DEATH BY NUMBERS:

New Zealand Mortality Rates in the 1918 Influenza Pandemic

2006 Fifth Year Medical students: Group E

Public Health Project

Abdullah Al Haidari, Abdullah Al Hazmi, Hassan Al Marzouq, Mark Armstrong, Atalie Colman, Nick Fancourt, Kirsten McSweeny, Mahendra Naidoo, Peter Nelson, Melinda Parnell, Diana Rangihuna-Winekerei, Yasotha Selvarajah, Sara Stantiall.

Supervisors: Dr Nick Wilson and Dr Michael Baker

Wellington School of Medicine & Health Sciences, Otago University

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Group E:
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EXECUTIVE SUMMARY

Influenza pandemics occur around three times per century. We should therefore aim to learn lessons from these past events as part of our pandemic planning process. It is particularly useful to identify risk and protection factors associated with mortality rates in past pandemics, as these could provide useful evidence to inform future plans. The 1918 influenza pandemic was quite virulent with estimates of over 100 million deaths worldwide. The New Zealand mortality topped 8500 people, with a national mortality rate of 7.7 per 1000 people. The Maori population were more severely affected with a mortality rate of 42.3 per 1000 people.

This report aims to statistically analyse data available from the 1918 influenza pandemic in New Zealand, focussing on the effects of population and density on mortality rates, the socioeconomic gradient, and lastly, to determine if an institutionalised population, such as Featherston military camp, can offer us further insights into influenza mortality and morbidity. For the first two sections of this report, we used the 1916 census to extract the relevant denominators. Our numerator was obtained from work compiled in the publication 'Black November' by Geoffrey Rice. The denominator for the third section of our report was derived from current proportions of military groups and calculated according to the total Featherston Military camp population during the 1918 pandemic. The numerator was obtained from the original transcript written by the Primary Medical Officer at Featherston Military camp during the outbreak.

The first section of the report investigates the effect of social distancing on mortality rates by investigating the relationship of population size of cities, towns and counties, the population density per square mile, and the number of occupants per dwelling. Our underlying hypothesis is that areas of high population size and density would have higher mortality rates. Our results show us that the more densely populated North Island had a higher mortality rate when compared with the South Island. Those living in cities and towns had increased mortality rates when compared with counties.

Interestingly however, larger towns and cities had a lower mortality rate when compared to small towns of a population less than 2000. We believe this effect could be due to the larger population centres having better access to medical and community care, offering them a degree of protection. Lastly, our results showed that in the lowest population density quintile, the mortality rate is well below the national rate, and significantly different from all other density quintiles, which suggests that living in areas of lowest population density in New Zealand may offer some protection from mortality due to influenza. There may be an effect of threshold of protection, above which the density of the population plays no role in increasing susceptibility to infection.

In the second section of the report, we attempt to identify a socioeconomic status (SES) gradient for influenza mortality rates. Our general hypothesis was that those with lower status occupations would have an increased mortality rate when compared with high status professionals. Indeed a relationship was found when occupations were ranked according to an estimated socioeconomic status. Those in employment considered low in SES had an increased mortality when compared with higher SES citizens. However, primary producers, who were considered of low SES, did not experience high mortality rates, potentially due to decreased exposure from living and working in a rural area. Finally, dependents were found to be relatively protected from mortality in this pandemic, perhaps due to remaining at home and thus not being exposed to the virus.

The final section of our report follows the devastation of the influenza pandemic in Featherston Military camp. Our interest in Featherston military camp was to use it as an example of the effects of an influenza outbreak in an institutionalised population. By using rank as a measure of both SES and housing density, we hypothesized that as the rank increases, the risk of mortality from influenza reduces. Surprisingly, we found that privates had a lower mortality rate when compared with officers and non commissioned officers. This finding may, in part, be explained by officers providing both a medical and supervisory role during the outbreak, increasing their exposure and thus mortality rate. When the epidemic curves were compared with towns of a similar population size, it was found that Featherston had a higher mortality rate and an earlier peak mortality.

Important implications valuable to the current day can be extracted from this report on the 1918 Influenza pandemic. Firstly, social distancing does provide a protective effect on mortality rates from an influenza pandemic but only if such distancing exceeds a threshold level. Living in country areas with a very low population per square mile may offer protection compared to living in a town or city. Secondly, a socioeconomic gradient as determined by occupations was found. Therefore, those of a lower SES may experience greater mortality rates in future pandemics. Lastly, our investigations of the Featherston Military camp suggests pandemics can spread very swiftly through institutionalised populations resulting in relatively high morbidity and mortality rates. This investigation also showed that those in leadership roles may demonstrate increased mortality rates from an influenza pandemic. This has implications for medical staff/caregivers, and coordinators/facilitators of action plans. Finally, New Zealand's current stockpile of the antiviral oseltamivir (Tamiflu™) can supply 21% of the population. There is, therefore, some justification to prioritise use of available antivirals, possibly focussing on communities with relatively higher mortality rates such as those of low SES, institutionalised populations, and those working in occupations associated with increased exposure, such as leadership roles and medical/caregivers.

INTRODUCTION

Influenza “flu” is an infection of the respiratory tract with one of the influenza viruses. It is a contagious disease among birds and mammals, caused by an RNA virus of the Orthomyxoviridae family. These viruses spread rapidly around the world infecting hundreds of thousands of people. They are usually characterised by a short incubation period, 1-3 days, causing symptoms like sore throat, cough, fever, headache, muscle aches “myalgia” and a generalized feeling of illness “malaise” [1]. Bacterial pneumonia is a recognised complication of influenza, especially in high-risk groups such as the elderly, very young or those with chronic respiratory problems, and thus worldwide pandemics of influenza cause significant rates of mortality, often by this secondary infection [2].

Influenza pandemics have been documented from as early as 412BC by Hippocrates and occur regularly, with an average of three pandemics per 100 years. Last century the world experienced three pandemics in 1918, 1957 and 1968. By far the most devastating of these was the 1918 pandemic, which latest figures would suggest caused upwards of 100 million deaths worldwide [3]. A key characteristic of the 1918 flu pandemic was that it affected predominately those aged from about 20-40 years, which is unlike the usual flu which tends to affect the young and the old. The strain had a particularly rapid onset - people were struck down with dizziness, weakness and pain while on duty or in the street, and a few hours later could be severely ill or dead [3].

Influenza is a virus native to avian aquatic species where it generally lives as a gastrointestinal commensal, though it can cause death if the infection becomes systemic. The strain of the virus believed to have caused the 1918 pandemic (strain A, subtype H1N1) has its natural home in the gastrointestinal tract of ducks, from where virus sharing all eight genomic segments with the 1918 strain can still be isolated today [1].

The deadly 1918 strain of the disease is believed to have originated in South China, and deaths were first documented from this illness at Camp Funston in Kansas in March 1918. From here the disease spread through France via American troops and in France there were the first cases of civilian infection. From here the flu spread across the Euro-Asian super continent to reach Japan by May. In June it had reached South America and India. In September it had reached Africa and by October Central America. In October the pandemic had reached New Zealand, with the peak occurring in November and by December flu cases were subsiding [3].

There is much speculation about the origins of influenza in New Zealand. For years many believed that it arrived on board the Niagara, which, despite carrying cases of influenza, was not quarantined, allegedly because Prime Minister Massey and Finance Minister Ward were on board. Subsequent study has yielded other potential sources of the disease, such as returning troop ships, or possibly even further mutation of the mild flu virus already present in New Zealand [3].

Influenza spread rapidly by rail and shipping routes in New Zealand so that Wellington, Christchurch and Dunedin had virtually simultaneous peaks of the disease in late November, approximately two weeks after Auckland [3].

New Zealand was hit hard by the pandemic with over 8,500 deaths out of a total population of around 1.15 million, yielding a total mortality rate of 7.7 per 1000. A significant contributor to this was the number of Maori deaths, believed to be 2,160 – a mortality rate of 42.3 per 1000 [3].

The pandemic had a massive influence on the communities of the time. Public meeting places such as schools, hotels, factories and theatres were closed down in an effort to stop the spread. Many buildings were turned into make shift hospitals staffed by volunteers [3]. World War One undoubtedly had a significant role in many aspects of the pandemic, such as its initial spread and how society responded to the disease. The peak of the disease in many communities occurred soon after the armistice of 11th November, leading many to speculate about the role of Armistice Day celebrations on the spread of disease.

The 1918 influenza pandemic can provide much information to today's society. In today's society, there is a widening gap between affluent and poorer communities. It is established that living in an area of higher deprivation, as calculated using

NZDEP96 index, is associated with poorer health. Indeed, the mortality rate doubles from approximately 400/100,000 to around 800/100,000 in New Zealand European as deprivation increases [4]. The living conditions, crowding, education and poor nutrition in more deprived areas may lead to an increased susceptibility to contracting illnesses and to general poor health. Maori experience a more dramatic increase in mortality rates when considering levels of deprivation. In the more affluent areas, the Maori population have comparable mortality rates to New Zealand European. These rates however, increase 5 fold as the deprivation increases, peaking at approximately 2000/100,000. These figures are likely due to more than 1/3 of the Maori population located in the poorest deciles [4].

These current living conditions in New Zealand may potentially place a portion of residents at an increased risk of being exposed to and acquiring influenza should a future pandemic occur. As the Treaty of Waitangi aims to create equity in terms of health for all residents of Aotearoa, it is necessary to investigate such inequalities, and understand what implications this may have should a future pandemic occur. Consequently, policy and reform may occur to lessen the inequalities, and bridge the health gap between low and higher SES and between Maori and non-Maori. In addition, an action plan can be created using the past as a platform for future pandemic waves [5]. For this reason, it is of great interest to investigate the 1918 influenza epidemic, to gain valuable insight into the social, ethnic and geographical patterns of spread.

The aim of this project is to statistically analyse the 1918 influenza mortality figures to more accurately describe influenza patterns in this country. Of interest to us are the respective roles that population density and socioeconomic status played in the spread of the disease. We also aim to use Featherston military camp as a model population where different population densities and ranks approximate socioeconomic status in this community. Lastly, we aimed to use the extracted information to provide recommendations to the public health system for future epidemics and pandemics.

SECTION 1

SPATIAL EPIDEMIOLOGY OF THE 1918 INFLUENZA PANDEMIC IN NEW ZEALAND

INTRODUCTION

Human to human contact is a known risk factor for spread of infectious disease [2], yet little statistical evidence exists on the role of social distancing on the mortality rate from influenza in New Zealand in the 1918 influenza pandemic. With reference to population size, population density and occupants per dwelling, we aimed to investigate and analyse the influence of social distancing on mortality rates from the disease.

HYPOTHESIS ONE: POPULATION SIZE

Cities and towns will experience higher mortality rates from influenza when compared to the counties.

HYPOTHESIS TWO: POPULATION DENSITY

Counties (inclusive of interior boroughs) with higher population densities will experience higher mortality rates from influenza when compared to counties with lower population densities.

HYPOTHESIS THREE: OCCUPANTS PER DWELLING

Counties, towns and cities with higher mean numbers of occupants per dwelling will experience higher mortality rates from influenza when compared to counties, towns and cities with lower numbers of mean occupants per dwelling.

METHODS

POPULATION SIZE

Data on deaths and population for New Zealand Europeans were obtained from the published work by Rice [3]. The numerator data was collected from death certificates and the denominator data from the 1916 census. To examine the effect of geography on mortality rates, the country was divided into cities, towns and counties. The data relating to individual counties were entered directly into Excel, while the data on cities and towns were divided by population: city (population >20 000), town >2 000 or town <2 000. Data were also split into North and South Islands and analysed separately to determine whether living on one island or the other had a confounding effect on the results. Statistical analyses were performed using OpenEpi (Emory University, 2005) and EpiInfo. Mortality rates for cities, towns (population >2000 or population <2000) and counties were then compared.

Data from 11 counties were excluded from evaluation where separating the towns and counties was required. The published data for these counties included deaths for some internal towns that could not be separated. These counties were Cook County (and Gisborne), Sounds County (and Picton), Woodville County (and Woodville), Waipukurau County (and Waipukurau), Eketahuna County (and Eketahuna), Wairoa County (and Wairoa), Rodney County (and Warkworth), Waipawa County (and Waipawa), Hobson County (and Dargaville), Waitemata County (and Hellensville) and Whangarei County (and Whangarei).

POPULATION DENSITY

Population density figures for counties inclusive of interior boroughs were obtained from the New Zealand 1916 census. The data on deaths and population for New Zealand Europeans published by Rice were mainly calculated for counties exclusive of interior boroughs, with the boroughs being listed separately (n=222 cities, towns and counties). In order to compare the two sets of data, the data published by Rice was converted to counties inclusive of interior boroughs, therefore boroughs were added to their respective counties so that the counties population matched that in the 1916 census. This allowed calculation of a mortality rate for counties inclusive of interior boroughs using the summed populations and number of deaths from Rice's

compiled data (n=106 counties). Mortality rates and population densities for counties inclusive of interior boroughs were then compared, graphed and regression analyses performed. Data was also divided into quintiles based on population density so that comparisons could be made. Finally, the data for North and South Island were separated to investigate any potential confounding effects. Statistical analyses were performed using OpenEpi and EpiInfo.

Nine counties were excluded from the analysis for various reasons. Counties in Rice's data that did not match the census data with respect to population after addition of their interior boroughs (e.g. Oxford County) were excluded due to uncertainty of total county population and contributing deaths. Several other counties had to be excluded as Rice had included towns in their total population that were not internal boroughs of that county. Because it was impossible to separate this interior borough from its incorrect county it was impossible to obtain a mortality rate for those counties (e.g. Sounds and Marlborough Counties were excluded as Sounds County and Picton are combined in the data published by Rice, though Picton is an internal borough of Marlborough County).

