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Epidemiological transition: a historical analysis of immigration patterns by country of origin (1861–1986) related to circulatory system diseases and all-cause mortality in twentieth-century Australia

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ABSTRACT

Background and objectives Circulatory system disease (CSD) patterns vary over time and between countries, related to lifestyle risk factors, associated in turn with socioeconomic circumstances. Current global CSD epidemics in developing economies are similar in scale to those observed previously in the USA and Australasia. Australia exhibits an important macroeconomic phenomenon as a rapidly transitioning economy with high immigration throughout the nineteenth and twentieth centuries. We wished to examine how that historical immigration related to CSD patterns subsequently.

Methods and setting We provide a novel empirical analysis employing census-derived place of birth by age bracket and sex from 1891 to 1986, in order to map patterns of immigration against CSD mortality rates from 1907 onwards. Age-specific generalised additive models for both CSD mortality in the general population, and all-cause mortality for the foreign-born (FB) only, from 1910 to 1980 were also devised for both males and females.

Results The percentage of FB fell from 32% in 1891 to 9.8% in 1947. Rates of CSD rose consistently, particularly from the 1940s onwards, peaked in the 1960s, then declined sharply in the 1980s and showed a strong period effect across age groups and genders. The main effects of age and census year and their interaction were highly statistically significant for CSD mortality for males ($p<0.001$, each term) and for females ($p<0.001$, each term). The main effect of age and year were statistically significant for all-cause mortality minus net migration rates for the FB females (each $p<0.001$), and for FB males, age ($p<0.001$) was significant.

Conclusions We argue our empirical calculations, supported by historical and socioepidemiological evidence, employing immigration patterns as a proxy for epidemiological transition, affirm the life course hypothesis that both early life circumstances and later life lifestyle drive CSD patterns.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ An original analysis employing census data and immigration patterns to reinterpret historical trends in circulatory system disease (CSD) in Australia.
⇒ Relevant to modern public health policy for population approaches to CSD prevention, also integrates life course and lifestyle drivers of trends.
⇒ Historical databases do not categorise either all-cause or CSD mortality according to country of origin.
⇒ However, data for foreign-born mortality were inferred using novel actuarial type calculations.
⇒ There are no second-generation data by country of origin, unlike in USA.

INTRODUCTION

Circulatory system disease (CSD) patterns, including ischaemic heart disease and stroke, are known to vary over time and between countries, related to lifestyle risk factors across the life course, associated in turn with underlying socioeconomic circumstances.[1–6] Current global epidemics in developing economies are similar in scale to those observed much earlier in the USA and Australia.[7 8] Since the mid-twentieth century in particular, international epidemiological studies have assessed how proximal lifestyle risk factors such as tobacco smoking and diet-related hypertension, obesity and lipoprotein profiles can explain the patterns of heart disease and stroke in given population.[7–13]

It is well established that in that period, patterns of heart disease were highest in countries such as the USA, Australia and Finland, intermediate in northern Europe and lowest in Mediterranean countries.[14] That pattern shifted, with steep declines observed first in the USA and Australia, followed by falls in all western countries from the early 1980s onwards.[14 15] The conventional, primarily period, explanation is that the balance of lifestyle change and introduction of rigorous clinical management protocols, including

coronary care and pharmacological management of hypertension and hypercholesterolaemia, drive these patterns.[5 7 11 16 17]

The concept of epidemiological transition posits that as countries move from patterns of poverty to patterns of affluence, changes in prevailing diseases occur, with first infectious diseases waning, followed by a rise in non-communicable diseases.[1 18 19] The American Heart Association describes four distinct stages of epidemiological transition at population level.[1]

The contemporary global burden of disease maps shows clearly the shifting patterns downwards in current western populations and upwards in parts of Asia and Africa.[20] Moreover, in a time of huge global challenge in relation to non-communicable disease and the rise of obesity in recent decades, it is necessary to understand at a mechanistic level how global epidemics occur to intervene effectively in their management.[21–24] An assessment of how historical patterns of disease occurred[4 5 7 21] can contribute to modern strategies to prevent non-communicable disease.[6]

The USA, Australia and New Zealand share an important macroeconomic phenomenon in that they were each rapidly developing transitional economies throughout the nineteenth and twentieth centuries, with large-scale immigration as a major driver of population growth and composition.[15] In both continents, the original immigrants were predominantly from Europe, initially from northern and then Central Europe, followed by Southern Europe and then latterly from Asia.[25–27] A previous analysis employing historical US census data showed a strong temporal association between the patterns of age-adjusted heart disease in both men and women and the proportion of foreign-born and of first-generation Americans between 38 and 50 years earlier.[15]

Our objective in this current analysis was to examine whether historical census immigration patterns in Australia by country or origin related to subsequent all-cause and CSD mortality patterns during the twentieth century and thereby establish if a pattern similar to that in the USA existed.

