

The equine influenza epidemic in Australia: spatial and temporal descriptive analyses of a large propagating epidemic

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ABSTRACT

Australia experienced a large outbreak of equine influenza in August 2007. We used spatial and temporal analytical techniques to describe the epidemic, to quantify important descriptors of the epidemic, and to generate hypotheses about how the epidemic progressed and which control tools assisted in eradication. Movement restrictions appeared to prevent long distance spread, but the epidemic peak had passed before emergency vaccination could have induced substantial immunity. We identified 37 clusters of disease. Spread distances were less than 10 km in most cases. Premises level transmission was extensive early in the epidemic with very high R_0 values, but rapidly reduced following implementation of the eradication campaign with each premises on average infected 2 other premises (effective reproductive rate (R_t) ~ 2) throughout the epidemic. Premises level cumulative incidence was less than 50% in all clusters indicating that spread between premises was constrained by control measures or spatial disconnectedness. Clusters in peri-urban landscapes had denser and larger populations of horse premises, longer epidemic durations and shorter spread distances compared with rural clusters. Perhaps surprisingly there appeared little difference between the incidence rate, cumulative incidence and R_T between rural and peri-urban regions. The relative impact of vaccination and national movement restrictions in controlling this epidemic needs further investigation.

INTRODUCTION

In August 2007 Australia – which had been previously free of Equine Influenza (EI) – experienced a large outbreak. The disease was associated with imported horses. Despite a rapid and effective disease eradication campaign that limited the spread of the disease to the two eastern states of New South Wales and Queensland, nearly 10 000 premises were infected during the epidemic. Following the last EI case in December 2007, extensive surveillance programs were unable to detect the virus in the Australian horse population, confirming the successful eradication of EI.

The program to eradicate EI was a coordinated national response with many facets, including:

1. Movement restrictions;
2. Public awareness and communication;
3. A risk-based zoning system (e.g. 'red' and 'purple' zones for infected areas);
4. Laboratory testing (more than 100 000 tests were conducted);
5. Disease tracing and surveillance;
6. Enhanced biosecurity;
7. Emergency vaccination of approximately 136 000 horses.

These measures contained and ultimately eradicated the epidemic, but it is important to determine which control tools were the most useful. This can be accomplished by post outbreak analysis.

In the event of an extensive epidemic, comprehensive outbreak data is rarely available in a timely fashion. In this situation, resources for tracing and epidemiological investigations can be rapidly overwhelmed and available data may only include locations of infected premises (IP) or animals (spatial data, with varying accuracy) and the date that clinical signs of disease began or were reported (temporal data). A substantial amount of research has been conducted on methods of analysing spatial and temporal disease data. The objective of this descriptive study was to analyse data from the Australian EI epidemic using a range of spatial and temporal methods to:

1. Describe spatial and temporal trends;
2. Quantify important epidemic descriptors; and
3. Generate hypotheses about disease spread and the effect of major control tools.

METHODS

This study first focused on describing the spatial and temporal features of the epidemic using spatio-temporal epidemic curves. The introduction of major control measures was then compared with the spatio-temporal epidemic curves to generate hypotheses about the efficacy of movement restrictions and emergency vaccination. The second phase of the study used common spatial and time characteristics to divide epidemiologically linked IPs into clusters of diseased premises. We then analysed these individual clusters to estimate key epidemiological parameters. Finally, clusters were pooled, based on whether they were in a peri-urban or rural region and standardised for different management zones ('purple' or 'red' zone), to provide summary statistics of the epidemic.

