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The new emerging H7N9 influenza virus indicates poultry as new mixing vessels

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Since 30 March 2013, the Chinese Center for Disease Control and Prevention (China CDC) has reported the first laboratory-confirmed cases of influenza A H7N9 virus human infection. This new pathogen caused 144 human infections with 47 fatal cases in year 2013, and continued to bring another wave in 2014, caused another 273 infections with 108 deaths as of 27th April. The H7N9 influenza virus has again raised the global concerns of the potential pandemics. Studies of the origin and evolution of this virus revealed that the novel H7N9 influenza virus was reassorted from H7, N9 and H9N2 subtypes of influenza viruses from domestic ducks, wild waterfowls and poultry, respectively; and the processes of genetic reassortments were sequential and dynamic [1–3].

Unlike H5N1 and the 2009 pandemic H1N1 (pH1N1), the H7N9 virus showed great genetic diversity since its emergence. This is essentially due to the frequent genetic reassortments of the internal genes with poultry H9N2 virus happened during poultry transportation and trade. With no doubt, the live poultry markets have played pivotal roles, as varied species of domestic birds from different regions are gathered together, even with mammals. Nevertheless, the surface proteins, hemagglutinin (HA) and neuraminidase (NA), experienced trivial changes, as the virus strains with mammalian-signature receptor-binding site and oseltamivir-sensitive site are dominantly circulated. The latest released H7N9 virus genomes still present great heterogeneous in their internal genes, and keep the HA and NA genes phylogenetically conservative. Many recent virus strains have one or more of their internal genes replaced by H9N2 virus genes from local poultry. This implies that following the H7N9 virus transmission into a new region, most probably via poultry transportations, the virus soon spread out to the local populations and reassorted with local H9N2 virus.

Human infections of H7N9 virus are still greatly affected by the methods of poultry feed and trade, so that the closedown of live poultry markets and centralized slaughtering has repeatedly been suggested [4,5]. Until recently, the H7N9 virus has not adapted to become a human virus, though several unsustained human-to-human case clusters were suspected. The 2009 pH1N1 influenza virus, on the contrary, was established from swine-origin triple-reassortant virus (human-, avian- and swine-origin), and quickly spread worldwide and caused millions of people infected. Every year, there are still thousands of people infected by pH1N1 and some of them died in China, and so as worldwide. It has long been believed that pigs are acting as the mixing vessels of different subtypes of influenza viruses, as swine respiratory tracts possessing both avian- and mammalian-receptors. The establishment of pH1N1 did exhibit pigs as mixing vessel for influenza virus evolution,

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as genetic reassortments of avian, swine and human viruses successively occurred. The outbreak of H7N9 virus warns us that beside the pigs, the poultry may also act as mixing vessel for the emerging of novel influenza viruses. The human-infecting H10N8 influenza virus was also established from poultry following the reassortment with H9N2 influenza virus.

In China, H9N2 influenza virus circulates predominantly across poultry populations, and this forms the environment for the emerging of novel influenza viruses. Avian influenza viruses from domestic birds and wild birds are possibly co-infected and reassorted with H9N2 virus and thus gain the ability to establish in poultry. Like the novel H7N9 and H10N8 viruses, most avian influenza viruses showed low pathogenicity to poultry, and therefore remain hidden until causing human infection. It is unknown whether the limited mammalian adaptation of the H7N9 and H10N8 viruses are because of the poultry but not pigs as the mixing vessel. However, the H7N9 virus has proven that avian influenza virus circulating in poultry could also possessing mammalian receptor-binding site, and could gain the substitutions for mammalian-host adaption.

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