



## Transmission dynamics of the 1918 influenza pandemic in New Zealand: analyses of national and city data

Understanding the detailed transmission dynamics of previous influenza pandemics is crucial for planning an appropriate response to future pandemics. Although the Southern Hemisphere is fairly unique in that the countries in this hemisphere experienced the so-called “autumn wave” of the 1918 influenza pandemic (caused by influenza A virus [H1N1]) in the spring season, the transmission potential has been empirically explored only in a confined (military camp) setting in New Zealand<sup>1</sup> and by using coarsely reported data in a city in Brazil.<sup>2</sup>

To quantify the transmissibility of the 1918 influenza pandemic in a community setting, we investigated more detailed historical data in New Zealand and estimated the reproduction number,  $R$ , the average number of secondary cases generated by a single primary case. This estimate is in a country that did not successfully apply widespread public health control measures,<sup>3</sup> and where the disease was possibly exacerbated by mass celebrations for the end of World War I.<sup>3</sup>

We analysed influenza-attributed mortality in five large population groups: daily number of deaths in the three largest cities (Auckland, Wellington and Christchurch, all based on individual records) and the daily mortality report (per 100,000) for the entire North and South Islands (see Table 1).<sup>3-5</sup> The data were for European populations only; unfortunately, the impact of the disease in the Maori population was not as well documented at this time (though Maori were nearly all residing outside of the main cities in 1918).

Figure 1 shows the observed temporal distributions from October-December, 1918. To estimate  $R$ , we investigated the initial growth phase (i.e. first 15 days). The historical data based on individual death certificates enabled us to ignore reporting delays and to assume exponential growth as seen in the onset of disease. During the initial growth phase, Malthusian (exponential) growth of death  $i(t)$  at time  $t$  with daily growth rate  $r$  is expected<sup>6</sup>, and we estimated  $r$  using the following likelihood, which was based on an explicit stochastic birth process<sup>7</sup>:

$$(1) L(r, i_0) = \prod_{t=1}^{15} \binom{i(t)-1}{i_0-1} \exp(-i_0 r t) (1 - \exp(-r t))^{i(t)-i_0}$$

for  $i_0 > 1$ , where  $i_0$  is the initial number of cases (which was jointly estimated).

Based on the detailed historical investigations by Geoffrey Rice,<sup>3</sup> epidemic time  $t = 1$  was counted from 17th October in Auckland and 1st November for other data. The generation time was assumed to be gamma-distributed with mean  $\mu = 2.92$  days and variance  $\sigma^2 = 5.57$  days<sup>2</sup>, respectively.<sup>8,9</sup> Solving an estimator of  $R$  given  $\mu$  and  $\sigma$ ,<sup>9</sup>  $R$  was estimated by replacing  $r$  in equation (1) by the right-hand side of

$$(2) r = \frac{\mu R^{\frac{\sigma^2}{\mu^2}} - \mu}{\sigma^2}$$

Since the generation time of influenza has yet to be fully clarified, we investigated the sensitivity of  $R$  to different  $\mu$ . Moreover, since the proportion of symptomatic infections among the total number of infected individuals still remains unknown, we estimated the proportion using  $R$  in three cities and examined the sensitivity to different case fatality proportions.

**Table 1. The reproduction number of the 1918 influenza pandemic in New Zealand**

Main island / city	$R^\dagger$ (95% CI <sup>‡</sup> )	AIC <sup>¶</sup>	Data source (Reference)
North Island	1.60 (1.47, 1.78)	77.1	5
South Island	1.47 (1.33, 1.68)	62.7	5
Auckland (North Island)	1.44 (1.33, 1.61)	68.7	4
Wellington (North Island)	1.55 (1.42, 1.76)	67.9	(Unpublished)*
Christchurch (South Island)	1.33 (1.22, 1.50)	50.5	(Unpublished)*

<sup>†</sup> $R$ , reproduction number; <sup>‡</sup>CI, confidence interval; <sup>¶</sup>AIC, Akaike information criterion ( $= -2 \times \text{LogLikelihood} + 2 \times \text{parameters}$ ); \* Daily unpublished individual level mortality data supplied by Geoffrey Rice, the author of a large historical study<sup>3</sup>; The 95% confidence intervals were derived from profile likelihood.

