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Migration and Pacific Mortality: Estimating Migration Effects on Pacific Mortality Rates Using Bayesian Models

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Abstract Pacific people living in New Zealand have higher mortality rates than New Zealand residents of European/Other ethnicity. The aim of this paper is to see whether Pacific mortality rates vary by natality and duration of residence. We used linked census-mortality information for 25- to 74-year-olds in the 2001 census followed for up to three years. Hierarchical Bayesian modeling provided a means of handling sparse data. Posterior mortality rates were directly age-standardized. We found little evidence of mortality differences between the overseas-born and the New Zealand–born for all-cause, cancer, and cardiovascular disease (CVD) mortality. However, we found evidence for lower all-cause (and possibly cancer and CVD) mortality rates for Pacific migrants resident in New Zealand for less than 25 years relative to those resident for more than 25 years. This result may arise from a combination of processes operating over time, including health selection effects from variations in New Zealand's immigration policy, the location of Pacific migrants within the social, political, and cultural environment of the host community, and health impacts of the host culture. We could not determine the relative importance of these processes, but identifying the (modifiable) drivers of the inferred long-term decline in health of the overseas-born Pacific population relative to more-recent Pacific migrants is important to Pacific communities and from a national health and policy perspective.

Keywords New Zealand · Pacific migrants · Mortality · Hierarchical Bayesian model · Duration of residence

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Introduction

Pacific peoples have been part of New Zealand society for over 100 years, and the country has enjoyed constitutional relationships with the Cook Islands, Niue, Tokelau, and (Western) Samoa since early last century (King 2004). Pacific migrants and their descendants have become a significant minority population in New Zealand and will constitute about 10 % of the national population by 2026 (Statistics New Zealand 2010). Thus, Pacific people will play an increasingly important role in determining the national health profile and demands on the New Zealand health system. However, because the Pacific population is small and relatively young, previous research into the health of Pacific New Zealanders has been hampered by sparse data. To reduce the impact of data sparseness, we used a Hierarchical Bayesian shrinkage estimator and an extensive mortality data set to investigate the health of New Zealand–born and overseas-born Pacific people, an approach that is applicable to analyses of other migrant groups for which sparse data is problematic.

The demographic characteristics of Pacific people in New Zealand today reflect distinct migration histories. The Pacific population of the country grew slowly until 1945, when the population stood at just over 2,000 (0.1 % of the national population). Labor shortages after World War II and during New Zealand's economic expansion in the 1960s and early 1970s resulted in increased migration from Pacific countries. A desire to control the flow of Pacific migrants inspired changes in immigration policy: for example, while Cook Islanders and Niueans (since 1901) and Tokelauans (since 1916) are considered New Zealand citizens, the Western Samoan Quota scheme was established to facilitate and regulate migration from Samoa (Appleyard and Stahl 1995; Beaglehole 2007). However, the demand for unskilled labor also determined how strictly immigration policies were enforced. In effect, when labor was short, the migration of Pacific people to New Zealand was relatively unrestricted (Beaglehole 2007). This resulted in a significant increase in the number of migrants from the Pacific Islands—mainly Samoa, Tonga, the Cook Islands, Niue, and Tokelau—and therefore in the number of Pacific people living in New Zealand (Cook et al. 1999). Following steady migration-driven growth until the early 1970s, the Pacific population of New Zealand reached approximately 65,000 people, or 2 % of the national population, by the 1976 census (Statistics New Zealand and the Ministry of Pacific Island Affairs 2010).

Following recession and a change in New Zealand's economic fortunes, a major review of immigration policy in 1974 led to the tightening of immigration policy for Pacific migrant groups other than those from the Cook Islands, Niue, and Tokelau, who were still considered New Zealand citizens. As a result, net migration from the Pacific region slowed, and it even became negative during the early 1980s (Cook et al. 1999). However, permanent or long-term migration from the Pacific increased significantly after major changes in immigration policy in 1986, which saw an emphasis on nationality and ethnic origin as the basis for admission replaced by educational, business, professional, age, and asset requirements. A military coup in Fiji in 1987 and growing demand for migrants in the late 1980s also (positively) impacted Pacific migration.

The New Zealand economy was drastically restructured in the late 1980s, causing a significant decline in unskilled and semi-skilled employment by the early 1990s.

Because Pacific employment was concentrated in these industries, a net migration loss to Polynesian countries followed. However, by 2001, Pacific people had found new ways to gain residency as New Zealand adopted more flexible rules relating to changes in visa/permit status. The overall result of these events is reflected in the following estimates (from Bedford 2007) for the number of long-term or permanent migrants of Pacific nationals: 10,281 (1986–1991), 3,732 (1991–1996), 11,114 (1996–2001), and 15,898 (2001–2006). By the 2006 census, the Pacific population in New Zealand numbered approximately 266,000, or 6.9 % of the national population (Statistics New Zealand and the Ministry of Pacific Island Affairs 2010). The five largest Pacific groups resident in New Zealand at this time were Samoan (131,103), Cook Island (58,008), Tongan (50,481), Niuean (22,476), and Fijian (9,861).

In numerical terms, though, the contribution of migration to New Zealand's Pacific population is relatively minor: since the late 1970s, less than 20 % of its growth has been attributable to immigration (Cook et al. 1999). As natural increase has taken over from migration as the main driver of population growth, the proportion of Pacific people born in New Zealand has steadily increased, reaching over 60 % by the 2006 census (Statistics New Zealand and the Ministry of Pacific Island Affairs 2010). In consequence, the New Zealand–born Pacific population has a more youthful age structure than the overseas-born Pacific population (Ministry of Pacific Island Affairs 2011b). The Pacific population is also diverse, with more than 20 ethnic communities, each with its own language, distinctive culture, and history of settlement in New Zealand (Ministry of Pacific Island Affairs 2011b). Linking this ethnic diversity, however, is a fair degree of shared ancestry and customs, as well as the common experience of migration, acculturation, and discrimination.

Migration has long been linked to health. For example, many studies from North America (Gushulak 2007; Hummer et al. 1999; Hyman 2001; Markides and Eschbach 2005; Morales et al. 2002; Singh and Siahpush 2001, 2002), Europe (Anson 2004; Deboosere and Gadeyne 2005; Williams et al. 1997), and Australia (Kouris-Blazos 2002; Strong et al. 1998; Taylor et al. 1999) have shown that immigrants to a new country are often in better health upon arrival in the destination country than their native-born counterparts, despite (often) lower socioeconomic status that might otherwise suggest poorer health profiles. Over time, however, the benefits of this positive health selection disappear, resulting in lower health status among long-standing migrants and their descendants than among new migrants (Gushulak 2007; Hyman 2001; Morales et al. 2002). A migrant health advantage does not hold for all populations and health outcomes, however. Some studies have shown that immigrants arrive in their host countries with a health disadvantage compared with the majority population in the host country, implying a higher risk of disease in their country of origin and the absence of any healthy migrant effect (Albin et al. 2005; Gadd et al. 2003; Harding et al. 2008; Jamrozik et al. 2001).