METHODS

Data

To examine the transitioning population and its subsequent pattern of CSD, we looked at both census data and epidemiological records and constructed a historical dataset based on digitised paper records. Census data on place of birth, age bracket and sex were retrieved from scanned paper copies from 1891 over the subsequent 95 years coming to 13 in total—every 10 years between 1891 and 1921, 1933, 1947, 1954 and then every 5 years from 1961 to 1986.[28] Prior to 1891, there was not a synchronised census year for all six pre-Federation colonies; however, an overall number of foreign-born residents were obtained for 1861, 1871 and 1881 (not by region).

We reconciled the country of birth data across changes of political entities into the following: Commonwealth of Australia (the six colonies/states, External Territories and New Zealand), UK and Ireland, northern and Central Europe (including Denmark, Sweden and Norway, Germany, The Netherlands, Belgium, France, Switzerland, Austria/Austria-Hungary, eastern Europe through to Russia), Southern Europe (all other countries with extensive Mediterranean sea coasts including Spain, Italy, Greece, Malta, Yugoslavia and other Balkan nations, Turkey, and Lebanon, in addition to Portugal, Romania and Bulgaria), with the Americas, Asia, Africa and Polynesia grouped into the Other group including the small number of persons unknown, or born at sea. The percentage of the total population which was born in each of the world regions was calculated. As the predominant immigrant population of Australia historically has been from the UK and Ireland, we also created an aggregated European and Other region, to include all non-UK/Irish immigration.

Age-specific and age-adjusted CSD mortality rates per 100 000 were available from 1907 to 2016 from online records of the General Record of Incidence of Mortality (GRIM; Australian Institute of Health and Welfare) book 0900.[29] The GRIM mortality information is obtained from (compulsory) death certificates, certified by a medical practitioner or coroner, and coded by the Australian Bureau of Statistics or precursors to that agency, to International Statistical Classification of Diseases and Related Health Problems (ICD). CSD currently encompasses ICD-10 codes I00-I99, including all forms of heart disease and stroke and is aligned with corresponding categories back to ICD-1 (1907–1917). Coronary heart disease deaths specifically (ICD-10 codes I20-I25) were considered but were only available starting in 1940. We obtained incidence of aggregated cerebrovascular disease (CVD) mortality from GRIM book 0904 (Australian Institute of Health and Welfare), encompassing ICD-10 codes I60 to I69. All mortality rates are age-standardised to the 2001 estimated resident Australian population.

Patient and public involvement statement

None. This is a historical analysis of archived publicly available datasets, with no patient or public involvement.

Analysis

Age-specific all-cause mortality rates for the whole Australian population are also compared with those foreign-born.[30 31] CSD mortality rates are available only for the whole Australian population, but there is justification that these are indicative of all-cause mortality rates, in the relevant period.[5 7 9 11] For each sex/age group/census year, mortality rates due to CSD in the Australian population were calculated by taking the ratio N_mort/N_total in the raw dataset.

Mortality rates for foreign-born due to circulatory disease or all causes are not readily available either, as country of origin was not recorded at death. However,

all-cause mortality rates for foreign-born were estimated from numbers of foreign-born in different age groups at different census years, using the following actuarial type/analytical method, set out in detail below. For example, let fb denote foreign-born, and taking a specific age and specific year, then

Number fb age 31 in 1948 = (Number fb age 30 in 1947) × (1− (r_1−r_2)), where r_1 is the mortality rate for fb age 30 in 1947 and r_2 is the inward migration rate for age 30 in 1947–1948. Let $r=r_1-r_2$ then we rewrite the above equation as:

$$\text{Number fb age 31 in 1948} = (\text{Number fb age 30 in 1947}) \times (1-r) \quad (1)$$

Thus, r is the mortality rate-inward migration rate for the fb age 30 in 1947.

Then

$$\text{Number fb age 32 in 1949} = (\text{Number fb age 31 in 1948}) \times (1-r) \quad (2)$$

where $r=r_1-r_2$ and r_1 is the mortality rate for fb age 31 in 1948–1949 and r_2 is the inward migration rate for age 31 in 1948–1949 and we assume these rates are unchanged from the 1947–1948 values.