OCCUPANTS PER DWELLING

Statistics on the mean number of occupants per dwelling for New Zealand towns and counties were obtained from the 1916 census (exclusive of those dwellings occupied only by Maori) and compared to mortality rates from those areas as calculated from Rice's published data. North and South Island and city and county data were also analysed separately to attempt to establish any potential confounding effects. This was graphed in Excel and linear regression performed. A total of 31 towns and counties were excluded from the analysis due to lack of data on mean number of occupants per dwelling for these areas.

NORTH VS. SOUTH ISLAND

Data on North and South Island deaths and total populations were obtained from *Black November* [3]. Mortality rates for the North and South Islands were calculated and these rates were compared and analysed for significance using OpenEpi. The Chatham Islands and Stewart Island were included in the South Island analysis.

RESULTS

POPULATION SIZE

Of the 96 counties included in the evaluation, the total number of deaths varied from 0 to 100 with a total of 1213, a mean number of deaths per county of 12.50 and an overall mortality rate of 3.3 per 1 000. Of the 85 smaller towns of population <2 000, the total number of deaths was 862 with a mean number of deaths per town of 10.14 and an overall mortality rate of 9.0 per 1000. For the 26 larger towns of population >2 000, the total number of deaths was 1150, a mean of 44.23 per town and overall mortality rate of 7.3 per 1000. The four major cities (Auckland, Wellington, Christchurch and Dunedin) had a total number of deaths of 2616 and a mortality rate of 6.5 per 1000 (Table 1.1).

Table 1.1: Mortality rates in cities, towns and counties

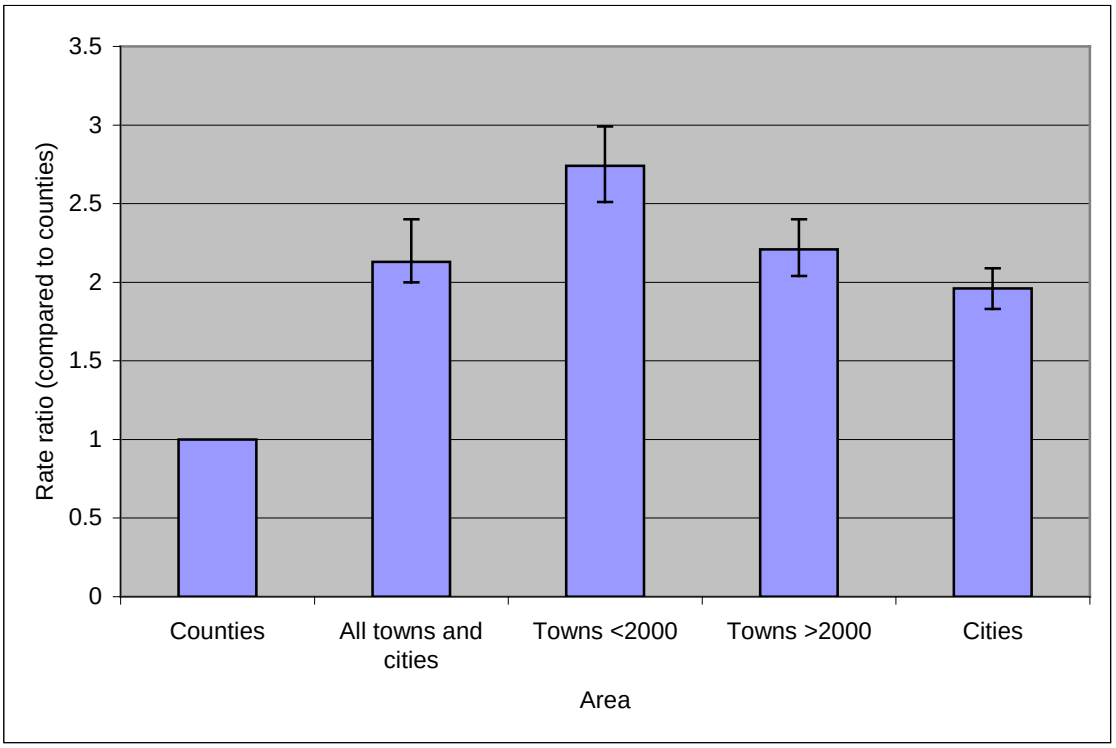
Population grouping (N)	*Deaths*	*Population*	*Mortality rate (per 1000)*
Cities (4)	2616	404 916	6.5
Larger towns (26)	1162	157 554	7.4
Smaller towns (85)	862	95 362	9.0
Counties (97)	1213	367 358	3.3

Table 1.2 shows rate ratios of mortality rates for the towns and cities combined vs. the counties, as well the individual rate ratio for smaller towns (population <2 000), larger towns (population >2 000), and cities vs. the counties. All of these results demonstrate an increased mortality rate in the towns and cities when compared to the counties. Interestingly, this apparent increased risk diminishes as the size of the town or city increases, such that the mortality rate ratio for mortality rates in larger towns compared to smaller towns was 0.81 (95% CI=0.74-0.88) and for cities compared to larger towns was 0.89 (95% CI=0.83-0.95) (Table 1.2, Figure 1.1).

Table 1.2: Rate ratios for mortality rates by cities, towns and counties

Area	*Mortality rate ratio*	*95% C.I*	*p-value*
All towns and cities vs. counties	2.13	2.00-2.27	<0.001
Smaller towns vs. counties	2.74	2.51-2.99	<0.001
Larger towns vs. counties	2.21	2.04-2.40	<0.001
Cities vs. counties	1.96	1.83-2.09	<0.001
Larger towns vs. smaller towns	0.81	0.74-0.88	<0.001
Cities vs. larger towns	0.89	0.83-0.95	<0.001

Figure 1.1: Rate ratios for mortality rates in towns and cities compared to counties.



In *Black November* Rice states that 'you were three times as likely to die in the flu epidemic if you lived in the city than if you lived in the country, and twice as likely to die if you lived in a town rather than if you lived on a farm'. Assuming that the counties are the 'farms', and counties and all towns (both large and small) are the 'country', the relative risks for mortality rates between these groups were calculated (Table 1.3).

Table 1.3: Rate ratios for mortality rates in cities vs. country and towns vs. counties.

Comparison	*Mortality rate ratio*	*95% C.I*	*p-value*
Cities vs. All Towns and Counties*	1.28	1.22-1.35	<0.001
All Towns vs. Counties	1.58	1.48-1.69	<0.001

** Includes the 11 counties previously excluded due to presence of interior borough data*

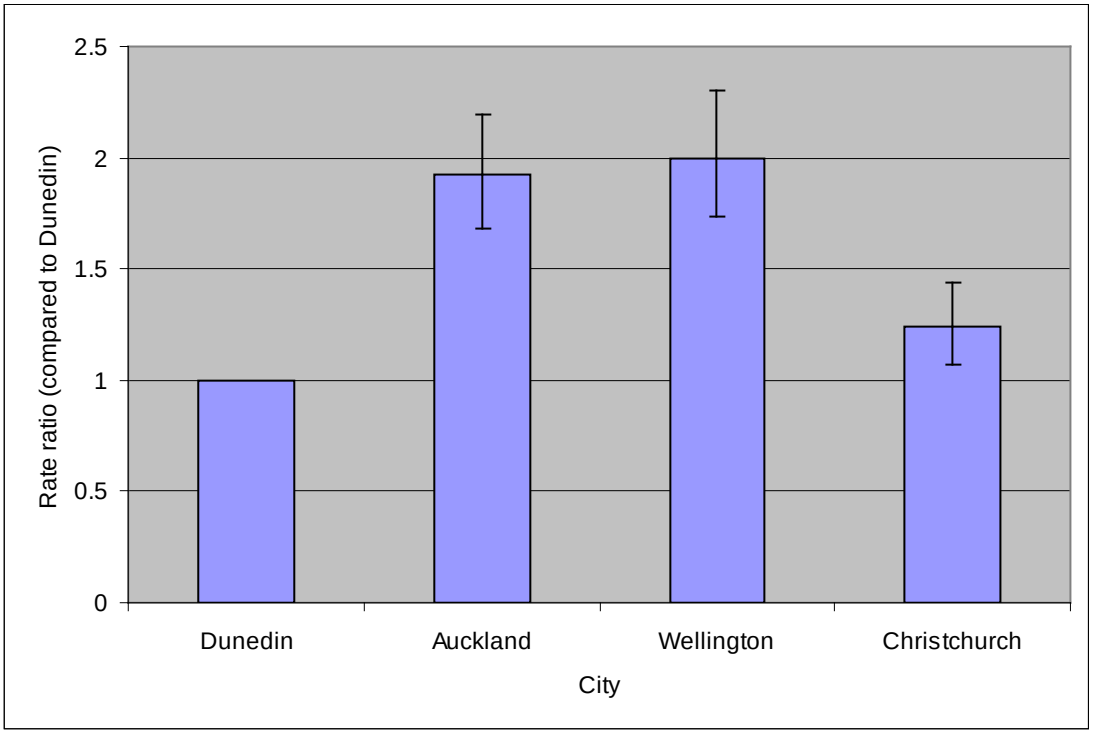
From this analysis, it would appear that people in 1918 were 25% more likely to die in the flu epidemic if they lived in the city than if they lived in the country, and 60% more likely to die if they lived in a town rather than if they lived on a farm. This finding shows the same trends as described in *Black November*, however when statistically analysed, the associations using our definitions are not as strong.

The mortality rates for the four cities were also compared. Mortality rates were highest in Wellington at 7.9 per 1000 and lowest in Dunedin at 4.0 per 1000. Statistically significant differences were observed between Auckland, Wellington and Christchurch when compared to Dunedin (Table 1.4, Figure 1.2).

Table 1.4: Mortality rates and rate ratios in the cities.

City	*Deaths*	*Population*	*Mortality rate (per 1000)*	*Mortality rate ratio*	*95% CI*	*p-value*
Auckland	1128	148 192	7.6	1.92	1.68-2.19	<0.001
Wellington	757	95 235	7.9	2.00	1.74-2.30	<0.001
Christchurch	458	92 773	4.9	1.24	1.07-1.44	0.004
Dunedin	273	68 716	4.0	1.00 (Ref)		

Figure 1.2: Rate ratios for city mortality rates compared to Dunedin.



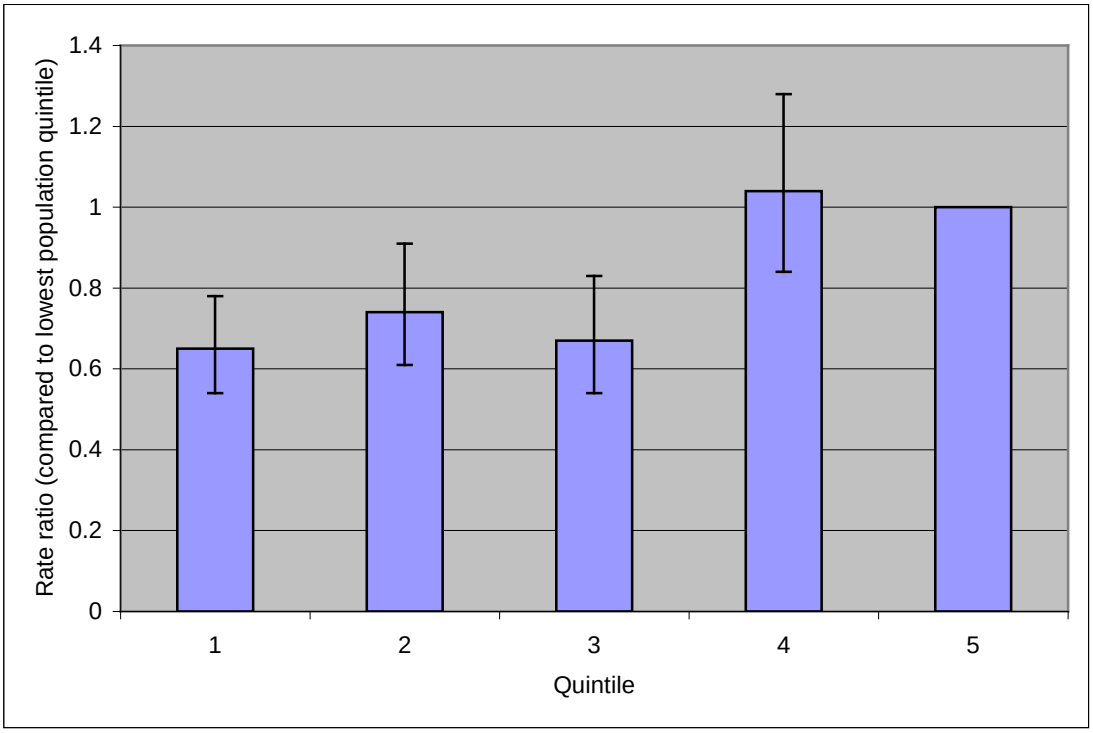
For the 111 towns studied, the mortality rates ranged from 0 to 46.0 per 1000 population. The towns were split into quintiles based on population size, with quintile one containing towns of largest population size, and quintile 5 containing towns of lowest population size. There is a pattern of lower mortality rates with increasing town size such that the mortality rate amongst the largest towns in quintile one was only 65% of that of the smallest towns in quintile five (Table 1.5, Figure 1.3).

Table 1.5: Mortality rates and rate ratios in towns by population size quintile.

Quintile	*Number of towns*	*Deaths*	*Population*	*Mortality rate (per 1000)*	*Mortality rate ratio*	*95% CI*	*p-value*
1	22	1 073	151 862	7.1	0.65	0.54-0.78	<0.001
2	22	343	42 539	8.1	0.74	0.61-0.91	0.003
3	23	255	30 867	8.3	0.67	0.54-0.83	<0.001
4	22	221	19 637	11.3	1.04	0.84-1.28	0.75
5	22	132	12 141	10.9	1.00 (Ref)		

Quintile 1=towns of largest population; quintile 5=towns of smallest population

Figure 1.3: Rate ratios for mortality rates compared to lowest population size quintile in towns.



