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Prevalence and incidence of frailty in Aboriginal Australians, and associations with mortality and disability

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ABSTRACT

Objectives: Frailty represents a loss of homeostasis, markedly increasing the risk of death and disability. Frailty has been measured in several ethnic groups, but not, to our knowledge, in Aboriginal Australians. We aimed to determine the prevalence and incidence of frailty, and associations with mortality and disability, in remote-living Aboriginal people.

Study design: Between 2004 and 2006, we recruited 363 Aboriginal people aged ≥45 years from 6 remote communities and one town in the Kimberley region of Western Australia (wave 1). Between 2011 and 2013, 182 surviving participants were followed-up (wave 2). We assessed frailty with an index, comprising 20 health-related items. Participants with ≥4 deficits (frailty index ≥0.2) were considered frail. Disability was assessed by family/carer report. Those unable to do ≥2 of 6 key or instrumental activities of daily living were considered disabled. We investigated associations between frailty, and disability and mortality, with logistic regression and Cox proportional hazards models.

Results: At wave 1 (W1), 188 participants (65.3%) were frail, and of robust people at W1 who participated in wave 2, 38 (51.4%) had become frail. Frailty emerged at a younger age than expected. A total of 109 people died (30.0%), of whom 80 (73.4%) were frail at W1. Frailty at W1 was not associated with becoming disabled, but was associated with mortality (HR = 1.9; 95% CI 1.2, 3.0).

Conclusions: Frailty in remote-living Aboriginal Australians is highly prevalent; substantially higher than in other populations. Research to understand the underlying causes of frailty in this population, and if possible, reverse frailty, is urgently needed.

1. Introduction

Aboriginal people comprise approximately 3% of the Australian population [1], and experience marked health disparity compared with the general community. Deficits are observed in nearly all areas of health, including life expectancy, chronic disease, mental health, and disability [2]. Risk factors such as smoking, substance abuse, poor nutrition, and physical inactivity, and psychosocial stressors including overcrowding, violence, and premature mortality of family members, are common [2,3]. The resulting accumulation of insults to the body likely accelerate the ageing process and predisposition to frailty [4].

Frailty is an unstable state where the normal age-related loss of physiological reserve is accelerated, and homeostatic systems begin to fail [5,6]. The ability to cope with even minor stressors (both endogenous and exogenous) is reduced, potentially inducing a progressive frailty cascade, with a concomitant increase in the risk of disability and death [5,7,8]. Frailty becomes more prevalent with increasing age, but is not a normal part of the ageing process and may be reversible [9]. Understanding how frailty might be prevented or ameliorated is therefore important, given its contribution to disability, mortality, and burden on health systems [10,11].

Frailty has been studied in a range of ethnic groups [5,10,12–16], and we have previously examined frailty and associated factors in a population-based sample of Western Australian men [17].

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Table 1
Demographic, lifestyle, and clinical characteristics of the cohort at wave 1 (2004–06) and at wave 2 (2011–13).