Several explanations exist for the ostensibly better health of new migrants. The healthy migrant effect provides one such explanation. Specifically, it posits that immigrants do not constitute a random sample of the source country population but are positively selected on health characteristics and so are healthier on average than those remaining in their country of origin. This selection of healthy persons from the source country could be due to the requirement that potential migrants undergo

medical screening (direct selection), or from immigration policies favoring tertiary education, occupational skills, and wealth (indirect selection). The extent of health selection for Pacific migration to New Zealand has differed among Pacific ethnic groups. For example, people from the Cook Islands and Niue—with constitutional rights to settle in New Zealand—have often experienced less health selection than those from Tonga or Samoa, though this difference in health selection "pressure" has varied over time. We return to this point in the discussion.

Complementing this "healthy immigrant" effect may be a corresponding "unhealthy emigrant" effect. This hypothesis has been proposed for Hispanic people in the United States, who have better-than-expected health and mortality compared with non-Hispanic whites despite having lower socioeconomic status. The theory is that some migrants who become (chronically) ill return to their home (source) country to die and are thus not counted in the mortality rate of the destination country (Abraido-Lanza et al. 1999; Franzini et al. 2001). Many factors could affect return migration: for example, distance from the country of origin, ease of return, eligibility for superannuation, and access to health care in the country of origin. Moreover, the impact of any "unhealthy emigrant" effect may be declining for the Pacific population in New Zealand as the proportion of the population that is native-born increases.

In the putative form of the healthy migrant effect, the relative health benefit from the initial selection process is highest at the time of arrival and erodes thereafter as migrant health status is influenced by conditions in the new country (acculturation). In reality, acculturation can have a positive or a negative impact on health, depending on the style of acculturation (Berry 1990), differences in health between the source and destination countries, and the strength of health selection for the particular migrant population.

Migrants are also vulnerable to discrimination because of immigrant status and/or ethnic origin (McKay et al. 2006). Discrimination is a determinant of an individual's state of health, which in turn is linked to social structure and hierarchy, socioeconomic class, gender, and ethnic group (Commission on Social Determinants of Health 2008). Discrimination can have direct adverse effects on health, or it can impact health through its relation to access to health care services (Gee et al. 2006; Williams and Mohammed 2009). All migrants are at some risk of experiencing discrimination, but characteristics such as belonging to a visible minority may place such migrant communities at additional risk (Williams and Mohammed 2009). Thus, the health status of the migrant population may be improved or disadvantaged relative to their counterfactual health status (i.e., had they not migrated) as a result of multiple processes working in different directions.

A full explanation for health inequalities between Pacific and non-Pacific New Zealanders would require consideration of all three migration-related processes (selection, acculturation, and discrimination) as well as New Zealand–specific factors e.g., differential socioeconomic position by ethnicity, or biological susceptibility to some diseases. However, focusing just on migration, it is difficult to estimate the independent effects of health selection for migration and return migration, the waning of the consequent health benefits (should they exist) over time, and the health impacts of acculturation, as well as the experience of interpersonal and systemic racial discrimination in the destination country. Nevertheless, by examining whether mortality rates of overseas-born Pacific people differ from mortality rates of their New

Zealand–born counterparts and by determining whether the mortality rates change as duration of residence increases, we can better understand the net effect of the factors that contribute to the health of Pacific communities. Duration of residence (0–9 years, 10–24 years, and 25 years or longer) is used here as a proxy measure to capture the net result of the waning of health selection effects, the effects of acculturation, and exposure to racial discrimination on the health of first-generation Pacific New Zealanders. We do not attempt to separately distinguish the effects of age, age at migration, and duration of residence because of the linear relationship between them.

Often, new migrants have low income relative to long-standing migrants who have had the opportunity to find better-paying jobs and become more settled. If improving income also reduces mortality, then lower mortality rates among the most recent migrants will become even more apparent after adjusting for income. We adjust for income, hypothesizing that mortality differences between new and long-standing migrants would increase if there was a significant income-health relationship for Pacific migrants.

As noted earlier, previous research into Pacific health has often been hampered by the small-cell (sparse data) problem: mortality is an uncommon outcome, and Pacific populations in New Zealand are small and relatively young. For this reason, the migrant status of Pacific peoples as a source of variation in health status and health inequalities has generally received limited attention. However, Hajat et al. (2010) used pooled data from the 1996–1999 and 2001–2004 New Zealand Census Mortality Study (NZCMS) to investigate all-cause mortality differences among New Zealand–born and overseas-born people of Asian, Pacific, and European/Other (non-Māori, non-Pacific, non-Asian) ethnicities. The main focus of that study was duration of residence (DoR), classified as less than 9 years, 10–19 years, 20–34 years, and 35 or more years. For overseas-born Pacific people, these authors found no statistically significant mortality differences by DoR relative to their New Zealand–born counterparts. Neither did they find a statistically significant difference in mortality between overseas-born and New Zealand–born Pacific subgroups. After considering several potential data biases, Hajat et al. concluded that there was no evidence for health selection for Pacific peoples migrating to New Zealand. However, because of the small-cell problem, they were unable to examine mortality effects by Pacific ethnicity or cause of death.

The current study advances the work of Hajat et al. in several respects. First, we use a Hierarchical Bayesian (HB) shrinkage estimator (Richardson et al. 2009) to calculate standardized posterior mortality rates: posterior mortality rates are "shrunk" toward a prior covariate structure, an approach that has significant advantages for the small-cell problem (Young et al. 2006). Other advantages include protection against model misspecification and overdispersion (extra-Poisson variability) that are common in observational data, good out-of-sample predictive capabilities (Gelman et al. 2004), realistic estimates of uncertainty without additional assumptions of asymptotic normality (Greenland 2008), and a consistent framework for realistically complex models. Thus, although we do not explicitly include measurement error, the model could be extended to include Bayesian sensitivity approaches, which would allow estimates of bias from this source. Second, in addition to all-cause mortality, we look at mortality differences for Pacific people by cardiovascular and cancer causes of death. Third, we use only the 2001–2004 NZCMS data set, reducing concerns about temporal effects. Fourth, we investigate the role of income as a potential mediator between duration of residence (or natality) and mortality.

