International Journal of Infectious Diseases 33 (2015) 130–131

Contents lists available at ScienceDirect

International Journal of Infectious Diseases

journal homepage: www.elsevier.com/locate/ijid

Short Communication

Isolation and characteristic analysis of a novel strain H7N9 of avian influenza virus A from a patient with influenza-like symptoms in China

Faming Chen [a], Junhua Li [b], Biancheng Sun [a], Hong Zhang [b], Rusheng Zhang [a], Jie Yuan [a], Xinhua Ou [a,*], Wen Ye [a], Jingfang Chen [a], Yunzhi Liu [b], Yiwei Huang [b]

[a] Changsha Center for Disease Control and Prevention, Changsha, Hunan, 410001, China
[b] Hunan Provincial Center for Disease Control and Prevention, Changsha, Hunan, China

ARTICLE INFO

Article history:
Received 9 August 2014
Accepted 14 January 2015

Corresponding Editor: Eskild Petersen, Aarhus, Denmark

Keywords:
Avian influenza virus
Genetic analysis
H7N9
Influenza surveillance
Mutations

SUMMARY

A novel H7N9 virus (A/Changsha/1/2013(H7N9)) identified through routine examination in the influenza network laboratory was analyzed retrospectively. The gene sequences of A/Changsha/1/2013(H7N9) were highly homologous to other viruses isolated in mainland China. Mutations of Q226L and G186 V were found in the hemagglutinin protein (HA). Amino acid deletions were found at positions 69–73 of the neuraminidase protein (NA) and 218–230 of the non-structural protein (NS1). All viral genes except PB1 were essentially identical to the sequences of other Chinese influenza A H7N9 isolates. Overall, A/Changsha/1/2013(H7N9) is highly homologous to other H7N9 avian influenza viruses isolated in mainland China.

1. Introduction

A new reassortant viral subtype of avian influenza virus named H7N9 was first reported in mainland China on March 27, 2013.[1] No evidence indicating human-to-human transmission has been found, but some studies have shown that the H7N9 avian influenza virus could be transmitted from birds to humans.[2]

In April 2013, a mild case of H7N9 infection in a human was identified during routine examination at the influenza network laboratory. We analyzed the genetic characteristics of this strain retrospectively.

2. Materials and methods

Throat swab specimens were collected from a 2-year-old child who had a fever and flu-like symptoms. The virus was isolated, cultured, and identified at the Chinese National Influenza Center (Changsha, China). The sequence homology of the hemagglutinin (HA), neuraminidase (NA), matrix protein (MP), nucleocapsid protein (NP), non-structural protein (NS), polymerase acidic (PA), polymerase basic (PB1), and PB2 genes were analyzed by online BLAST analysis. Phylogenetic trees were constructed using MEGA 5.0 software.

3. Results

The online BLAST analysis showed that the HA, NA, MP, NP, NS, PA, PB1, and PB2 sequences of A/Changsha/1/2013(H7N9) were highly homologous (homology >99%) to other viruses isolated in mainland China between March and April, 2013. The mutations in the A/Changsha/1/2013(H7N9) virus were Q226L and G186 V in the HA protein, S31N in the M2 protein, P42S in the NS1 protein, N30D and T215A in the M1 protein, and L89 V in the PB1 protein. Amino acid deletions were found in the NA protein at positions 69–73 and in the NS1 protein at positions 218–230. Phylogenetic trees showed that all viral genes except PB1 were essentially identical to the sequences of other Chinese influenza A H7N9 isolates. The PB1 gene formed a single sub-branch (Figure 1).

4. Discussion

A/Changsha/1/2013(H7N9) and other new H7N9 viruses isolated in mainland China and Taiwan in 2013 were in the same branch in the phylogenetic trees. All viral genes except PB1 were essentially identical to the sequences of other Chinese influenza A H7N9 isolates. The PB1 gene of A/Changsha/1/2013(H7N9) formed

* Corresponding author. Tel.: +86 0731 84712291; fax: +86 0731 84726810.
E-mail address: xhouteam@163.com (X. Ou).