Thus, by substituting equation (1) into equation (2), we have

$$\text{Number fb age 32 in 1949} = (\text{Number fb age 30 in 1947}) \times (1-r)^2 .$$

Proceeding in this way we have

$$\text{Number fb age 37 in 1954} = (\text{Number fb age 30 in 1947}) \times (1-r)^7$$

Therefore

$$(\text{Number fb age 37 in 1954}/\ \text{Number fb age 30 in 1947}) = (1-r)^7$$

and therefore

$$r = 1 - (\text{Number fb age 37 in 1954}/\text{Number fb age 30 in 1947})^{1/7} \quad (3)$$

Thus, r is the mortality rate-inward migration rate for the fb age between 30 and 36 per year in the interval 1947–1954.

r is estimated from the data by

$$r = 1 - (\text{N\_AllFB age 35} - 45 \text{ in } 1954/\ \text{N\_AllFB age } 25 - 35 \text{ in } 1947)^{1/7} \quad (4)$$

This calculation was repeated for each age group and census interval. Only raw data by age group totals are available from the censuses. Note that r can be negative if the inward migration rate exceeds the mortality rate. Thus, for all age groups, r will underestimate the mortality rates but it is used here as a proxy for the mortality rates. Essentially, equations (1)–(4) use a geometric mean of 1–r over an age group/census interval to estimate r, and this is repeated for different age groups/census intervals.

The estimates of r over time and across age groups were calculated as above. These estimates were interpolated and smoothed over all ages and census years using a tensor product smooth model[32] and are displayed in 3D plots. This is an extension of a generalised additive model (gam)[33] to three dimensions and was fitted using the mgcv package in R with the ti function. This was done separately for males and females. This smooth model was decomposed into a functional analysis of variance (ANOVA) structure with main effects of age and year and their interaction, and p values for these effects were obtained. The CSD mortality rates per 100 000 for the whole population were also fitted using a tensor smooth model and displayed in 3D plots.

Note that the raw data are very variable both by census year and age group. For example, the number of foreign-born female age group 30 in 1921 was 74 913, while it was 100 385 in 1933, and for age group 40 in 1921, it was 93 089. So, all interpolating models must be interpreted with caution.

However, model checking was carried out using the gam.check function in the mgcv package in R, and this function was also used to find the best basis dimension for the terms in the model. In additional model validation, the standard deviation (σ) of the estimated rates r from equations (1)–(4) were computed separately for males and females and the rates were then perturbed by adding a uniform random number on (0, σ) and the analysis repeated on the perturbed rates. This was repeated 20 times.

RESULTS

The exact population numbers from 1891 to 1986 from census data by region of origin are presented in table 1. This shows respectively the population numbers born in Australia or abroad or the birthplace of origin as a percentage of the total population, from 1891 onwards. There are two distinct patterns observed. In 1891 when the population numbered just over three million, the proportion foreign-born was relatively high. The contribution made to the population composition by those UK or Irish-born is initially very considerable but tails off until the 1940s. A new surge in migration beginning after World War II (WW2) is apparent, initially comprising an increase in European (mostly Southern) immigration. Asian populations in particular were the predominant group in the large 'Other' category after the 1970s.

The Australian population composition mapped against the age-adjusted CSD mortality is shown in figure 1A. This illustrates the change in the proportions of the population foreign-born by region of origin (left-hand vertical axis), alongside the change in incident mortality due to all CSD (red line, right-hand vertical axis). This shows first that the percentage of the population foreign-born was over 60% in 1861 and declined steeply over the next 80 years so that it was just 10% by the mid-1940s. Thereafter, there was again a rise in immigration, climbing to over 20% of the population in the early 1980s. The figure also shows the contribution to this pattern made by the UK/Ireland, northern and Southern European groups. The predominant early group is of UK and Irish origin, dropping steeply by the mid-1940s and plateauing at approximately 7%. The other two European groups are much smaller in proportion and reached their peak in the 1960s.

Table 1 Descriptive statistics of Australian population composition by country of birth, from census data 1891–1986