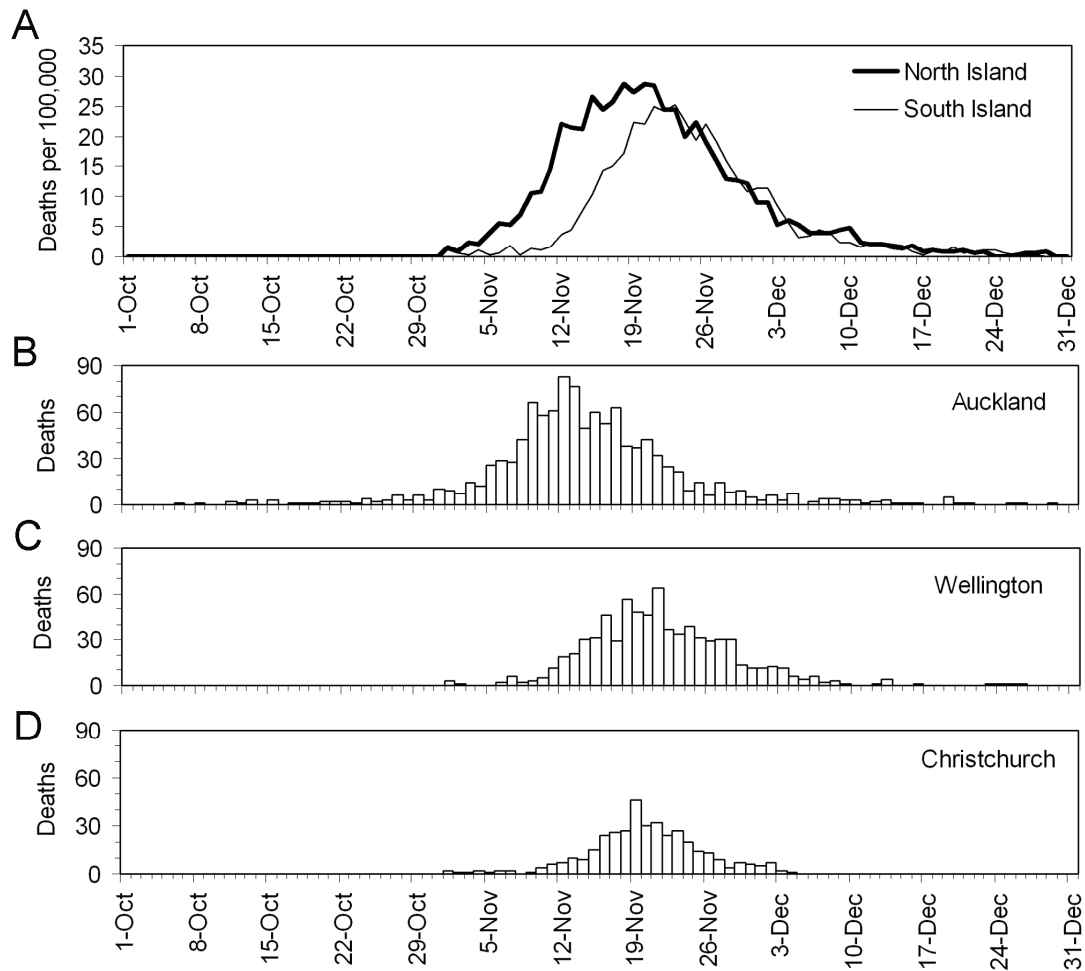
Figures 2A-C show the observed and predicted number of influenza deaths during the initial growth phase. The goodness-of-fit was assessed by Akaike Information Criterion, and the deviation did not significantly differ between the datasets (Table 1). Table 1 summarises the maximum likelihood estimates of  $R$  for five populations. The expected values ranged from 1.3 to 1.6. Among the two cities in the North Island, Wellington yielded higher estimate (1.6) than that in Auckland (1.4), though the difference was not statistically significant.

The estimate for the South Island city of Christchurch was the lowest (1.3). The comparative size-relationship of  $R$  was consistent with the crude mortality by the end of pandemic (795, 761 and 494 per 100,000 for Wellington, Auckland and Christchurch, respectively<sup>3</sup>). It should be noted that  $R$  for Auckland and Wellington was smaller than that for the entire North Island (1.6), indicating the importance of spatial spread at a local level.

Figure 2D shows the sensitivity of  $R$  to different mean generation times in the plausible range. Given that the mean generation time  $\mu$  ranges from 2 to 4 days,<sup>6,8,9,11</sup>  $R$  may lie in the range of 1.2 to 1.8. This brief work is the first to report  $R$  for

community transmission in the Southern Hemisphere for the 1918 pandemic with detailed daily data. The estimates were consistent with those in the Northern Hemisphere and were close to the lower bound among previous published estimates.<sup>12-14</sup>

