

ORIGINAL RESEARCH

Chronic condition risk factor change over time in a remote Indigenous community

AUTHORS



Kylie Strate¹ MB BS FRACGP, GP *

Julie Brimblecombe² PhD, Associate Professor Public Health and Nutrition

Louise Maple-Brown³ MB BS FRACP PhD, Senior Principal Research Fellow and Endocrinologist

Joanne Garrngulkpuy⁴ Diploma of Education, Community researcher

Elaine Maypilama⁵ PhD, Research Fellow

Maria Scarlett⁶ BSSc, Retired



Kerin O'Dea⁷ PhD, Professor

Elizabeth LM Barr⁸ PhD, Research Fellow

CORRESPONDENCE

*Dr Kylie Strate kyliestrate@hotmail.com

AFFILIATIONS

¹ Menzies School of Health Research, Charles Darwin University, Casuarina, Australia; and Miwatj Aboriginal Health Corporation, PO Box 519, Nhulunbuy, NT 0881, Australia.

² Department of Nutrition, Dietetics and Food, Monash University, Victoria, Australia

³ Menzies School of Health Research, Charles Darwin University, PO Box 41096, Casuarina, NT 0811, Australia; and Division of Medicine, Royal Darwin Hospital, Darwin, Australia

^{4,5} Yalu Marnggithinyaraw Centre, Galiwin'ku community, NT 0822, Australia

⁶ Menzies School of Health Research, Charles Darwin University, PO Box 41096, Casuarina, NT 0811, Australia

⁷ Centre for Population Health Research, University of South Australia, Adelaide, Australia

⁸ Menzies School of Health Research, Charles Darwin University, PO Box 41096, Casuarina, NT 0811, Australia; and Baker Heart and Diabetes Institute, PO Box 6492, Melbourne, Vic., Australia

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ABSTRACT:

Introduction: This study sought to determine change in chronic condition risk factors in a remote Indigenous community following a 3-year period of community-led health promotion initiatives.

Methods: Data were compared between two cross-sectional surveys of Indigenous Australian community residents before and after health promotion activities, and longitudinal analysis of participants present at both surveys using multilevel mixed-effects regression.

Results: At baseline, 294 (53% women; mean age 35 years) participated and 218 attended the second survey (56% women, mean age 40 years), and 87 attended both. Body composition, blood pressure and urinary albumin-to-creatinine ratio remained stable between baseline and follow-up. After adjusting for age and sex, haemoglobin A_{1c} significantly increased (from 57 to 63 mmol/mol (7.5% to 8.1%), $p=0.021$) for those with diabetes.

Increases were also observed for total cholesterol (from 4.4 to 4.6 mmol/L, $p=0.006$) and triglycerides (from 1.5 to 1.6 mmol/L, $p=0.019$), and high-density lipoprotein cholesterol levels improved (from 0.98 to 1.02 mmol/L, $p=0.018$). Self-reported smoking prevalence was high but stable between baseline (57%) and follow-up (56%). Similar results were observed in the longitudinal analysis to the cross-sectional survey comparison.

Conclusion: Community-led health promotion initiatives may have had some benefits on chronic condition risk factors, including stabilisation of body composition, in this remote Indigenous community. Given that less favourable trends were observed for diabetes and total cholesterol over a short time period and smoking prevalence remained high, policy initiatives that address social and economic disadvantage are needed alongside community-led health promotion initiatives.

Keywords:

Australia, chronic disease, health promotion, Indigenous Australians.

FULL ARTICLE:

Introduction

Chronic conditions including cardiovascular disease and type 2 diabetes mellitus contribute an estimated 80% to the significant gap between Indigenous and non-Indigenous life expectancy in Australia. These conditions appear to have been rare or non-existent prior to European colonisation¹. Despite the efforts of the Close the Gap campaign, chronic conditions continue to increase in Indigenous communities in Australia^{2,3}. Intergenerational social inequity and economic disadvantage underpin the rise in chronic conditions because they impact all aspects of life, including health-related behaviours, and access to health and other services^{1,4}. Furthermore, an imposed food system has resulted in a rapid change for Australian Indigenous peoples from the traditional lifestyle – characterised by high levels of physical activity and a moderate energy, nutrient-dense diet – to a more ‘westernised’ lifestyle, characterised by a high-calorie, nutrient-poor, processed diet and low physical activity^{5,6}.

Traditional risk factors for these chronic conditions are those identified from the Framingham Heart Study and include smoking, total cholesterol, low high-density lipoprotein (HDL) cholesterol, systolic blood pressure (BP) and diabetes⁷. These risk factors have been reported to be more prevalent in Indigenous Australians compared to non-Indigenous Australians⁸; however, risk of future coronary heart disease in Indigenous Australians has been significantly underestimated when only traditional risk factors were

included in risk prediction models⁹. Hence, additional risk factors need to be considered in determining risk. These include body composition and body fat distribution, albuminuria, dyslipidaemia (high triglycerides and low HDL cholesterol), impaired fasting glucose and impaired glucose tolerance. Socioeconomic and environmental structural and psychosocial factors also play a significant role in increasing risk of chronic disease in Indigenous populations⁸.

The first nationwide survey of chronic condition risk factors prevalence among Indigenous Australians was recently undertaken in 2012/13, but no follow-up data describing change in risk factors are available yet¹⁰. Prior to this national survey, the only information on risk factors for chronic conditions were from a small number of surveys among selected, mostly remote, Indigenous communities¹¹⁻²¹, with very few studies reporting temporal changes in risk factors²²⁻²⁴. Thus, there is a critical gap in knowledge regarding the follow-up of chronic condition risk factors in Indigenous Australians, particularly in relation to community-wide health promotion campaigns.

The Healthy Lifestyle project²⁵ was established in one remote community in Arnhem Land, in the Northern Territory in Australia, to improve community-led health promotion initiatives that focused on healthy eating and physical activity to prevent diabetes and related chronic conditions²⁵. Arnhem Land is a region of about 79 000 km² in the north-eastern part of the Northern Territory,

with a largely remote Indigenous population (approximately 12 000).

Findings from the baseline survey showed low body mass index (BMI) and absence of diabetes among those aged less than 30 years, but higher BMI and high prevalence of diabetes among middle-aged participants. These findings informed a program of health promotion initiatives that were developed in partnership with community, health and government agencies, and aimed to maintain leanness among younger people and delay the onset of diabetes and related conditions. To measure the impact of these initiatives, a second follow-up survey was undertaken. The authors now report on changes in risk factors between the baseline and follow-up surveys. The aims were to (i) compare risk factor prevalence from two cross sectional surveys at baseline and at follow-up, and (ii) perform a longitudinal analysis of change in risk factors among participants who attended both surveys.

