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Elevated fasting glucose and albuminuria may be a marker for all-cause mortality in Indigenous adults in North Queensland - a follow up study, 1998–2006

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ABSTRACT

Aims: To document risk factors of all-cause mortality in a cohort of indigenous Australians from 23 communities of North Queensland during 1998–2006.
Methods: Among 2787 indigenous adults, baseline weight, waist circumference, blood pressure, fasting glucose, lipids, gamma-glutamyl transferase, urine albumin creatinine ratio, smoking, alcohol intake and physical activity were measured in 1998–2000. Deaths were ascertained from State Registry of Deaths, hospitalization and clinical records till 2006. Mortality risk factors were assessed using a Cox proportional-hazards model.
Results: The standardized all-cause mortality rate was 23.2/1000 person–years (95% CI 20.3–26.3/1000 pys). After adjusting for age, sex, and ethnicity, baseline plasm fasting glucose > = 5.5 mmol/L was associated with a 50% increased risk of death (HR 1.5, 95% CI 1.2–2.0). Albuminuria was associated with all-cause mortality with a hazards ratio of 1.4 for microalbuminuria (95% CI 1.0–1.9) and 2.6 (95% CI 1.8–3.7) for macroalbuminuria. Gamma-glutamyl transferase > = 50 IU was associated with an increased risk of all-cause mortality by 40% (95% CI 1.04–1.8).
Conclusions: Fasting glycaemia, albuminuria, and gamma-glutamyl transferase, may be a marker for all-cause mortality within this cohort.

1. Introduction

Australia enjoys a high life expectancy of 80.9 years for men and 84.8 years for women at birth in 2015 (WHO, 2016). There are, however, significant inequalities between indigenous and non-indigenous Australians regarding incidence and prevalence of chronic conditions and mortality (Australian Institute of Health and Welfare-AIHW, 2014). Indigenous Australians are the Aboriginal and Torres Strait Islander people (TSI) of Australia who descended from groups that existed in Australia and surrounding islands prior to European colonization (collectively called indigenous people in this paper). Based on the AIHW National Mortality Database of deaths registered during 2009–2011, all-cause mortality in indigenous people was twice that of non-indigenous Australians, despite an overall decrease in the death rate of 6% between 2001 and 2011. There were large regional differences among indigenous people, with those in remote locations suffering higher mortality than those in urban and regional areas (AIHW, 2014). Among the indigenous populations of Australia, New Zealand, Canada, and the US, Australia has the largest mortality gap between indigenous and non-indigenous people, based on national mortality data (Bramley, Hebert, Jackson, & Chassin, 2004; Hill, Barker, & Vos, 2007). Although national mortality data are useful for cross-country comparisons and for broad trend estimates, significant bias in both indigenous identification in state data collections, and census estimates of denominator populations coupled with known regional differences in morbidity and mortality make local interpretation difficult (Williams, Najman, & Clararino, 2006). Cohort studies provide an opportunity to make more accurate estimates of mortality risk, as well as enabling an examination of important and modifiable risk predictors for premature death.

The aim of this study was to investigate all-cause mortality and its associated risk factors in a cohort of indigenous Australians from 23 communities of North Queensland during 1998–2006.

2. Methods

2.1. Study population

Baseline data were collected on participants belonging to 23 rural communities in Far North Queensland (the northernmost part of the state of Queensland, Australia) from a community wellness screening project between 1998 and 2000. Methods for this study have been

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reported in detail elsewhere (Miller et al., 2002). All indigenous residents of the communities aged 13 years and over, were invited to attend a health check. Based on the local census data, the study achieved a participation rate of 44.5% with greater participation noted in smaller communities, and participants overall were not different demographically from the Australian indigenous population as a whole (Miller et al., 2002). Written informed consent was obtained from participants. The study protocols were approved by the Cairns Base Hospital Human Research Ethics Committee with support from the peak Indigenous Health Organizations, Apunipima Cape York Health Council and the Torres Strait and Northern Peninsula Area Health Council. For the purposes of this analysis, 2787 indigenous adults were included. Adults were defined as biological adults that are aged 15 years and over.

2.2. Measurements

Participants wearing light clothing were weighed to the nearest 0.1 kg. Height and waist circumference (WC) were recorded to the nearest centimeter with the latter measured by the same technician at the level of the umbilicus. Blood pressure (BP) was the average of three measurements using a Dinamap model 800 automated blood pressure monitor taken while seated after 10 min rest (Critikon, Tampa FL, US).

Fruit and vegetable intake, tobacco and alcohol consumption were assessed using a methodology from the National Nutrition Survey 1995 and categorized by the Australian dietary guidelines (National Health and Medical Research Council, 2013). Alcohol drinkers were those people who had consumed any alcohol within 7 days prior to the survey. Physical activity was measured using a 7-day recall method and was categorized by the WHO criteria in which 'enough' means doing moderate to vigorous physical activity for more than 30 min/day for 5 days in the week before the survey.