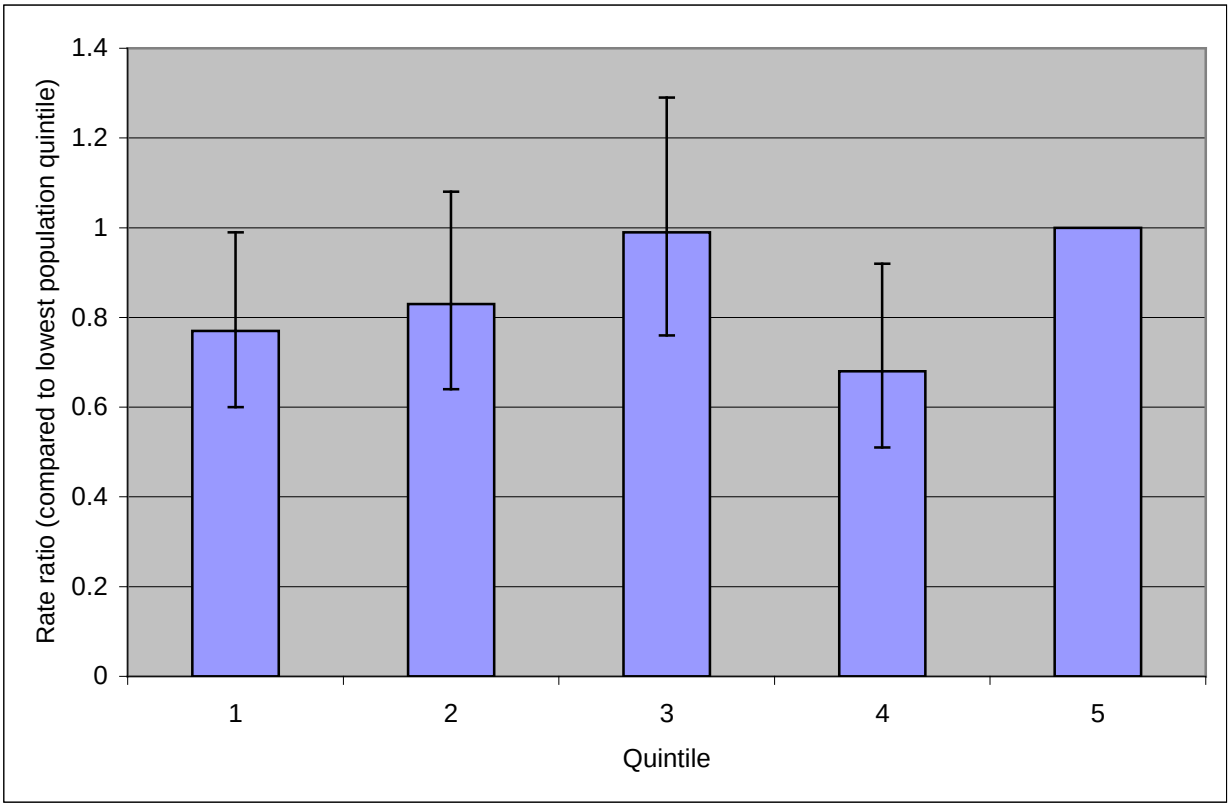
For the 96 counties studied, the mortality rates ranged from 0 to 10.1 per 1 000 population. While there was no consistent pattern of declining mortality rates with increasing county size, there were significantly lower mortality rates in two of the quintile groupings (including for those counties in the highest population size quintile) when compared to the lowest population quintile (Table 1.6, Figure 1.4).

Table 1.6: Mortality rates and rate ratios in counties by population size quintile.

Quintile	*Number of counties*	*Deaths*	*Population*	*Mortality rate (per 1000)*	*Mortality rate ratio*	*95% CI*	*p-value*
1	20	517	166 556	3.1	0.77	0.60-0.99	0.038
2	19	282	84 267	3.3	0.83	0.64-1.08	0.16
3	19	231	58 201	4.0	0.99	0.76-1.29	0.92
4	19	111	40 437	2.7	0.68	0.51-0.92	0.011
5	20	72	17 897	4.0	1.00 (Ref)		

Quintile 1=counties of largest population; quintile 5=counties of smallest population

Figure 1.4: Rate ratios for mortality rates compared to lowest population size quintile in the counties.



When the data were split into North and South Islands, and the same analysis applied, the trends remained the same for both islands: a protective effect was observed in the counties when smaller towns, larger towns, and cities were compared to the counties.

This protective effect diminished as the size of the town increased (Tables 1.7 and 1.8).

Table 1.7: Rate ratios for mortality rates by counties, towns and cities in the North Island.

Area	*Mortality rate ratio*	*95% CI*	*p-value*
Towns and cities vs. counties	2.15	1.98-2.33	<0.001
Smaller towns vs. counties	2.59	2.31-2.90	<0.001
Larger towns vs. counties	2.06	1.85-2.28	<0.001
Cities vs. counties	2.10	1.92-2.28	<0.001
Larger towns vs. smaller towns	0.79	0.71-0.89	<0.001
Cities vs. larger towns	0.77	0.74-0.80	0.668

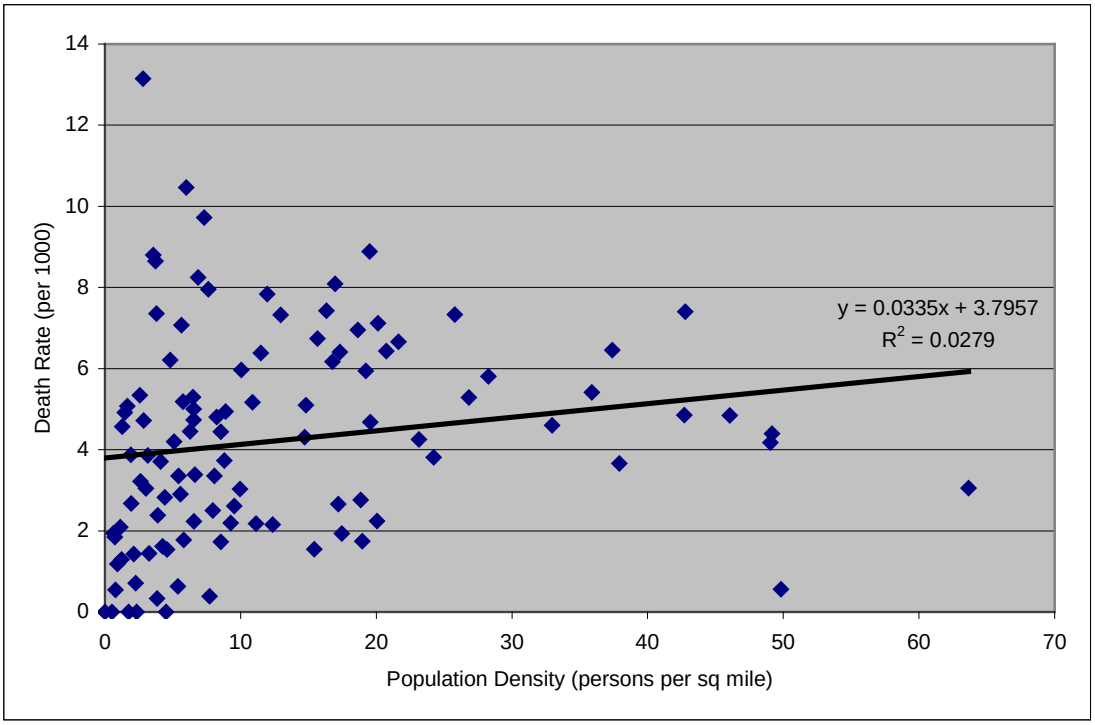
Table 1.8: Rate ratios for mortality rates by counties, towns and cities in the South Island.

Area	*Mortality rate ratio*	*95% CI*	*p-value*
Towns and Cities vs. Counties	2.00	1.81-2.21	<0.001
Smaller towns vs. Counties	2.78	2.42-3.19	<0.001
Larger towns vs. Counties	2.46	2.17-2.78	<0.001
Cities vs. Counties	1.58	1.41-1.78	<0.001
Larger towns vs. Smaller towns	0.88	0.77-1.01	0.071
Cities vs. Larger towns	0.64	0.58-0.72	<0.001

POPULATION DENSITY

The relationship between population density and the mortality rate for counties (inclusive of interior boroughs) was also examined. Simple linear regression for population density and mortality rates gave a result of $r^2 = 0.028$ which was not statistically significant (p=0.11) (Figure 1.5). Weighting by population size resulted in $r^2 < 0.01$ (i.e. completely removing any relationship).

Figure 1.5: Relationship between population density and mortality rate (n=106)



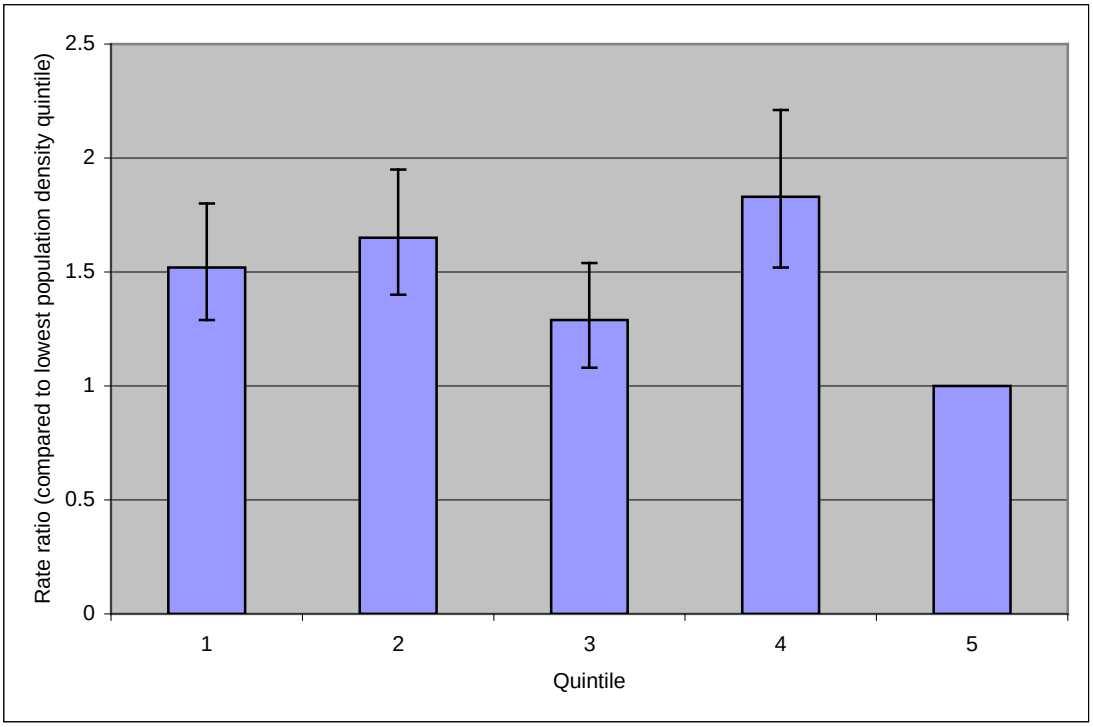
The counties were categorised into population density quintiles and mortality rates and rate ratios (compared to lowest density quintile) calculated (Table 1.9 and Figure 1.6). While there was no obvious trend of increasing mortality rate with increasing population density, there was a statistically significant difference in mortality rates for each of quintiles one to four when compared to the lowest population density quintile five. This difference was not statistically significant when the two highest density quintiles (1 and 2) were compared to the two lowest density quintiles (4 and 5): mortality rate ratio = 1.08, (95% CI=0.99-1.19, p=0.096).

Table 1.9: Mortality rates and mortality rate ratios by population density quintiles

Quintile	*Number of counties*	*Deaths*	*Population*	*Mortality rate (per 1000)*	*Mortality rate ratio*	*95% CI*	*p-value*
1	21	1023	202 653	5.1	1.52	1.29-1.80	<0.001
2	21	1211	221 086	5.5	1.65	1.40-1.95	<0.001
3	22	573	133 558	4.3	1.29	1.08-1.54	0.004
4	21	365	60 085	6.1	1.83	1.52-2.21	<0.001
5	21	156	47 037	3.3	1.00 (Ref)		

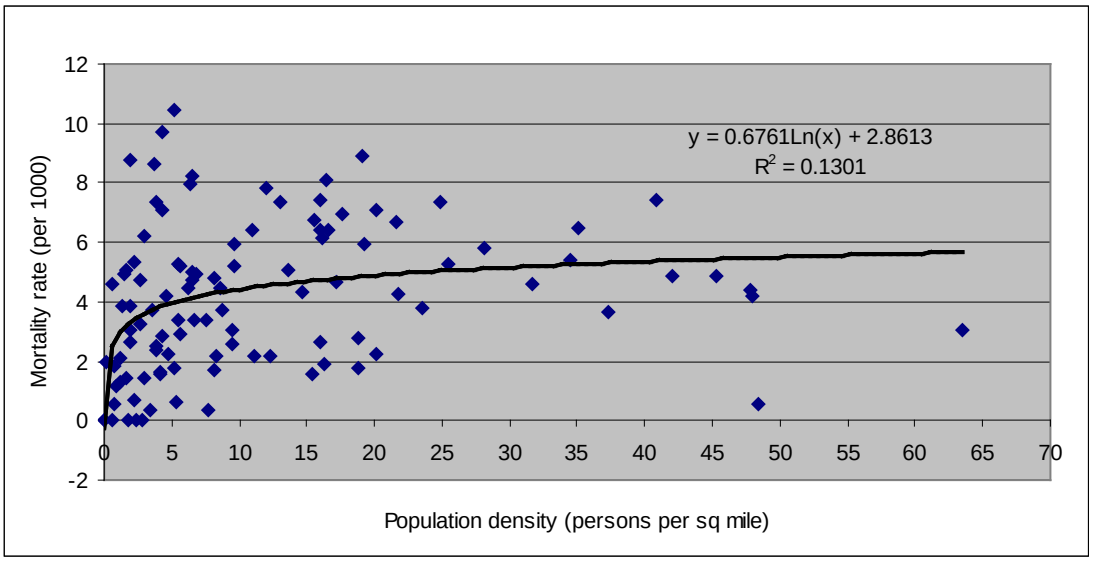
Quintile 1=counties of highest population densities; quintile 5=counties of lowest population densities

Figure 1.6: Rate ratios for mortality rates compared to lowest population density quintiles (n=106)



Quintiles one to four all had significantly higher mortality rates when compared to quintile five, raising the possibility of a threshold effect. A non-linear regression line was plotted on the scatter plot of population density vs. mortality rate to investigate this possibility. The r^2 value for this analysis was 0.13, which suggests a better fit than the linear regression (Figure 1.7).