Methods

Study population/participants

In response to community-wide concerns about the increasing impact of chronic diseases, the Healthy Lifestyle project was established as a joint program between a local community organisation and Menzies School of Health Research²⁵. It was the first community-wide screening for chronic condition risk factors in this remote centralised community in north-eastern Arnhem Land, and focused on type 2 diabetes and cardiovascular disease prevention. Methods for the baseline survey have been previously described¹². In brief, all Indigenous residents aged 15 years or more were invited to participate using several recruitment methods. The first survey was conducted between September 2001 and March 2002 and follow-up data collected between July 2003 and July 2004. Median time between baseline and follow up was 1.9 years (698 days).

Between the first and second surveys, 131 discrete community-based cardiovascular disease and diabetes prevention activities were implemented over 3 years by more than 20 different organisations, including the local health centre, Menzies School of Health Research, government and community agencies as previously reported²⁵. Many of the activities were repeated across multiple years, which meant that more than 200 activities were undertaken²⁵. In order to promote a community-directed approach, the prevention activities were based on existing community activity, which was identified by a network of community members and researcher facilitators, and then supported and strengthened. Examples of activities included employing and training community project officers, physical activity workshops, workshops and materials for community gardens, a yearly week-long healthy lifestyle festival and improvements in fruit and vegetable display and quality at the local store.

A community census of residents aged 15 years or more was conducted in April 2002. Of the 706 residents, 378 (54%) participated in the baseline survey. In terms of sex distribution, participants were representative of the census population;

however, older women were over-represented¹². For the present analysis, survey data were available for 294 participants at baseline, and for 218 participants at the second survey. Of these, 87 attended both surveys and had longitudinal data on risk factors. Participants were excluded if they were pregnant ($n=7$ at baseline, $n=3$ at follow-up), and thus 425 participants contributed data to this analysis.

Measures

Local Aboriginal researchers administered a health questionnaire in local language and undertook the clinical examinations with other members of the research team. The same protocol was used for the baseline and follow-up surveys. Height, weight, waist circumference, hip circumference and BP were measured. Self-reported smoking was recorded¹².

Blood samples were obtained for measurement of glucose and lipids after an overnight fast and analysed at a central laboratory (Flinders Medical Centre, Adelaide, South Australia) accredited in the US Centers for Disease Control Lipid Standardisation Program. Haemoglobin A_{1c} (HbA_{1c}) was assayed using cation exchange high-performance liquid chromatography using a Pharmacia MonoS column; and insulin by immunoassay (Abbott AxSYM). Spot early-morning urine samples were collected to determine urinary albumin-to-creatinine ratio (ACR)¹².

Statistical analysis

Diabetes was classified as HbA_{1c} ≥ 48 mmol/mol (6.5%)^{26,27} or if a medical record confirmed diabetes. Impaired glycaemia was defined as HbA_{1c} of 39–48 mmol/mol (5.7–6.5%)²⁶. Hypertension was defined as systolic BP ≥ 140 mmHg and/or diastolic BP ≥ 90 mmHg²⁸. High cholesterol was defined as ≥ 5.5 mmol/L, high triglycerides as ≥ 2.0 mmol/L and low HDL cholesterol ≤ 1.0 mmol/L. Microalbuminuria was defined as ACR ≥ 3 mg/mmol and < 30 mg/mmol; macroalbuminuria was defined as ≥ 30 mg/mmol²⁹. BMI was classified into four groups: < 20 kg/m², 20– < 25 kg/m², 25– < 30 kg/m² and ≥ 30 kg/m²³⁰. Elevated waist-to-hip ratio (WHR) was defined as ≥ 0.90 for men and ≥ 0.85 for women³⁰.

To analyse change in risk factors, multilevel mixed-effects logistic regression was used for categorical variables and multilevel mixed-effects linear regression for continuous variables. These methods took into account that the follow-up included some repeat participants from baseline as well as new participants. HbA_{1c}, fasting glucose, triglycerides, ACR and WHR were not normally distributed and thus were log-transformed before analysis. Models were adjusted for age and sex. Change in prevalence of risk factors was stratified according to three age groups (15–29 years, 30–49 years and > 50 years) and sex. Change in HbA_{1c} was stratified into those with diabetes and those without diabetes. Longitudinal change of risk factors among those individuals who attended both surveys was also assessed using similar regression techniques. Statistical analysis was performed using Stata v14.0 (StataCorp; <http://www.stata.com>).

Ethics approval

Participants provided informed consent and the study was approved by the Joint Human Research Ethics Committee of the Northern Territory Department of Health and Community Services and Menzies School of Health Research (HREC 00-63) following approval from the Aboriginal sub-committee of the HREC, which has veto rights over applications involving Indigenous participants. The local Community Council, who comprise Indigenous Elders, endorsed the study in 2001, and the authors worked in collaboration with researchers.

Results

Cross-sectional analysis

In the first survey, 53% of participants were women and the mean (range) age was 35 (15–75) years. In the second survey, 56% of participants were women and mean (range) age was 40 (15–76) years. Between surveys, self-reported smoking prevalence rates were stable but high (57% in survey 1 and 56% in survey 2, unadjusted, Table 1). When smoking prevalence was stratified by age and sex, smoking tended to increase for women although confidence intervals overlapped, but it remained stable in men for all age groups (Fig1).

The median HbA_{1c} increased from 35 mmol/mol (5.4%) at the first survey to 37 mmol/mol (5.5%) in the second survey (unadjusted, Table 1), which showed borderline significance after adjusting for age and sex ($p=0.047$) (Table 2). The magnitude of increase in the geometric mean of HbA_{1c} was the same in the younger and older age groups, although this change was only significant in the younger age group ($p=0.037$) after adjusting for sex. Significant age-adjusted increases in the geometric mean of HbA_{1c} were observed in women ($p=0.019$), not men ($p=0.786$). When the cohort was stratified into those with and without diabetes, there was no increase in the age- and sex-adjusted geometric mean of

HbA_{1c} in those without diabetes (from 39 to 41 mmol/mol (5.4% to 5.4%), $p=0.339$), and a significant increase in those with diabetes (from 57 to 63 mmol/mol (7.5% to 8.1%), $p=0.021$). The prevalence of diabetes was similar between the first survey (19%) and the second survey (20%); however, impaired glucose tolerance increased from 18% to 22% (Table 1).

Table 2 presents the change in risk factors after adjusting for age and sex. Although there was a significant increase in total cholesterol, there was no significant change in the prevalence of high total cholesterol. There was a small but significant increase in HDL cholesterol levels, and a significant decrease in the prevalence of low HDL cholesterol levels. Triglyceride levels significantly increased, but prevalence of high triglycerides did not change. These results varied between sexes and age groups (Fig1). As expected, the prevalence of high cholesterol increased with age for both men and women. The prevalence of low HDL cholesterol decreased in younger men (from 47% to 34%), women aged 31–50 years (from 74% to 66%), older men (from 72 to 56%) and older women (from 80% to 52%) but it remained stable in younger women (from 47% to 48%) and men in the middle age group (31–50 years) (from 57% to 53%) (Fig1).