**Figure 1. Epidemic curves of the 1918 influenza pandemic in New Zealand**

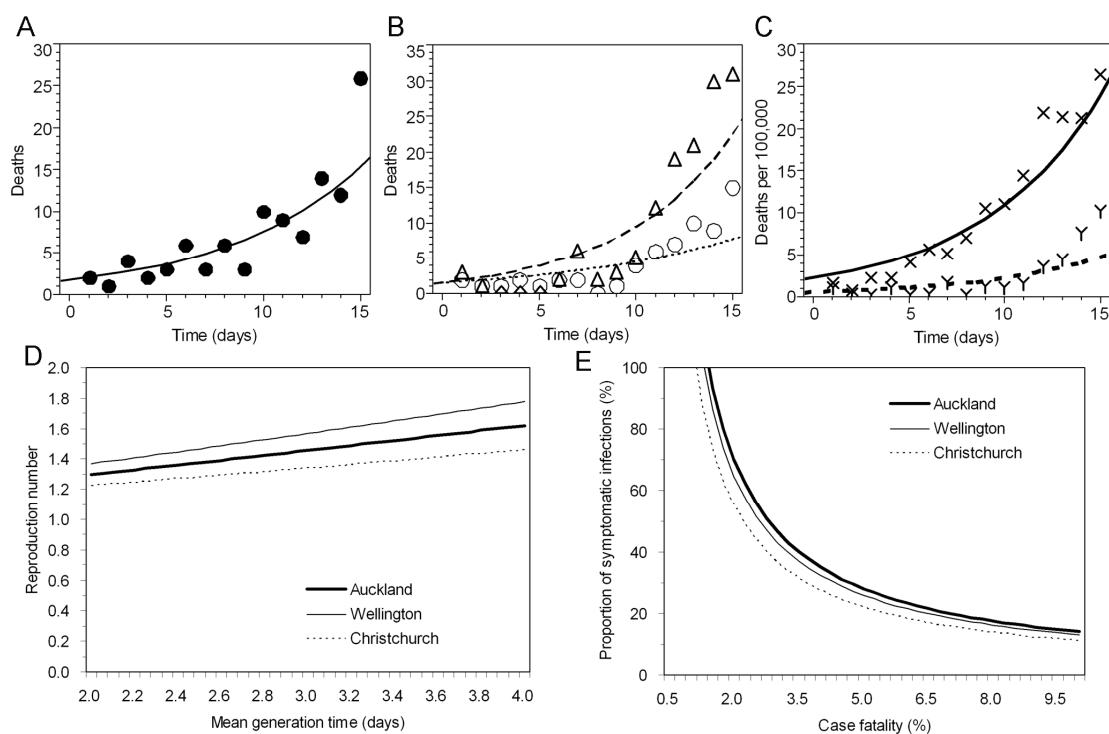


(A) Reported daily mortality (per 100,000 inhabitants) in North and South Islands and (B-D) the absolute number of influenza deaths in the 3 largest cities: Auckland, Wellington and Christchurch (see Table 1 for data sources).

The tendency to be smaller than the estimates in the Northern Hemisphere may be related to virus-fitness to spring weather and social contact patterns in New Zealand. Within-country variations in  $R$  indicated the importance of the detailed spatial and other heterogeneous patterns of spread. In particular, given that  $R$  for the entire North and South Islands were greater than those for their major cities alone, detailed contact patterns (rather than crude measures such as urbanization and population density) have to be explored to elucidate the mechanism to yield different  $R$  estimates.

Figure 2E shows the proportion of symptomatic infections as a function of the case fatality proportion. According to the available literature, we assumed that the case fatality proportion of the 1918 influenza pandemic ranged from 0.5-10.0% with 2.0% as the most plausible community estimate.<sup>6,15</sup> If the case fatality proportion is 1.5%, then 73.2-93.3% of infected individuals will develop symptoms. If 2.0%, then 54.9-70.0% of infected individuals are symptomatic. The probability of asymptomatic infection being up to 30.0-45.1% is consistent with a published estimate (at 33.3%) based on an analysis of meta-data of experimental infection with seasonal influenza viruses and also with the common epidemiological assumption in other studies.<sup>8,9</sup>

**Figure 2. Transmission dynamics of the 1918 influenza pandemic in New Zealand**



(A-C) Observed (markers) and predicted (lines) numbers of influenza deaths during the early stage of the 1918 influenza pandemic in New Zealand. (A) Auckland; (B) Wellington (triangles and dashed line) and Christchurch (circles and dotted line); (C) North (X and thick continuous line) and South (Y and thick dashed line) Islands. (D) Sensitivity of the reproduction number to the different mean generation times; (E) Sensitivity of the proportion of symptomatic infections to the different case fatality proportion estimates.

Our estimates of  $R$  for the 1918 pandemic in New Zealand appeared to be broadly consistent with previously suggested estimates for Northern Hemisphere settings<sup>12-14</sup> and were close to the reported lower bound. Although  $R$  for the 1918 pandemic is therefore not exceptionally large, it should be noted that the generation time is as short as 3 days and the proportion of asymptomatic infection is as large as 45%. The former characterises the rapidity of spread, and so pandemic plans have to involve the

rapid and effective implementation of both non-pharmaceutical (e.g. social distancing measures) and pharmaceutical interventions (e.g. antivirals and possibly pandemic vaccines).

The issue of asymptomatic transmission further complicates control and so pandemic plans may need to consider this issue (e.g. media messages that encourage social distancing for all people; and stockpiles of rapid diagnostic tests to assist with case finding). But more research to clarify the relevance of asymptomatic infection in pandemic influenza is critical as although it might be common, its public health relevance (in terms of transmission) is far from clear.

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**Acknowledgement:** The authors thank Professor Geoffrey Rice (University of Canterbury, Christchurch) for providing individual-level daily mortality data.

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