

9 March 2010

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Chem-Bio News – Pandemic Influenza Edition #102

1. EVOLUTIONARY PATTERN OF PANDEMIC INFLUENZA (H1N1) 2009 VIRUS IN

THE LATE PHASES OF THE 2009 PANDEMIC: *“The relevant evolutionary steps for the new virus to adapt to human populations occurred very early during the pandemic, before the end of April. Of the several resulting clades or clusters, clade 7 appeared later and proved more successful, substituting all other early clades before the bulk of the worldwide infections occurred.”*

2. GENOMIC SIGNATURE AND MUTATION TREND ANALYSIS OF PANDEMIC (H1N1)

2009 INFLUENZA A VIRUS: *“All these mutant residues, except that at NA-91, are located in the viral functional domains, suggesting that they may play roles in the human adaptation and virulence of 2009 H1N1pdm.”*

3. OBSERVED ASSOCIATION BETWEEN THE HA1 MUTATION D222G IN THE 2009 PANDEMIC INFLUENZA A(H1N1) VIRUS AND SEVERE CLINICAL OUTCOME,

NORWAY 2009-2010: *“Infection with the recently emerged pandemic influenza A(H1N1) virus causes mild disease in the vast majority of cases, but sporadically also very severe disease. A specific mutation in the viral haemagglutinin (D222G) was found with considerable frequency in fatal and severe cases in Norway, but was virtually absent among clinically mild cases. This difference was statistically significant and our data are consistent with a possible causal relationship between this mutation and the clinical outcome.”*

4. DEVELOPMENT OF A MULTIPLEX REAL-TIME RT-PCR THAT ALLOWS UNIVERSAL DETECTION OF INFLUENZA A VIRUSES AND SIMULTANEOUS TYPING OF

INFLUENZA A/H1N1/2009 VIRUS: *“It was the aim of this study to develop a real-time RT-PCR that can detect all influenza A viruses and offer simultaneous typing for influenza A/H1N1/2009. This would be a useful addition to existing diagnostic protocols for influenza A.”*

CB Daily Report

Chem-Bio News

EVOLUTIONARY PATTERN OF PANDEMIC INFLUENZA (H1N1) 2009 VIRUS IN THE LATE PHASES OF THE 2009 PANDEMIC

By Maria Beatrice Valli, Silvia Meschi, Marina Selleri, Paola Zaccaro et al (7 authors)

PLoS Currents Influenza

March 05, 2010

"Influenza A(H1N1)v has spread rapidly in all parts of the globe in 2009 as a true pandemic, although fortunately a clinically mild one. The relevant evolutionary steps for the new virus to adapt to human populations occurred very early during the pandemic, before the end of April. Of the several resulting clades or clusters, clade 7 appeared later and proved more successful, substituting all other early clades before the bulk of the worldwide infections occurred."

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"Conclusions

Our study demonstrates that pandemic (H1N1) 2009 virus has evolved worldwide, shifting from an initial mixed clade pattern to the predominance of one clade (clade 7) during the course of the pandemic. The virus constituting this clade was therefore responsible for most of the pandemic burden worldwide. After its origin, which remains obscure, clade 7 virus has been subjected to strong purifying selection, with the exception of the earliest phases of its evolution, behaving later as a well-fit virus, similar to viruses circulating in swine or seasonal influenza in humans. Interestingly, the highest Ka/Ks values were associated to HA and NS, key proteins for virus-host interactions, suggesting adaptation to the new host species. As yet, no pathogenetic correlate of this evolution has emerged, since no clear trend in the clinical aspects could be observed between the early and the late peaks of the epidemic [9]. Neither was a clear clinical impact demonstrated for HA variants which occurred on clade 7: D222/G/N or E, (WHO report, 28th December 2009). The hypothesis that clade 7 virus enjoyed a marked advantage, in terms of transmissibility, over other early clades is intriguing, but has yet to be demonstrated."