Figure 1.7: Relationship between population density and mortality rate (n=106).



These data were separated according to North and South Islands to assess the possible effect of this as a confounder. When comparing quintiles of the North and South Islands individually, statistically significant differences between the mortality rates in quintiles one to four were observed when compared to quintile five for both Islands, but no real trend is observed. When the two highest density quintiles and the two lowest density quintiles were compared, the result was statistically significant for the North Island (rate ration (RR)=1.17 [95% CI=1.03-1.33]; p=0.01, Table 1.10), but not for the South Island (RR=0.91, [95% CI= 0.79-1.05]; p=0.2, Table 1.11).

Table 1.10: Mortality rate ratios by population density quintiles in North Island counties (n=63).

Quintile	*Number of Deaths*	*Population*	*Mortality rate (per1000)*	*Mortality rate ratio*	*95% CI*	*p-value*
1	728	146125	5.0	1.27	1.03-1.58	0.03
2	731	122272	6.0	1.53	1.23-1.90	<0.001
3	391	77486	5.0	1.29	1.03-1.61	0.03
4	190	37083	5.1	1.31	1.02-1.67	0.03
5	94	23981	3.9	1.00		

Quintile 1=counties of highest population densities; quintile 5=counties of lowest population densities

Table 1.11: Mortality rate ratios by population density quintiles in South Island counties (n=43)

Quintile	*Number of Deaths*	*Population*	*Mortality rate (per1000)*	*Mortality rate ratio*	*95% CI*	*p-value*
1	157	42265	3.7	2.41	1.44-4.03	<0.001
2	543	108760	5.0	3.24	1.97-5.33	<0.001
3	250	58493	4.3	2.78	1.68-4.60	<0.001
4	228	37563	6.1	3.94	2.38-6.54	<0.001
5	16	10391	1.5	1.00		

Quintile 1=counties of highest population densities; quintile 5=counties of lowest population densities

There were statistically significant differences between the mortality rates at the level of all but one quintile for mortality rates in the North vs. South Island (Table 1.12).

Table 1.12: Mortality rate ratios for North vs. South Island counties by population density quintiles.

Quintile	*NI mortality rate (per 1000)*	*SI mortality rate (per 1000)*	*Mortality rate ratio*	*95% CI*	*p-value*
1	5.0	3.7	1.34	1.13-1.59	<0.001
2	6.0	5.0	1.20	1.07-1.34	0.001
3	5.0	4.3	1.18	1.01-1.38	0.04
4	5.1	6.1	0.84	0.70-1.02	0.08
5	3.1	1.5	2.55	1.5-4.32	<0.001

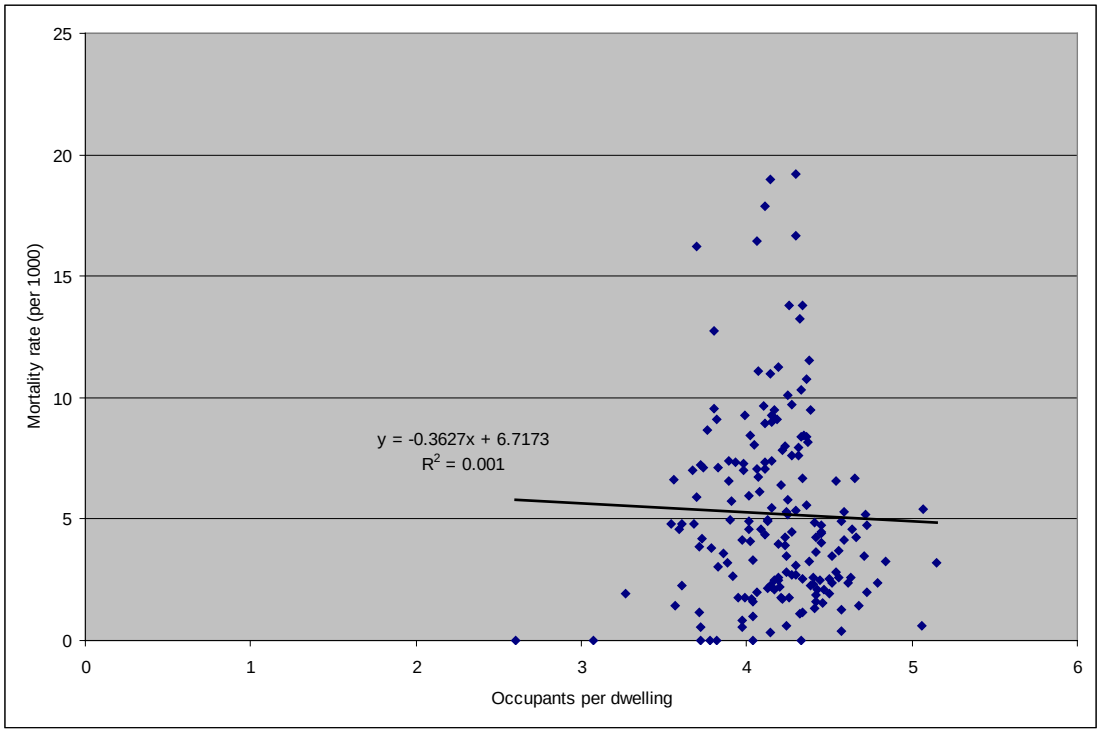
Quintile 1=counties of highest population densities; quintile 5=counties of lowest population densities

OCCUPANTS PER DWELLING

Data from the 1916 census was also evaluated for evidence of the average number of occupants per dwelling. These data were available for 180 counties, towns and cities with a mean of 4.18 ± 0.34 (SD) occupants per dwelling (range 2.6 to 5.15). A total of 31 counties and towns did not have details of the average number of occupants per dwelling and could not be included in the analysis. It should be noted that the total

occupants given for a county or town often did not match their total population as stated in other areas of the census. The data was plotted on a scatter graph against mortality rates per 1000 population (Figure 1.8). Simple linear regression gave a result of $r^2<0.01$, discounting any relationship.

Figure 1.8: Relationship between number of occupants per dwelling and mortality rate.



North and South Island and city and county data were plotted separately and regression analysis performed for each. This failed to show any relationship between mean number of occupants per dwelling and mortality rate for any one of these subgroups.

NORTH VS. SOUTH ISLAND

This evaluation found a significantly increased mortality rate in the North Island when compared to the South Island (Table 1.13).

Table 1.13: Mortality rates and rate ratios for North and South Island

Island	*Number of deaths*	*Population*	*Mortality rate (per 1000)*	*Mortality rate ratio*	*95% CI*	*p-value*
North	4051	651 072	6.2	1.37	1.29-1.44	<0.001
South	2040	447 503	5.5	1.00 (Ref)		

DISCUSSION

POPULATION SIZE

Significant protective associations were found to exist for those who lived in the counties as compared to those who lived in towns or cities. These results must be interpreted with care because they are comparing crude mortality rates based on total population alone, with no reference to actual density of population in these areas or to the age-sex structure of the populations. The 1916 census states that 'the counties contain what is understood to be the rural population, but this is not strictly correct, as some of the towns not municipalized and forming parts of the counties have considerable populations. On the other hand, the population of a few of the smaller boroughs should strictly be counted as rural. Similarly, these data have not been adjusted for age and sex, and we hypothesise that this might alter them considerably due to the unique demographics of New Zealand at the time of the 1918 influenza pandemic. One characteristic of the 1918 influenza pandemic that was relatively unique to New Zealand was that it had a propensity toward killing men over women. While this illness was seen to kill many young people worldwide, the overall mortality rates between men and women were noted to be almost equal. New Zealand was still at war when the pandemic arrived and hence it is likely that sex distribution especially varied greatly throughout the country. Data from the 1916 census puts the number of females per 100 males high at 115.08 in metropolitan area, where the total for New Zealand was 99.26, suggesting that there was a higher proportion of males living rurally at this time [6]. In light of the fact that influenza killed a higher proportion of men than women in New Zealand, adjusting for sex in these groups would probably cause the observation of lower mortality rates in the counties to be strengthened.

Adjusting for sex may also remove the effect of a few small towns (population <2 000) with high mortality rates that significantly increased the overall mortality rate for that group. Examples include the Southland mining towns of Nightcaps and Otautau (mortality rates 46.0 and 25.1 per 1 000 respectively) which were presumably also predominantly male [3].

The increased mortality rate in smaller (population <2 000) towns compared to larger (population >2 000) towns and the cities was unexpected. While we can find no

definitive reason to explain this finding, it is speculated that the populations in the larger centres may have had access to better care. Thus, while the incidence may have been higher or comparable in these areas, the overall mortality rate was lower. Factors such as better hospitals and more organised community care may have benefited those in the larger centres. It is also possible that the smaller towns (<2 000 population) contained relatively more men (such as mining and forestry towns) which would cause an apparent increase in mortality rate in this group as men were more likely to die from influenza.

A possible confounding factor for the observed result of higher mortality rates amongst cities and towns is the increased likelihood that these centres were exposed to more cases of disease by way of shipping ports or the railway network, when compared to the more rural counties.

POPULATION DENSITY

No significant association between population density and mortality rate was observed when the results were plotted graphically and linear regression performed. A statistically significant association was observed when the counties and towns were split into quintiles based on increasing population density and the lowest and highest quintiles were compared for the whole country, and for the North and South Islands separately. An association was also seen between increasing mortality rate and increasing population density in the North Island when the top two population density quintiles were compared to the bottom two. It can be concluded that there was a protective effect of living in an area with very low population density, but mortality rates were no higher at higher density areas than in moderately population dense areas. This is perhaps due to a threshold effect where very low population density is protective, but above the limit of the lowest population density quintile cut-off, there was no benefit (in terms of decreased mortality rate) of living in a low versus high density area. At a national level, the upper limit of the 5^{th} quintile of population density data was 2.34 persons per square mile. This apparent protective effect of living in the lowest density quintile was still observed when the data were split into North and South Islands and analysed for both, even though the upper limit of the 5^{th} quintile differed greatly between the islands (3.51 and 1.22 persons per square mile respectively). This implies that the threshold could in fact be higher than that

suggested by the national data and that more analysis would be required to establish its true value.

OCCUPANTS PER DWELLING

There was no association found between mortality rates from influenza and mean number of occupants per dwelling. It is speculated that the primary reason for this was the lack of variation in exposure (number of occupants per dwelling) with great variation in the corresponding mortality rates for these areas. There appears to be no geographical variation in the mean number of occupants per dwelling, and separating the data by major island did not yield any significant association between number of occupants per dwelling and mortality rate.

NORTH VS. SOUTH ISLAND

A significantly increased mortality rate from influenza was observed in the North Island when compared to the South Island. Several factors have been identified which could have contributed to this finding. The population density of the North Island was higher at 14.75 persons per square mile than the South Island at 7.58 persons per square mile, and as already discussed, there is evidence for an association between increasing population density and increasing mortality rate from influenza. The peak of the epidemic in the South Island was about a fortnight after its peak in Auckland, and it is speculated that many of the South Island towns and cities (particularly Christchurch and Dunedin) may have had an action plan prepared by the time that the disease reached them.

It was decided that Maori data should be excluded from this evaluation for several reasons. At the time of the 1916 census and the 1918 influenza epidemic, many Maori tribes were boycotting officialdom in resistance to wartime conscription [3]. For this reason it is generally accepted that the 1916 census is an underestimate of Maori population at the time. To complicate matters, the census figures concerning Maori are not part of the official census, but are attached as an appendix and do not contain many of the figures of interest to us for this analysis, such as population density and number of occupants per dwelling. These limitations to the 1916 census would cause the gathering of denominator data to be much more complicated than for non-Maori.

As part of their boycott of officialdom at this time, many Maori were also refusing to register births, deaths and marriages, leading to potential problems with numerator data. Rice has largely addressed this issue with a detailed search of all death certificates and newspaper sources until the end of the first quarter of 1919. It is felt that a detailed analysis of Maori data is desirable as part of future research and would be of great interest as we have very few answers to the question of why Maori suffered such great losses in the 1918 influenza pandemic.

SECTION 2

THE SOCIOECONOMIC GRADIENT OF THE 1918 INFLUENZA PANDEMIC IN NEW ZEALAND

INTRODUCTION

The 1918 pandemic has, until recently, been referred to as a socially neutral disease. Recently, and for the first time, it was suggested that social class may act as a factor contributing to mortality from the influenza pandemic [7]. This Norwegian study found that mortality rates for those in the 'upper class' was significantly lower than those in 'lower class.' New Zealand is currently experiencing a widening socioeconomic gap and so it is of interest to investigate the effects of socioeconomic status (SES) on mortality rates in the 1918 influenza pandemic in this country. We aimed to analyse the occupational mortality rates recorded in *Black November* [3]. Unfortunately we could not find any denominator data from the 1916 Census to make valid statistical analyses. Thus we aimed to deduce socioeconomic conclusions by focussing on the occupational data contained within *Black November*.

