial hydrophobicity plots, we have identified two putative fusion peptides (SARSWW-I and SARSWW-II) at the N terminus of the SARS-CoV S2 subunit. Both peptides are hydrophobic and rich in alanine, glycine, and/or phenylalanine residues and contain a canonical fusion tripeptide along with a central proline residue. Only the SARSWW-I peptide strongly partitioned into the membranes of large unilamellar vesicles (LUV), adopting a -sheet structure. Likewise, only SARSWW-I induced the fusion of LUV and caused membrane leakage of vesicle contents at peptide/lipid ratios of 1:50 and 1:100, respectively. The activity of this synthetic peptide appeared to be dependent on its amino acid (aa) sequence, as scrambling the peptide rendered it unable to partition into LUV, assume a defined secondary structure, or induce both fusion and leakage of LUV. Based on the activity of SARSWW-I, we propose that the hydrophobic stretch of 19 aa corresponding to residue

# HAI = Hemagglutination inhibition

Xxiv Ciclo, Director Prof, Bruno Masala, Tutor Prof, Hbov Human Bocavirus, Hcov-hku Human Coronavirus- Hku, Hcov-oc Human Coronavirus- Oc, Hcov-nl Human Coronavirus- Nl,

2015-10-29

# SUMMARY

Struetura Polypeptides, Coronavirus Ibv, David Cavanagh,

1980

Avian infectious bronchitis virus (IBV) was grown and radiolabelled with 35S-methionine, 3H-leucine and 3H-glucosamine in de-embryonated chicken eggs. Approximately 12 different polypeptides were clearly detected by SDS-polyacrylamide gel electrophoresis of virus preparations. Growth of IBV in chorioallantoic membrane cells labelled with 35S-methionine dicated that most of these polypeptides, and additional ones, some of which were gtycosylated, were host components. Five polypeptides appeared to be virus-coded, with apparent tool. wt. o

# The rank of pathogen detection rate and co-pathogen rate in 4242 pediatric patients with ARI in Guangzhou from July 2009 to June 2012.

Wen Kuan Liu (559440), Qian Liu (135614), De Hui Chen (559441), Huan Xi Liang (559442), Xiao Kai Chen (559443), Mei Xin Chen (559444), Shu Yan Qiu (559445), Zi Yeng Yang (559446), Rong Zhou (138287),

2014-05-05T03:18:57Z

# CONTENT ALERTS

Fathy Gaber, Sanjay Kapil, Coronavirus Bovine,

1999

# Alignment of the 3′-terminal 55-nt sequence within the individual subgroups of betacoronaviruses.

Wei-Yu Liao (300267), Ting-Yung Ke (438913), Hung-Yi Wu (409276),

2014-05-22T04:00:10Z

<p>The conserved sequence within individual subgroups is identified by shading. Position numbers −1 and −55 below the sequence indicate the first and 55th nt counted from the poly(A) tail, respectively. Abbreviations: CoV, coronavirus; BCoV, bovine coronavirus-Mebus; OC43, human coronavirus-OC43; HECoV-4408, human enteric coronavirus-4408; HEV, porcine hemagglutinating encephalomyelitis virus-TN11; ECoV, equine coronavirus-NC99; MHVA59, mouse hepatitis virus-A59; MHVJ, mouse hepatitis virus-JHM; PV, puffinosis virus; SARS-CoV, SARS coronavirus; B-S-HKU3, bat SARS coronavirus HKU3; B-S-HKU4, bat SARS coronavirus HKU4; B-S-HKU5, bat SARS coronavirus HKU5; BCHKU9-1, bat coronavirus HKU9-1; BCHKU9-2, bat coronavirus HKU9-2; BCHKU9-3, bat coronavirus HKU9-3; BCHKU9-4, bat coronavirus HKU9-4. GenBank Accession Nos. for the sequences studied here are as follows: U00735 for BCoV-Mebus, AF523847 for HCoV-OC43, AF523848 for HECoV-4408, AF523849 for HEV-TN11, AF523850 for ECoV-NC99, NC001846 for MHV-A59, X00990 for MHV-JHM, AJ544718 for PV, NC004718 for SARS-CoV, NC009694 for B-S-HKU3, NC009019 for B-S-HKU4, NC009020 for B-S-HKU5, EF065513.1 for BCHKU9-1, EF065514.1 for BCHKU9-2, EF065515.1 for BCHKU9-3, and EF065516.1 for BCHKU9-4.</p

# CONTENT ALERTS

Fathy Gaber, Sanjay Kapil, Coronavirus Bovine,

1999

# Effects of Toll-Like Receptor Stimulation on Eosinophilic Infiltration in Lungs of BALB/c Mice Immunized with UV-Inactivated Severe