Characteristic	Wave 1 (n = 363)			Wave 2 (n = 182)		
	Total n (%)	Non-frail n (%)	Frail n (%)	Total n (%)	Non-frail n (%)	Frail n (%)
Age (years)						
45–49	71 (19.6)	32 (25.4)	39 (16.5)	0 (0.0)	0 (0)	0 (0)
50–59	124 (34.2)	45 (35.7)	79 (33.3)	79 (43.4)	31 (52.5)	48 (39.0)
60–69	71 (19.6)	25 (19.8)	46 (19.4)	42 (23.1)	15 (25.4)	27 (22.0)
70–79	67 (18.5)	19 (15.1)	48 (20.3)	38 (20.9)	10 (17.0)	28 (22.8)
80+	30 (8.3)	5 (4.0)	25 (10.6)	23 (12.6)	3 (5.1)	20 (16.3)
Sex						
Male	165 (45.5)	68 (54.0)	97 (40.9)	72 (39.6)	21 (35.6)	51 (41.5)
Female	198 (54.5)	58 (46.0)	140 (59.1)	110 (60.4)	38 (64.4)	72 (58.5)
Some formal schooling	219 (60.3)	88 (69.8)	131 (55.3)	118 (64.8)	42 (71.2)	76 (61.8)
Drink alcohol[35,5]	134 (36.9)	58 (46.0)	76 (32.1)	50 (27.5)	18 (30.5)	32 (26.0)
Smoke tobacco[35,4]	127 (35.0)	55 (43.7)	72 (30.4)	50 (27.5)	19 (32.2)	31 (25.2)
Chew tobacco[35,5]	140 (38.6)	41 (32.5)	99 (41.8)	63 (34.6)	19 (32.2)	44 (35.8)
Poor vision[18,3]	209 (57.6)	57 (45.2)	152 (64.1)	79 (43.4)	10 (17.0)	69 (56.1)
Poor hearing[18,5]	61 (16.8)	6 (4.8)	55 (23.2)	37 (20.3)	2 (3.4)	35 (28.5)
Prior stroke[19,8]	35 (9.6)	3 (2.4)	32 (13.5)	26 (14.3)	0 (0)	26 (21.1)
Diabetes[19,12]	135 (37.2)	31 (24.6)	104 (43.9)	90 (49.5)	20 (33.9)	70 (56.9)
Hypertension[19,34]	134 (36.9)	19 (15.1)	115 (48.5)	71 (39.0)	12 (20.3)	59 (48.0)
Heart problem[19,16]	58 (16.0)	5 (4.0)	53 (22.4)	50 (27.5)	5 (8.5)	45 (36.6)
Kidney problem[19,17]	47 (13.0)	5 (4.0)	42 (17.7)	45 (24.7)	7 (11.9)	38 (30.9)
Poor mobility[19,5]	139 (38.3)	17 (13.5)	122 (51.5)	80 (44.0)	9 (15.3)	71 (57.7)
Pain[20,8]	199 (54.8)	43 (34.1)	156 (65.8)	100 (55.0)	17 (28.8)	83 (67.5)
Recent fall[20,10]	71 (19.6)	6 (4.8)	65 (27.4)	45 (24.7)	4 (6.8)	41 (33.3)
Head injury with loss of consciousness[21,7]	173 (47.7)	43 (34.1)	130 (54.9)	56 (30.8)	10 (17.0)	46 (37.4)
Incontinence[20,9]	60 (16.5)	6 (5.0)	54 (22.8)	50 (27.5)	7 (11.9)	43 (35.0)
KICA-Cog ≤35[1,32]	107 (29.5)	20 (15.9)	87 (36.7)	41 (22.5)	9 (15.3)	32 (26.0)
Feels happy most of the time[26,67*†]	312 (86.0)	122 (96.8)	190 (80.2)	112 (61.5)	45 (76.3)	67 (54.5)
Sleeping well at night[27,71*†]	302 (83.2)	118 (93.7)	184 (77.6)	102 (56.0)	42 (71.2)	60 (48.8)
Sleeping all the time[26,67*]	36 (9.9)	9 (7.1)	27 (11.4)	14 (7.7)	2 (3.4)	12 (9.8)
Eating properly[25,67*†]	319 (87.9)	122 (96.8)	197 (83.1)	107 (58.8)	45 (76.3)	62 (50.4)
Can still do own work[9,48*†]	281 (77.4)	118 (93.7)	163 (68.8)	84 (46.2)	39 (66.1)	45 (36.6)
Can still do activities they enjoy[11,48*†]	312 (86.0)	123 (97.6)	189 (79.8)	94 (51.7)	41 (69.5)	53 (43.1)
Can shower themselves[11,47*†]	317 (87.3)	122 (96.8)	195 (82.3)	121 (66.5)	45 (76.3)	76 (61.8)
Frail (frailty index ≥0.2)	237 (65.3)	0 (0)	237 (100)	123 (67.6)	0 (0)	123 (100)
Disabled (cannot do ≥2 I/ADLs)[11,51]	54 (15.3)	2 (1.6)	52 (22.7)	38 (29.0)	5 (11.4)	33 (37.9)

Note: Percentages calculated without excluding missing data (i.e., denominator is entire sample), except for disability, which was not calculated when disability data were missing. Column percentages are shown. Numerals in superscript denote number of people with missing data for that variable, for wave 1, and then wave 2. Items denoted (*) reported by family or carer. Items denoted (†) are scored inversely in the frailty index. I/ADLs = Instrumental Activities of Daily Living and/or Activities of Daily Living.