HYPOTHESIS ONE: PRIMARY PRODUCTION VS. PROFESSIONALS

People in 'Primary Production' will experience higher mortality rates from influenza than those working as 'Professionals.'

In formulating this hypothesis we have made the assumption that the class of 'Professionals' is of a significantly higher socioeconomic status than those working in 'Primary Production'.

HYPOTHESIS TWO: ARMED FORCES VS. PROFESSIONALS

The 'Armed Forces' will experience higher mortality rates from influenza than those working as 'Professionals'.

HYPOTHESIS THREE: ALL OCCUPATIONS VS. DEPENDANTS

Those having an occupation will experience higher mortality rates from influenza than 'Dependants'.

HYPOTHESIS FOUR: TRANSPORT AND COMMUNICATIONS VS. PROFESSIONALS

People working in 'Transport/Communication' will have higher mortality rates from influenza then those working as 'Professionals'.

METHODS

We calculated mortality rate ratios from Rice's data in *Black November* using the online statistical program 'OpenEpi'. These rate ratios compared each individual occupational group (exposure group) to our reference (non-exposure) group: 'Professionals.' The reasoning for using 'Professionals' as a reference point was because we were absolutely certain that this group was of the highest SES.

When comparing 'all occupations' to 'Dependants' we combined all those groups we considered as having jobs into one broad category, consisting of the sub groups: Professional and Public Service (excl. defence); Armed Forces; Accommodation & Domestic Service; Commercial; Financial and Retail; Transport and Communications; Industry and Manufacture; Trades and Construction; and Primary Production.

We initially thought we could compare SES data from the major cities (Auckland, Christchurch, Wellington, Dunedin) but Rice's data on SES status of the victims did not correlate with the classification system of the 1916 census. Effectively we could not find matched denominator data. Thus we directed our efforts towards estimating SES from Rice's occupational mortality data, which correlated with the 1916 census 'denominator' values.

Each occupational group was described in the 1916 census as follows [6]:

- "Professional & Public Service: Persons mainly engaged in government and defence of the country and in satisfying the moral, intellectual, and social wants of its inhabitants."

  Rice has further sub-divided the 'Professional' group into 'Armed Forces,' so certain aspects of the above definition also apply to this sub-group.

- "Domestic: Persons engaged in the supply of accommodation, and in rendering personal services for which remuneration is usually paid."

- "Commercial: People directly connected with the hire, sale, transfer, distribution, storage, and security of property and materials."

- "Transport and Communications: People engaged in the transport of persons or goods or in effecting communications."

- "Industrial: People, who are principally engaged in various works of utility or in the specialities connected with the manufacture, construction,

modification, or various uses of man, but excluding so far as possible, all who are mainly or solely engaged in the service of commercial interchange."

Rice has subdivided the 'Industrial' group into 'Industry & Manufacture' and 'Trades & Construction.'

- "Agricultural: agricultural, pastoral, mineral, and other primary producers. This includes all persons mainly engaged in the cultivation or acquisition of food products and in obtaining other raw materials from natural sources."

  This group is the equivalent of Rice's 'Primary Production' group.

- "Dependants: Persons dependent upon relatives or natural guardians, including wives, children, and others not otherwise engaged in pursuits for which remuneration is paid. Also including people dependent upon private charity or whose support is a burden on the public revenue."

- "Undefined: All people of whom the nature of their work cannot be exactly determined."

We used data from *Black November* to confer SES rankings to each occupational group [3]. These data, in conjunction with the description of occupations from the 1916 census, were used to rank the occupations according to SES. Each rank was designated a score of 1-7 (1 being the highest SES, 7 being the lowest) as follows:

SES rank	Occupation
1	Professional and Public Service (excludes Defence)
1b	*Armed Forces*
2	Commercial, Financial, and Retail
3	Accommodation and Domestic Service
4	Industry and Manufacture
5	Trades and Construction
6	Transport and Communications
7	Primary Production

RESULTS

By using professionals as our reference group, we were able to assess many occupational relationships with the outcome of pandemic influenza mortality. The 'Armed Forces' (mortality rate ratio (RR) [95% CI]; = 3.74 [3.12-4.49]) 'Transport & Communications' (1.59 [1.34-1.88]) and those in 'Trades & Construction' (1.30 [1.08-1.56]) had an increased mortality rate when compared with 'Professionals'. Interestingly, 'Dependents' had a reduced mortality rate compared with professionals (0.45 [0.38-0.52]) and 'all occupations' (2.54 [2.4-2.7]) (Table 2.1).

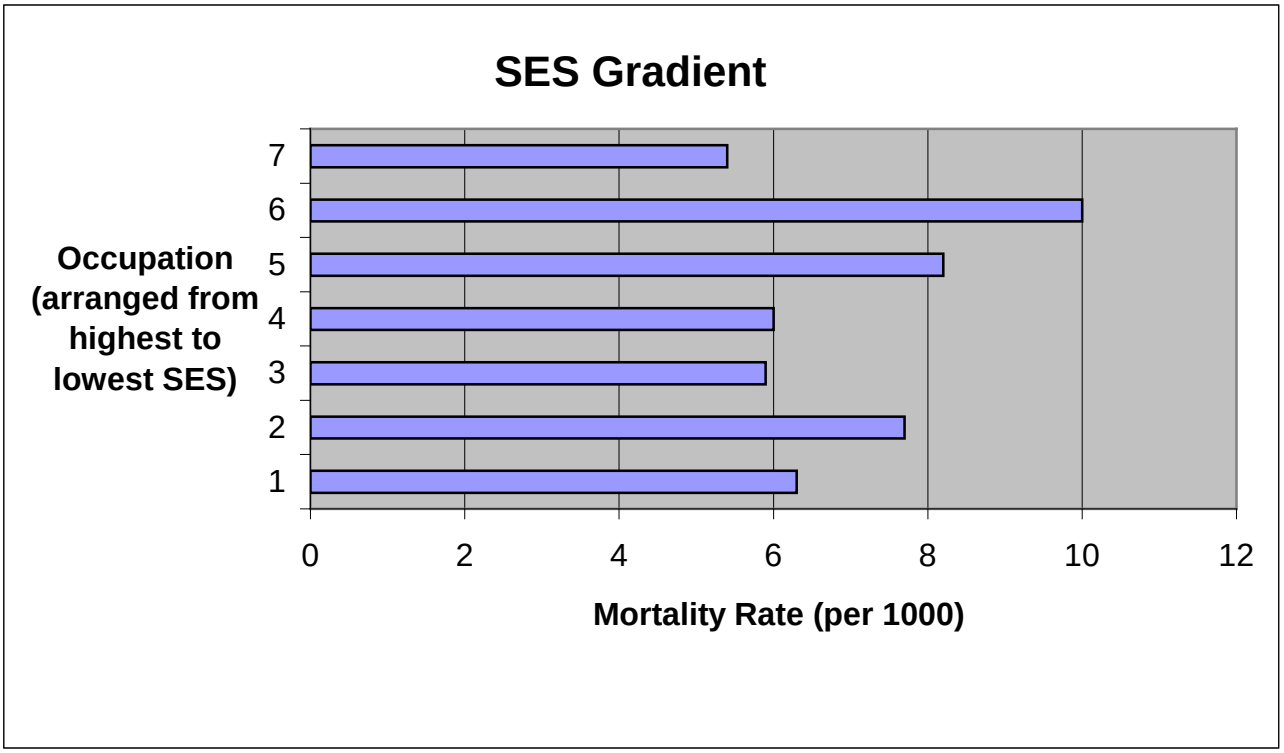
Table 2.1: Comparison of mortality rate by all occupational groups (reference occupation: professionals)

	Deaths	*Population*	*Mortality Rate (per 1000)*	*Mortality Rate Ratio*	*95% C.I.*	*p-value*
Armed Forces	**286**	**12 131**	**23.6**	**3.74**	**3.12-4.49**	**<0.01**
Accommodation & Domestic Service	249	41 915	5.9	0.94	0.78-1.14	0.53
Commercial, Financial, & Retail	**510**	**66 204**	**7.7**	**1.22**	**1.03-1.44**	**0.018**
Transport & Communications	**426**	**42 522**	**10**	**1.59**	**1.34-1.88**	**<0.001**
Industry & Manufacture	484	80 237	6	0.96	0.81-1.13	0.6
Trades & Construction	**300**	**36 682**	**8.2**	**1.30**	**1.08-1.56**	**0.0048**
Primary Production	716	132499	5.4	0.86	0.73-1.01	0.058
Dependants	**1774**	**631033**	**2.8**	**0.45**	**0.38-0.52**	**<0.001**
Pensioners & Independents (e.g. widows)	158	23937	6.6	1.05	0.85-1.30	0.67
Undefined (e.g. 'returned soldier' & Inmates)	**208**	**2319**	**89.7**	**14.22**	**11.73-17.24**	**<0.001**
All Occupations	**3160**	**442160**	**7.1**	**2.54**	**2.4-2.7**	**<0.001**
Professionals (Reference group)	189	29970	6.3	1.00 (Ref)		

- Results in bold are statistically significant
- Population is for Europeans only (excludes Maori)

The armed forces had a considerable mortality rate (23.6 per 1000) when compared with the reference group (professionals, 6.3 per 1000). As the armed forces were predominantly male, and are likely to have lived in a relatively closed population in unusually close living quarters, it was decided to exclude this group from further socioeconomic analysis. Each occupation group was designated a rank for socioeconomic status, with 1 being highest SES status (professionals) and 7 the lowest SES status. Following extended Mantel-Haenszel Chi Square analysis, a significant association was found for this socioeconomic gradient, where the higher SES status offers protection from influenza mortality (table 2.2: linear trend= 4.78; p=0.029).

Figure 2.1: The mortality rate for each occupation according to SES rank



Key: **1.** Professional and Public Service (excludes Defence) **2.** Commercial, Financial, and Retail **3.** Accommodation and Domestic Service **4.** Industry and Manufacture **5.** Trades and Construction **6.** Transport and Communications **7.** Primary Production

Table 2.2: Mantel-Haenszel Summary Mortality Ratios & Crude Mortality Ratios for Each Exposure Level

Exposure (vs. L 0)	MH Summary Mortality Ratio	Crude Mortality Ratio
Level 0	1	1
Level 1	1.22	1.22
Level 2	0.94	0.94
Level 3	0.96	0.96
Level 4	1.3	1.3
Level 5	1.6	1.6
Level 6	0.86	0.86

KEY for exposure levels: **0.** Professional and Public Service (excludes Defence) **1.** Commercial, Financial, and Retail **2.** Accommodation and Domestic Service **3.** Industry and Manufacture **4.** Trades and Construction **5.** Transport and Communications **6.** Primary Production

DISCUSSION

PRIMARY PRODUCTION VS. PROFESSIONALS

Results showed a reduced rate of mortality in 'Primary Production' compared to 'Professionals', with a mortality rate ratio of 0.86. Although this was a non-significant result, due to the confidence interval including 1.0, we felt this warranted discussion as it was not what we expected. It is possible that the professional workforce may have been more exposed to influenza than those working in 'Primary Production'. Furthermore, it is highly likely that an urban-rural disparity exists in the makeup of the 'Primary Production' group. Using the 1916 census data, we determined the proportion of males working as primary producers in the four main urban areas of NZ (Auckland, Wellington, Christchurch and Dunedin) was 3.88%. This compares to 31.34% for the remainder of "the Dominion", showing a strong urban-rural difference. It is likely that living/working in a rural area may have a protective effect (see elsewhere in this report). Other confounding factors such as age and gender may also affect the mortality rates.

ARMED FORCES VS. PROFESSIONALS

There was a significantly higher mortality risk ratio of 3.74 for **'**Armed Forces' when compared to professionals. The high population densities in army barracks, resulting in high exposure could explain these results. It is important to keep in mind that while some members of the 'Armed Forces' were officers and so have a correspondingly high SES, many of the personnel recruited may have come from a wide range of SES backgrounds (given that compulsory conscription was in place at this time).

ALL OCCUPATIONS VS. DEPENDANTS

There is strong evidence to support this hypothesis, with those being officially employed having an increased mortality rate (rate ratio: 2.54) when compared with those who were 'Dependants'. A possible reason for this could be related to the greater exposure to the disease from the workforce. However, we must also take into account that such exposures would possibly be transferred to these dependants from the respective 'breadwinners' of the family household. We cannot be sure of the exact reasons for the mortality rate ratio observed between these two groups, but the association is definitely present. One thing that needs to be kept in mind is that the

‘Dependant’ group makeup is heterogeneous. It could contain those who are sick, housewives and undoubtedly many children. All these groups fall outside the ‘typical’ victim of the 1918 flu pandemic: ‘fit male aged 20-40’.

TRANSPORT AND COMMUNICATIONS VS. PROFESSIONALS

We found support for our hypothesis when comparing ‘Transport/Communications’ against ‘Professionals’ with a significant mortality risk ratio of 1.59. Intuitively this is expected, as the former group would have received greater exposure to influenza due to the nature of their work. It would have been interesting if data were available for mortality rate differences between those specifically in ‘Transport’ versus those in ‘Communications’. The ‘Communications’ subgroup may be masking an even stronger association.

