Monthly Newsletter
May 2013

ANSORP *NOW*

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Dear ANSORP Investigators

Greetings from Seoul !
I hope all ANSORP investigators are doing well and wish you and your family the New Year filled with joy and happiness.
This is the 2013 May issue of ANSORP NOW. It provides update information and current status of ANSORP activities. "ANSORP NOW" is a monthly newsletter, delivered to all ANSORP investigators by e-mail and website of APFID (www.apfid.org).

Please read this ANSORP NOW carefully to update our international collaboration. If you have any ideas, opinions, or issues that can be shared with other ANSORP investigators, please send us e-mails or FAX.

I always appreciate your active participation in the ANSORP activities.

Jae-Hoon Song, MD, PhD
Organizer, ANSORP
Founder & Chairman, APFID

Sixty-six World Health Assembly

The Sixty-sixth World Health Assembly was held on May 20-28, 2013 in Geneva, as officials from 194 Member States have begun their annual review of the activities of WHO and set new priorities for the future. The Health Assembly has discussed specific health topics like non-communicable diseases, universal health coverage, women and children's health, as well as the program budget, administration, reform and management matters of WHO.

During the World Health Assembly, the United Kingdom and Sweden co-hosted a World Health Assembly lunchtime event on 21 May 2013 to consider **'Antibiotic resistance - a threat to global health security'**. The event aimed to consider the risks we face and the challenges we need to overcome; and to share learning about what is being done at a national and global level to address the issue and to discuss what needs to happen next. At this lunchtime event, APFID and ANSORP's efforts and experiences to control and prevent antimicrobial resistance in the Asian region since 1996 were introduced and shared with the Member States.

Contact Information

Jae-Hoon Song, MD, PhD
Organizer, ANSORP / Chairman, APFID
Samsung Medical Center
Tel: 82-2-3410-0320, FAX: 82-2-3410-0041
E-mail: ansorp@gmail.com or
songjh@skku.edu

Doo Ryeon Chung, MD, PhD
Coordinator, ANSORP
Samsung Medical Center
Tel: 82-2-3410-0323, FAX: 82-2-3410-0041
E-mail: iddrchung@gmail.com or
drchung@skku.edu

So Hyun Kim, DVM, PhD
Project Manager, ANSORP
Asia Pacific Foundation for Infectious Diseases
Tel: 82-2-3410-6826, FAX: 82-2-3410-6667
E-mail: shkim@ansorp.org or
shkim.ansorp@gmail.com

Interesting papers

Origin and diversity of novel avian influenza A H7N9 viruses causing human infection: phylogenetic, structural, and coalescent analyses

Lancet. 2013 May 1. [Epub ahead of print]

Liu D, Shi W, Shi Y, Wang D, Xiao H, Li W, Bi Y, Wu Y, Li X, Yan J, Liu W, Zhao G, Yang W, Wang Y, Ma J, Shu Y, Lei F, Gao GF.

ABSTRACT

BACKGROUND: On March 30, 2013, a novel avian influenza A H7N9 virus that infects human beings was identified. This virus had been detected in six provinces and municipal cities in China as of April 18, 2013. We correlated genomic sequences from avian influenza viruses with ecological information and did phylogenetic and coalescent analyses to extrapolate the potential origins of the virus and possible routes of reassortment events.

METHODS: We downloaded H7N9 virus genome sequences from the Global Initiative on Sharing Avian Influenza Data (GISAID) database and public sequences used from the Influenza Virus Resource. We constructed phylogenetic trees and did 1000 bootstrap replicates for each tree. Two rounds of phylogenetic analyses were done. We used at least 100 closely related sequences for each gene to infer the overall topology, removed suspicious sequences from the trees, and focused on the closest clades to the novel H7N9 viruses. We compared our tree topologies with those from a bayesian evolutionary analysis by sampling trees (BEAST) analysis. We used the bayesian Markov chain Monte Carlo method to jointly estimate phylogenies, divergence times, and other evolutionary parameters for all eight gene fragments. We used sequence alignment and homology-modelling methods to study specific mutations regarding phenotypes, specifically addressing the human receptor binding properties.

FINDINGS: The novel avian influenza A H7N9 virus originated from multiple reassortment events. The HA gene might have originated from avian influenza viruses of duck origin, and the NA gene might have transferred from migratory birds infected with avian influenza viruses along the east Asian flyway. The six internal genes of this virus probably originated from two different groups of H9N2 avian influenza viruses, which were isolated from chickens. Detailed analyses also showed that ducks and chickens probably acted as the intermediate hosts leading to the emergence of this virulent H7N9 virus. Genotypic and potential phenotypic differences imply that the isolates causing this outbreak form two separate subclades.

INTERPRETATION: The novel avian influenza A H7N9 virus might have evolved from at least four origins. Diversity among isolates implies that the H7N9 virus has evolved into at least two different lineages. Unknown intermediate hosts involved might be implicated, extensive global surveillance is needed, and domestic-poultry-to-person transmission should be closely watched in the future.

Genomic signature and protein sequence analysis of a novel influenza A (H7N9) virus that causes an outbreak in humans in China

Microbes Infect. 2013 Apr 27. [Epub ahead of print]

Liu Q, Lu L, Sun Z, Chen GW, Wen Y, Jiang S.

ABSTRACT

Very recently, a new avian flu outbreak in humans, which is caused by a novel H7N9 influenza A virus (AIV), was reported in China. As of April 13, 2013, 49 confirmed cases (mainly middle-aged to elderly males), including 11 deaths, were reported in China. Here we analyzed the genomic signatures and protein sequences of the human H7N9 AIVs. We found that the genomic signatures of A(H7N9) had high and low identity to avian and human IAVs, respectively, suggesting its avian origin. The signature amino acids of A(H7N9) had high identity to 1997 H5N1 and 2009 H1N1, but low identity to those influenza strains that caused pandemics before 1980. One of the key signature amino acids at 627 in PB2 mutated to lysine, which is associated with mammalian adaptation and increased virulence of the highly pathogenic avian influenza A(H5N1) virus. Besides, several other human-like signatures, including PB2-44S, PA-100A, PA-356R, and PA-409N are also found in this avian-origin A(H7N9) virus. The HA protein has the Q226L mutation, which is associated with increased binding to mammalian-like receptors bearing alpha 2,6 receptor in the human upper airway. The M2 protein contains the N31S mutation, suggesting its resistance to the M2 channel blockers amantadine and rimantadine. These findings suggest that this avian-origin AIV gains its bird-to-human, i.e., zoonotic, transmissibility and increased virulence, as well as drug-resistance, by mutating key signature amino acid residues and those in the functional domains of the viral proteins. Therefore, it is prudent to monitor the evolution of A(H7N9), as well as develop strategies to combat any potential epidemic or pandemic.

If you need PDF version of the papers, please contact ANSORP Project Manager (Dr. So Hyun Kim, shkim@ansorp.org).

We always appreciate your active contribution to ANSORP activities.
If you have any questions, or issues that can be shared with other ANSORP investigators, please let us know them at any time.