Mean BMI and WHR remained essentially unchanged between baseline and follow-up (Table 2). The prevalence of a high WHR increased in the younger age group in men (from 19% to 41%) and women (from 65% to 73%) but decreased in the older age group in men (from 100% to 81%) and women (from 95% to 74%) (Fig1).

Mean ACR levels and prevalence of albuminuria were similar for the first and second surveys (Tables 1,2). Although systolic and diastolic BP were similar in the first and second surveys, the prevalence of hypertension significantly reduced (odds ratio (OR)=0.31, $p=0.019$).

Table 1: Cohort characteristics at first and second surveys: the Healthy Lifestyle Study

Characteristic	Survey 1: 2001/2		Survey 2: 2003/4	
	Value (n (%), mean (SD) or median (25th, 75th percentile))	Total n	Value (n (%), mean (SD) or median (25th, 75th percentile))	Total n
Age, years	35 (13)	294	40 (13)	218
Men, n (%)	139 (47%)	294	96 (44%)	218
Women, n (%)	155 (53%)	294	122 (56%)	218
Diabetes, n (%) [†]	55 (19%)	294	44 (20%)	218
Impaired glycaemia, n (%) [‡]	52 (18%)	294	49 (22%)	218
Current smoking, n (%)	155 (57%)	273	121 (56%)	218
HbA _{1c} , mmol/mol	35 (32, 40)	294	37 (34, 41)	218
HbA _{1c} , %	5.4 (5.1, 5.8)	294	5.5 (5.3, 5.9)	218
Fasting glucose, mmol/L	5.2 (4.9, 5.6)	290	4.9 (4.7, 5.5)	166
Cholesterol, mmol/L	4.4 (0.9)	294	4.7 (1.0)	218
Triglyceride, mmol/L	1.4 (0.9, 2.1)	294	1.8 (1.2, 2.5)	218
HDL cholesterol, mmol/L	1.0 (0.3)	294	1.0 (0.3)	218
Cholesterol:HDL cholesterol	4.7 (1.6)	294	5.0 (1.5)	218
High cholesterol, n (%)	32 (11%)	294	35 (16%)	218
Low HDL cholesterol, n (%)	176 (60%)	294	117 (54%)	218
High triglycerides, n (%)	83 (28%)	294	84 (39%)	218
Albumin-to-creatinine ratio	1.1 (0.5, 3.5)	294	1.0 (0.5, 3.4)	218
Microalbuminuria, n (%) [§]	58 (20%)	294	44 (20%)	218
Macroalbuminuria, n (%) [‡]	19 (6%)	294	16 (7%)	218
Height, m	1.68 (0.08)	294	1.68 (0.08)	218
Weight, kg	64 (15)	294	66 (16)	218
Waist circumference, cm	86 (14)	294	87 (16)	218
Hip circumference, cm	93 (9)	294	93 (10)	218
Systolic BP, mmHg	118 (22)	294	120 (24)	218
Diastolic BP, mmHg	70 (11.5)	294	71 (11)	218
Hypertension, n (%) [‡]	49 (17%)	294	28 (13%)	218
BMI, kg/m ²	23 (5)	294	23 (6)	218
BMI categories, kg/m ²				
<20	105 (36%)		63 (29%)	
20–<25	96 (33%)		80 (37%)	
25–<30	69 (23%)		52 (24%)	
≥30	24 (8%)		23 (11%)	
Waist-to-hip ratio	0.9 (0.9, 1.0)	294	0.9 (0.9, 1.0)	218
Elevated waist-to-hip ratio, n (%)	194 (66%)	294	164 (75%)	218

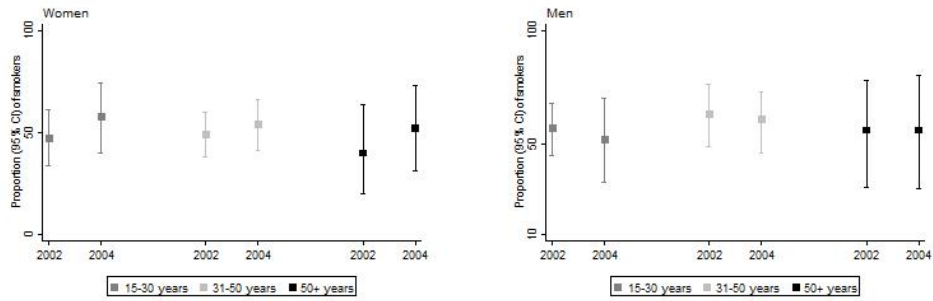
[†] Diabetes classified as HbA_{1c} ≥48 mmol/mol (6.5%), participant self-reported diabetes or their medical record confirmed diabetes.
[‡] Impaired glycaemia defined as HbA_{1c} between 38 mmol/mol (5.7%) and 48 mmol/mol (6.5%).
[§] Microalbuminuria defined as albumin-to-creatinine ratio ≥3 mg/mmol and <30mg/mmol.
[‡] Macroalbuminuria defined as albumin-to-creatinine ratio ≥30 mg/mmol.
[‡] Hypertension defined as systolic ≥140 mmHg or diastolic ≥90 mmHg.
 BMI, body mass index. BP, blood pressure. HbA_{1c}, haemoglobin A_{1c}. HDL, high-density lipoprotein. SD, standard deviation.

Table 2: Change in chronic disease risk factors between first and second surveys: the Healthy Lifestyle Study (Data are coefficient for continuous variables and odds ratios for categorical variables. All models adjusted for age and sex)