In this article, we present aggregated posterior standardized mortality rates of Pacific people by migrant status in two ways. First we compare mortality rates by natality, specifically rates for those who are New Zealand–born (NZB) versus those who are overseas-born (OSB). Second, we compare DoR effects within the OSB population. We use linked census-mortality data for 2001–2004, thereby reducing the problems of a noncomparable collection of Pacific ethnic groups between census and mortality data (i.e., numerator-denominator bias; Tan et al. 2010). Note that DoR periods 0–9 years, 10–24 years, and 25 years or longer correspond to arrival times after 1991, between 1976 and 1991, and before 1976, respectively.

Methods

We use linked census-mortality data from the 2001–2004 cohort of the NZCMS. Details of the linkage, weighting for incomplete linkage of mortality data to census data, and variable specifications can be found elsewhere (Fawcett et al. 2008). Briefly, 72.8 % of eligible 2001–2004 Pacific death registration records were linked to the national 2001 census (82.2 % for European/Other). Because not all mortality records were linked to a corresponding census record, correction for any linkage bias and consequent underestimation of mortality rates is necessary. Weights are calculated based on variables that are predictors of linkage in logistic regression analyses: age at census, sex, prioritized ethnicity, rurality, residential mobility of area unit, Territorial Authority, New Zealand deprivation index, months since census night at death, and cause of death. Having a valid household income variable requires that all adults were at their usual residence on census night *and* that they all responded to questions about income. Consequently, of all follow-up times available for this analysis, 28 % did not have a valid income variable.

Because income is probably more a mediator than a confounder of the relationship between natality (or DoR) and mortality, our main results are derived from models in which income is not included in the model or in the underlying data. The data set for this analysis (identified as the "large" Pacific data set) are from census-mortality records agnostic to the presence or absence of income. However, we are also interested in the possible mediating effects of income, so to compare models that include or exclude income, a second data set (the "small" Pacific data set) is extracted from records for which income was not missing. The following description relates to the analysis that includes income (using the small Pacific data set), and we leave to the reader the simplifications required for models applied to the large Pacific data set.

For the OSB-NZB comparison, data are restricted to 176,523 person-years of follow-up for 25- to 74-year-old Pacific people with nonmissing data on follow-up time and covariates. Analyses are conducted on data for the 120 strata formed by cross-classifying sex (dichotomous) by age (10 groups) by income (tertiles) by natality (two groups). For the comparison of OSB Pacific mortality rates by DoR, analyses are conducted on data for the 240 strata formed by cross-classifying sex (dichotomous) by age (10 groups) by income (tertiles) by DoR (four groups: 0–9, 10–24, and 25+ years, plus NZB).

The ethnicity variable is classified using a "total count" definition (Statistics New Zealand 2005). For example, with this definition all of the following people are categorized as Pacific (i.e., self-identifying as Samoan, Cook Islands, Tongan, Niuean, Fijian, or other Pacific ethnicity): self-identified Pacific only; self-identified Pacific and New Zealand European; and self-identified Pacific and Māori (the indigenous people of New Zealand).

For both the OSB-NZB and DoR analyses, we use 10 age groups: 25–29, 30–34, 35–39, 40–44, 45–49, 50–54, 55–59, 60–64, 65–69, and 70–74 years, centered at 45–49 years and scaled so that each unit increase in scaled age corresponds to an actual increase of 10 years. Thus, the age ranges are represented by their endpoints, which become −2.0, –1.5, –1.0, –0.5, 0.0, 0.5, 1.0, 1.5, 2.0, and 2.5 after centering and scaling. To allow for the nonlinear increase in mortality with age, a linear spline for age with knots at age groups 45–49 and 60–65 is included in the prior mean (Eq. (3)).

We use equivalized household income, categorized into tertiles within strata of sex and (five-year) age groups and based on income for the total New Zealand Pacific population. Income ranks are median-centered. Consequently, income ranks (coded as 1, 2, 3) are transformed to (−1, 0, 1).

Hierarchical Bayesian Poisson Regression

We largely follow the hierarchical Bayesian methods used previously by Blakely et al. (2009). In brief, the method for the OSB-NZB analysis is as follows (with DoR models for the Pacific OSB population similar in structure, but with strata of DoR substituted for natality). Assuming that death is a Poisson process such that for natality $i(= 1, 2)$ and stratum $j(= 1, \ldots, 60)$ with deaths d_{ij}, mortality rate λ_{ij}, and person-years at risk P_{ij}, and using the notation $x \sim D\ [a, b]$ to represent a random variable x distributed as D with mean a and variance b, a three-level Poisson model is defined by

$$d_{ij} \mid \lambda_{ij}, P_{ij} \sim \text{Poisson}\left[\lambda_{ij} P_{ij}, \lambda_{ij} P_{ij}\right], \tag{1}$$

$$\lambda_{ij} \mid \mathbf{x}_{ij}, \boldsymbol{\beta}, \varsigma \sim \text{gamma}\left[\mu_{ij}, \mu_{ij}^2 / \varsigma\right], \tag{2}$$

$$\log(\mu_{ij}) = \mathbf{x}_{ij}^T \boldsymbol{\beta}, \tag{3}$$

$$\boldsymbol{\beta}, \varsigma \sim \pi. \tag{4}$$

The mortality rate λ_{ij} has a gamma distribution with mean μ_{ij} and variance μ_{ij}^2 / ς, and the structural mean μ_{ij} depends on covariate vector $\mathbf{x}_{ij}$ and parameter vector $\boldsymbol{\beta}$ through a log-link function. Second-level parameters, $\boldsymbol{\beta}$ (the regression hyper-parameters) and ζ (the mortality rate variance or "shape" hyper-parameter) are assigned independent prior distributions ("hyper-priors") at the third level of the hierarchy. A standard approach is adopted for $\boldsymbol{\beta}$, with proper but noninformative prior distributions for each component.