Year	UK/Ireland n (%)	Northern Europe n (%)	Southern Europe n (%)	Other place of origin n (%)	All foreign-born n (%)	Australian born n (%)	Total
1891	821 166 (26%)	75 145 (2.4%)	5307 (0.2%)	113 799 (4%)	1 015 417 (32%)	2 158 975 (68%)	3174 392
1901	679 159 (18%)	67 291 (2.0%)	7382 (0%)	111 666 (3%)	865 498 (23%)	2 908 303 (77%)	3773 801
1911	591 729 (13%)	63 594 (1.4%)	9348 (0.2%)	122 664 (3%)	787 335 (17.7%)	3 667 670 (82.3)	4455 005
1921	676 387 (12%)	52 503 (1.0%)	15 539 (0.3%)	109 118 (2%)	853 547 (15.7%)	4 581 663 (84.3%)	5435 210
1933	732 674 (11%)	48 125 (0.7%)	44 680 (0.7%)	94 860 (1%)	920 339 (13.8%)	5 726 566 (86.2%)	6646 905
1947	542 910 (7%)	50 180 (0.7%)	58 516 (0.8%)	90 995 (1%)	742 601 (9.8%)	6 835 171 (90.2%)	7577 772
1954	664 205 (7%)	294 667 (3.3%)	196 192 (2.2%)	39 756 (0%)	1 194 820 (13.4%)	7 700 064 (86.6%)	8894 884
1961	755 402 (7%)	433 999 (4.1%)	406 791 (3.9%)	178 612 (2%)	1 774 804 (16.9%)	8 729 406 (83.1%)	10 504 210
1966	908 664 (8%)	430 574 (3.7%)	554 273 (4.8%)	237 409 (2%)	2 130 920 (18.4%)	9 419 542 (81.6%)	11 550 462
1971	1 088 210 (9%)	475 095 (3.7%)	633 173 (5.0%)	382 840 (3%)	2 579 318 (20.2%)	10 176 320 (79.8%)	12 755 638
1976	1 117 600 (8%)	460 673 (3.4%)	632 550 (4.7%)	508 209 (4%)	2 719 032 (20.1%)	10 829 617 (79.9%)	13 548 649
1981	1 132 599 (8%)	471 278 (3.2%)	678 445 (4.7%)	900 143 (6%)	3 182 465 (21.8%)	11 393 865 (78.2%)	14 576 330
1986	1 127 191 (7%)	488 825 (3.1%)	662 124 (4.2%)	1 213 554 (8%)	3 491 694 (22.4%)	12 110 456 (77.6%)	15 602 150

The pattern of circulatory diseases shows a steep rise throughout the early twentieth century and reaches a plateau by 1950–1970 and then declines sharply from then onwards. By 1990, rates had fallen again to those previously seen in 1920 and continued to decline before levelling off currently.

Figure 1B shows the contrast between males and females in age-adjusted CSD mortality, this time in relation to the population foreign-born of both sexes from UK and Ireland. There is an approximate time lag of 40 years in the fall-off of immigration from UK and Ireland and that of age-adjusted CSD in the general population. Female and male rates began to diverge in the 1940s–1950s, with female rates declining earlier, and male rates not beginning to decline until the 1980s. By the late 1990s, the rates had converged again. The pattern in figure 1C showing the CVD mortality by sex aligns with this, although with negligible sex differences: an increase as the late nineteenth century immigrants reach old age, a plateau from the 1950s to 1970s, followed by steep decline thereafter.

Figure 2A, B show mortality rates due to circulatory disease in the Australian population by age group and year during the period 1910 to 1990 for males and females separately. Mortality rates increase with age as expected and can be seen from the left scales. In all age groups and in both males and females, rates of circulatory disease rose consistently, particularly from the 1940s onwards, peaked in the late 1960s and then began to decline sharply by the 1980s. An interaction effect between age and year is clearly visible, with differences between ages changing with year. The main effects of age and census year and their interaction were statistically significant in the model for males (p<0.001, each term) and for females (age, interaction, p<0.001 each term, year, p=0.01). The adjusted R^2 was 98% for the male model and 96.4% for the female model.

Figure 3A, B show all-cause mortality-net migration rates for the foreign-born only, separately for both males and females. The main effect of age and year were statistically significant in the model for females (p<0.001 for both) and the interaction term (p=0.040) was also significant. For males, the age term (p<0.001) was significant. The adjusted R^2 was 51.1% for the male model and 72.5% for the female model. No model inadequacies were found using the function gam.check. The model validation using perturbed rates found that statistical conclusions remained the same in 20 checks for males. For females, the main effects were significant in the 20 checks, while the interaction term was borderline significant (p<0.10 in 18 of 20 checks). Thus, the statistical conclusions are unchanged.

The number of non fb at each census year and age group was found by subtracting the N_AllFB from N_total. Equations (1)–(4) were then carried out for the non fb, and the models were fitted on these data. Figure 4A, B show all-cause mortality-net migration rates for the non-foreign-born only, separately for both males and females. The main effect of age was statistically significant in the model for females (p<0.001) and the interaction term (p<0.001) was also significant. For males, all three terms age, year and the interaction (p<0.001 for each) were significant. The adjusted R^2 was 71.3% for the male model and 81.1% for the female model. No model inadequacies were found using the function gam.check. The model validation using perturbed rates found that statistical conclusions remained the same in 20 checks.