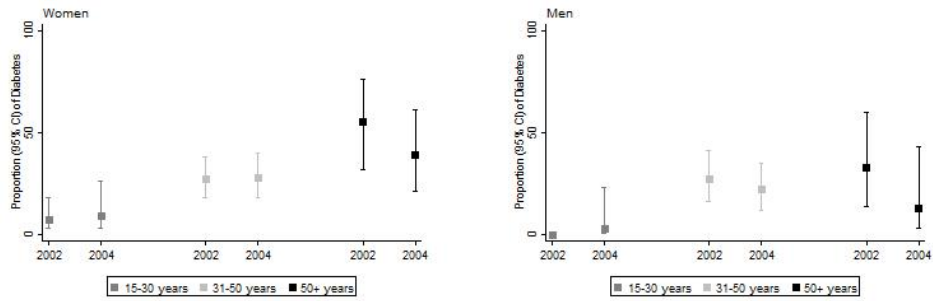
Characteristic	Coefficient or OR	95%CI	p-value	Round 1 mean	Round 2 mean
Diabetes [†]	OR 0.98	0.34, 2.78	0.968	–	–
Current smoking	OR 1.10	0.55, 2.22	0.782	–	–
HbA _{1c} , mmol/mol [‡]	0.03	0, 0.06	0.047	38	39
HbA _{1c} , % [‡]	0.02	–0.001, 0.04	0.066	5.7	5.8
Fasting plasma glucose, mmol/L [‡]	–0.05	–0.1, –0.01	0.028	5.7	5.4
Cholesterol, mmol/L	0.2	0.05, 0.3	0.006	4.4	4.6
Triglycerides, mmol/L [‡]	0.09	0.02, 0.17	0.019	1.5	1.6
HDL cholesterol, mmol/L	0.04	0.01, 0.07	0.018	0.98	1.02
Cholesterol:HDL cholesterol	–0.04	–0.2, 0.1	0.617	4.8	4.8
High cholesterol (≥5.0 mmol/L)	OR 1.43	0.66, 3.08	0.367	–	–
Low HDL cholesterol (<1.0 mmol/L)	OR 0.42	0.21, 0.83	0.012	–	–
High triglyceride (≥2.0 mmol/L)	OR 1.62	0.87, 3.01	0.124	–	–
Albumin-to-creatinine ratio [‡]	–0.1	–0.2, 0.1	0.504	1.6	1.5
Albuminuria [§]	OR 0.79	0.33, 1.85	0.592	–	–
Height, m	0.005	0.001, 0.008	0.008	1.68	1.68
Weight, kg	0.3	–0.5, 1.0	0.531	63.9	64.2
Waist circumference, cm	–1.0	–2.8, 0.8	0.280	86.6	85.6
Hip circumference, cm	–1.4	–2.4, –0.4	0.006	92.9	91.5
Systolic BP, mmHg	–1.6	–4.5, 1.3	0.279	119.6	118.0
Diastolic BP, mmHg	–0.8	–2.3, 0.7	0.311	70.9	70.1
Hypertension [‡]	OR 0.31	0.12, 0.83	0.019	–	–
BMI, kg/m ²	–0.1	–0.3, 0.2	0.714	22.7	22.6
Waist-to-hip ratio [‡]	–0.0001	–0.01, 0.02	0.985	0.93	0.93
Elevated waist-to-hip ratio [‡]	OR 1.06	0.55, 2.06	0.859	–	–

[†] Diabetes classified as HbA_{1c} ≥48 mmol/mol (6.5%), participant self-reported diabetes or their medical record confirmed diabetes.
[‡] Natural logarithm taken for each variable before entering into the model. Geometric mean for rounds 1 and 2 are provided.
[§] Albuminuria defined as urine albumin-to-creatinine ratio of ≥3 mg/mmol.
[‡] Hypertension defined as systolic ≥140 mmHg or diastolic ≥90 mmHg.
[‡] Elevated waist-to-hip ratio defined as ≥0.90 for men and ≥0.85 for women.
 BMI, body mass index. BP, blood pressure. CI, confidence interval. HbA_{1c}, haemoglobin A_{1c}. HDL, high-density lipoprotein. OR, odds ratio.

A: Current smoking

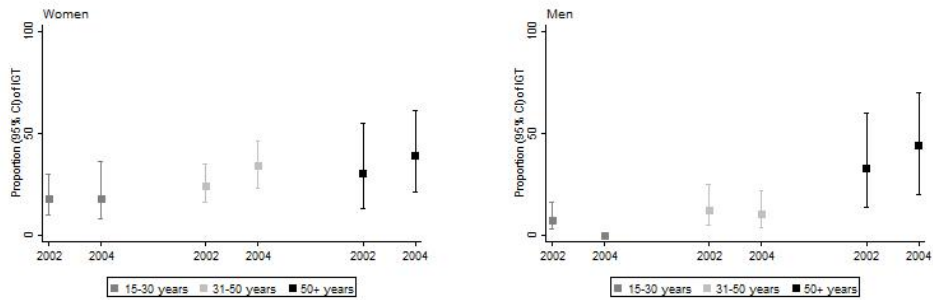


B: Diabetes



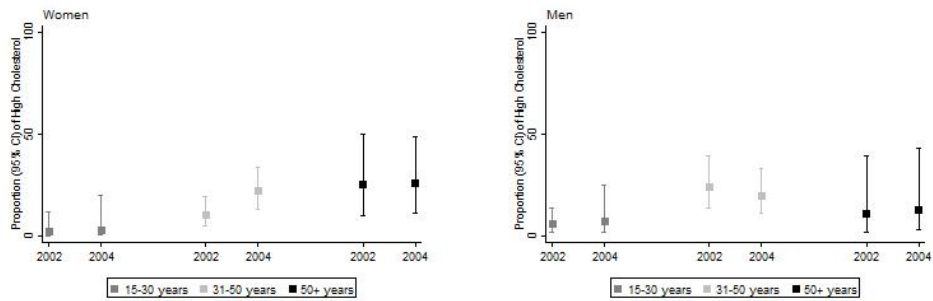
Diabetes classified as $HbA_{1c} \geq 48$ mmol/mol (6.5%), participant self-reported diabetes or their medical record confirmed diabetes.

C: Impaired glucose tolerance

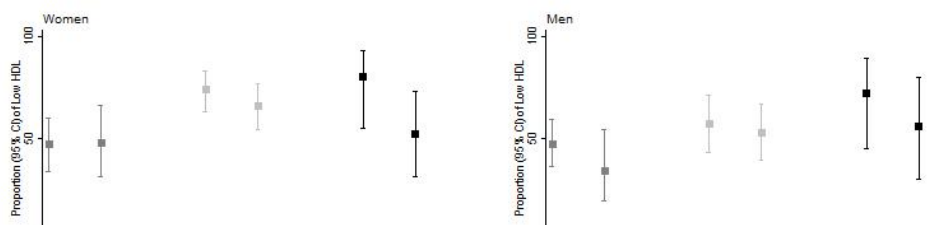


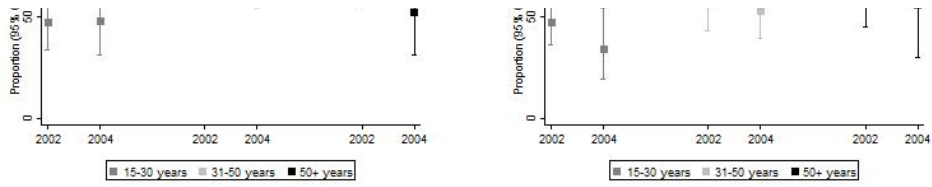
Impaired glycaemia defined by HbA_{1c} between 38 mmol/mol (5.7%) and 48 mmol/mol (6.5%).