SOCIOECONOMIC TRENDS FROM OCCUPATIONAL RANKING

When ranking the occupational groups (and excluded the “Armed Forces”) we found a statistically significant trend of increased mortality with declining SES. This removal of armed forces would be justified as the overcrowded nature of the occupation (i.e. living in army barracks) could have potentially confounded our analysis. There is some ambiguity regarding whether the ‘Armed Forces’ group should be designated to their respective SES level. This arose when our colleagues’ subdivided ‘Armed Forces’ into ranks, ranging from ‘privates’ to ‘majors’. At this time in New Zealand, there was compulsory military conscription for men over the age of 18 years. Thus men from all socioeconomic classes were recruited, and it may be unjustified in assuming that those in the ‘Armed Forces’ were mostly of high SES. We must also acknowledge that although ‘Primary Production’ mortality odds appear low, there may exist a ‘rural protective’ confounding effect that is not adjusted for, possibly skewing the SES trend toward the null.

A limitation in analysing the data was the lack of depth in the description of the characteristics of each occupational group. We have made limited generalisations from these definitions regarding the relative levels of exposure amongst each occupational group.

It is important to realise that while we are trying to compare SES to mortality, based around occupation, there was some problems in doing this. Our initial idea was that a

higher SES would correlate with a lower mortality rate, but it could be seen even before we began analysing that the ‘Professional’ group had a mortality rate of 6.3 per 1000 compared to the national average of 5.5 per 1000. We shared similar difficulties with Rice in allocating occupation to SES, but we feel that we ranked them reasonably well. We have excluded ‘Dependants’ as being part of our occupationally-based SES analysis for trends, as we cannot be sure whether they were part of a ‘breadwinning’ family or not. If available, this information would have been invaluable as dependants make up a large proportion of influenza deaths.

It was interesting to note that there was no significant finding for the ‘Pensioners and Independents’ group. We would expect that pensioners and independents (including ‘widows’), despite the time period of 1918, would be relatively less exposed to the pandemic and subsequently have significantly reduced mortality rates. This is a broad assumption considering there are no formal definitions in the 1916 data for this particular group.

A group that we didn’t anticipate investigating was the ‘Undefined’ group, which turned out significant results. The composition of this group has been divided into two broad categories: ‘returning soldier’ and ‘inmates’. The pandemic was brought into New Zealand from overseas, thus it is no surprise that soldiers returning from war would have relatively high rates of influenza. High rates would also be expected amongst inmates due the crowded nature of the facilities.

SECTION 3

INFLUENZA AND THE FEATHERSTON MILITARY CAMP

INTRODUCTION

Following an influenza outbreak in 1915, a government inquiry sought recommendations to improve standards of living within military camps in New Zealand. The conditions that contributed to the development of respiratory disease, noted from the inquiry, were specifically overcrowding, poor facilities including poor laundry drying facilities, and canvas accommodation. At that time the main army camp was in Trentham, north of Wellington, while Featherston, in South Wairarapa, was one of four reinforcement camps.

Up to this time, the Featherston Camp was a canvas camp in Tauherenikau. A camp of purpose built huts, capable of accommodating 4500 men, was established close to the canvas facilities, which in turn became overflow accommodation, as did the original camp at Tauherenikau. The rifle-range at Papawai, north of Featherston Camp, also provided temporary canvas accommodation.

At the time of the 1918 influenza pandemic, Featherston Camp had approximately 8000 military personnel, which was comparable to the size of a large regional centre [8]. The first wave of the influenza hit in September, but it was the second wave in November that overwhelmed the camp. The weather conditions were stated to be the worse ever witnessed since early settlers arrived in New Zealand [9]. The second wave was exacerbated by atrocious weather conditions, which flattened the canvas tents, thereby contributing to the overcrowding of huts.

Both Henderson and Rice stated that the epidemic was swift and only lasted 2-3 weeks [3, 9]. The morbidity rates were nearly half the camp population. A total of 3220 personnel were treated at various hospitals and 314 developed severe pneumonic symptoms and mortality rates were significant. The death toll in Featherston was subsequently reported as 177, including 18 Maori [3]. The mortality rate was recorded as 22.6 per 1000 – one of the highest for Pakeha anywhere in New Zealand.

AIMS

To determine whether the Featherston Military Camp could be used as a sample population, to make influenza mortality comparisons, using rank as an indicator of close quarter living and socioeconomic status.

To establish whether Featherston Military Camp as a closed community could be used as a reference population for making comparisons to the wider community.

HYPOTHESIS ONE: EFFECT OF MILITARY RANK

That as the rank increases, the risk of influenza decreases.

HYPOTHESIS TWO: COMPARISON WITH CIVILIAN TOWNS

That Featherston Military Camp can be used to make influenza mortality comparisons to the wider community.

METHODS

We conducted a descriptive study into the influence of environmental and social factors on influenza incidence and mortality within Featherston Military Camp.

1. We conducted a literature search for background information regarding the 1918 influenza epidemic within Featherston Military Camp. Rice's book *Black November* [3] was primarily used as the principle resource for planning a literature review.

2. We searched through the material in the National Library, Archives New Zealand, and made contact with the Ministry of Defence for information regarding the Featherston Military Camp.

3. The inclusion criteria consisted of morbidity and mortality of Featherston military camp personnel between the dates of October 28th to December 11th 1918, which were defined by information gathered from Henderson's official report [9].

4. The numerator information was derived initially from Henderson's report, which stated that there were 3220 hospital admissions and 177 deaths from pneumonic influenza [9]. We then refined the numerator to 163 from the information from the Principal Medical Officer's (PMO) report on the epidemic [10]. Access to the Commonwealth War Graves Commission website provided specific individual influenza mortality data which further defined the numerator by rank [11].

5. The denominator was derived from the PMO's report that stated that the camp had a 'complement' of approximately 8000. We interpreted the term complement to be inclusive of all permanent staff and trainees. Administrative records assisted with determining an estimate of permanent staff numbers in 1918. Whilst camp personnel could have changed in the interim, we made the assumption that any change would not be statistically significant.

   Data on the capacity of the main camp in Featherston provided additional denominator information. The two sources provided us with information of 2/3 of the compliment at the time. The remaining 1/3 was estimated from information provided by the New Zealand Army.

6. The current makeup of a generic infantry battalion was applied to the 1918 layout of the Featherston Camp. Based upon this, an estimate was then applied to the known population of the main camp at the time. This strategy produced a figure that was consistent with the known hut population. This gave us the confidence to estimate the remaining one third.

   The following information breakdown assists in understanding the formula:

- A *Section* is equivalent to 24 Privates plus 1 Corporal/Lance Corporal
- A *Platoon* comprises 3 Sections (72 Privates + 1 Corporal/Lance Corporal) plus 1 Sergeant.
- A *Company* comprises 3 Platoons (216 Privates + 9 Corporals/Lance Corporals) + 3 Sergeants + 1 Warrant Officer
- All *hutments* comprise exactly 20 Companies

Upon applying the formula to all non-permanent non-commissioned camp staff we were left with the following distribution:

Privates	6607
Corporals/Lance Corporals	275
Staff Sergeants/Sergeants	92
Warrant Officers	30

Additional numbers were added to these totals using Officers under training (160) and the camp permanent staff (835). The total denominator was then estimated as:

Privates	6717
Corporals/Lance Cpls	275
Staff Sergeants/Sgts	92
Warrant Officers	30

7. In total there were 17 different job descriptions including 12 different ranks. For ease of comparison, we divided all the ranks into three main subgroups – Privates, Non-Commissioned Officers and Commissioned Officers.

   - *Commissioned Officers* include (from junior to most senior ranking):
     - Sister
     - Second Lieutenant
     - First Lieutenant
     - Captain
     - Major
     - Lieutenant Colonel
   - Under the commissioned officers, were the *Non-Commissioned Officers*. This comprised of (from junior to most senior ranking):
     - Sergeant
     - Staff Sergeant
     - Company Sergeant Major (= Warrant Officer Class 2)
     - Regimental Sergeant Major (= Warrant Officer Class 1)

- The last subgroup, which we generally termed *Private* consisted of (from junior to most senior ranking):
    - Nurses
    - Privates
    - Drivers
    - Gunners
    - Troopers
    - Lance Corporal*
    - Corporal*

*Lance Corporal and Corporals were placed in the last subgroup as they were in the same dwellings as the privates, therefore subject to the same living conditions.

8. With the collated data, we used Excel to produce relevant tables and epidemic graphs. OpenEpi was used to produced 2×2 tables to compare rate ratios of the three subgroups of army personnel.

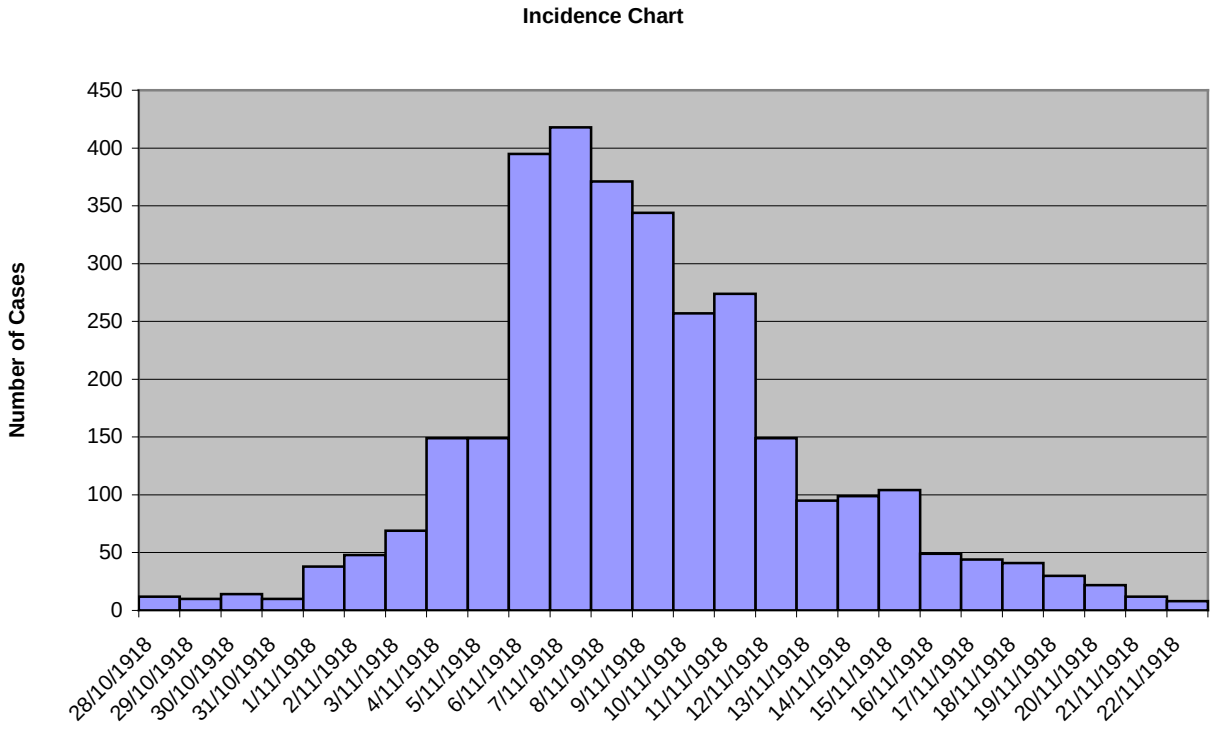
RESULTS

Information regarding the daily incidence of cases of influenza was already tabled [10] and is included to highlight the timeframe of the epidemic (Table 3.1). The data shows that the epidemic started on 28th October 1918, rendering 3220 hospital admissions throughout the main Featherston Camp and its satellites (Tauherenikau and Papawai). An epidemic curve was constructed (Figure 3.1) to show the impact of the influenza on the camps and shows a peak incidence on 7th November 1918 with 418 new cases of influenza on that day. From the 3rd November to 7th November a rapid increase in the incidence of influenza was observed in the camp. The average number of new cases per day was 70.2. The incidence also decreased rapidly, however, on the 11th and 15th November the incidence inclined again.

Table 3.1: Daily Incidence of Influenza in Featherston Military Camp

Date (in 1918)	New Cases Reported (N)
October 28	12
29	10
30	14
31	10
November 1	38
2	48
3	69
4	149
5	149
6	395
7	418 ▲
8	371
9	344
10	257
11	274
12	149
13	95
14	99
15	104
16	49
17	44
18	41
19	30
20	22
21	12
22	8
23-30	9
TOTAL	**3220**

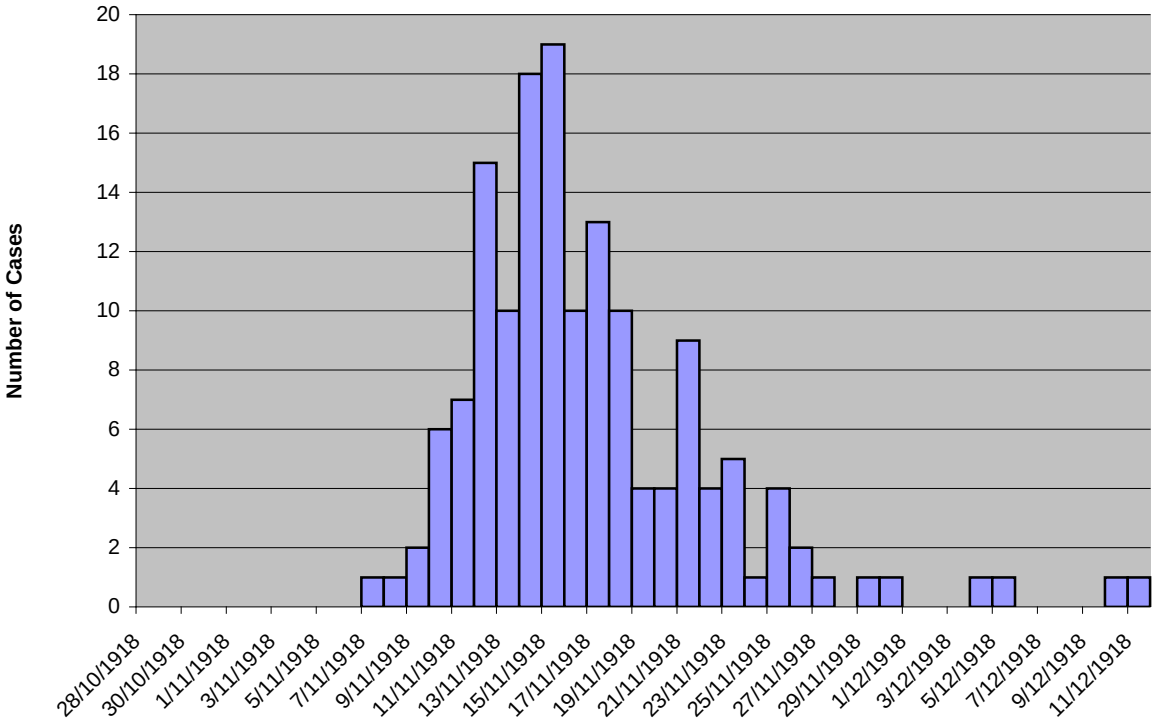
Figure 3.1: Daily Incidence Curve of Influenza in the Featherston Military Camp