Acute Respiratory, Syndrome-related Coronavirus Vaccine,

2016-09-28

Severe acute respiratory syndrome-related coronavirus (SARS-CoV) is an emerging pathogen that causes severe respiratory ill-ness. Whole UV-inactivated SARS-CoV (UV-V), bearing multiple epitopes and proteins, is a candidate vaccine against this virus. However, whole inactivated SARS vaccine that includes nucleocapsid protein is reported to induce eosinophilic infiltration in mouse lungs after challenge with live SARS-CoV. In this study, an ability of Toll-like receptor (TLR) agonists to reduce the side effects of UV-V vaccination in a 6-month-old adult BALB/c mouse model was investigated, using the mouse-passaged Frankfurt 1 isolate of SARS-CoV. Immunization of adult mice with UV-V, with or without alum, resulted in partial protection from lethal doses of SARS-CoV challenge, but extensive eosinophil infiltration in the lungs was observed. In contrast, TLR agonists added to UV-V vaccine, including lipopolysaccharide, poly(U), and poly(I·C) (UV-VTLR), strikingly reduced excess eosinophilic infil-tration in the lungs and induced lower levels of interleukin-4 and-13 and eotaxin in the lungs than UV-V-immunization alone. Additionally, microarray analysis showed that genes associated with chemotaxis, eosinophil migration, eosinophilia, and cell movement and the polarization of Th2 cells were upregulated in UV-V-immunized but not in UV-VTLR-immunized mice. In particular, CD11b cells in the lungs of UV-V-immunized mice showed the upregulation of genes associated with the induction of eosinophils after challenge. These findings suggest that vaccine-induced eosinophil immunopathology in the lungs upon SARS-CoV infection could be avoided by the TLR agonist adjuvants

# Seasonal distribution of the pathogens in 4242 pediatric patients with ARI in Guangzhou from July 2009 to June 2012.

Wen Kuan Liu (559440), Qian Liu (135614), De Hui Chen (559441), Huan Xi Liang (559442), Xiao Kai Chen (559443), Mei Xin Chen (559444), Shu Yan Qiu (559445), Zi Yeng Yang (559446), Rong Zhou (138287),

2014-05-05T03:18:57Z

# Coronavirus diversity, phylogeny and interspecies jumping

Huang, Y, Yuen, KY, Woo, PCY, Lau, SKP,

2009

The SARS epidemic has boosted interest in research on coronavirus biodiversity and genomics. Before 2003, there were only 10 coronaviruses with complete genomes available. After the SARS epidemic, up to December 2008, there was an addition of 16 coronaviruses with complete genomes sequenced. These include two human coronaviruses (human coronavirus NL63 and human coronavirus HKU1), 10 other mammalian coronaviruses [bat SARS coronavirus, bat coronavirus (bat-CoV) HKU2, bat-CoV HKU4, bat-CoV HKU5, bat-CoV HKU8, bat-CoV HKU9, bat-CoV 512/2005, bat-CoV 1A, equine coronavirus, and beluga whale coronavirus] and four avian coronaviruses (turkey coronavirus, bulbul coronavirus HKU11, thrush coronavirus HKU12, and munia coronavirus HKU13). Two novel subgroups in group 2 coronavirus (groups 2c and 2d) and two novel subgroups in group 3 coronavirus (groups 3b and 3c) have been proposed. The diversity of coronaviruses is a result of the infidelity of RNA-dependent RNA polymerase, high frequency of homologous RNA recombination, and the large genomes of coronaviruses. Among all hosts, the diversity of coronaviruses is most evidenced in bats and birds, which may be a result of their species diversity, ability to fly, environmental pressures, and habits of roosting and flocking. The present evidence supports that bat coronaviruses are the gene pools of group 1 and 2 coronaviruses, whereas bird coronaviruses are the gene pools of group 3 coronaviruses. With the increasing number of coronaviruses, more and more closely related coronaviruses from distantly related animals have been observed, which were results of recent interspecies jumping and may be the cause of disastrous outbreaks of zoonotic diseases. Copyright © 2009 by the Society for Experimental Biology and Medicine.link\_to\_subscribed\_fulltex

# The patterns of pathogen distribution among different pediatric age groups in 4242 pediatric patients with ARI in Guangzhou from July 2009 to June 2012.

Wen Kuan Liu (559440), Qian Liu (135614), De Hui Chen (559441), Huan Xi Liang (559442), Xiao Kai Chen (559443), Mei Xin Chen (559444), Shu Yan Qiu (559445), Zi Yeng Yang (559446), Rong Zhou (138287),

2014-05-05T03:18:57Z

# Human coronavirus types

Coronaviruses are named for the crown-like spikes on their surface. There are four main sub-groupings of coronaviruses, known as alpha, beta, gamma, and delta.Human coronaviruses were first identified in the mid-1960s. The seven coronaviruses that can infect people are:Common human coronaviruses1. 229E (alpha coronavirus)2. NL63 (alpha coronavirus)3. OC43 (beta coronavirus)4. HKU1 (beta coronavirus)Other human coronaviruses5. MERS-CoV (the beta coronavirus that causes Middle East Respiratory Syndrome, or MERS)6. SARS-CoV (the beta coronavirus that causes severe acute respiratory syndrome, or SARS)7. 2019 Novel Coronavirus (2019-nCoV)People around the world commonly get infected with human coronaviruses 229E, NL63, OC43, and HKU1.Sometimes coronaviruses that infect animals can evolve and make people sick and become a new human coronavirus. Three recent examples of this are 2019-nCoV, SARS-CoV, and MERS-CoV.2020715

# Coronavirus SARS-CoV-2: informació sobre les actuacions preventives en referència al nou coronavirus SARS-CoV-2 en l’àmbit de la residència [fullet]

2020-03-19

# Detection of respiratory tract pathogens from 4242 pediatric patients with ARI by real-time PCR.

Wen Kuan Liu (559440), Qian Liu (135614), De Hui Chen (559441), Huan Xi Liang (559442), Xiao Kai Chen (559443), Mei Xin Chen (559444), Shu Yan Qiu (559445), Zi Yeng Yang (559446), Rong Zhou (138287),

2014-05-05T03:18:57Z