D: High total cholesterol (≥ 5.5 mmol/L)

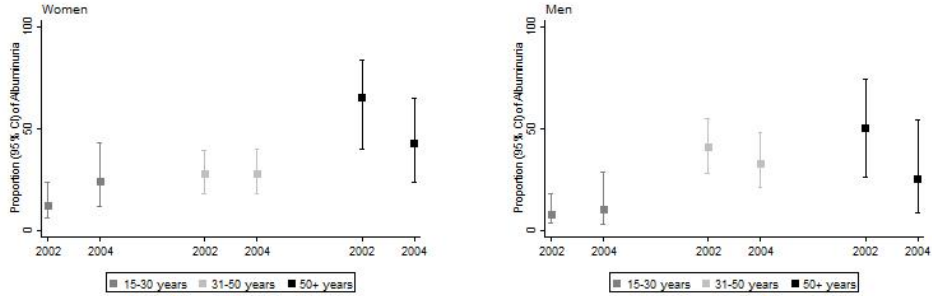


E: Low HDL cholesterol (< 1.0 mmol/L)

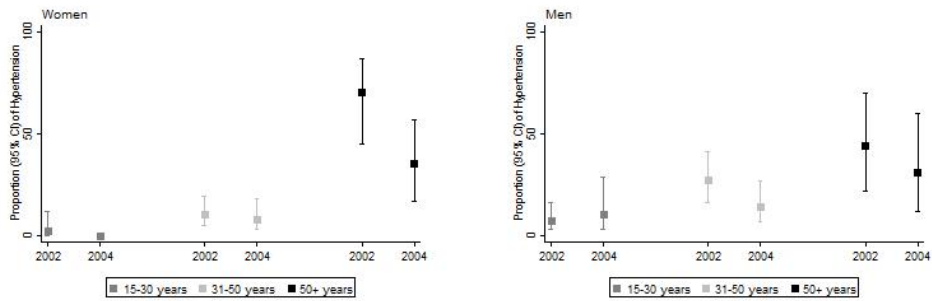




F: Albuminuria (urine albumin-to-creatinine ratio >3 mg/mmol)

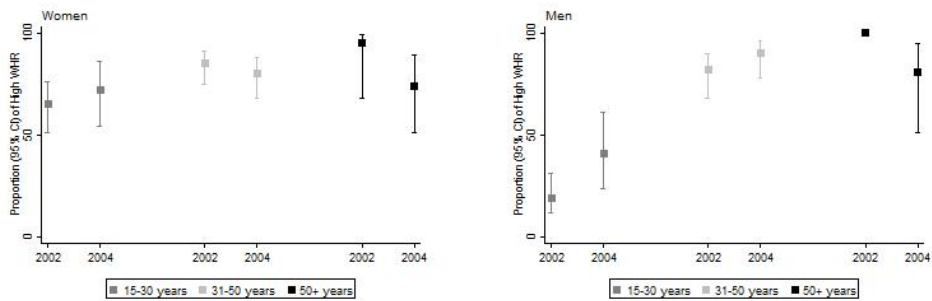


G: Hypertension



Hypertension defined as systolic ≥ 140 mmHg or diastolic ≥ 90 mmHg

H: High waist-to-hip ratio (≥ 0.90 for men and ≥ 0.85 for women)



I: Overweight/obesity according to body mass index (≥ 25 kg/m²)

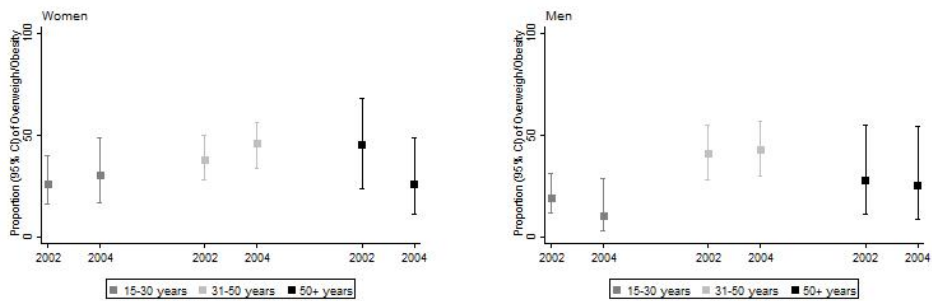


Figure 1: Prevalence of risk factors according to age groups in women and men at first and second surveys.

Longitudinal analysis

Findings from the longitudinal analysis ($n=87$) are reported in Table 3. Age- and sex-adjusted means for HbA_{1c}, glucose, cholesterol, ACR, weight, waist circumference, BMI and WHR were

higher than the cross-sectional results in both the first and second surveys. However, the change in risk factors over time in the longitudinal analysis reflected the same general trends as observed from the cross-sectional analysis, except for HbA_{1c}, which showed a greater increase across the two surveys.

Table 3: Longitudinal analysis of chronic disease risk factors in participants present at both first and second surveys ($n=87$) (Data are coefficient for continuous variables and OR for categorical variables. All models adjusted for age and sex)

Characteristic	Coefficient or OR	95%CI	p-value	Round 1 mean	Round 2 mean
Diabetes [†]	OR 6.57	0.26, 166.64	0.254	–	–
Current smoking	OR 0.82	0.30, 2.25	0.707	–	–
HbA _{1c} , mmol/mol [‡]	0.047	0.01, 0.08	0.012	41	43
HbA _{1c} , % [‡]	0.03	0.01, 0.06	0.016	5.9	6.1
Fasting glucose, mmol/L [‡]	–0.03	–0.10, 0.03	0.309	6.0	5.8
Cholesterol, mmol/L	0.1	–0.03, 0.3	0.135	4.6	4.7
Triglyceride, mmol/L [‡]	0.09	–0.01, 0.20	0.089	1.6	1.8
HDL cholesterol, mmol/L	0.04	0, 0.07	0.051	0.95	0.99
Cholesterol:HDL cholesterol	–0.1	–0.3, 0.1	0.615	5.1	5.0
High cholesterol (≥5.5 mmol/L)	OR 0.87	0.29, 2.53	0.795	–	–
Low HDL cholesterol (<1.0 mmol/L)	OR 0.33	0.13, 0.86	0.024	–	–
High triglycerides (≥2.0 mmol/L)	OR 1.57	0.67, 3.66	0.298	–	–
Albumin-to-creatinine ratio [§]	0.001	–0.19, 0.19	0.994	2.2	2.2
Albuminuria [§]	0.96	0.27, 3.43	0.951	–	–
Height, m	0.004	0.0004, 0.009	0.032	1.68	1.68
Weight, kg	0.2	–0.7, 1.2	0.661	70.2	70.4
Waist circumference, cm	–1.07	–3.4, 1.3	0.372	93.1	92.0
Hip circumference, cm	–1.7	–2.9, –0.5	0.006	96.3	94.7
Systolic BP, mmHg	–1.8	–5.7, 2.0	0.358	120.9	119.0
Diastolic BP, mmHg	–0.8	–2.9, 1.1	0.391	71.5	70.6
Hypertension [‡]	OR 0.33	0.10, 1.14	0.081	–	–
BMI, kg/m ²	–0.1	–0.4, 0.3	0.695	24.9	24.8
Waist-to-hip ratio [¶]	0.004	–0.02, 0.02	0.733	0.96	0.96
Elevated waist-to-hip ratio [¶]	OR 0.64	0.21, 1.95	0.428	–	–

[†] Diabetes classified as HbA_{1c} ≥48 mmol/mol (6.5%), participant self-reported diabetes or their medical record confirmed diabetes.