The full article can be found at: <http://knol.google.com/k/maria-beatrice-valli/evolutionary-pattern-of-pandemic/ql0y91xwcnj/1?collectionId=28qm4w0q65e4w.1&position=1#>

[Return to Top](#)

GENOMIC SIGNATURE AND MUTATION TREND ANALYSIS OF PANDEMIC (H1N1) 2009 INFLUENZA A VIRUS

By Chungen Pan, Byron Cheung, Suiyi Tan, Chunling Li, Lin Li, Shuwen Liu, Shibo Jiang

PLoS One

March 08, 2010

"Abstract

A novel swine-origin pandemic influenza A(H1N1) virus (H1N1pdm, also referred to as S-

OIV) was identified as the causative agent of the 21st century's first influenza pandemic, but molecular features conferring its ability of human-to-human transmission has not been identified. Here we compared the protein sequences of 2009 H1N1pdm strains with those causing other pandemics and the viruses isolated from humans, swines and avians, and then analyzed the mutation trend of the residues at the signature and non-signature positions, which are species- and non-species-associated, respectively, in the proteins of H1N1pdm during the pandemic of 2009. We confirmed that the host-specific genomic signatures of 2009 H1N1pdm, which are mainly swine-like, were highly identical to those of the 1918 H1N1pdm. During the short period of time when the pandemic alert level was raised from phase 4 to phase 6, one signature residue at the position of NP-100 mutated from valine to isoleucine. Four non-signature residues, at positions NA-91, NA-233, HA-206, and NS1-123, also changed during the epidemic in 2009. All these mutant residues, except that at NA-91, are located in the viral functional domains, suggesting that they may play roles in the human adaption and virulence of 2009 H1N1pdm."

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"Summary

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"In summary, our study confirms that the 2009 H1N1pdm virus has much closer linkage to the 1918 H1N1pdm than any other pandemic influenza viruses. We identified one dominant mutation at the signature position (NP-100) and four dominant mutations at the non-signature positions (NA-91, NA-233, HA-206, and NS1-123). Except NA-91, all these mutant residues are located in the viral functional domains, suggesting that they may play roles in the human adaption and virulence of 2009 H1N1pdm."

The full article can be found at: <http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0009549>

[Return to Top](#)

OBSERVED ASSOCIATION BETWEEN THE HA1 MUTATION D222G IN THE 2009 PANDEMIC INFLUENZA A(H1N1) VIRUS AND SEVERE CLINICAL OUTCOME, NORWAY 2009-2010

By A Kilander, R Rykkvin, S G Dudman, O Hungnes

Eurosurveillance

March 04, 2010

"Infection with the recently emerged pandemic influenza A(H1N1) virus causes mild disease in the vast majority of cases, but sporadically also very severe disease. A specific mutation in the viral haemagglutinin (D222G) was found with considerable frequency in fatal and severe cases in Norway, but was virtually absent among clinically mild cases. This difference was statistically significant and our data are consistent with a possible causal relationship between this mutation and the clinical outcome."

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"Conclusions

To our knowledge, this is the first identification of a change in the pandemic virus that correlates with a severe clinical outcome. However, whereas our data lend statistically significant support to an association between the D222G mutation and severity, the number of mild cases would need to be larger to determine whether mutant viruses are indeed circulating at a very low frequency also in non-severe cases. Provided that D222G mutant viruses are not circulating, i.e. that they are less transmissible, the immediate public health impact of this finding is limited. However, it may have implications for the management of severe cases where the virus, if transmitted through massive exposure, may be more virulent than the commonly circulating variant. Furthermore, it may serve as a reminder that the generally very low virulence of the current pandemic virus is not a fixed characteristic, and that there is no reason for complacency in carrying out measures that limit infection with this virus at individual and population level.

Further virological, clinical and epidemiological investigations are needed to ascertain the role of this and other mutations that may alter the virulence and transmissibility of the pandemic influenza A(H1N1) virus."

The full article can be found at: <http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19498>

[Return to Top](#)

DEVELOPMENT OF A MULTIPLEX REAL-TIME RT-PCR THAT ALLOWS UNIVERSAL DETECTION OF INFLUENZA A VIRUSES AND SIMULTANEOUS TYPING OF INFLUENZA A/H1N1/2009 VIRUS

Hospital Business Week
February 28, 2010

"It was the aim of this study to develop a real-time RT-PCR that can detect all influenza A viruses and offer simultaneous typing for influenza A/H1N1/2009. This would be a useful addition to existing diagnostic protocols for influenza A."

The full article can be found at: (R. Gunson, et. al., "Development of a multiplex real-time RT-PCR that allows universal detection of influenza A viruses and simultaneous typing of influenza A/H1N1/2009 virus". Journal of Virological Methods, 2010; 163(2): 258-61). Link not available.

[Return to Top](#)

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