However, to our knowledge, no studies have investigated frailty in Aboriginal Australians. In this study of remote-living Aboriginal people, we aimed to: (i) determine the prevalence and incidence of frailty; and, (ii) describe associations between frailty, and disability and mortality. We hypothesised the prevalence and incidence of frailty would be higher than observed in other populations and be present at a younger age, and that frailty would be associated with increased mortality and development of disability.

2. Methods

2.1. Setting and participants

This was a cohort study of Aboriginal people living in the remote Kimberley region of Western Australia (WA). A total of 363 participants aged ≥45 years were originally recruited to investigate dementia in this population between 2004 and 2006 (wave 1; W1) from the communities of Ardyaloon, Junjuwa, Looma, Mowanjum, Warmun, and Wirrimanu, and from the town of Derby (response fraction: 94.3%). Because dementia is rare in younger individuals, only those aged ≥45 years were eligible to participate. While enrolment to vote is compulsory in Australia, Aboriginal people are under-represented on the electoral roll. It was therefore not possible to obtain a random sample of Aboriginal people living in the Kimberley. Instead, one town and a number of communities representative of the region were identified, and with the assistance of local Aboriginal health and community services, a sampling frame was constructed reflecting the 5 major language families of this region. Participants were added to the sampling frame if they were resident in the selected communities for at least 6 months of the year. All such participants living in the remote communities were approached to participate, while one-third of the town's eligible population were randomly selected from the sampling frame and invited to take part. The age distribution of participants was representative of the general population, and the study protocol is described in depth elsewhere [18,19]. Between 2011–2013 (wave 2; W2), 238 surviving participants were invited to participate in a follow-up study, since 109 (30.0%) had died and 16 could not be located. Of 238 people invited, 189 participated, and 182 (76.5%) provided sufficient information to assess frailty.

2.2. Study design

At each wave, research assistants administered a culturally-appropriate questionnaire to participants and their family members/carers (as applicable). Twenty items assessing health status common to both waves were selected to construct a frailty index (FI), following the methodology described by Searle and colleagues [13]. As per Searle et al.'s guidance, deficits were defined as "symptoms, signs, disabilities and diseases", the prevalence of which must increase with age [13]. The FI was constructed by summing the number of health deficits present in an individual, and dividing this