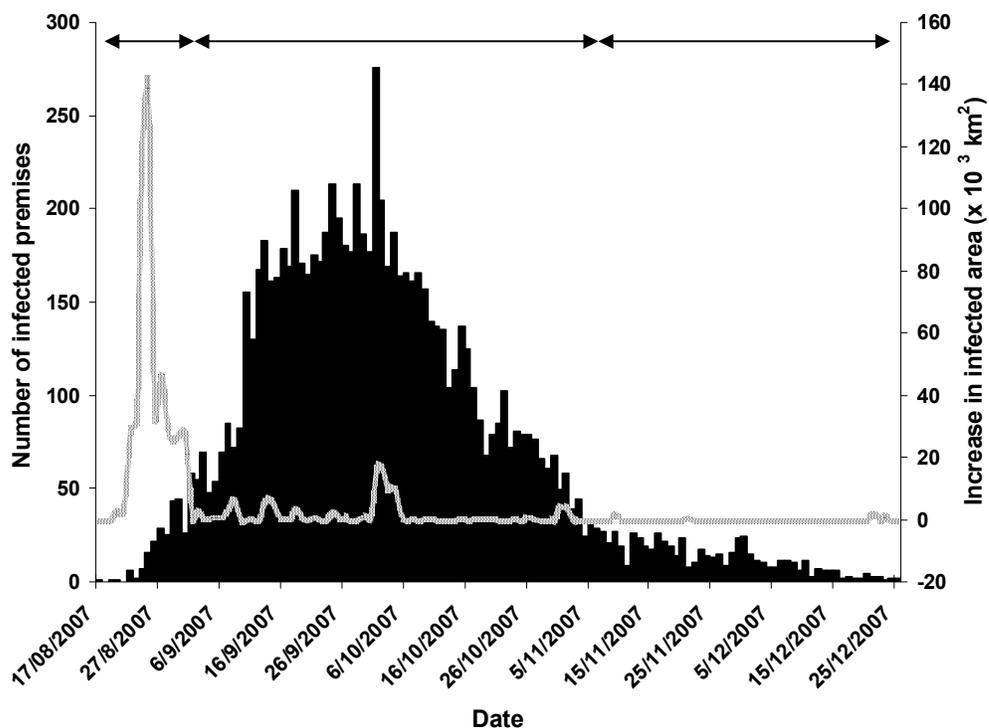
RESULTS

Spatio-temporal epidemic curves revealed that there were three phases in the epidemic: dispersal, local spread and disease fade-out. Spatial dispersal of infection rapidly declined immediately after national movement restrictions were introduced (See Figure 1). The epidemic peak had passed before emergency vaccination could have induced substantial immunity in the horse population.

Thirty seven clusters of epidemiologically linked infected premises were delineated using an interpolated surface of date of onset of clinical signs, geographic data and location of infected premises. These clusters were analysed individually to parameterise key epidemic measures: cumulative incidence, incidence rate, effective reproduction rate, nearest neighbour spread distances, epidemic length and the number of infected premises. Cumulative incidence was less than 50% for all clusters and effective reproductive rates were approximately 2. Spread distances were generally less than 10 km. These measures were summarised by landscape (rural versus peri-urban location) and standardised by disease management zone. Compared to rural areas, peri-urban areas appeared to have a higher density of horse premises (4.66 versus 0.80 premises km²), longer epidemics (95 versus 87 days), more infected premises (393 versus 339) and a shorter spread distance (1.27 versus 2.38 km). However, effective reproduction rates (2.04 versus 1.99), cumulative incidence (27.4 versus 26.9) and incidence rates (1.36 versus 1.54) were similar.

Figure 1: The spatio-temporal epidemic curve for the Australian equine influenza epidemic.

The vertical columns represent the number of new infections by estimated first clinical signs each day, whilst the polygon represents the increase in the total area of infected land every two days. Each phase of the epidemic is represented by an arrow (with arrows from left to right indicating dispersal, local spread and epidemic fade out phases).



DISCUSSION

Greater than 80% of the total infected area was infected by less than 300 IPs (3% of the final IP tally) in the several days immediately before the first diagnosis of EI in Australia (25 August 2007). However, spatial dispersal rapidly decreased and had largely ceased by 1 September 2007. The principal control measures implemented within the eradication program to this stage was a national horse standstill (implemented 25 August 2007). This suggests that one of the key steps in controlling the epidemic was the movement standstill.

The role that vaccination played in the containment and eradication of EI in Australia is unclear. However, an examination of the temporal overlap of earliest immunity following vaccination, in relation to epidemic curves suggests that the peak of the epidemic had already been reached before substantial immunity from vaccination could have developed.

This descriptive analysis also aimed to summarise data and estimate parameters that might be useful for future modelling studies. Epidemiological tracing of horses in the early phase of the epidemic from two key known horse events before the control program began revealed a very high premises level basic reproductive rate (R_0) in the early phases of the epidemic. However, we estimated much lower premises level effective reproduction rates (R_T) for clusters over the course of the epidemic (~2). In other words, once the control program had been implemented, there were few instances of an IP infecting large numbers of other premises. This indicates that the measures put in place (including movement restrictions, disinfection and biosecurity precautions) were highly effective in reducing the spread of infection.

Spread distances estimated in this descriptive analysis can inform contingency plans for future epidemics of equine disease in Australia. Our analysis indicates that 95% of spread distances were less than 10 km in all but one cluster. This implies that the size of the restricted area was adequate (minimum restricted area size 10 km). Another parameter of interest in this study was the cumulative incidence over the course of each cluster's epidemic (range 6-47%). These values are lower than may intuitively be expected for such an infectious disease. Partially, this may be explained by spatial disconnectedness between premises, limiting the spread of infection. However, reductions in movements between premises and other interventions would have played a role in limiting risk.

CONCLUSIONS

The relative impact of vaccination and national movement restrictions in controlling this epidemic needs further investigation. The paper of Garner et al. elsewhere in these proceedings reports on a simulation modelling approach to explore the effect of emergency vaccination on epidemic characteristics.