From Eqs. (1) and (2), the observed deaths d_{ij}, conditional on hyper-parameters $\boldsymbol{\beta}$ and ζ, have a marginal negative binomial distribution; in this sense, our structural model resembles the approach of Hajat et al. (2010). However, the hierarchical Bayesian model goes further by allowing the covariate structure to influence the mean of the posterior rate, but with a degree of influence that depends on the overall support for the covariate structure in the data as well as on how much local information is available. Given the structure of the model defined by Eqs. (1) and (2) and conjugacy of the Poisson and gamma distributions, the conditional posterior distribution for the mortality rate is also gamma with mean

$$E\left[\lambda_{ij} \mid y_{ij}, \boldsymbol{\beta}, \varsigma\right] = B_{ij}\mu_{ij} + (1 - B_{ij})y_{ij}, \tag{5}$$

where $y_{ij} = d_{ij} / P_{ij}$ is the observed mortality rate in the ith natality of the jth stratum, and

$$B_{ij} = \varsigma \Big/ \left(\varsigma + \mu_{ij} P_{ij}\right). \tag{6}$$

Thus, the conditional posterior mean for λ_{ij} is a weighted average of the structural mean μ_{ij} and the observed mortality rate (y_{ij}). The B_{ij}, which lie between 0 and 1, are known as shrinkage parameters because larger values shrink the conditional posterior mean mortality rates toward the structural mean. The gamma shape parameter ζ provides a measure of the influence of the structural mean. We adopt the relatively uninformative uniform shrinkage prior of Christiansen and Morris (1997) for ζ (see also Daniels 1999).

Following previous NZCMS work (Blakely et al. 2009; Young et al. 2006), we use interactions of (a) age and income and (b) sex and income as predictors of the structural mean mortality rate. Thus, for models that use income, the components of the regression hyper-parameters in Eq. (3) are

$$\boldsymbol{\beta} = \left(\beta_0, \beta_{\text{sex}}, \beta_{\text{age}}, \beta_{\text{nat}}, \beta_{\text{inc}}, \beta_{\text{age}\times\text{inc}}, \beta_{\text{sex}\times\text{inc}}\right), \tag{7}$$

where subscripts *nat* and *inc* denote natality and income, respectively. To allow comparison across OSB and NZB groups by age, posterior mortality rates are directly standardized to the follow-up times of the total Pacific population. Thus, for each age-sex-income stratum, follow-up times are fixed at the total Pacific (NZB + OSB) follow-up time in that (age-sex-income) stratum. Standardized rates are then computed as follows:

$$\lambda^{*}_{nat,\, age} \sim \sum_{sex,\, inc} \lambda_{nat,\, age,\, sex,\, inc} \times \frac{\sum_{nat} P_{nat,\, age,\, sex,\, inc}}{P_{Tot}}, \tag{8}$$

where $P_{nat,age,sex,inc}$ is follow-up time in strata of natality, age, sex, and income, and P_{Tot} is follow-up time summed over all strata. Standardization is often used in epidemiology to compare summaries of observations (e.g., mortality rates) that depend on strata that are not of direct interest (e.g., sex, income), and we follow that tradition here using posterior mortality rates. When it is necessary to compare rates or rate ratios across strata or models, we use stratum- or model-specific posterior samples to compute posterior estimates of differences, for which mean rates and credible intervals are computed.

All analyses and plots are done using the R environment (http://www.r-project.org) for statistical computation version 2.13.0, available from the Comprehensive R Archive Network (CRAN) website (http://cran.r-project.org) or SAS 8.2 (SAS Institute Inc., Cary, North Carolina). All hierarchical Bayesian analyses use WinBugs 1.4, available online (http://www.mrc-bsu.cam.ac.uk/bugs/winbugs/contents.shtml), and the R2WinBUGS package version 2.1-16. The WinBugs and R code used for this analysis are available from the authors on request.

Results

Total follow-up times and deaths (weighted and unweighted) by age and sex for the large Pacific data set are given in Table 1. The sparseness of data in this highly stratified data set is evident by the occurrence of the number 6 in the table (the minimum publishable value permitted for counts under Statistics New Zealand's confidentiality rules), despite the relatively wide age categories used for this table. As expected, the majority of OSB deaths tend to come from the 25+ years DoR group.

We first discuss models that exclude income and use the large Pacific data set. Posterior age-specific rate ratios, averaged across all age strata, are shown in Table 2. Averaging is a useful strategy in this case since although all-cause, cancer, and CVD mortality rates for both OSB and NZB groups vary by age (see Table 3), there is no evidence of age heterogeneity in the posterior rate ratios of OSB relative to NZB. For all causes of death, mortality rate ratios are close to unity, and all credible intervals include 1.0: the all-cause mortality rate ratio is 1.00, with a 95 % credible interval of 0.85 to 1.17.

Within the OSB population, there is evidence of lower mortality rates for migrants living in New Zealand for less than 24 years than for longer-standing migrants. All-cause mortality rates for Pacific people living in New Zealand for less than 10 years are 0.75 (0.62 to 0.91) times that for people living 25 or more years in the country. The corresponding estimate for people resident in New Zealand for 10–24 years is 0.78 (0.66, 0.92). Mean posterior estimates for cancer and CVD mortality rate ratios are less than unity in both 0–9 years and 10–24 years DoR strata, but credible intervals exclude the null only for cancer mortality in the 10–24 years stratum. Tables 4, 5, and 6 provide the age-specific rates and rate ratios that underlie these averaged results.

Adjusting for income reveals slightly smaller protective effects of recent migration in age-averaged rate ratios. For example, the all-cause rate ratio for 0–9 years of residence compared with 25+ years of residence decreased from 0.84 to 0.75 after adjusting for income, and the 95 % credible interval excludes the null. However, none of the differences between models that include or exclude income are significant either within age strata or averaged across them.

Mean age-specific NZB mortality rates are often intermediate between mean rates for the 25+ years DoR stratum, and the 0–9 and 10–24 years DoR strata; averaged age-specific rate ratios for these DoR strata relative to the NZB stratum are not significant (rate ratios available on request).

We previously described the hierarchical Bayesian model as a shrinkage estimator, and it is of interest to see how shrinkage affects posterior rates. We provide an illustration in Fig. 1, which shows smoothed plots of mean observed Pacific mortality

Table 1 Person-years and deaths (unweighted (UW) and weighted (W)) by sex, age, DoR, and NZB 2001–2004 cohort, "total" Pacific definition of ethnicity