DISCUSSION

In this analysis, we have examined whether the immigration pattern in Australia, as one form of proxy for epidemiological transition, related to patterns of both CSD and all-cause mortality in the population, as had been observed previously in the USA.[15] We demonstrate a temporal association of age-adjusted CSD with immigration approximately a half century earlier from predominantly the UK and Ireland, which changes as

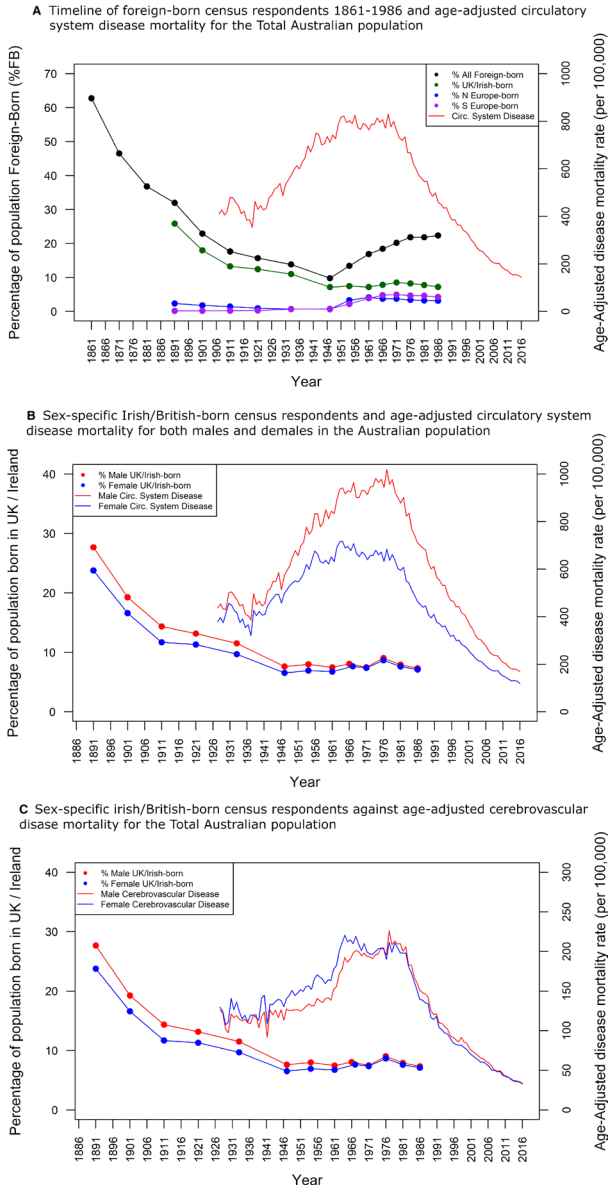

Figure 1 (A) Timeline of foreign-born census respondents 1861–1986 and age-adjusted circulatory system disease (CSD) mortality for the total Australian population. (B) Sex-specific Irish-born/British-born census respondents and age-adjusted CSD mortality for both males and females in the Australian population. (C) Sex-specific Irish-born/British-born census respondents against age-adjusted cerebrovascular disease mortality for the total Australian population.

the population stabilises economically and newer waves of immigration from lower-risk Mediterranean and later Asian countries occur.

We also show in age-specific models that assess both CSD and all-cause mortality, a strong age and period effect for the general Australian population and a highly significant interaction between age and census year in CSD mortality. Both foreign-born male and female populations showed a strong age effect for all-cause mortality, while for females the interaction term for age and year was also significant. A similar period impact is also seen for the general population less the migrant groups. These novel findings do not contradict the period impact on cardiovascular disease shown in a previous age-period-cohort analysis undertaken[5] for the general Australian population, but is unlike that analysis in showing a clear interaction effect, and for the first time looks specifically also at the foreign-born population.

STRENGTHS

This is an original analysis employing census data and immigration patterns to reinterpret historical trends in CSD in Australia. It is relevant to modern public health policy for population approaches to CSD prevention and also integrates life course and lifestyle drivers of trends. We demonstrate in essence, for Australia, similar temporal findings to those observed in the USA.

