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Could avian H9N2 influenza viruses become a threat to swine? Lessons from experimental studies in the pig.

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Introduction: It is a classical but unproven hypothesis that pigs can serve as intermediate hosts between birds and humans in the generation of novel pandemic influenza viruses. Yet the single pandemic virus of likely swine origin is the 2009 pandemic H1N1 virus (2009 pdm), a virus with genetic components of swine, human and avian origin (reassortant). The latter virus has become well adapted to pigs and humans and is now widespread in both species. Avian H9N2 viruses are endemic in Eurasian poultry and they are considered potential pandemic candidates because they cause sporadic dead-end infections in pigs and humans. However, H9N2 viruses are not adapted to mammals since they lack capacity to spread within the human or swine population. We and other researchers have previously examined whether H9N2 viruses could become adapted to pigs by two strategies: serial pig passages which are known to force viruses to mutate; and reassorment replacing H9N2 internal genes by those of a swine adapted influenza virus (SIV), the 2009 pdm. Both approaches slightly enhanced virus replication and transmission, but transmission was still inefficient when compared with SIV. Thus, we aimed to examine whether such reassortant H9N2 virus could become better adapted to swine after serial pig passages.
Materials and Methods: We performed four pig transmission experiments with four different viruses: a non-passaged reassortant virus containing A/quail/Hong Kong/G1/97 (H9N2) surface genes and A/California/04/09 (2009 pdm) internal genes; another reassortant virus with the same genetic constellation but passaged seven times in pigs and both parental viruses, the avian H9N2 and the 2009 pdm SIV. In each experiment 3 animals were individually housed and intranasally inoculated with the selected virus. Two days later, 2 direct contact animals were co-housed with each inoculated pig. Nasal swabs were collected daily from all pigs for virus titration.
Results: All inoculated pigs excreted medium to high amounts of virus during at least 5 days. The 2009 pdm was the single virus for which all 6 contact pigs shed high amounts of virus during 5 days. As expected, the parental H9N2 and the non-passaged reassortant H9N2 viruses were not efficiently shed by any contact pig. In contrast, the pig-passaged reassortant H9N2 virus was shed in high amounts by 4 of the 6 contact pigs.
Conclusion: Our data suggest that serial passages induced mutations in the H9N2 reassortant virus, which improved its replication and transmission. Genetic analysis of this virus is still pending. We demonstrated that adaptation of avian H9N2 viruses to pigs is a complex multi-step process. H9N2 could pose a threat for pigs and humans if this process would occur in nature.

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