http://dx.doi.org/10.1016/j.ijid.2015.01.013

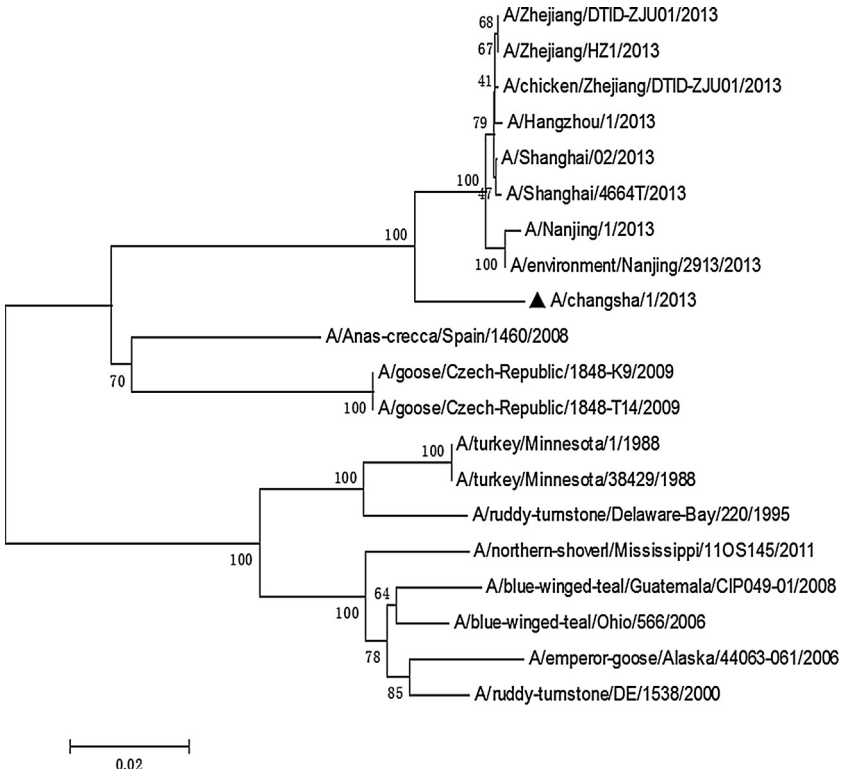

Figure 1. Phylogenetic tree for the PB1 gene of the H7N9 virus isolated from a patient in Hunan Province, China. ▲ = the sequence of the H7N9 virus in our study.

a separate sub-branch. The HA protein harbored Q226L and G186 V mutations, which have been reported to enhance binding to human receptors.[3] Amino acid deletions were found in the NA protein at amino acids 69–73 and in the NS1 protein at amino acids 218–230. As reported previously, the amino acid deletions at positions 69–73 in the NA protein may increase viral virulence in mammals,[4] whereas the amino acid deletions at 218–230 in the NS1 protein may reduce virus virulence in mammals.[5]

In conclusion, the A/Changsha/1/2013(H7N9) investigated in this study is highly homologous to the other H7N9 avian influenza viruses isolated in mainland China.

Acknowledgements

This work was supported by the Medical and Health Research Project of Hunan Province, China (grant number B2013-126).

Conflict of interest: None.

References

1. Gao R, Cao B, Hu Y, Feng Z, Wang D, Hu W. Human infection with a novel avian-origin influenza A(H7N9) virus. *N Engl J Med* 2013;**368**:1888–97.
2. Chen Y, Liang WF, Yang SG, Wu N, Gao H, Sheng J, et al. Human infections with the emerging avian influenza A H7N9 virus from wet market poultry: clinical analysis and characterisation of viral genome. *Lancet* 2013;**381**:1916–25.
3. Nidom CA, Takano R, Yamada S, Sakai-Taqawa Y, Daulay S, Aswadi D, et al. Influenza A(H5N1) viruses from pigs. *Indonesia Emerg Infect Dis* 2010;**16**: 1515–23.
4. Matsuoka Y, Swayne DE, Thomas C, Rameix-Welti MA, Naffakh N, Warnes C, et al. Neuraminidase stalk length and additional glycosylation of the hemagglutinin influence the virulence of influenza H5N1 viruses for mice. *J Virol* 2009;**83**: 4704–8.
5. Jackson D, Hossain MJ, Hickman D, Perez DR, Lamb RA. A new influenza virus virulence determinant: the NS1 protein four C-terminal residues modulate pathogenicity. *Proc Natl Acad Sci U S A* 2008;**105**:4381–6.