Our findings can be situated in the earlier history of that transitioning Australian development. To understand any cohort effect, we contend the historical developments influencing early childhood must be considered. Of the three classical risk factors, active individual smoking is strongly period driven,[7 9] whereas dietary intake, which drives both lipoprotein and blood pressure patterns, can be influenced both by short-term and longer-term programming factors.[6 23 24]

LIMITATIONS

We acknowledge there are clear limitations to this current analysis in that the historical databases do not categorise either all-cause or CSD mortality according to country of origin and there are no available second-generation data by country of origin. However, data for foreign-born were inferred using actuarial type calculations, and a three-dimensional model, representing a novel approach and model validation, was carried out with unchanged conclusions.

One would expect an impact on foreign-born, but also their children born into a transitioning economic environment, especially urban disadvantage. In the previous analysis in the USA, data were available both for foreign-born and native-born of foreign parents and that afforded a fit which explained both the rise and fall of the epidemic;[15] however, we do not have second-generation data in this analysis.

DEMOGRAPHIC TRANSITION

The actuarial approach we have taken is in keeping with the demographic evidence base. Borrie provided a comprehensive analysis of the transitioning Australian and New Zealand populations over a 200-year period from 1788 to 1988.[34] Those who migrated into Australia from Europe[35–37] included First Fleeter and other colonists, transported convicts and assisted passage individuals who were supported to emigrate from their countries of origin. The Irish and British were the predominant early migrating group. A total of over 800 000 immigrants came

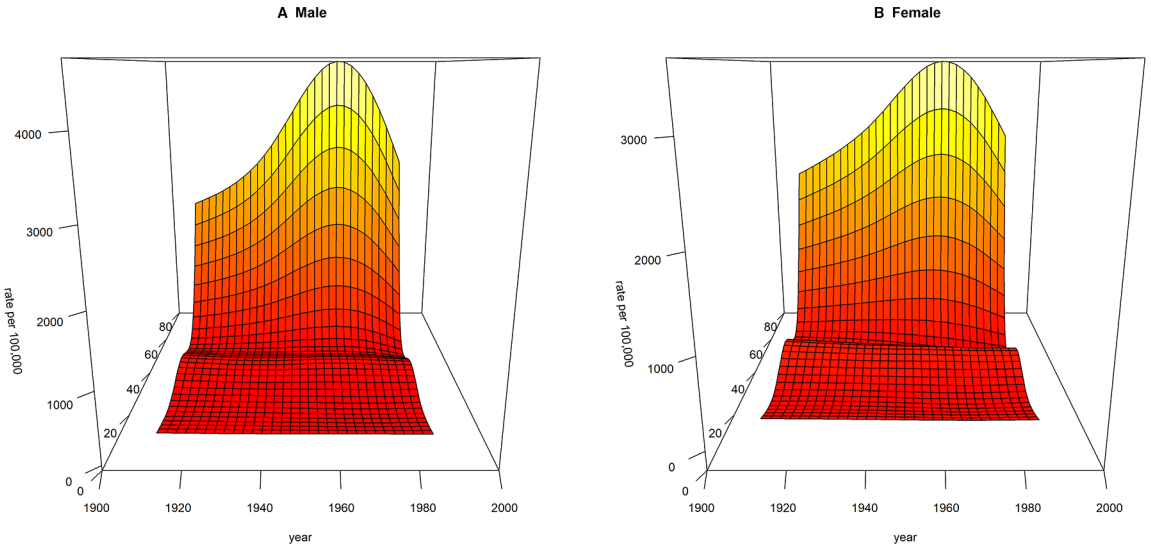

Figure 2 Circulatory system disease mortality rates per 100 000 by age and year in the general Australian population, (A) males and (B) females. The unlabelled axis is age in both plots.

also through Government Assistance programmes established 1861–1865 to 1900, tapering off sharply during that time. Irish-assisted emigration to Australia was related to workhouse programmes established during and after the Great Irish famine.[36 37] The impact of historical circumstances, including the famine in Ireland in the 1840s, has been examined previously in an Irish context, both at home and in relation to its huge emigrant diaspora in the USA.[38 39] An analysis of birthplace of all brides marrying in Australia between 1908 and 1979 shows the transition to Australian-born mothers almost fully completed by 1979[34] and family size fell from 5.6 to 2.8.