		Total			DoR: 0–9 Years			DoR: 10–24 Years			DoR: 25+ Years			NZB		
Sex	Age	Person-Years	Deaths (UW)	Deaths (W)	Person-Years	Deaths (UW)	Deaths (W)	Person-Years	Deaths (UW)	Deaths (W)	Person-Years	Deaths (UW)	Deaths (W)	Person-Years	Deaths (UW)	Deaths (W)
Males and Females	25–74	245,985	969	1,314	37,977	105	141	60,621	150	204	78,771	591	789	68,619	135	180
	25–39	120,045	90	144	24,915	15	15	32,817	21	36	13,440	18	27	48,876	39	66
	40–54	82,716	249	339	7,908	27	36	22,062	48	66	36,465	141	186	16,281	42	51
	55–74	43,227	630	834	5,151	63	84	5,742	81	105	28,866	435	576	3,462	51	63
Males	25–74	115,932	570	777	18,063	51	75	26,892	75	108	37,521	357	474	33,453	81	117
	25–39	55,635	57	93	12,123	6	12	13,833	12	27	6,144	6	9	23,538	30	48
	40–54	39,900	150	210	3,891	18	21	10,650	27	39	17,130	84	108	8,232	30	39
	55–74	20,394	357	477	2,049	30	45	2,412	39	51	14,250	267	357	1,683	24	30
Females	25–74	130,056	402	537	19,917	48	66	33,723	72	96	41,250	231	315	35,163	48	63
	25–39	64,407	33	48	12,795	6	6	18,981	6	12	7,293	12	18	25,338	9	18
	40–54	42,813	102	129	4,017	12	15	11,415	18	24	19,335	57	75	8,049	9	15
	55–74	22,833	267	360	3,105	33	45	3,327	42	57	14,622	165	219	1,779	30	33

Notes: All counts are random rounded to a near multiple of three with a minimum cell size of 6, as per Statistics New Zealand protocols. Follow-up time is shown as (weighted) person-years.

Table 2 Posterior all-cause mortality rate ratios (95 % credible intervals) by cause of death for natality (relative to NZB) and duration of residence (relative to 25+ years)

Cause of Death	OSB Compared With NZB	Rate Ratios by DoR Among OSB Only		
		DoR: 0–9 Years	DoR: 10–24 Years	DoR: 25+ Years
Not Adjusted for Income[a]				
All-cause	1.00 (0.85, 1.17)	0.75 (0.62, 0.91)	0.78 (0.66, 0.92)	1
Cancer	0.99 (0.73, 1.34)	0.82 (0.56, 1.12)	0.71 (0.51, 0.97)	1
Cardiovascular disease	1.06 (0.78, 1.44)	0.80 (0.55, 1.12)	0.89 (0.66, 1.18)	1
All-Cause, Adjusted for Income[b]	1.03 (0.86, 1.23)	0.75 (0.57, 0.97)	0.79 (0.63, 0.97)	1
All-Cause, Not Adjusted for Income[b]	1.05 (0.88, 1.24)	0.84 (0.64, 1.08)	0.83 (0.67, 1.02)	1

[a] Models were fitted to the large Pacific data set.

[b] Models were fitted to the small Pacific data set (nonmissing income).

rates alongside posterior estimates of structural mean rates and shrinkages by age and DoR. Because standardization by gender has no advantages when follow-up time is similar for males and females, posterior estimates are simply averaged by gender. For ages under about 40 years, mean shrinkages are typically between 0.7 and 0.9, suggesting that information from the structural model dominates posterior rates. Shrinkages generally tend to reduce as age increases, reflecting the greater number of deaths and higher mortality rates at older ages. This is particularly marked for the Pacific people in the longest DoR group (25 years or longer): for ages greater than 50 years, mean shrinkages are less than 0.5, suggesting that observed rates strongly influence posterior estimates. In contrast, for the DoR groups 0–9 and 10–24 years, the structural means appear to dominate posterior rate estimates at all ages. Such "shrinkage" behavior of hierarchical Bayesian models provides some protection against model misspecification and, together with other desirable properties of the hierarchical Bayesian approach described previously, explains why it is an improvement on the methods of Hajat et al. (2010) for the small-cell problem. Checks of posterior predictive distributions of mortality rates against empirical estimates produce no evidence of model lack-of-fit.

Discussion

The first aim of this study was to investigate the existence of a healthy immigrant effect for Pacific people in New Zealand using a Bayesian shrinkage estimator to reduce the impact of data sparseness. We found little evidence of a mortality advantage (for any cause of death) for the OSB population over the NZB Pacific population. This result is consistent with the conclusion of Hajat et al. (2010), who also found no evidence for a difference in all-cause mortality rates between OSB and NZB using a combined 1996–1999 and 2001–2004 NZCMS data set.

A decline in health with DoR has previously been observed in many migrant populations, including Asian and European/Other immigrants in New Zealand, but

Table 3 Posterior mortality rates (and 95 % credible intervals) by natality, cause-of-death, and age

	OSB			NZB		
Age	All-Cause	Cancer	Cardiovascular Disease	All-Cause	Cancer	Cardiovascular Disease
25–29	14.73 (9.94, 20.86)	2.07 (0.92, 3.87)	2.34 (0.89, 4.69)	14.36 (10.15, 19.61)	1.88 (0.85, 3.45)	2.45 (1.07, 4.61)
30–34	20.51 (14.74, 26.85)	3.58 (1.86, 5.75)	4.91 (2.50, 8.45)	21.96 (16.52, 29.02)	3.94 (2.23, 6.45)	3.69 (1.78, 6.32)
35–39	29.39 (22.24, 36.83)	7.00 (4.48, 10.26)	7.13 (4.12, 10.92)	31.35 (24.27, 40.32)	6.89 (4.36, 10.13)	7.11 (4.09, 11.29)
40–44	36.34 (29.18, 45.11)	10.63 (7.54, 14.72)	10.26 (6.46, 15.05)	31.96 (23.56, 40.95)	9.59 (6.04, 13.54)	9.06 (5.13, 14.05)
45–49	37.63 (28.78, 46.85)	14.55 (9.85, 19.91)	12.39 (7.64, 18.38)	40.53 (29.96, 53.41)	16.15 (10.17, 24.08)	13.16 (7.27, 21.02)
50–54	55.30 (46.34, 65.85)	17.76 (13.08, 22.80)	22.84 (16.55, 30.59)	50.47 (38.33, 65.05)	19.42 (12.92, 28.26)	17.98 (10.66, 28.50)
55–59	67.83 (57.51, 80.37)	23.80 (18.60, 30.95)	21.06 (15.24, 27.62)	61.96 (46.45, 80.98)	22.52 (14.84, 32.83)	20.71 (12.02, 32.55)
60–64	82.56 (69.68, 96.36)	26.61 (19.96, 34.13)	27.67 (20.58, 35.88)	78.77 (56.91, 104.30)	26.80 (16.19, 40.70)	27.21 (15.10, 43.83)
65–69	83.21 (70.26, 96.19)	27.17 (21.29, 33.77)	27.73 (20.45, 35.50)	77.76 (55.71, 101.20)	26.52 (16.86, 38.69)	29.74 (17.40, 46.32)
70–74	84.84 (71.62, 99.51)	24.44 (17.79, 31.99)	35.63 (27.14, 45.43)	74.74 (52.68, 100.40)	21.94 (11.79, 33.81)	25.56 (12.90, 41.94)