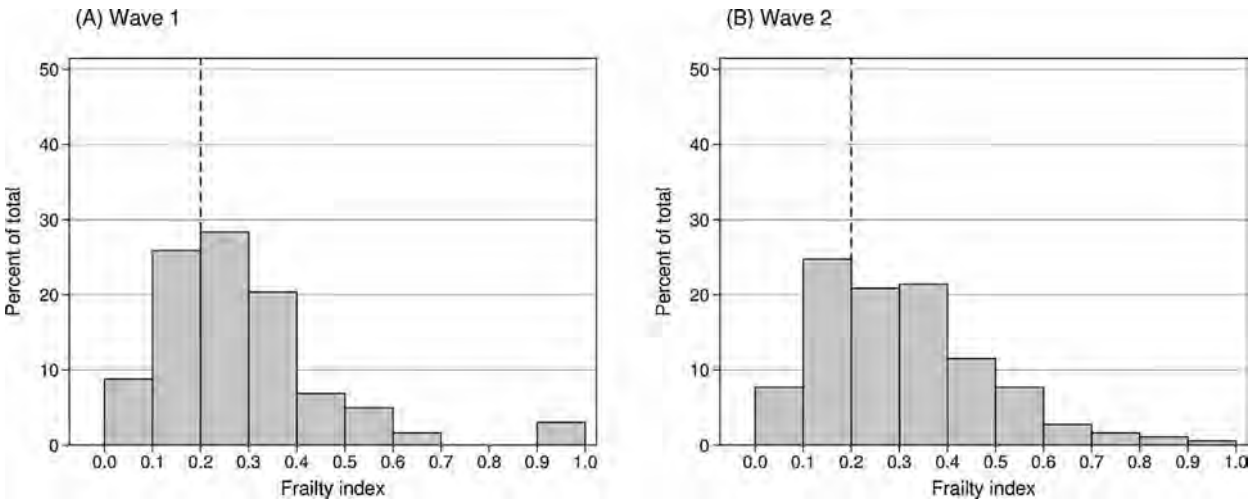

Fig. 1. Distribution of the frailty index at wave 1 (A) and wave 2 (B).

Note: Dashed line denotes cut-off for frailty (frailty index ≥0.2)

by the number of possible deficits (n = 20). If missing data existed for an item, the denominator was reduced by 1 accordingly. The items chosen to construct the FI, along with the scoring schema, are provided as supplementary material (Supplementary Tables 1 and 2). These items, excluding those which are not health deficits (age, sex, schooling, alcohol and tobacco use, and the number of frail and disabled people), are also shown in Table 1. Those with ≥4 deficits (FI ≥0.2) were considered frail.

Disability was assessed by counting the number of 6 basic activities of daily living and/or instrumental activities of daily living (I/ADLs) common to both waves that participants were unable to perform (Supplementary Tables 3 and 4). Those unable to do ≥2 I/ADLs were considered to have a disability.

Approval to conduct this study was obtained from the communities involved; the Kimberley Aboriginal Medical Services Council; Kimberley Aged and Community Services; the Kimberley Aboriginal Health Planning Forum Research Subcommittee; the Human Research Ethics Committee of the University of Western Australia; the WA Aboriginal Health Ethics Committee; and the Department of Health WA Human Research Ethics Committee. All participants provided written informed consent.

2.3. Primary outcome measures

We examined the FI at both time-points to determine the prevalence of frailty at W1 and W2, and incidence of frailty at W2. We calculated prevalence and incidence of disability similarly. We obtained mortality data from the WA Data Linkage System, which links together the state's population health collections, including all deaths registered in the state [20]. We obtained death records to August 2013—approximately 2 months after W2. From these data, we performed survival analyses and calculated mortality incidence. One person could not be linked; mortality analyses were therefore restricted to the remaining 362 participants.

2.4. Statistical analysis

We analysed the data with the Stata statistical package, version 11.2 (StataCorp, College Station, Texas). Summary statistics for demographic and clinical data for each wave are presented as the number and proportion of people who answered in the affirmative for each particular variable. When calculating proportions with 95% confidence intervals, we used the binomial (exact) method. We used binary logistic regression to investigate whether frailty at W1 was associated with becoming disabled at W2, and Cox proportional hazards models to ascertain whether frailty at W1 was associated with all-cause mortality. We assessed the Schoenfeld residuals to confirm the proportional hazards assumption. We initially performed univariate binary logistic and Cox regression to determine variables associated with the outcomes of interest. In addition to frailty status at W1, variables tested comprised the following lifestyle and demographic factors: age; sex; education; alcohol use; smoking; and chewing tobacco. We then entered all variables significant in univariate analyses into multivariate models, and subsequently removed non-significant variables in a manual, backwards manner. We considered p values < 0.05 statistically significant.

3. Results

Sociodemographic and clinical characteristics of the cohort are shown in Table 1. The mean follow-up time for the assessment of frailty (n = 182) was 6.7 ± 0.7 years (range: 5.2–7.9 years), while for mortality (n = 362), it was 6.8 ± 2.2 years (range: 0.1–9.0 years). The mean age of participants was 60.7 ± 11.9 years (range: 45–96 years) at W1, and 65.6 ± 10.7 years (range: 50–93 years) at W2.

