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Transmission of Japanese encephalitis virus in Hong Kong

Key Messages

1. Pigs are likely to be the main amplifying host for Japanese encephalitis virus.
2. The success of a swine vaccination programme depends on the timing of the loss of maternal antibody protection and seasonal dynamics of the vector population.
3. Vaccination may be ineffective in the face of strong natural infection because of the variability in timing of the loss of maternal antibody protection.
4. Evidence in support of swine vaccination as a human health intervention was not found.

Background

Local acquisition of Japanese encephalitis virus (JEV) was a regular, if not widespread, occurrence in Hong Kong prior to 1991. Although there have been only seven such cases since then, six of them occurred in 2003/4. The JEV is an indirectly transmitted zoonotic virus. In Hong Kong, the vector is likely to be *Culex tritaeniorhynchus* and the vertebrate amplifying hosts are pigs and wild wetland birds (egrets and herons). Humans are non-amplifying hosts for JEV; they are not able to infect susceptible mosquitoes. Transmission between these species is the main concern for an effective JEV control policy.

Based on a mathematical model, the role of transmission involving the vector and amplifying hosts was assessed.¹ Pigs are the principal source of JEV infection in mosquitoes. This suggests swine vaccination may be a strategy for preventing human infection. However, the seasonal variation in mosquito population drives most of the dynamics of JEV transmission. The lack of vector data precludes prediction of the impact of any vaccination programme.

We aimed to describe quantitatively the role of mosquitoes, pigs, and wild birds in the transmission of JEV in Hong Kong, and assess more accurately the likely impact of swine vaccination.

Methods

This study was conducted from 1 September 2006 to 17 August 2007. Four distinct deterministic compartmental models were used, one each for mosquitoes, pigs, wild birds, and humans with various transmission processes: susceptible, exposed, infectious, and recovered. Transmissions were linked by the mosquito-biting event.

Seasonal variation in the mosquito population was assumed to have exponential growth and decay in spring and fall, respectively. Decrease in maternal antibody protection in pigs and their age structure were modelled to allow realistic assessment of swine vaccination. Transmission under two scenarios (different duration of summer peak vector season) was simulated, consistent with mosquito data.

Relevant estimates from the literature were used for parameters such as lifespan of JEV vectors, their expected number of blood meals, and duration of infectiousness in pigs. Local ovitrap data for the species *Aedes albopictus* were used as a proxy for the seasonal variation in JEV vector population.

Data for JEV seroprevalence in pigs were collected, which showed marked seasonal trends over years. These were used to calibrate the transmission model after which the impact of a swine vaccination programme was assessed.

Results

An optimised two-dose swine vaccination strategy is likely to be effective if the peak summer period is very short and the winter vector population is large. However, swine vaccination is likely to have little effect if the summer season is much longer and the winter vector population is smaller. In both scenarios,

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excess human JEV infection attributed to transmission by wild birds is <20%.

Conclusions

Interaction of a maternal antibody protection in a non-seasonal pig population and a highly seasonal vector population makes the likely success or failure of swine vaccination difficult to predict. Nonetheless, an optimised two-dose vaccination for pigs would have a dramatic decrease on the number of human infection caused by pigs if the peak vector season is short. Further field studies should systematically quantify the abundance of potential vectors, their rates of viral carriage, and identification of the mosquito blood meals. This would enable a direct estimate of the relative biting probabilities in the model. Moreover, the contribution of wild bird in human JEV infection is minor.

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Reference

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