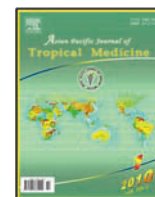


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Swine flu pandemic: a global concern

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ABSTRACT

Recently WHO has again declared Influenza pandemic due to the outbreak of H1N1 which has infected over 254,206 people in 80 countries, with 625 deaths. Our lab has started working on the proteome analysis of H1N1, 2009 out break strains.

The recent influenza outbreak in humans swine flu is due to a new H1N1 strain of influenza virus A that was probably originated by re-assortment of human and swine influenza virus genome [1]. Our lab has started working on the proteomic analysis of H1N1, 2009 out break strains. We have already analyzed the genome of H5N1 strains from Vietnam, 2004 outbreak and 2006 outbreaks occurred in India [2–6]. The common subtypes of influenza A virus are H1N1, H3N2, and H1N2 although H3N1, and H2N3 has also been reported earlier [7–9]. The H1N1 subtype was exclusively prevalent among swine populations before 1998 in USA. However, by the end of August 1998, H3N2 subtype has also been isolated from pigs. As of 2004, H3N2 virus isolates in US swine and turkey stocks were supposed to have genes from human (HA, NA, and PB1), swine (NS, NP, and M), and avian (PB2 and PA) as a result of triple re-assortment as shown in figure 1 [10].

It is expected that A/Veracruz/2009 (H1N1), the new strain of swine influenza A (H1N1) involved in the 2009 out break in humans, is a result of re-assortment of several strains of influenza A virus subtype H1N1 that are usually found separately, in humans, birds and pigs (Figure 1). Preliminary data suggest that the hemagglutinin (HA) gene was similar to that of swine flu viruses present in United States pigs since 1999, but the neuraminidase (NA) and matrix protein (M) genes were found to be similar to strains present in European pigs. Viruses with this genetic make up had not previously

been found to be circulating in humans or pigs, although no proper surveillance system has been established to determine the circulation of viruses among pigs in the United States [11]. Although preliminary studies showed that 6 genomic segments were close to North American Swine viruses and 2 were from Eurasian origin [12].

Recently WHO has again declared Influenza pandemic due to the outbreak of H1N1. The disease has infected over 254,206 people in 80 countries, with 625 deaths. But it has been most severe in Mexico, which has reported the highest number of fatalities. The role of pigs in the evolution of new pandemic influenza strains appears to be more restricted as it was thought previously. It has also been described earlier that pigs are more susceptible to AI viruses than humans and they serve as ‘mixing vessels’ for the transmission of AI viruses to humans. Major genetic changes are required for consistent pig-to-human transmission of such viruses. Our lab has started to gain some insights into the nature of these genetic changes [2, 6]. We also need detailed studies on the pathogenesis of influenza viruses from birds and other species in pigs.

People working in poultry and swine, especially those with intense exposures, are at increased risk of zoonotic infection with influenza virus endemic in these animals, and form a population of human hosts in which zoonosis and reassortment can occur to gather [13].

Zoonosis and reassortment are insufficient for the generation of a pandemic influenza virus and so additional genetic changes are likely to be involved in the process of antigenic shift [14]. Therefore, we take this opportunity to analyze two surface proteins (HA and NA). The amino acids Asp-190 and Asp-225 were known specific for the NeuAc α 2,6 Gal linkage (personal communication). Although Mexican (A/

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maxican/3955/09) H1 isolate had Gly225 which was found similar to isolates from Swine Influenza Virus during 1980 and 1997 outbreaks in USA. Mutation at position 225 may effect the binding to NeuAc α 2, 6 Gal SA but it needs to be validated by further experimental approaches. Furthermore, our NA analysis concludes that no mutation was detected in the

conserved region of NA which further corroborates the efficacy of existing drugs against Influenza A Virus.

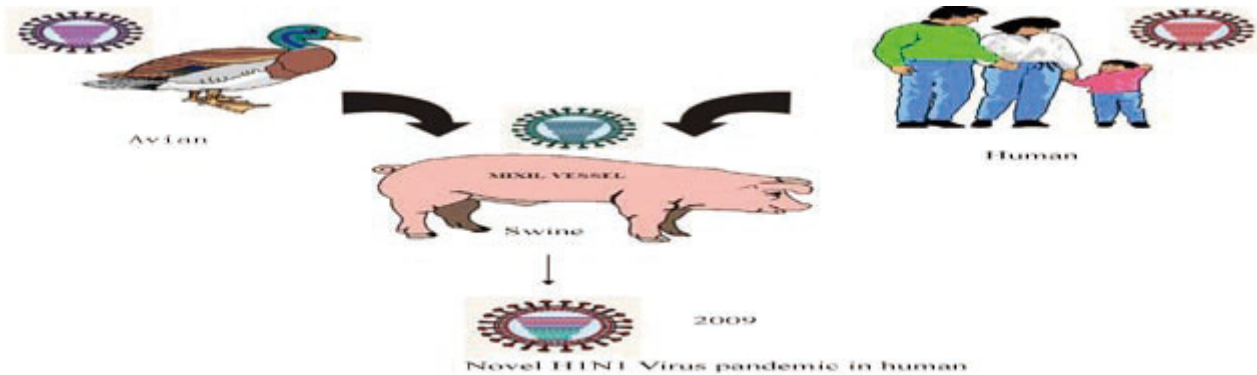


Figure 1. A proposed model mechanism of triple re-assortment influenza A virus resulted a novel H1N1 strain causing ‘Swine Flu’

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