[‡] Natural logarithm taken for each variable before entering into the model. Geometric mean for rounds 1 and 2 are provided.

[§] Albuminuria defined as urine albumin-to-creatinine ratio of ≥3 mg/mmol.

[‡] Hypertension defined as systolic ≥140 mmHg or diastolic ≥90 mmHg.

[¶] Elevated waist-to-hip ratio defined as ≥0.90 for men and ≥0.85 for women.

BMI, body mass index. BP, blood pressure. CI, confidence interval. HbA_{1c}, haemoglobin A_{1c}. HDL, high-density lipoprotein. OR, odds ratio.

Discussion

This is one of the few studies to report change in the prevalence of chronic condition risk factors over time in an Australian Indigenous community who experienced community-wide health promotion activities over 3 years. The authors observed stable body composition and albuminuria levels, and an increase in HDL cholesterol over time. These findings suggest that the health promotion activities may have had some positive impact. However, given that there was no change in the high smoking prevalence rates, an increase in HbA_{1c} levels for those with diabetes, an increase in the prevalence of impaired glycaemia, and small increases in total cholesterol and triglyceride levels between baseline and follow-up, further efforts to improve health and wellbeing are still required to minimise risk of chronic conditions.

Smoking prevalence in this community was high, but rates were consistent with 2002 national data¹⁰. The findings showed no significant changes in smoking rates. This is inconsistent with national data and other literature that report a reduction in smoking rates over time. The Australian Bureau of Statistics has reported a consistent decline in smoking rates in the Indigenous population: from 49% in 2002 to 41% in 2012/13¹⁰. A study by

Thomas et al also reported a significant decline in national Indigenous smoking prevalence between 1994 and 2008³¹. This equates to less than 1% absolute change in prevalence each year. Similarly, in another Indigenous community, Burgess et al reported a 5% decrease in smoking rates over a 1-year follow up period, following institution of Aboriginal health checks¹³. The health promotion activities in this community predominantly focused on nutrition and physical activity to align with type 2 diabetes prevention²⁵, and thus further programs targeting smoking cessation are required, and may also need to target young women, given that the authors observed increasing trends in smoking rates for this group.

Diabetes prevalence was high in this remote community and remained stable over the time period. Rates were higher than those reported from national data¹⁰, but diabetes prevalence rates are higher in remote than urban Indigenous populations^{13,19,32,33}. The higher mean HbA_{1c} observed in the second survey was largely driven by the higher HbA_{1c} levels among those with diabetes, rather than those without diabetes. These findings indicate that further strategies to improve the management and prevention of diabetes are warranted. Other longitudinal studies of people with

diabetes have demonstrated significant reductions in HbA_{1c} after instituting strategies to improve medical management. An 18-month follow-up study of a community-based health worker outreach program reported a 1% decrease in HbA_{1c}³⁴. Furthermore, there was a reported decrease in HbA_{1c} in the urban-based Fremantle Diabetes Study in Western Australia, consistent with improved diabetes treatment¹⁴. There are challenges, however, to the successful implementation of chronic disease primary health care for Indigenous people, particularly in remote settings. Strategies that identify and address the enablers and barriers that present to healthcare professionals, patients and their communities are required³⁵.

Overall, the authors observed little change in the prevalence of lipidaemia in this community other than an improvement in HDL cholesterol, which may have resulted from store-based health promotion activities focused on improving fruit and vegetable intake. Whilst prevalence of high total cholesterol was relatively lower than national rates¹⁰, rates of dyslipidemia (low HDL cholesterol and high triglycerides) were high. The inertia observed in the prevalence of lipidaemia is surprising given that several of the health promotion activities focused on reducing the consumption of high-fat foods. For example, one program advocated a reduction in the availability of fried foods at the local stores. Other studies of remote communities have reported reductions in total cholesterol levels in the population after the introduction of a community-based nutrition awareness and healthy lifestyle program^{22,23}. However, compared to the activities undertaken in the community in the present study, the programs were undertaken over a longer follow-up period, which may indicate that longer term initiatives are required to achieve change.^{10,22,23,36,37}

Overall, no increase in the prevalence of overweight or obesity was observed over the follow-up period. This contrasts with findings of an increase in BMI over time^{24,38}. The present findings suggest that the health promotion activities that focused on improving nutrition and physical activity may have had some impact maintaining stable mean BMI and diabetes prevalence in this community. However, the follow-up period was relatively short in this study; longer follow-up is required to ascertain whether the positive impact of these activities persists over time.

Although it is concerning that a number of the other chronic disease risk factors assessed worsened over time, it is difficult to conclude whether or not these results would have been even worse without the health promotion campaigns. This is because there was not a control group that was not exposed to these preventive initiatives. Potential reasons for the incomplete improvements in chronic condition risk factors in this community may have been attributable to limited sustainable impact in the areas of organisational and community-level policy²⁵. Despite the involvement of multiple agencies in the community-wide health promotion and integration of health-promoting initiatives in service delivery, the impact of community-level change may have been limited by insufficient short-term funding affecting long-term capacity development among community members and growth of governance structures to coordinate agencies to develop policy²⁵.

Further, community-led health promotion initiatives alone are unlikely to achieve sustained change in the health and wellbeing of populations. It will be important to simultaneously address social and economic disadvantages experienced by many Indigenous communities. Future efforts to improving structural issues such as education, employment, housing, food affordability and public spaces to promote physical activity are required, in conjunction with policies and programs that address intergenerational grief, mental health and racism⁴. Consideration needs to be given to the provision of dedicated funding for community-driven and governed health promotion initiatives and supporting policy to further the benefits observed from the health promotion activities that occurred during this project.

The following limitations of this study should be considered when interpreting the findings. First, the analysis was based on around 50% of the community population, and the follow-up survey under-represented younger people and men. This may have affected the generalisability of the study findings to the whole community. Recruitment to studies in remote communities may be hampered by high mobility of the population: people travel between communities and remote centres for cultural and family reasons. Nonetheless, the first survey population was representative of the community population¹² and attempts to account for age and gender response bias were undertaken in the statistical analyses. Additionally, repeat analysis in the longitudinal cohort showed similar trends in risk factors over time. Second, the authors were not able to compare the results to a comparison control community not undertaking health promotion activities. Third, smoking was based on self-report and thus subject to recall bias, which may have led to inaccurate prevalence estimates, although high rates reflect rates reported from other Indigenous populations¹⁰. Fourth, higher mean values for several risk factors were observed among the longitudinal cohort compared to the cross-sectional analysis, which likely reflected the selection bias of those participants with more chronic disease risk factors being interested in their health and presenting to both surveys. Finally, the data were collected between 2001 and 2004 and may not represent current chronic conditions rates in the community, although the program of work and study processes could still inform future programs. The implementation of electronic medical records data collection in recent years may provide an opportunity to use contemporary data to re-measure rates in this community and provide information on longer term trends.