The salmon bias evaluated in other period migrations suggests that older emigrants with established illness returning home might impact mortality.[40] There is no information on those who might have left Australia in the electronic databases, though our calculations are based on net migration between census points which include both incoming and outgoing migrants as estimated at the time. Borrie[34] reports some such movement, particularly younger, working males who followed employment across Australia after the goldrush period, but because of the distance from Europe and the various costly transportation and assisted passage schemes, returning numbers to country of origin would have been limited in scale. There was no accurate measure of settler flows until after WW2. Until then, according to Borrie, 'arrivals minus departures' between 1860 and 1986 showed a net positive migration of 4.77 million inwards.[34]

Whitewell *et al* conducted a detailed economic and public health review of Australia from 1860 to 1940 which illustrates how and when population changes occurred which might have influenced longer-term health outcomes into the twentieth century.[41] Height, as a proxy measure for public health impact of standards of living, was initially high, dipped in the mid-nineteenth century

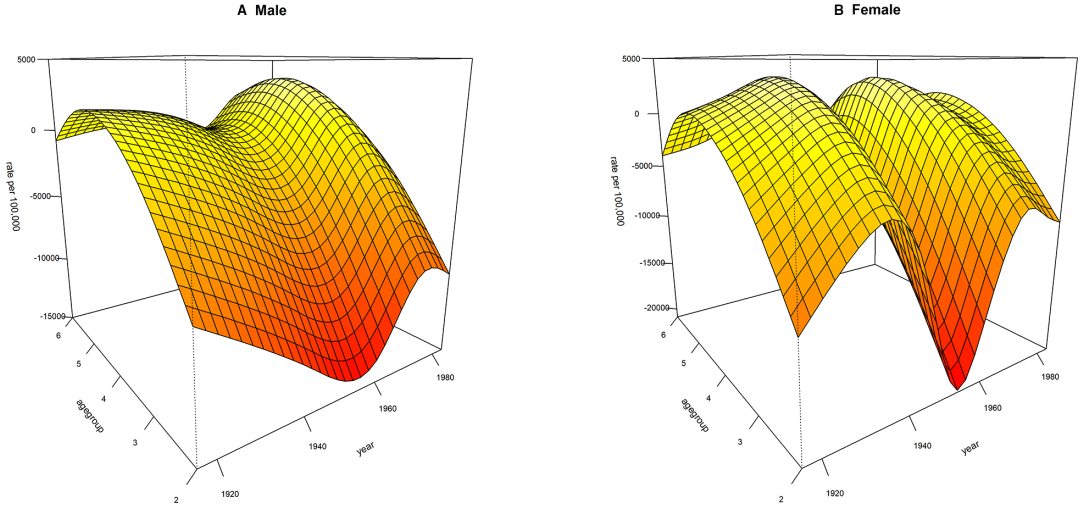

Figure 3 All-cause mortality-net migration rates per 100 000 by age group and year for Australian foreign-born population, (A) males, (B) females. Age groups 2–6 correspond to age midpoints 20, 30,40, 50 and 60 years old, respectively. Both plots have been rotated with an angle of −35 degrees to permit a better view.

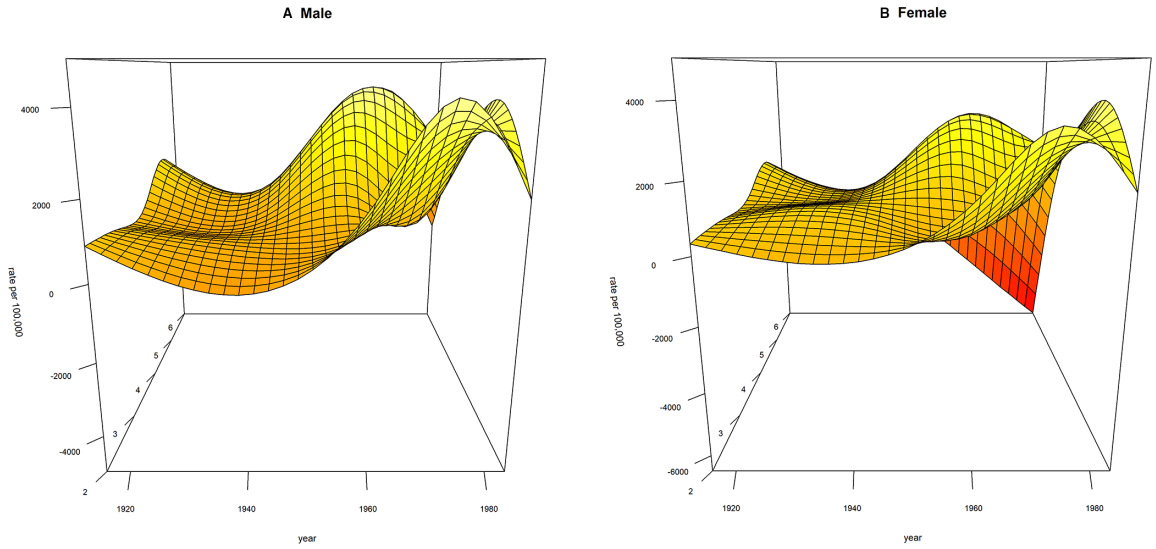

Figure 4 All-cause mortality-net migration rates per 100 000 by age group and year for the Australian non-foreign-born population, (A) males, (B) females. Age groups 2–6 correspond to age midpoints 20, 30, 40, 50 and 60 years old, respectively.