Blood glucose, total cholesterol, high density lipoprotein (HDL), triglycerides, and Gamma-glutamyl transferase (GGT), were measured on fasting blood collected in the early morning by a medical officer, registered nurse or trained phlebotomist (Miller et al., 2002). Plasma glucose and lipids were measured using photometric enzyme endpoint assay with Cobas Integra 700/400 (Roche Diagnostics, www.roche-diagnostics). Fasting plasma glucose was categorized using 5.5 mmol/L as a cut-off point, because elevated glucose (> = 5.5 mmol/L) was associated with increased risk of diabetes, hypertension, and coronary heart diseases incidence in this population (Li, McCulloch, & McDermott, 2012; Li & McDermott, 2015; McDermott, Li, & Campbell, 2010; McDermott, McCulloch, & Li, 2011). Additional analysis with glucose quintile, quartiles or tertiles revealed a threshold effect of the level on mortality. GGT was measured using the kinetic photometric procedure with Cobas Integra 800 (Roche Diagnostics, www.roche-diagnostics). Elevated GGT was defined as GGT > = 50 IU and normal as a GGT < 50 IU for those aged 4 months to 120 years old by Queensland Pathology Service, the main provider of public sector pathology services in Queensland (http://www.health.qld.gov.au/qhcss/qhps/default.asp).

Urine specimens provided by participants in sterile 50 mL containers from the first morning void were obtained. The samples were tested using dipstick urinalysis (Combur-test, Roche) for protein, pH, nitrites, leucocytes and blood. Urine albumin creatinine ratio (UACR) was measured by immunoassay in mg/mmol. Microalbuminuria was defined as UACR > = 2.5 mg/mmol for males and > = 3.5 for females, and macroalbuminuria as UACR > 25 mg/mmol for males and >35 mg/mmol for females (Johnson et al., 2012). All pathology testing was conducted at the Cairns Base hospital and the coefficient of variation of glucose and lipids ranged between 1.8% and 3.9%.

Hypertension was defined as BP > = 140/90 mmHg, or an established hypertension diagnosis in clinic files. Diabetes was defined as either clinical diagnosis verified by the participants' medical records or a 2 h glucose tolerance test, or fasting plasma glucose level > = 7.0 mmol/L. Overweight was defined as BMI > = 25 Kg/m^2 and obesity as BMI > 30 Kg/m^2. Abdominal overweight was defined as WC > = 80 cm in females and 94 cm in males, and obesity as WC > = 88 cm in females and 102 cm in males.

2.3. Record matching and outcome determination

Death and hospitalization records for consenting participants were identified via a manual search (by a registered nurse with experience working in the region) and linked with baseline data by probabilistic matching method using name, date of birth, and gender. Matching of death records was performed manually at the Queensland Registry of Births, Deaths and Marriages. Date and cause of death were extracted in combination with hospitalization records. Unregistered deaths were confirmed where life extinct forms were observed at health centers because delays in registration of deaths are more common for aboriginal and TSI people.

The censor date for follow-up was 1 January 2006, as this marked the commencement of the matching of WPHC identification numbers to hospital unit record numbers and matching of death records. The leading cause of death was generated using a WHO recommended method (Becker, Silvi, Fat, L'Hours, & Laurenti, 2006).

2.4. Statistical analysis

The follow up period for death was the time from the baseline study to death. For 65 deaths without death date, a random date was assigned because the missing was random by baseline measurements. For the rest, the follow up period was the interval between the date of baseline survey and the follow up or the censor date. The age- and sex-specific incidence rate stratified by ethnicity was calculated by dividing the number of new cases by the total follow-up person–years of the corresponding subgroups. Direct standardization was conducted using the 2007 Australian Bureau of Statistics (ABS) national data as the reference population. Cumulative mortality was also calculated for a range of baseline variables, including ethnicity, BMI, WC category, fasting glycaemia category, diabetes, hypertension, fasting triglyceride, HDL, UACR, smoking and drinking category, and compared using log-rank tests. Hazard ratios (HR) were generated from Cox proportional-hazards model after testing proportional-hazards assumption using Schoenfeld residuals and graphic assessment by "stphplot" command. Adjusted HRs were obtained by including baseline age, sex, and ethnicity in Cox models. Adjusted population attributable fractions for significant risk factors were generated using the "punafcc" function in Stata, assuming death and the factors are causal. The "punafcc" estimates parameters for general population by normalizing and variance-stabilizing transformations to generate confidence limits after the parameters of a regression model has been estimated (Newson, 2013). All analyses were conducted using Stata v13 (StataCorp, College Station, Texas, USA).

3. Results

3.1. Characteristics of the study cohort and death during the follow up

Baseline health status differed significantly between TSIs and aboriginal adults. Specifically, the TSIs had higher BMI, WC, BP, fasting plasma glucose, but lower levels of triglycerides, HDL, and GGT. The TSIs also had lower self-reported prevalence of smoking and alcohol drinking (Appendices Table A.1).

During an average of 6 years of follow up, 232 individuals (8.3%) were identified as deceased, with a mean baseline age of 52.6 years. Of those deceased, 126 (54.3%) were male and 155 (66.8%) were