Further studies on trends in chronic disease risk factors in other communities are required to develop a more complete picture of the changing health status in Indigenous Australians. This may now be possible with the advent of electronic medical records data collection, which will permit assessment of population-level trends in risk factors and conditions over time. Current health promotion activities in Indigenous communities also need to be assessed in terms of their effectiveness from a cultural perspective³⁵. There is limited evidence available to inform policy and practice decisions regarding health promotion initiatives in Indigenous communities, and further research is critical³⁹. The Good Food Planning Tool, developed with the input of this study community, offers a

participatory multi-sector continuous improvement approach to support community level decision-making with regards to food security⁴⁰. Consideration, however, needs to be given to the establishment of dedicated funding of community-based health promotion agencies, whose focus is the development of culturally appropriate, effective and long-term public health initiatives, supported by appropriate government policy to enable the community environment to be a health-promoting one.

Conclusion

This study is an important contribution to the knowledge of chronic disease risk factor trends in a remote Indigenous community on a background of community-wide and community-led health promotion activities. The stability of body composition and increase in HDL cholesterol perhaps reflect the benefits of multisectoral community-wide health promotion initiatives. Nonetheless, the high rates of smoking and dyslipidaemia, the increase in impaired glucose tolerance and increase in HbA_{1c} for those with diabetes over a short period of time require ongoing attention. This analysis provides important quantitative assessment of work undertaken in the community, and it will inform future policy and health promotion initiatives and chronic conditions management that aim to reverse current trends in chronic diseases for Indigenous Australians.

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REFERENCES:

- 1 Australian Institute of Health and Welfare. *The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples 2015*. Canberra: Australian Institute of Health and Welfare, 2015.
- 2 Paradies Y, Cunningham J. Placing Aboriginal and Torres Strait Islander mortality in an international context. *Australian and New Zealand Journal of Public Health* 2002; **26(1)**: 11-16. <https://doi.org/10.1111/j.1467-842X.2002.tb00264.x> PMID:11895018
- 3 Holland C, Close the Gap Campaign Steering Committee. *Progress and priorities report 2016*. Sydney: Australian Human Rights Commission, 2016.
- 4 Marmot M. Social determinants and the health of Indigenous Australians. *Medical Journal of Australia* 2011; **194(10)**: 512-513. <https://doi.org/10.5694/j.1326-5377.2011.tb03086.x> PMID:21644897
- 5 O'Dea K. Westernization and non-insulin-dependent diabetes in Australian Aborigines. *Ethnicity & Disease* 1991; **1(2)**: 171-187.
- 6 Luke JN, Ritte R, O'Dea K, Brown A, Piers LS, Jenkins AJ, et al. Nutritional predictors of successful chronic disease prevention for a community cohort in Central Australia. *Public Health Nutrition* 2015; 1-9.
- 7 Anderson KM, Wilson PW, Odell PM, Kannel WB. An updated coronary risk profile. A statement for health professionals. *Circulation* 1991; **83(1)**: 356-362. <https://doi.org/10.1161/01.CIR.83.1.356> PMID:1984895
- 8 Brown A, Carrington MJ, McGrady M, Lee G, Zeitz C, Krum H, et al. Cardiometabolic risk and disease in Indigenous Australians: the heart of the heart study. *International Journal of Cardiology* <https://doi.org/10.1161/01.CIR.83.1.356> PMID:1984895
- 9 Wang Z, Hoy WE. Is the Framingham coronary heart disease absolute risk function applicable to Aboriginal people? *Medical Journal of Australia* 2005; **182(2)**: 66-69. <https://doi.org/10.5694/j.1326-5377.2005.tb06577.x> PMID:15651963
- 10 Australian Bureau of Statistics. *Australian Aboriginal and Torres Strait Islander Health Survey: first results, Australia, 2012-13*. 2016. <http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/39E15DC7E770A144CA257C2F00145A66?opendocument> (Accessed 10 April 2016).
- 11 Bradshaw PJ, Wilkes ET, Thompson PL. Determinants of carotid intima-medial thickness in an urban Australian Aboriginal population. *Atherosclerosis* 2007; **192(1)**: 218-223. <https://doi.org/10.1016/j.atherosclerosis.2006.05.013> PMID:16860327
- 12 Brimblecombe J, Mackerras D, Garnngulkpuy J, Maypilama E, Bundhala L, Dhurrkay R. Leanness and type 2 diabetes in a