3.1. Prevalence of frailty and disability

More than half the cohort was frail at both time-points (Fig. 1). The mean FI was 0.3 ± 0.2 at both W1 and W2, exceeding the cut-off of 0.2. The prevalence of frailty at W1 was 65.3% (95% CI 60.1, 70.2%), while at W2 it was 67.6% (95% CI 60.3, 74.3%). Although the prevalence of frailty between time-points was almost unchanged, mortality was high among people frail at baseline. Between W1 and W2, 30.0% (n = 109) of the original sample died, and of these, 73.4% (n = 80) were frail at W1.

In contrast, a marked difference in disability was observed between waves. At W1, the prevalence of disability was 15.3% (95% CI 11.7, 19.5%). At W2, this had increased to 29.0% (95% CI 21.4, 37.6%). As with frailty, mortality was also high in those disabled at W1, with 76.2% (n = 32) of these individuals dying before W2 (Table 2).

3.2. Prevalence of frailty and disability by age

The prevalence of frailty was high in all age groups at baseline. Even in the youngest age group (45–49 years), 54.9% met

Table 2
Prevalence of frailty and disability by age group, at wave 1 (2004–06) and at wave 2 (2011–13).

Age (years)	Prevalence of frailty	
	Wave 1 (n = 363) % (95% CI)	Wave 2 (n = 182) % (95% CI)
45–49	54.9 (42.7, 66.8)	N/A
50–59	63.7 (54.6, 72.2)	60.8 (49.1, 71.6)
60–69	64.8 (52.5, 75.8)	64.3 (48.0, 78.4)
70–79	71.6 (59.3, 82.0)	73.7 (56.9, 86.6)
80+	83.3 (65.3, 94.4)	87.0 (66.4, 97.2)
Age (years)	Prevalence of disability	
	Wave 1 (n = 352) % (95% CI)	Wave 2 (n = 131) % (95% CI)
45–49	2.9 (0.3, 9.9)	N/A
50–59	8.5 (4.2, 15.2)	14.5 (6.5, 26.7)
60–69	18.8 (10.4, 30.1)	20.7 (8.0, 39.7)
70–79	19.7 (10.9, 31.3)	37.9 (20.7, 57.7)
80+	53.3 (34.3, 71.7)	72.2 (46.5, 90.3)

Note: Prevalence of disability calculated only in those individuals without missing I/ADL data (i.e., all 6 key I/ADL measures were completed in the questionnaire). No participants were aged 45–49 years at wave 2. CI = confidence interval.

Table 3
Change in frailty and disability between wave 1 (2004–06) and wave 2 (2011–13).

	Frailty status at wave 2		
Frailty status at wave 1	Not frail n (%)	Frail n (%)	Deceased n (%)
Not frail	36 (34.9)	38 (36.9)	29 (28.2)
Frail	23 (12.2)	85 (45.2)	80 (42.6)
	Disability status at wave 2		
Disability status at wave 1	No disability n (%)	Disabled n (%)	Deceased n (%)
No disability	86 (45.3)	30 (15.8)	74 (38.9)
Disabled	2 (4.8)	8 (19.0)	32 (76.2)

Note: Row percentages are shown.

the criteria for frailty. As expected, frailty prevalence was higher with older age, reaching 83.3% in those aged ≥80 years. Data were similar at W2. The association between age and disability was similar to frailty, although less pronounced. Disability was present in 2.9% of those aged 45–49, reaching 53.3% in the oldest age group. However, unlike frailty, the prevalence of disability rose between time-points. For example, increasing from 53.3% at W1, to 72.2% at W2 in those aged ≥80 years.

3.3. Incidence of frailty and disability

Stability of frailty and disability between waves is shown in Table 3. People who were not frail at W1 were almost equally likely to remain robust, become frail, or die. However, those who were already frail at baseline were unlikely to revert to a non-frail state (n = 23; 12.2%), and were almost equally likely to die (n = 80; 42.6%) or remain frail (n = 85; 45.2%). The incidence proportion of frailty was 51.4%.