Table 4 Posterior all-cause mortality rates (95 % credible intervals) by DoR (0–9 years, 10–24 years, and 25+ years), NZB, and age for the model including age and sex (but not income), and rate ratios for DoR (relative to 25+ years) by age (95 % credible intervals)

	Posterior All-Cause Mortality Rates				Rate Ratios	
Age	DoR: 0–9 Years	DoR: 10–24 Years	DoR: 25+ Years	NZB	DoR: 0–9 Years	DoR: 10–24 Years
25–29	10.28 (6.64, 14.45)	12.29 (8.24, 17.76)	16.04 (10.91, 23.72)	13.29 (9.52, 17.81)	0.66 (0.38, 0.94)	0.78 (0.52, 1.11)
30–34	16.54 (11.70, 22.00)	17.35 (12.60, 23.00)	22.03 (15.42, 29.35)	21.40 (16.28, 27.74)	0.77 (0.51, 1.11)	0.80 (0.54, 1.16)
35–39	24.34 (17.65, 32.08)	23.56 (17.34, 29.84)	33.86 (25.74, 44.09)	29.75 (23.07, 37.21)	0.73 (0.49, 1.03)	0.71 (0.47, 0.95)
40–44	30.99 (22.85, 41.41)	30.24 (23.55, 37.69)	40.81 (32.04, 51.58)	33.19 (24.52, 41.99)	0.77 (0.52, 1.08)	0.75 (0.54, 1.00)
45–49	39.40 (28.55, 54.19)	34.23 (24.88, 44.43)	46.15 (37.07, 55.83)	42.22 (31.61, 54.44)	0.86 (0.61, 1.25)	0.75 (0.53, 1.00)
50–54	45.96 (33.57, 60.70)	51.13 (39.55, 66.67)	63.41 (53.98, 74.00)	56.32 (42.37, 72.62)	0.73 (0.51, 0.99)	0.81 (0.60, 1.09)
55–59	50.97 (35.08, 67.11)	57.18 (42.98, 72.88)	79.45 (68.92, 91.85)	68.65 (52.53, 88.56)	0.65 (0.43, 0.87)	0.72 (0.52, 0.94)
60–64	72.54 (54.97, 95.22)	65.01 (47.18, 84.11)	85.01 (73.00, 97.61)	81.83 (60.90, 107.70)	0.86 (0.64, 1.16)	0.77 (0.56, 1.01)
65–69	70.63 (53.06, 89.53)	82.65 (64.76, 105.90)	95.90 (83.10, 109.20)	87.83 (65.66, 113.00)	0.74 (0.54, 0.97)	0.87 (0.66, 1.16)
70–74	79.00 (61.04, 99.40)	87.45 (68.85, 110.60)	103.14 (88.46, 119.00)	93.71 (68.55, 122.60)	0.77 (0.58, 1.00)	0.85 (0.66, 1.11)

Table 5 Posterior cancer mortality rates (95 % credible intervals) by DoR (0–9 years, 10–24 years, 25+ years), NZB, and age for the model including age and sex (but not income), and rate ratios for DoR (relative to 25+ years) by age (95 % credible intervals)

	Posterior Cancer Mortality Rates				Rate Ratios	
Age	DoR: 0–9 Years	DoR: 10–24 Years	DoR: 25+ Years	NZB	DoR: 0–9 Years	DoR: 10–24 Years
25–29	1.42 (0.58, 2.75)	1.39 (0.59, 2.70)	2.21 (0.95, 4.40)	1.60 (0.69, 2.98)	0.69 (0.27, 1.29)	0.67 (0.30, 1.24)
30–34	2.98 (1.41, 5.24)	2.59 (1.29, 4.47)	3.74 (1.80, 6.51)	3.86 (2.08, 6.48)	0.85 (0.38, 1.68)	0.74 (0.34, 1.45)
35–39	5.88 (3.12, 9.81)	4.54 (2.44, 7.12)	7.67 (4.49, 12.56)	6.80 (3.97, 10.60)	0.81 (0.37, 1.53)	0.63 (0.29, 1.11)
40–44	8.55 (4.54, 13.73)	7.40 (4.39, 11.13)	14.65 (9.41, 22.74)	9.07 (4.89, 14.00)	0.61 (0.27, 1.05)	0.53 (0.25, 0.89)
45–49	14.86 (8.10, 24.50)	12.00 (6.94, 18.45)	15.41 (10.15, 21.56)	17.09 (10.38, 26.32)	0.99 (0.51, 1.79)	0.80 (0.43, 1.38)
50–54	18.00 (10.22, 28.72)	16.21 (9.92, 24.86)	19.24 (13.87, 25.16)	22.74 (14.28, 34.77)	0.96 (0.51, 1.65)	0.86 (0.49, 1.46)
55–59	17.54 (9.04, 28.10)	17.71 (10.48, 27.01)	28.16 (21.84, 35.58)	25.87 (15.72, 40.16)	0.63 (0.31, 1.05)	0.64 (0.36, 1.02)
60–64	20.03 (10.18, 32.07)	20.83 (12.25, 32.52)	27.34 (20.88, 34.76)	27.88 (15.98, 44.11)	0.74 (0.37, 1.22)	0.77 (0.44, 1.26)
65–69	24.74 (15.06, 36.92)	22.96 (14.03, 34.38)	35.12 (27.67, 43.74)	30.32 (17.74, 46.41)	0.71 (0.41, 1.11)	0.66 (0.38, 1.04)
70–74	33.71 (21.91, 49.50)	24.36 (15.14, 36.48)	29.48 (21.72, 38.32)	28.93 (15.42, 46.64)	1.17 (0.70, 1.88)	0.84 (0.50, 1.33)

Table 6 Posterior cardiovascular disease (CVD) mortality rates (95 % credible intervals) by DoR (0–9 years, 10–24 years, 25+ years), NZB, and age for the model including age and sex (but not income), and rate ratios for DoR (relative to 25+ years) by age (95 % credible intervals)