MORBIDITY AND MORTALITY

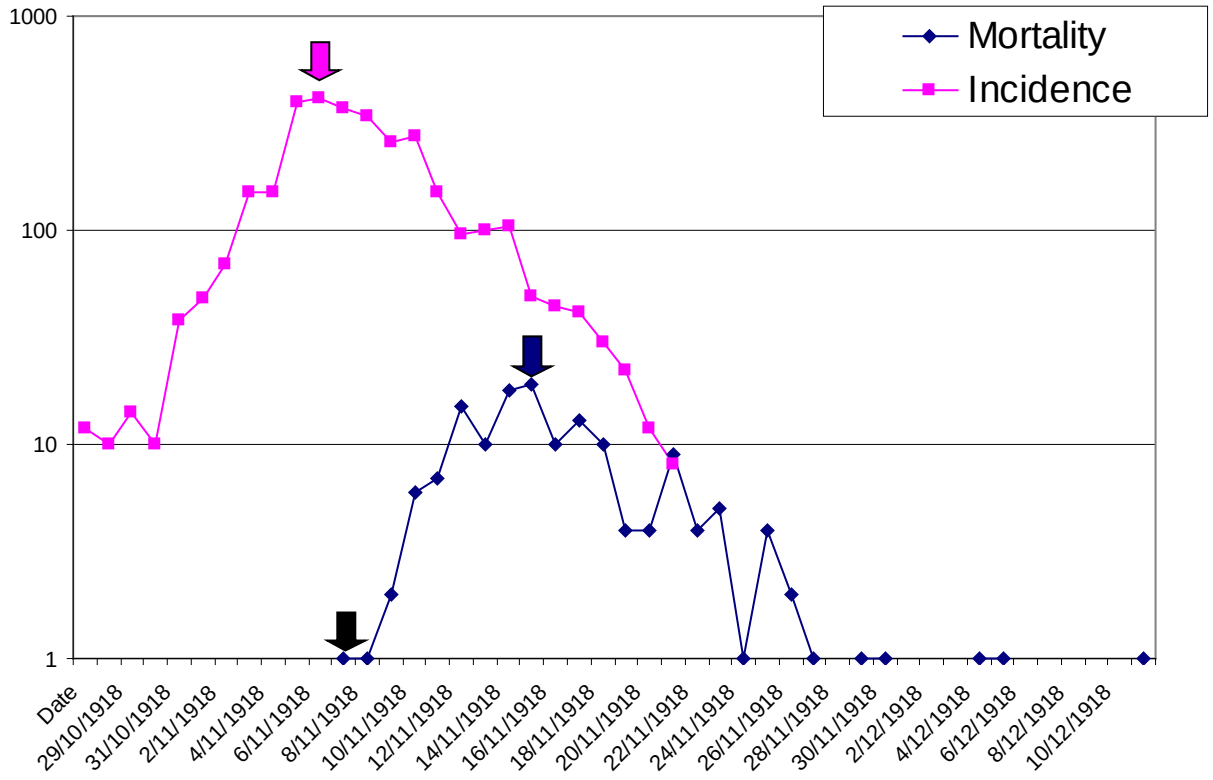
The first death that occurred after the increase in morbidity that defined the second wave, was on 7th November 1918. The incidence peaked on 15th November with 19 deaths and the last recorded death was on 11th December. The average daily mortality was found to be 3.62 deaths (Figure 3.2).

Figure 3.2: Daily Mortality in the Featherston Military Camp



Daily incidence and mortality rates were plotted on a log scale to determine any findings and appreciable trends in the data (Figure 3.3). The graph shows that the peak mortality (Blue Arrow; Figure 3.3) happened 8 days after the peak incidence (Pink Arrow; Figure 3.3). The first death (Black Arrow; Figure 3.3) and peak incidence occurred on the 7th of November. The scale also shows a case fatality of 5.06%.

Figure 3.3: Daily incidence and Mortality



EFFECT OF RANK

The Non-Commissioned Officers and Officers subgroup had higher mortality rates than that of the privates. The total mortality rate is 20.5 per 1000 (Table 3.2).

Table 3.2: Deaths of Army Personnel by broad categories of rank

Broad Category	Deaths	Total denominator population	Death rate per 1000 population
Privates	128	7481	17.11
Non-Commissioned Officers	15	301	49.83
Officers	11	217	50.69
Unknown by rank or date of death	10		
Total	**164**	**7999**	**20.5**

Officers were 2.9 times at higher risk of dying from influenza than the privates (RR = 2.9 [95% CI = 1.62, 5.20]; $p < 0.05$; Table 3.3). Non-Commissioned Officers (NCOs) had 2.8 times higher risk of dying from influenza than the privates (RR = 2.8 [95% CI = 1.71, 4.58]; $p < 0.05$; Table 3.3). When comparing Non-Commissioned Officers with Officers, no significant differences were found (RR = 1.01 [95% CI = 0.47,2.17]; p= not significant ; Table 3.3).

Table 3.3: Mortality rate ratios of Army Personnel by broad categories of rank

Broad Category	Rate Ratio	Confidence Interval	p-value
Officers vs. Privates	2.90	1.62, 5.20[1]	0.0023
Officers vs. NCOs	1.01	0.47, 2.17	0.95
NCOs vs. Privates	2.80	1.71, 4.58	0.00051

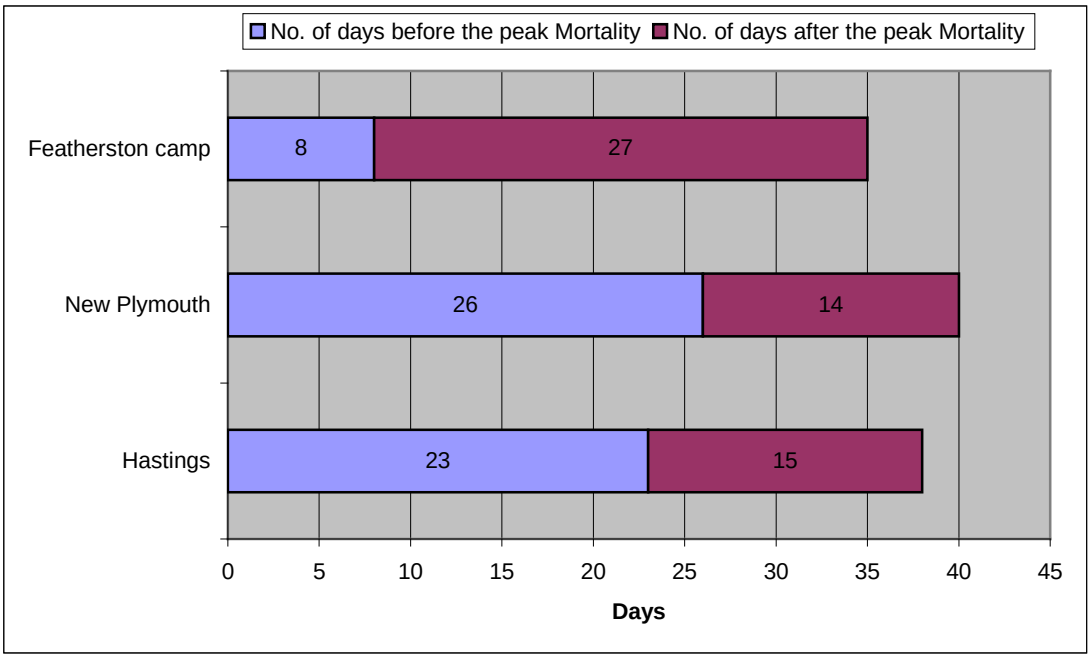
COMPARISON WITH CIVILIAN TOWNS

When comparing the influenza outbreak in the Featherston Military Camp (population = 7999) with that of North Island towns of similar population sizes such as New Plymouth (population = 8704) and Hastings (population = 7918), Featherston demonstrated the highest mortality rate of 20.5 per 1000 compared to 4.59 per 1000 for New Plymouth and 9.21 per 1000 for Hastings (Table 3.4). Featherston displayed the shortest outbreak duration of 35 days (from the time from the first to last reported death), compared with 40 days in New Plymouth and 38 days in Hastings. Featherston also demonstrates the shortest period of 8 days before mortality peak – from the time from the first reported death (Figure 3.4).

Table 3.4: Mortality rates of Featherston, New Plymouth and Hastings

Town / Camp	Deaths	Population	Death rate per 1000
Featherston Military Camp	164	7999	20.5
New Plymouth	40	8704	4.59
Hastings	73	7918	9.21

Figure 3.4: Outbreak duration after the first death and to the last death for Featherston Military Camp, and the towns of New Plymouth and Hastings



DISCUSSION

LENGTH OF OUTBREAK AT THE CAMP

The outbreak has previously been recorded as occurring during November. Records show that 46 personnel were infected with influenza during the closing days of October, and that the last death occurred on 11 December. Therefore we defined the timeframe as being 28 October to 11 December 1918. The first date of death provided by the PMO was 22 October, which he attributed to a pneumonic complication of "ordinary" influenza. The next recorded death, and the first that the PMO attributed to the epidemic, occurred on 7 November. We acknowledged his account and based our analysis upon the assumption that the influenza epidemic in Featherston Military Camp started about 28 October 1918. There are 12 deaths for which a date cannot be determined, but as the dates of the first and last deaths have been stated in the epidemic report submitted by the PMO, we are confident that the incomplete date information will not affect our timeframe definition.

LIVING ARRANGEMENTS

We found data that contradicts our initial hypothesis that those of a higher rank would be at a lower risk of dying from influenza. Our initial assumption was based on the fact that the living arrangements of the Privates made them more susceptible to overcrowding. They were accommodated in huts that had a capacity of 25 men. Officers, by comparison, were billeted at 10 per hut. However, the extent of the effects of close quarter living arrangements could not be estimated, as the data were not adequate. Some of the staff were billeted in training huts, some in staff huts and as the hut camp was operating beyond its capacity, over 2500 soldiers were living in tents. Although a map of the camp was obtained, the number of tents and occupants per tent could not be found.

Allocating the ranks into the three categories therefore came to be defined by sleeping quarters as well as our understanding of their status and roles in the camp. This resulted in the Corporals and Lance Corporals, who are Junior Non-Commissioned Officers, being analysed with the Privates. This meant that the independent influences of rank and sleeping arrangements became blurred and our research moved away from analysing close quarter living arrangements and focussed on occupation/rank within

the camp and whether this could be used as a sample for comparison with towns or similar populations.

Data analysis demonstrated a protective effect for being in the "Privates" group, and that being a member of either of the other two groups was a disadvantage. The officers and the NCOs were nearly three times more likely to die from influenza than the privates. There were no differences between officers and NCOs. Possible reasons for the apparent increased mortality amongst the higher ranks include:

- Medical personnel, who with their occupation are more likely to be exposed to the disease, are therefore more likely to die.

- That the finding was not a true association and that our estimate of the relevant denominator populations were inaccurate (see below).

EXTERNAL COMPARISONS

It is important to note the population being described in this project is unique in that the majority were male and between the ages of 20-40 years. This therefore makes it difficult to compare with the wider community. With the lack of information regarding age, gender, socioeconomic status and privileges, these factors could not be adjusted for.

When comparing the outbreak duration in Featherston Camp with that of towns of similar population numbers such as New Plymouth and Hastings, we found that the Featherston Camp displays a higher mortality rate of almost four times that of New Plymouth and almost double that of Hastings. Featherston had the shortest outbreak duration of 35 days after the first death occurred, as opposed to 40 days in New Plymouth and 38 days in Hastings. Featherston also demonstrated a shorter period before mortality peaked, which suggests the rapid spread of infection. Although all known measures of outbreak prevention were reinforced in Featherston Camp, this finding is consistent with the fact that military camps were particularly severely affected by the 1918 flu pandemic relative to towns in New Zealand.

An attempt was made to further compare mortality figures in these places. We tried to apply epidemic curves for the mortality distribution to compare these towns with

Featherston Camp. However a lack of daily mortality data, as well as not knowing the peak number of deaths for the towns was a limiting factor.

LIMITING FACTORS IN THIS ANALYSIS

Assumptions

As noted in the methods section, the numerator information was incomplete. There were 10 casualties for whom there was no information concerning either rank or date of death. As the PMO noted the deaths of Medical Corps Officers by name, but only mentioned the loss of other members of his team, we assumed that at least some of those 10 would be medics below the rank of Sergeant. As there were no victims with Maori names buried locally we also assumed that some of the unknowns could be Maori who travelled home for their tangi (Maori funeral rites), and that these soldiers would most likely be below the rank of Sergeant as well. If these assumptions were incorrect, they would have little impact on the rates for the "Privates" group, but the affect upon the other two groups could be significant.