Those without a disability fared better, although mortality remained high at 38.9%. However, those already disabled at baseline were unlikely to revert to a non-disabled state (n = 2; 4.8%), and the majority (76.2%) died. The incidence proportion of disability was 25.9%.

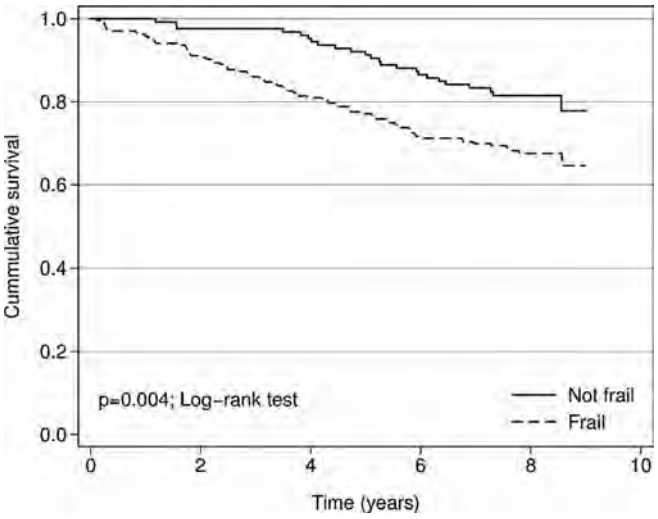

Fig. 2. Kaplan-Meier survival curves showing the association between frailty and all-cause mortality.

3.4. Association between frailty and disability

In binary logistic regression models, being frail at W1 was not a statistically significant predictor of developing a disability before W2 (odds ratio [OR] = 1.1; 95% CI 0.5, 2.6), nor was male sex (OR = 1.7; 95% CI 0.7, 3.9), alcohol use (OR = 0.7; 95% CI 0.3, 1.9), smoking (OR = 0.7; 95% CI 0.3, 1.7), or chewing tobacco (OR = 1.2; 95% CI 0.5, 2.8). However, confidence intervals were large owing to the modest number of participants in these models (n = 122–126). Increasing age (entered as a continuous variable) was associated with becoming disabled (OR = 1.09; 95% CI 1.05, 1.14), as was formal schooling (OR = 0.2; 95% CI 0.1, 0.5). However, after adjustment, increasing age was the sole predictor of disability.

3.5. Mortality incidence and association with frailty

Frailty at baseline was associated with all-cause mortality (Fig. 2). In univariate Cox proportional hazards models, frailty at W1 was associated with mortality (hazard ratio [HR] = 1.9; 95% CI 1.2, 3.1), as was increasing age (HR = 1.05; 95% CI 1.03, 1.07), male sex (HR = 2.1; 95% CI 1.4, 3.1), formal schooling (HR = 0.5; 95% CI 0.3, 0.7), and chewing tobacco (HR = 1.5; 95% CI 1.0, 2.2). Drinking alcohol (HR = 1.0; 95% CI 0.7, 1.6), and smoking (HR = 1.1; 0.7, 1.7) were not significantly associated. In a multivariate model, frailty at W1 (HR = 1.9; 95% CI 1.2, 3.0), age (HR = 1.05; 95% CI 1.04, 1.07), and male sex (HR = 2.6; 95% CI 1.7, 3.9) remained associated with mortality. After adjustment for age and sex, each 1-unit increase in the number of deficits was associated with a 14% increase in mortality risk (95% CI 1.1, 1.2).

The overall incidence of all-cause mortality in the cohort was 40.4 deaths (95% CI 33.2, 49.1) per 1000 person-years (2,475.3 person-years total). The 5-year mortality risk for those who were not frail at W1 was 8.7% (95% CI 4.9, 15.2%), while for frail individuals, it was 22.9% (95% CI 18.0, 28.8%).