- population of indigenous Australians, et al. *Diabetes Research and Clinical Practice* 2006; **72(1)**: 93-99. <https://doi.org/10.1016/j.diabres.2005.09.014> PMID:16260061
- 13** Burgess CP, Bailie RS, Connors CM, Chenhall RD, McDermott RA, O'Dea K, et al. Early identification and preventive care for elevated cardiovascular disease risk within a remote Australian Aboriginal primary health care service. *BMC Health Services Research* 2011; **11**: 24. <https://doi.org/10.1186/1472-6963-11-24> PMID:21281520 PMCid:PMC3045287
- 14** Davis TM, Hunt K, McAullay D, Chubb SA, Sillars BA, Bruce DG, et al. Continuing disparities in cardiovascular risk factors and complications between Aboriginal and Anglo-Celt Australians with type 2 diabetes: the Fremantle Diabetes Study. *Diabetes Care* 2012; **35(10)**. 2005-2011. <https://doi.org/10.2337/dc12-0225> PMID:22815295
- 15** Gracey M, Burke V, Martin DD, Johnston RJ, Jones T, Davis. Assessment of risks of 'lifestyle' diseases including cardiovascular disease and type 2 diabetes by anthropometry in remote Australian Aborigines. *Asia Pacific Journal of Clinical Nutrition* 2007; **16(4)**: 688-697.
- 16** Hoy WE, McFarlane R, Pugsley DJ, Norman R, Mathews JD. Markers for cardiovascular and renal morbidity: expectations for an intervention programme in an Australian Aboriginal community. *Clinical and Experimental Pharmacology & Physiology* 1996; **23(8)**: S33-S37. <https://doi.org/10.1111/j.1440-1681.1996.tb03039.x> PMID:8886511
- 17** Li M, McDermott RA. Using anthropometric indices to predict cardio-metabolic risk factors in Australian indigenous populations. *Diabetes Research and Clinical Practice* 2010; **87(3)**: 401-406. <https://doi.org/10.1016/j.diabres.2009.12.004> PMID:20034692
- 18** O'Dea K, Cunningham J, Maple-Brown L, Weeramanthri T, Shaw J, Dunbar T, et al. Diabetes and cardiovascular risk factors in urban Indigenous adults: results from the DRUID study. *Diabetes Research and Clinical Practice* 2008; **80(3)**: 483-489. <https://doi.org/10.1016/j.diabres.2008.02.008> PMID:18359533
- 19** McDonald SP, Maguire GP, Hoy WE. Nephrology, dialysis. *Transplantation* 2003; **18(8)**: 1555-1561. <https://doi.org/10.1093/ndt/gfg199> PMID:12897094
- 20** Rowley KG, Gault A, McDermott R, Knight S, McLeay T, O'Dea K. Reduced prevalence of impaired glucose tolerance and no change in prevalence of diabetes despite increasing BMI among Aboriginal people from a group of remote homeland communities. *Diabetes Care* 2003; **23(7)**: 898-904. <https://doi.org/10.2337/diacare.23.7.898> PMID:10895838
- 21** Thompson PL, Bradshaw PJ, Veroni M, Wilkes ET. Cardiovascular risk among urban Aboriginal people. *Medical Journal of Australia* 2003; **179(3)**: 143-146. <https://doi.org/10.5694/j.1326-5377.2003.tb05473.x> PMID:12885283
- 22** McDermott R, Rowley KG, Lee AJ, Knight S, O'Dea K. Increase in prevalence of obesity and diabetes and decrease in plasma cholesterol in a central Australian Aboriginal community. *Medical Journal of Australia* 2000; **172(10)**: 480-484. <https://doi.org/10.5694/j.1326-5377.2000.tb124071.x> PMID:10901770
- 23** Rowley KG, Su Q, Cincotta M, Skinner M, Skinner K, Pindan B, et al. Improvements in circulating cholesterol, antioxidants, and homocysteine after dietary intervention in an Australian Aboriginal community. *American Journal of Clinical Nutrition* 2001; **74(4)**: 442-448. <https://doi.org/10.1093/ajcn/74.4.442> PMID:11566641
- 24** Wang Z, Scott J, Wang Z, Hoy WE. Trends in health status and chronic disease risk factors over 10-14 years in a remote Australian community: a matched pair study. *Australian and New Zealand Journal of Public Health* 2014; **38(1)**: 73-77. <https://doi.org/10.1111/1753-6405.12181> PMID:24494950
- 25** Cargo M, Marks E, Brimblecombe J, Scarlett M, Maypilama E, Dhurrkay JG, et al. Integrating an ecological approach into an Aboriginal community-based chronic disease prevention program: a longitudinal process evaluation. *BMC Public Health* 2011; **11**: 299. <https://doi.org/10.1186/1471-2458-11-299> PMID:21569320
- 26** American Diabetes Association. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes - 2018. *Diabetes Care* 2018; **41(Suppl1)**: S13-S27. <https://doi.org/10.2337/dc18-S002> PMID:29222373
- 27** World Health Organization. *Use of glycated haemoglobin (HbA1c) in the diagnosis of diabetes mellitus*. Geneva: World Health Organization, 2011.
- 28** Gabb GM, Mangoni AA, Anderson CS, Cowley D, Dowden JS, Gollidge J, et al. Guideline for the diagnosis and management of hypertension in adults - 2016. *Medical Journal of Australia* 2016; **205(2)**: 85-89. <https://doi.org/10.5694/mja16.00526> PMID:27456450
- 29** National Kidney Foundation. *KDOQI Clinical Practice Guidelines for Chronic Kidney Disease: Evaluation, Classification, and Stratification 2002*. Available: http://www2.kidney.org/professionals/KDOQI/guidelines_ckd/p5_lab_g5.htm (Accessed 29 June 2016).
- 30** World Health Organization. *Obesity. Preventing and managing the global epidemic*. Report of a WHO Consultation on Obesity. Geneva. World Health Organization. 1997.
- 31** Thomas D. National trends in Aboriginal and Torres Strait Islander smoking and quitting, 1994-2008. *Australian and New Zealand Journal of Public Health* 2012; **36(1)**: 24-29. <https://doi.org/10.1111/j.1753-6405.2011.00817.x> PMID:22313702
- 32** McDermott RA, Li M, Campbell SK. Incidence of type 2 diabetes in two Indigenous Australian populations: a 6-year follow-up study. *Medical Journal of Australia* 2010; **192(10)**: 562-565. <https://doi.org/10.5694/j.1326-5377.2010.tb03636.x> PMID:20477730
- 33** Minges KE, Zimmet P, Magliano DJ, Dunstan DW, Brown A, Shaw JE. Diabetes prevalence and determinants in Indigenous Australian populations: a systematic review. *Diabetes Research and Clinical Practice* 2011; **93(2)**: 139-149. <https://doi.org/10.1016/j.diabres.2011.06.012> PMID:21742398
- 34** McDermott RA, Schmidt B, Preece C, Owens V, Taylor S, Li M, et al. Community health workers improve diabetes care in remote Australian Indigenous communities: results of a pragmatic cluster randomized controlled trial. *BMC Health Services Research* 2015; **15**: 68. <https://doi.org/10.1186/s12913-015-0695-5>

PMid:25884300

35 Gibson O, Lisy K, Davy C, Aromataris E, Kite E, Lockwood C, et al. Enablers and barriers to the implementation of primary health care interventions for Indigenous people with chronic diseases: a systematic review. *Implementation Science* 2015; **10**: 71. <https://doi.org/10.1186/s13012-015-0261-x> PMid:25998148

36 Shemesh T, Rowley KG, Piers LS, Best JD, O'Dea K. Low high-density lipoprotein-cholesterol is the most prevalent metabolic abnormality for Australian Aboriginal men and women even when lean. *European Journal of Cardiovascular Prevention and Rehabilitation* 2008; **15**(1): 49-51. <https://doi.org/10.1097/HJR.0b013e3282f0e327> PMid:18277185

37 Maple-Brown LJ, Brimblecombe J, Connelly PW, Harris SB, Mamakeesick M, Zinman B, et al. Similarities and differences in cardiometabolic risk factors among remote Aboriginal Australian and Canadian cohorts. *Diabetes Research and Clinical Practice* 2013; **100**(1): 133-141. <https://doi.org/10.1016>

[/j.diabres.2012.12.017](https://doi.org/10.1016/j.diabres.2012.12.017) PMid:23312484

38 McDermott R, Campbell S, Li M, McCulloch B. The health and nutrition of young indigenous women in north Queensland - intergenerational implications of poor food quality, obesity, diabetes, tobacco smoking and alcohol use. *Public Health Nutrition* 2009; **12**(11): 2143-2149. <https://doi.org/10.1017/S1368980009005783> PMid:19519970

39 Gibson OR, Segal L. Limited evidence to assess the impact of primary health care system or service level attributes on health outcomes of Indigenous people with type 2 diabetes: a systematic review. *BMC Health Services Research* 2015; **15**: 154. <https://doi.org/10.1186/s12913-015-0803-6> PMid:25889993

40 Brimblecombe J, van den Boogaard C, Wood B, Liberato SC, Brown J, Barnes A, et al. Development of the good food planning tool: a food system approach to food security in indigenous Australian remote communities. *Health Place* 2015; **34**: 54-62. <https://doi.org/10.1016/j.healthplace.2015.03.006> PMid:25912518

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