and then rose again from the late 1890s to 1918. From 1890 onwards, the sanitary conditions in the booming cities, which initially made for an unhealthy environment in which children were especially vulnerable, greatly improved. Inwood *et al* show an association between growing incomes and height in Tasmania related to a decline in food cost and increased per capita domestic product.[42] Cumpston[25] documented the improvements in public health in Australia that would translate into reduced susceptibility to strokes and heart disease in these individuals as older adults. Taylor *et al* reviewed both all-cause mortality in Australia from 1788 to 1990[43] and more recent cause-specific mortality from 1907 to 1990.[44] They showed a precipitous fall in infant mortality from 1900, similar to European countries, but then a stagnation during the mid-twentieth century, attributable to the epidemic in CSD mortality.

There is now a strong body of recently assembled historical retrospective cohort studies that afford an opportunity to review social and economic circumstances in the late nineteenth and early twentieth centuries when Australia was becoming established as a modern economy and when cardiovascular diseases incidence and mortality were rising.[45–50] McCalman *et al* linked medically validated morbidity and mortality records to original maternity records from the Melbourne lying-in hospital[45 46] showing expected demographic predictors of infant mortality. These included reduced odds of that outcome if of a higher social group and of Australian birth and higher odds if a first-born child, younger mother, preterm and of relatively lower birth weight.[46] Early results from the Victoria Diggers to Veterans longitudinal study of World War I enlisters showed a strong social gradient according to military rank for height, and among survivors to 1922, farmers were significantly less likely to die compared with others.[50]

LIFESTYLE HYPOTHESIS

As in the USA, there was considerable research interest in Australia and New Zealand in the aetiology of non-communicable disease from the mid-1970s onwards.[51–59] Seminal epidemiological analyses document the rising rates of smoking in the population and changes in food availability, exposing a transitioning population to prevailing adverse lifestyle.[7 9] McMichael was an early proponent that general population drivers explained the rise in cardiovascular diseases including food supply factors, underpinned by industrialisation.[59]

Those studies at the height of the epidemic focused not primarily on the early British migrants but on the latterly arrived mainly Southern European immigrants who initially carried the reduced risk associated with their Mediterranean lifestyle into a now economically stable Australian society (online supplemental table 1). Italian and Greek immigrants to cities like Melbourne and Sydney initially had lower blood pressure and lipid profiles than their Australian-born contemporary neighbours but had higher risk factor profiles than their home countries.[51–54] The classical healthy migrant hypothesis posits that immigrants move from low to intermediate to high risk as they transition into a new environment[60] though migrants usually form a relatively small part of the population, which is more typical of the later immigration patterns seen since the mid-twentieth century in Australia. The post-WW2 immigrants assume the risk of the prevailing environment as they enter middle and old age.

The observed differences in mortality rates between men and women[3–5] have been variously attributed to a biological female hormonal advantage and less adverse lifestyle, but this difference remains unexplained.[21] It is likely that the effect of smoking is underestimated in these early epidemiological analyses. A key factor, active

smoking in the post-WW2 period, was extraordinarily high, particularly in men in both Europe, the USA and Australia.[61 62] Exposure to high levels of unrestricted active smoking would increase the risk of passive smoke exposure in public places and in households and transport, as with contemporary data.[63]

AN INTEGRATED APPROACH

The epidemiological focus has moved more recently to a more integrated life course explanation for development of adult non-communicable disease that takes account of early life exposures as well as personal health behaviours.[6 14 21–23] Policy should be directed at underlying drivers of population health as well as individual risk factor modification, as exemplified by current guidelines for management of hypertension.[6] Current rates of CSDs are now much higher in Aboriginal citizens.[64] Emerging contemporary data from newer studies support the life course hypothesis also.[65–69] Data from a historical New Zealand cohort have modelled the significant effects of birth weight on subsequent adult blood pressure.[68]

In conclusion, this analysis provides a contextual reassessment of the historical drivers of the patterns of CSD in Australia. These different immigrant groups all experienced a period effect in relation to CSD, but also brought with them cohort effects of differing magnitude, which impacted CSD rates in the general population. These findings may be particularly relevant to public health policy planning for contemporary movements of peoples.

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