4. Discussion

In this study of remote-living Aboriginal Australians, we found a very high prevalence and incidence of frailty and disability, at a much younger age than has been observed in the general population. Frailty was a strong predictor of all-cause mortality, but not disability. However, the high mortality of frail individuals at baseline may explain this. i.e., once frail, participants were likely to

die before they could reach a disabled state, or proceeded quickly through a disabled state to death before follow-up.

Notable was the strong protective association between education and disability, although this was attenuated by age. Education is an important social determinant of health, ultimately affecting other key factors such as access to employment, income, and enabling people to make better informed health decisions (particularly with regard to smoking and nutrition). Only 60% of the cohort had received some formal education, the majority of which was limited to primary education. With regard to Australia as a whole, only half of Aboriginal and Torres Strait Islander students completed their twelfth year of schooling in 2011, compared with 81% of non-Indigenous students [3]. Retention rates in the Kimberley region are markedly lower. There is an urgent need to improve the quality of, and access to education services in this region, and to develop strategies to address cultural and other barriers to improve retention rates.

We previously analysed the prevalence of frailty in a population-based cohort of older men living in Perth, the state capital [17]. In that study, in which 3616 men aged 70–88 years were followed for a mean of 5 years, 15.2% were frail at baseline, increasing to 23.0% at follow-up (ages 76–93 years). In contrast, in this study, the prevalence of frailty in the youngest age group (45–49 years) was 54.9% – more than twice the figure observed at follow-up (age ≥76 years) in the general male population [17]. Comparisons to other non-Aboriginal populations are similar. Other studies of frailty in men, women, and both sexes aged ≥60 years, report prevalence data ranging from 6.9% to 19% [5,10,12,14,21,22], although a review of frailty in developing countries report rates up to 31% in Brazil and China [23] that supports the influence of socioeconomic factors [24]. In one of the largest studies of frailty, Fried and colleagues studied 5317 men and women aged ≥65 years, and reported a prevalence of 6.9%, and four-year incidence proportion of 7.2% [5].

Why, then, is the prevalence and incidence of frailty so much higher in Aboriginal Australians? Higher rates of chronic disease and accidents [2] are likely contributors, as are psychosocial stressors. Poor psychological well-being and limited sense of control are associated with frailty and may potentiate the association with mortality [25,26]. Additionally, health deficits begin in this population in utero. e.g., Aboriginal babies are twice as likely to be of low birthweight than their non-indigenous counterparts [2], and have reduced renal volume [27]. Additionally, conditions now seldom seen in the general population (such as rheumatic heart disease and trachoma), remain endemic in remote Aboriginal communities [2]. Barriers to health services [3], could also contribute. Remote-living Aboriginal people often lack access to medical services such as dentistry, probably reflected in the high prevalence of dental caries, periodontal disease, and tooth loss in this population [28,29]. Poor dentition has been postulated as a risk factor in the frailty cascade, leading to decreased appetite, malnutrition, and sarcopaenia [5,8]. Barriers to health care are further compounded when disability is present. Approximately half of Aboriginal people with a severe core activity limitation report difficulty accessing medical, dental, legal, transport, and employment services [2,30]. Cohort effects must also be considered, given health services available to remote communities have hitherto been more limited, affecting morbidity. The aetiology of frailty is almost certainly multifactorial, likely resulting from accumulated insults to the body, together with other factors. Some argue that frailty better characterises the ageing phenotype than age itself [15]. Our results, together with the observation that many chronic diseases emerge earlier in Aboriginal Australians [2], suggest the ageing process is accelerated in this population.

Frailty is consistently associated with mortality, being estimated that up to 5% of deaths might be delayed if frailty could be prevented [31]. Mitnitski et al. reported that for each additional health deficit, mortality risk increased by 4% [32]. In our study, the risk was 14% per deficit, implying that frailty is associated with a greater burden of mortality in an Aboriginal population.