	Posterior CVD Mortality Rates				Rate Ratios	
Age	DoR: 0–9 Years	DoR: 10–24 Years	DoR: 25+ Years	NZB	DoR: 0–9 Years	DoR: 10–24 Years
25–29	1.51 (0.62, 2.91)	1.73 (0.72, 3.29)	2.06 (0.84, 3.97)	1.99 (0.93, 3.55)	0.78 (0.35, 1.50)	0.90 (0.40, 1.74)
30–34	3.51 (1.78, 6.08)	3.43 (1.78, 5.90)	3.79 (1.81, 6.60)	3.37 (1.75, 5.56)	1.00 (0.47, 2.01)	0.97 (0.47, 1.84)
35–39	5.38 (2.78, 8.97)	6.26 (3.72, 9.48)	7.48 (4.27, 11.81)	6.81 (4.06, 10.36)	0.75 (0.34, 1.37)	0.88 (0.45, 1.56)
40–44	8.44 (4.69, 13.53)	8.30 (4.92, 12.37)	12.70 (8.23, 18.76)	9.52 (5.65, 14.30)	0.69 (0.34, 1.19)	0.68 (0.34, 1.13)
45–49	13.36 (7.28, 22.00)	13.02 (7.41, 19.79)	16.12 (10.86, 22.24)	14.82 (8.68, 22.86)	0.85 (0.44, 1.49)	0.83 (0.44, 1.35)
50–54	18.13 (10.46, 29.35)	21.77 (13.94, 32.57)	25.84 (19.69, 33.13)	21.40 (13.10, 32.32)	0.71 (0.39, 1.20)	0.85 (0.52, 1.34)
55–59	20.79 (11.79, 32.65)	27.21 (17.45, 40.94)	25.81 (19.73, 32.57)	25.43 (14.99, 38.78)	0.82 (0.44, 1.36)	1.07 (0.65, 1.74)
60–64	33.15 (20.06, 52.02)	27.75 (16.65, 41.78)	30.13 (23.01, 37.95)	32.60 (19.33, 51.03)	1.12 (0.64, 1.85)	0.93 (0.54, 1.47)
65–69	22.40 (12.57, 33.81)	33.10 (21.35, 49.32)	31.60 (24.46, 39.43)	33.19 (20.36, 50.89)	0.72 (0.39, 1.14)	1.06 (0.65, 1.67)
70–74	21.10 (11.99, 32.29)	27.11 (17.04, 39.77)	37.93 (29.39, 47.76)	26.72 (14.34, 42.73)	0.56 (0.30, 0.90)	0.72 (0.44, 1.09)

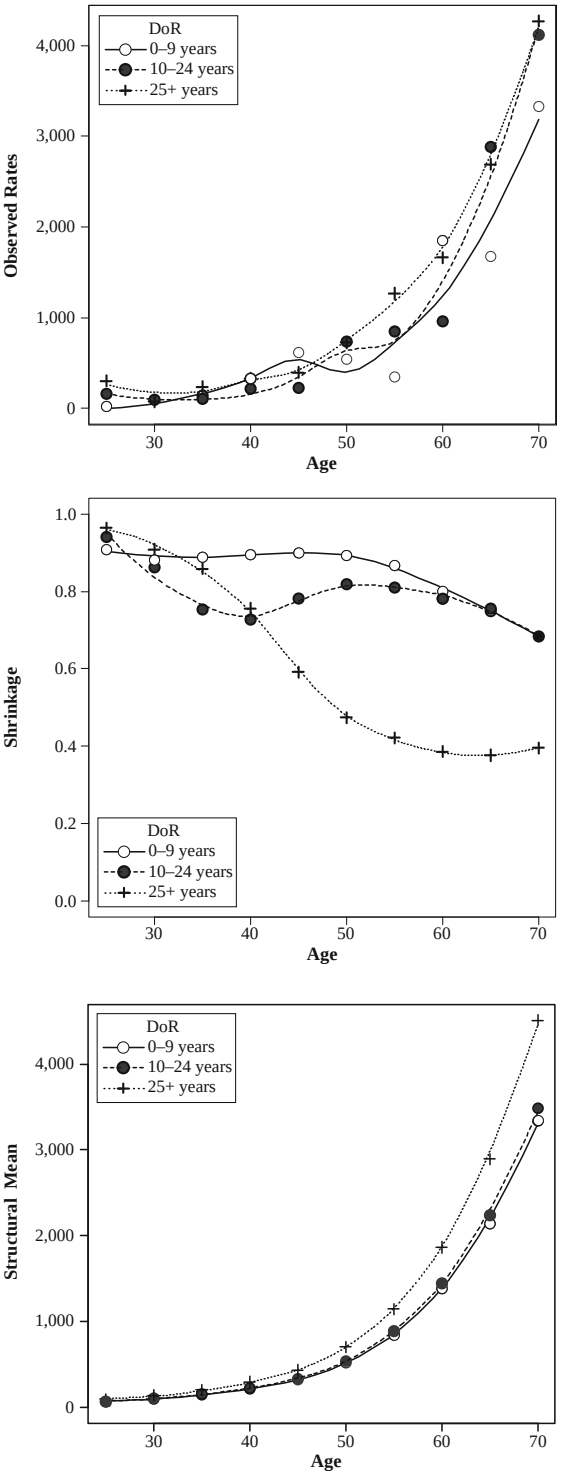

Fig. 1 Observed gender-averaged Pacific mean mortality rates (× 100,000) and posterior estimates of the shrinkage parameter and structural mean rates from the hierarchical Bayesian model. The lines were computed using spline interpolation between computed gender-averaged posterior means

not for Pacific people (Hajat et al. 2010; Richardson et al. 2012). Likewise, we found evidence for higher Pacific all-cause mortality rates for Pacific migrants resident in New Zealand for 25 or more years. The same may be true for cancer and CVD mortality: mean posterior rate ratios for the DoR strata 0–9 and 10–24 years (relative to 25 years or longer) were similar to corresponding all-cause mean posterior rate ratios. However, rate ratio credible intervals excluded the null only for cancer mortality in the DoR stratum 10–24 years, although even when credible intervals included the null, most of the posterior distributions remained less than unity. Confirmation of this result will require further work.

Other researchers have noted that Pacific migrants are more likely to have less-skilled occupations and lower incomes than other ethnic groups in New Zealand (Hajat et al. 2010; Statistics New Zealand 2002). Coupled with their position in the social hierarchy, this may elevate OSB mortality rates so that they are similar to those of their NZB counterparts. However, including income in the models did not produce any statistically significant changes in rates or rate ratios, suggesting that income plays a minor role in mediating the relationship between natality (or DoR) and mortality.