The denominator was also incomplete. Camp returns filed in January 1918 provided us with an estimate of the camp permanent staff by rank and duty. Records of the capacity of the main hut camp gave us the population of trainees and corporals in the huts. The shortfall between these totals and the given camp total were the men in the tents for whom we had no data, which needed an estimate. Despite taking considerable care over this estimate, this could cause an inaccurate denominator to have been calculated.

The total camp population at the time has been variously quoted as either 7800 or 8000 depending on the various sources. The mortality figures also vary by author. It therefore became necessary for us to justify the choice of a dataset upon which to base our analysis. The dataset we chose was the epidemic report submitted by the PMO. Using his mortality figure of 164 for the period concerned, and his figure of 8000 for the camp population, we arrived at our mortality rate of 20.5 per thousand. This is markedly above that of the general population, but less than the previously quoted rate of 22.6 per thousand.

Interpretation

Careful consideration must be taken when interpreting the findings from this study. This was not an isolated population and there are many possible explanations to account for the higher incidence. The high number of recruits entering the camp from many parts of the country may have contributed to increased exposure within the camp. The PMO suggested that a recent batch of recruits from Auckland might have been responsible for introducing the second wave of influenza to the camp.

Overcrowding appears to have been a likely contributor to the spread of disease in the camp. The sudden increase in morbidity followed a period of weekend leave. Travel to and from the camp was in crowded troop trains that were already a notorious cause for the spread of disease. Within the camp the men lived in very close quarters: the camp covered just 70 acres. The hut camp had a capacity of 4660 trainees of all ranks and a capacity of approximately 835 permanent staff. The tented overflow was accommodated over the road, as well as at Tauherenikau, and Papawai. This went against the recommendations from the government inquiry following the 1915 flu epidemic concerning the prevention of respiratory disease. Limited data made it impossible to conduct a detailed analysis of the effects of close quarter living.

The Featherston experience was also influenced by a severe storm that struck Wairarapa on 7 November. In his report the PMO commented that in his opinion the effects of influenza would not have been so devastating had that weather event not occurred. The storm flattened many tents and therefore placed additional stresses on accommodating men in huts that were already full, and that were also starting to be requisitioned as overflow wards to the main camp hospital. Although we could not account for density, the significance of overcrowding cannot be ignored.

No conclusions could be reached concerning the relationship between rank and morbidity as no data was found. Explanations regards the higher risk of mortality by rank is difficult to substantiate without the morbidity data. For example some army personnel i.e. medical officers would be more exposed and therefore at an increased risk, this could result in higher morbidity and mortality rate for that group.

Implications of the Featherston Camp Experience to Understanding Pandemic Influenza Epidemiology

Analysis of morbidity showed that the Camp influenza outbreak was brief and brutal. Daily incidence was greater than that of the general population and the ‘flu took hold much quicker than was the case in civilian communities. Whilst we cannot draw any inferences of incidence of infection with respect to rank and the role of close quarter living, it is clear that close quarter living does not influence mortality in the way that we had previously thought likely. Certainly the group with the highest mortality were the Privates, but they also comprised by far and away the largest group. This group’s mortality rate was actually considerably lower than those of the other 2 groups. It would be useful to investigate the dynamics of life within institutions in New Zealand, be they military barracks, schools, or prisons, etc. as there seem to be other factors at play.

The group that appeared to suffer the highest rate of mortality were the Officers of the New Zealand Medical Corp. There were 24 officers listed as being in either the Medical Corps or the Dental Corps. Unfortunately we do not have a breakdown between the two groups, but the death of three Officers has implications for the management of patients in epidemics of infectious disease, as well as for how we as a society care for the welfare of those upon whom we rely in such situations. This in turn has implications for the planning of epidemic management and healthcare prioritisation.

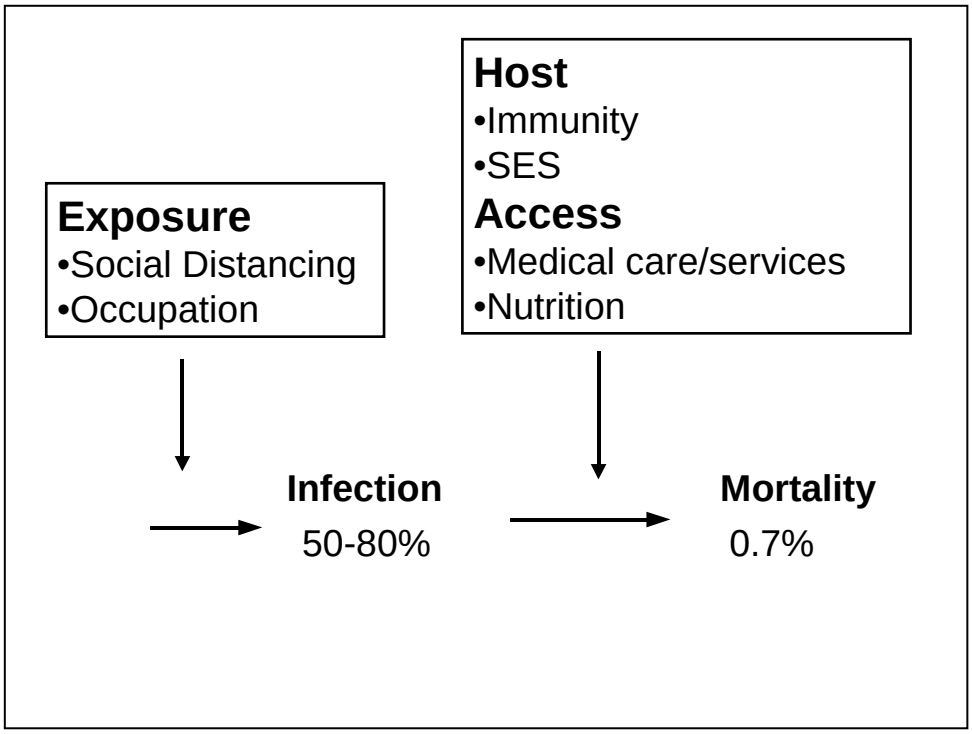
DISCUSSION

Given the current threat of an influenza pandemic in humans (possibly derived from the current avian influenza pandemic), this research provides insights into how a major influenza pandemic could spread in New Zealand. This report offers, for the first time in this country, statistical analysis of mortality rates from influenza during the 1918 pandemic. It focuses on geographical population and population density, occupation as a measure of socioeconomic status, and attempts to understand the pattern of spread of influenza in an institutionalised population using the Featherston military camp as an example. These important lessons from history may give us insights that are useful today, as well as recommendations for future research.

The 1916 census was utilised in this study to provide denominator data. Thus, there were inaccuracies that cannot be corrected due to the time between the collection of the denominator and the mortality numerator data. This however, could not be avoided. It is unfortunate that the 1916 census did not provide us with accurate or complete data on the Maori population. As such, we were not able to analyse ethnic susceptibility to mortality from influenza. We were, however, able to utilise the remaining data to draw some important conclusions.

Our analysis hypothesises that the observed patterns of pandemic influenza mortality was a product of two steps: infection and illness (which was largely unrecorded except in the Featherston Camp outbreak) followed by death in a minority of cases (which was generally well recorded, at least for Pakeha New Zealanders). Each of these steps had separate risk factors (Figure 4.1) which may potentially have had contradictory effects. For example, living in a large city might have increased the risk of infection from contact with large numbers of people, but decreased the risk of mortality because of better health care.

Figure 4.1: Factors hypothesised to have contributed to observed influenza morbidity and mortality in the 1918 pandemic



The influenza pandemic first reached New Zealand in Auckland in October 1918. Two weeks later, likely via train and sea transport systems, the pandemic simultaneously hit the other major cities of New Zealand, Wellington, Christchurch and Dunedin. Interestingly, the North Island had higher mortality (6.2 per 1000) than the South Island (5.5 per 1000; $p<0.001$). This may have been due to a higher population density, which is still apparent today, or that the peak of the epidemic occurred two weeks later in South Island, (whereby action plans may have already been implemented to help curb mortality). In addition, those living in cities and towns had increased mortality rates due to influenza when compared with counties (rate ratio = 2.13 [95% CI = 2.00-2.27]; $p<0.001$). These findings are not adjusted for age and sex, however it is expected that following these adjustments, the findings may be further enhanced as data from the 1916 census show that counties had a higher proportion of males, who are known to have had a higher mortality rate than females (for the national European population of New Zealand during this pandemic). These findings are supported by the comparison between North and South Islands, in suggesting that a higher population may result in increased deaths from influenza. However, we also demonstrated that larger towns and cities had a lower mortality rate when compared to small towns of a population less than 2000. We suggest that larger population centres may have provided their citizens with better access to medical or

community care, offering them a degree of protection from death. With these findings in mind, it could be suggested that higher density populations may increase the mortality rate from influenza, due to increased exposure. Our data shows that in the lowest population density quintile (for counties), the mortality rate is well below the national rate, and significantly different from all other density quintiles. Thus it could be suggested that the most remote areas of New Zealand may have offered some protection from mortality due to influenza, and that there may be a threshold at which the population density offers no further protective benefits. Unfortunately we could not demonstrate a relationship between number of occupants per dwelling and mortality rate due to the small amount of variation in exposure (occupants per dwelling) with large variation in outcome (mortality rate).

While many have labelled influenza a ‘classless’ illness, it has been previously suggested that social class may influence the mortality rates [7]. Our data shows that

those in a lower socioeconomic groupings, as determined by occupation status, had increased mortality rates when compared with those in professional employment. We did however, find some exceptions to this rule. For example, primary producers, who were considered to be low socioeconomic status were relatively protected, potentially due to living and working in a country area (with the associated protection provided by rurality). Finally, being a dependent offered protection from the pandemic, potentially due to remaining at home and therefore having reduced exposure to the virus.

In the institution we studied (the Featherston Military Camp), the peak wave of morbidity from the influenza virus was followed by a mortality wave peak approximately 8 days later. The length of the epidemic in this camp was relatively short, with peak morbidity at day 11 and mortality at day 19. While we compared the camp to other towns in New Zealand of the same population, these results must be viewed with caution, as the military camp was comprised predominantly of men aged between 20-45, the most susceptible age/sex group to mortality in this influenza pandemic. It would seem that while the peak mortality appeared earlier in the camp

outbreak when compared to both Hastings and New Plymouth, the outbreak was of shorter duration in the camp when measured from the first death compared with community exposure. Featherston Camp showed mortality rates almost four times higher than that of New Plymouth and almost two times higher than that of Hastings, which probably reflects the fast and severe effect of the outbreak on an institutional population of this type. In addition to these comparisons, we also investigated mortality rates within the Featherston Camp. We were surprised by our findings that privates had a lower mortality rate when compared with officers, and non commissioned officers. We hypothesised that privates would be living in poorer conditions than officers, thus representing a lower socioeconomic status, and would thereby have a higher mortality rate when compared to officers believed to be of higher socioeconomic status. Instead, our data reflect the opposite, and we suggest this may be an example of officers providing a medical or supervisory role during the outbreak, and thus increasing their exposure and consequently mortality rate.

While New Zealand is a relatively small nation, we will always have quite remote areas of low population density. In these regions, it could be expected that should a new influenza epidemic occur, that these regions might offer some protection for their residents. Smaller rural locations may also experience lower mortality rates compared with larger towns and the major city centres. Additionally, as the South Island has a lower population, lower mortality rates could be observed in this region. We currently also have a large divide in socioeconomic status, where it is well established that those of a lower socioeconomic status suffer poorer health. Thus it would be expected that these groups would have an increased morbidity and mortality should a future outbreak occur. These results should therefore strengthen current plans to minimise the current deprivation gaps, thus decreasing the susceptibility of many residents of this country. In this respect, as Maori make up a larger proportion of those in disadvantaged groups and experience poorer health outcomes within the same socioeconomic groups, Maori could be further targeted to minimise inequality gaps. Our information gathered from the Featherston Military Camp also provides us with a useful tool that can be applied to today's society. In this somewhat closed population, we found that the senior officers had an increased mortality rate when compared with the privates. Thus, any socioeconomic class difference that may suggest close quarter and poorer living conditions for the privates did not result in an increased mortality

rate in that group. These data suggests a vulnerability of senior military staff which may be equated to a potential vulnerability for today's caregivers should a future outbreak occur.

KEY IMPLICATIONS

- Social distancing does provide a protective effect on mortality rates from an influenza pandemic. However larger cities may offer some protection (relative to larger towns), perhaps due to better access to medical services and organised community care.
- Lower socioeconomic status groups may experience higher mortality rates in a future influenza pandemic. In addition, occupations which involve travel or interaction with many people, may experience increased mortality rates.
- Institutional populations may be more susceptible to increased morbidity and mortality compared to the wider community. This has implications for institutional service action planning, to combat the anticipated higher disease burden among such populations.
- New Zealand's current stockpile of antivirals can supply 21% of the population. There is therefore some justification for prioritising the provision of antivirals to the population groups at increased risk of influenza mortality.

RECOMMENDATIONS FOR FURTHER RESEARCH

- Carry out an individual-record-level analysis to adjust our results for age and sex to further clarify our current findings. This could be performed by analysing the complete numerator data set [3] including individual characteristics, using denominator data from the 1916 Census (this numerator data currently resides on paper forms with Professor Rice).
- Detailed analysis of Maori data, including: mortality rates in North vs. South Islands; mortality rate in areas of high and low Maori and total population; mortality rates in areas of high and low total population density.
- Comparisons of the mortality rates in towns and counties that had exposure to railways and ports along with other potentially important factors such as demographic composition, community support and access to health services.
- Comparison of mortality rate vs. population density and mean number of occupants per dwelling for suburbs within the four main cities.
- Analysis of any data on education status (if this could be obtained).
- Analysis of the risk to health professionals. While this group may be of high socioeconomic status, that they are likely to have been at increased occupational exposure to infection.

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