Strengths of our study include a representative sample, high response fractions (94.3% at W1 and 76.5% at W2), electronic record linkage to capture mortality, minimum follow-up of 5 years, and use of questionnaires specifically developed and/or adapted for this population. To our knowledge, this is the first such study of frailty in any Indigenous population in the world. Limitations include the modest sample size (given the difficulty and cost of field work in remote locations this is difficult to overcome), and the possibility of recall and response bias. Aspects of the latter (e.g., acquiescence bias) were hopefully minimised by use of trained Aboriginal research assistants in the field. Non-response bias (specifically at W2) is possible, and thus we may have somewhat overestimated prevalence and incidence of frailty. However, non-response was relatively low, particularly at W1 (when the prevalence of frailty was already very high). Furthermore, we have recently shown that non-response probably results in the underestimation of frailty in the general population, as non-respondents are more likely to be frail [33]. Whether this is also true for Aboriginal people is unknown, although in this study non-responders were more likely to be frail at baseline than responders (67.7% vs. 59.8%). However, this was based on relatively small numbers and did not reach statistical significance. An additional limitation of this study is our use of a frailty index comprising only 20 items. Adding further items to the index will improve its precision, up to a point [13]. Nonetheless, given the purpose of the original study (to investigate dementia in this population) we had a limited number of general health-related items to choose from. Of these, we selected all that were relevant. However, at the second wave of the study, participants answered a questionnaire assessing a wider range of health domains. At this time-point, it was possible to construct a larger frailty index (comprising 8 additional items). Owing to the longitudinal nature of this study, we were required to use the same frailty index at both waves, and hence could not use the larger frailty index. However, to investigate what the effect of using a larger frailty index might be, we compared the prevalence of frailty at wave 2 using the two indexes. Reassuringly, there was no statistically significant difference in the prevalence of frailty (69.8% with the larger index vs. 67.6% with the original), suggesting that our original 20-item index is a robust measure. Some participants had missing data for the frailty index, but excluding these participants left our results essentially unchanged. At wave 1 there were 30 people (8.3%) with more than 4 missing items in the frailty index, while at wave 2 there were 69 people (37.9%) with more than 4 missing items. Excluding these participants changed the proportion of frail individuals at wave 1 from 65.3% to 63.1%, while at wave 2, the proportion changed from 67.6% to 62.8%. A final issue to consider is our choice of a cut-off of 0.2 to indicate frailty. We followed Searle et al.'s guidance in this regard, although cut-points as high as 0.25 have been suggested [12]. If we were to apply this higher cut-off to our sample, the prevalence of frailty at wave 1 changes from 65.3% to 49.0%. While significantly lower, the prevalence remains astonishingly high given the age of our sample, being comparable to either a very elderly or institutionalised population. We acknowledge that the prevalence of frailty reported may have been influenced by the measurement tool used. It is particularly difficult to speculate on the Fried frailty phenotype as it relies on grip strength and we did not measure this factor, nor are there norms in Aboriginal populations. Both the Frailty Index and the Fried scale have been shown to comparably identify older people at risk of death and correlate well with each other, [12,34] but there are large variation in population prevalence even for similar types of populations [35]

In conclusion, remote-living Aboriginal Australians are more likely to be, or become frail, than the general population. Frailty was also associated with very high mortality. The prevalence and

incidence of disability were also high, but less so than frailty, suggesting there may be greater need for programs targeting frailty and ageing-related support than disability. Our work highlights an urgent need for further research with Aboriginal people to determine the factors associated with the development of frailty (which may be different from those in the general population), and to investigate frailty in Aboriginal Australians in urban settings and other indigenous populations. Finally, research to develop culturally-appropriate programs to prevent or perhaps even reverse frailty in this population should be prioritised.

Disclosure statement

The authors have no conflicts of interest to declare.

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Author contributions

Dina Lo Giudice, Leon Flicker, Kate Smith, Stephen Fenner, and David Atkinson conceived and designed the study. Zoë Hyde performed the statistical analyses and wrote the initial draft of the manuscript. Dina Lo Giudice, Leon Flicker, Stephen Fenner, Linda Skeaf, and Roslyn Malay collected the data. All authors reviewed and revised the manuscript for intellectual content, and provided approval for its submission.

Peer review

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.maturitas.2016.02.013.

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