Results from this study suggest that the healthy migrant effect, in its simplest form, does not apply to New Zealand's Pacific people. Those born outside the country had, on average, no measurable health advantage relative to those born in New Zealand. Additional analyses not reported here showed that this was also true for any duration of residence at any age. Moreover, as can be seen from Tables 4–6, NZB mean posterior mortality rates were typically intermediate between rates of the OSB resident in New Zealand for more than 25 years and those resident for less than 25 years.

We have shown that there was a DoR effect for Pacific migrants who resided in New Zealand for less than 25 years, and some indication that this might also be true for cancer and CVD mortality. Several explanations situated within the social, political, and cultural position of migrants in New Zealand could be responsible for higher all-cause mortality in the 25+ years DoR group. The role of government migration policy over the past several decades may provide one such explanation. As mentioned in the introduction, Pacific groups (and particularly the three main sources of Pacific migration: Fiji, Samoa, and Tonga) have had restricted entry to New Zealand, whereas others (e.g., from the Cook Islands, Niue, and Tokelau) have had unrestricted access. Nevertheless, between World War II and the mid-1970s, there was strong demand for semiskilled and unskilled labor in factories and in the service sector, and although there were regulatory attempts to control the resultant increased flow of Pacific migrants (e.g., through short-term visas and annual quotas), governments were not inclined to enforce them. Migration-driven growth saw New Zealand's Pacific population increase from 0.1 % of the population in 1945 to 2.0 % in 1976, and because access was relatively unrestricted, it seems reasonable to suppose that health-selection barriers for Pacific migrants were, in practice, low. It also seems reasonable to suppose that after the tightening of New Zealand migration policy in the mid-1970s in response to less favorable economic conditions, health criteria were more diligently enforced, particularly for people from Fiji, Samoa, and Tonga. Changes to migration policy in the mid-1980s, which emphasized skills and assets, may have added indirect forms of health selection to these direct (medical screening) forms. Under these assumptions, a significant proportion of Pacific migrants with duration of residence less than 25 years have been subject in practice to

more intense health-selection pressures than those with longer durations of residence, resulting in overall lower average mortality rates for those who settled in New Zealand after the mid-1970s compared with earlier migrants.

Other factors may also contribute to the higher mortality of OSB Pacific people who resided in New Zealand for more than 25 years relative to those who lived there less than 25 years. One is related to the position of Pacific people in the New Zealand social hierarchy: increasing years since migration may be associated with increasing (accumulated) exposure to discrimination (Gee et al. 2009; Harris et al. 2006), with attendant negative consequences for health. Another contributor to higher mortality rates of healthy migrants over time might be a change of diet and/or lifestyle—for example, abandonment by Pacific migrants of traditional diets in favor of high-fat, high-salt fast foods or an increase in drinking and illicit drug use. Evidence in support of this conjecture is limited and somewhat equivocal. Ulijaszek's (2005) survey of published information covering the period 1961–2000 found that energy intake and energy density of diets in five Pacific nations (including Western Samoa and the Cook Islands) had increased, probably in response to dietary/lifestyle changes. Nevertheless, differences in body mass index (BMI) were still observable depending on adherence to traditional lifestyles: in general, mean BMI was larger among urbanized Pacific people than among those leading traditional lifestyles. Notably, mean BMI was higher among migrants from these countries who were living in industrialized countries (e.g., New Zealand) than for Pacific people who had not migrated. Similar patterns were observed in the prevalence of Type 2 diabetes in a number of Pacific nations, including the Cook Islands and Samoa (Foliaki and Pearce 2003). In this case, though, prevalence was not always lower for those who had not migrated to countries like New Zealand.

Further research will be required to determine the relative importance of these hypotheses. For example, the abandonment of negative health behaviors that are more prevalent in the source population, coupled with generally improving health and health care in the host country, might lead to an improvement in migrant health over time. It is also possible that discrimination is more acute for recent arrivals, and even if not, it is plausible that health effects might manifest in Pacific migrants resident in New Zealand longer than, say, 10 rather than 25 years. The impact of discrimination over time may in fact be even more complex, perhaps intensifying in the mid-1970s as a consequence of the 1974 review of immigration policy. The review distinguished between migrants with a legal right to stay permanently in New Zealand and those who had stayed longer than their visitor or temporary permits allowed. Enforcing the distinction led to a period of police raids directed at "illegal overstayers" and it is interesting and perhaps relevant to note that the prosecution rates for overstaying were much higher for Pacific people (86 %) than for nationals of the United States or United Kingdom (5 %), even though these two groups were similar in size (Beaglehole 2007). The health-selection hypothesis, at least, can be readily explored by comparing Pacific mortality rates at a subgroup level using the methods used in this work.

Whatever the reason for the changes in mortality rate by duration of residence, our observation that mortality rates in the overseas-born change over time is sufficient to explain our other observation that there is no difference by natality. The longest DoR group has, on average, slightly higher mortality rates than Pacific people born in New

Zealand, whereas the shorter DoR groups have slightly lower mortality rates. Combining the overseas-born into one group ignores this heterogeneity and results in an overall mortality rate that is not measurably different from that of the NZB.

By 2026, 10 % of New Zealand's population is expected to be Pacific (Ministry of Pacific Island Affairs 2011a), and good health is essential to ensuring that the social and economic potential of Pacific communities is fully realized. Additionally, Pacific people (along with other migrant groups) will play a key role in determining the national health profile and demands on the health system. Future work to clarify the importance of modifiable drivers of the inferred long-term decline in health of the overseas-born Pacific population (relative to more recent migrants) is important not only to Pacific communities but also from a national health and policy perspective.

Limitations

There is significant and potentially important heterogeneity in the analysis of the health selection pressures on Pacific people, which has not been explicitly dealt with here but will be the subject of future work. Other potential sources of bias include linkage bias, differential return migration, and differential census undercounting (Blakely et al. 2009), but these would have to be unexpectedly large to change our conclusions regarding the absence of a mortality advantage for overseas-born Pacific people (relative to New Zealand–born) and the mortality advantage for the DoR groups 0–9 and 10–24 years relative to the longer-duration group. Finally, we have not attempted to separate the effects of duration of residence and age at migration, which potentially impact the health of Pacific migrants in different ways.

Policy implications

This work has demonstrated that although there is little difference in mortality rates between OSB and NZB Pacific groups, for up to 24 years after migration all-cause (and possibly cancer and CVD) mortality rates are reduced, a mortality advantage that disappears more than 25 years after migration. The reasons for this may be complex and not fully discernible with the current analysis. Specific policy recommendations therefore await an improved understanding of the process. In particular, designing effective policy interventions will require identification of modifiable determinants of the inferred long-term decline in health of overseas-born Pacific people relative to more recent Pacific migrants to New Zealand.

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