Original Article

Lack of folate improvement in high risk indigenous Australian adults over an average of 6.5 years: a cohort study

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Socioeconomically vulnerable groups in developed countries suffer excess chronic disease due in large part to an energy dense but nutrient poor diet. Low folate can be a marker of poor dietary quality and is also affected by smoking and chronic alcohol intake, all of which cluster in groups with a low socioeconomic position. A 4.5 to 9 year follow-up study of 567 indigenous adults from remote communities in far north Queensland, Australia, from 1998 to 2007 was conducted. Analysis of the effects of demographic factors, smoking, risky alcohol drinking, fruit and vegetable intake and waist circumference on changes in red cell folate (RCF) status was conducted. Prevalence of low red cell folate doubled in the cohort from a high baseline over this seven year period: 36.9% deficient in 2007, 15.9% at baseline (p<0.001). Smoking was associated with lower folate levels. People with a normal RCF were less likely to be smokers, and were more likely to have a greater number of serves of vegetables (RR 1.06, 95% CI 1.02-1.10) than those who were deficient at follow-up. The introduction of voluntary folate fortification since 1995 does not appear to have impacted on the already poor folate status of this cohort of adults. The increased prevalence of low folate has occurred despite improvements in the food supply, indicating the need for nutrition promotion, and subsidies for healthy food in remote communities. The impact of mandatory folate fortification of flour since 2009 should be assessed in this high risk population.

Key Words: folic acid, indigenous population, policy, nutrition policy, cohort studies

INTRODUCTION

The health benefits of a diet rich in fresh fruit and vegetables are well known. A low fruit and vegetable intake accounts for 2.1% of the total burden of disease in Australia, mainly through increasing the risk of cardiovascular disease and a range of cancers, including lung and stomach cancer.[1] Additionally, fruit and vegetable intake contributes to health inequality, with people from a lower socioeconomic position (SEP) having less access to fruit and vegetables.[2,3] This is thought to result from relatively higher costs for purchasing fruit and vegetables over cheaper, energy dense foods, but also lower quality and availability in shops in lower socioeconomic suburbs or towns.[4,5] For some Aboriginal and Torres Strait Islander communities, low fruit and vegetable access is compounded by living in remote areas, with both an increased cost and a reduced availability.[6,7] In addition, poor housing, particularly poor kitchen facilities may reduce the consumption of vegetables even if they are available to purchase.[8]

Across the population, red cell folate (RCF) levels can indicate the level of fruit and vegetable consumption as well as the general nutritional quality of the diet, given folate can also be found in high quantities in nuts, lentils and liver.[9] Additionally, a program of voluntary fortification of a number of foods including bread and breakfast cereals started in 1995 in Australia, expanding the range of foods that are rich in folate.[10] This program was reported to have led to halving the prevalence of low folate by 2001 in a general Australian population cohort study,[11] but it has not been evaluated for its effectiveness in remote Aboriginal or Torres Strait Islander populations. Folate levels are thought to also be influenced by health behaviours other than dietary intake, including tobacco smoking and chronic alcohol consumption. It is still unclear whether the effects of smoking are through an association between low dietary quality and tobacco smoking alone, or contributed to by direct and indirect metabolic effects of tobacco on folate.[12] There is evidence, however, that alcohol has direct effects on folate by impairing intestinal folate absorption and the bioavailability of folate.[13] Smoking, chronic alcohol intake and low dietary quality tend to adversely cluster in populations with a lower SEP,[14] indicating potentially complex causal path-

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ways to development of folate deficiency for people from a lower SEP and its serious concomitant risks, including neural tube defects.[15]

Aboriginal and Torres Strait Islanders have a well-documented higher prevalence of various risky health behaviours, as well as an alarming increased prevalence of cardio-metabolic diseases, cancer and premature death, relating to a low SEP and the effects of colonization.[16] The Well Person's Health Check (WPHC) cohort study in North Queensland conducted in 19 remote communities has previously documented high levels of tobacco smoking, risky alcohol drinking and also high prevalence of obesity in the predominately Aboriginal and Torres Strait Islander population, all of which may affect folate levels.[17] To explore trends in dietary intake of this high risk population, we analysed data from a small cohort who were followed up from the original WPHC between 4.5 and 9 years. The study was approved by the Cairns and Hinterland Health Service District Ethics Committee, with support from the relevant peak Indigenous health councils.

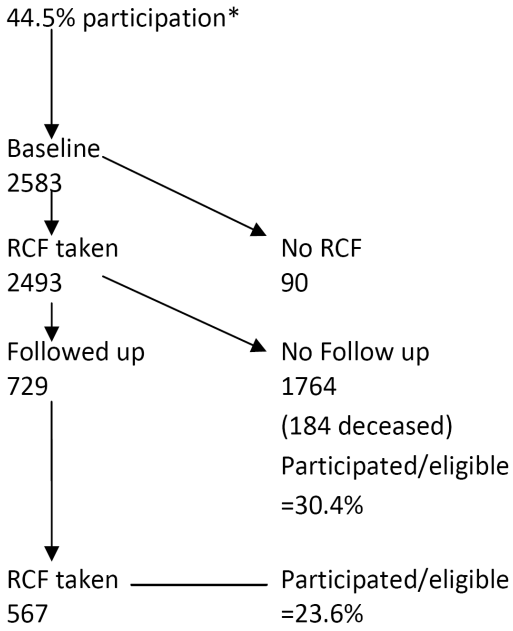

Figure 1. Response rate for Well Person's Health Check, 1998-2007. *Estimated from Census data

MATERIALS AND METHODS

Study population

Baseline data were collected from 2583 people in 19 rural Indigenous communities in Far North Queensland, who participated in the "Well Person's Health Check" between 1998 and 2000 (methods for this cross-sectional study have been reported in detail elsewhere).[18] Based on the local census data, the study achieved a participation rate of 44.5% with greater participation noted in smaller communities. The follow up data were collected between 2004 and 2007 from 729 participants. The sample for this study was taken from those who participated both in the baseline and follow up survey (n=729) and had an RCF level taken both at baseline and follow-up (n= 567, the 'folate cohort'). Details of the response rate at each stage are included in Figure 1.

Variables

Fruit and vegetable intake was assessed as the reported number of serves of fruit and vegetables consumed in the 24 hours prior to the survey, using a pictorial guide to illustrate serves.[18] Alcohol intake was recorded and those who drank alcohol were asked to recall the types and quantity of alcohol consumed in the previous seven days. 'Risky' drinking was defined as more than an average of two standard drinks per day or more than four on any single occasion for women and an average of more than four standard drinks per day or more than six on any single occasion for men.[19] Current smokers were defined as report of current smoking of at least one cigarette daily.

Height and weight were measured, with weight recorded to the nearest 0.1 Kg and height recorded to the nearest centimetre. Body mass index was calculated as weight (kg) divided by the height squared (m^2) and categorised by using WHO definitions of overweight and obesity.[20] Waist circumference was measured to the nearest centimetre by the same technician midway between the iliac crest and bottom of the rib cage.[21]

Fasting venous blood specimens for RCF were collected by trained health staff in a four-millilitre ethylenediamine tetra-acetic acid (EDTA) vacuum tube. Blood tubes were sealed, packed in refrigerated eskies, and transported by air to the laboratory. Red cell folate at baseline was measured using the Bayer Advia Centaur automated immunoassay system (Bayer, Australia) by Queensland Health Pathology Service in Brisbane (reference range for this assay is 295-1800 nmol/L) with RCF deficiency defined as less than 295 nmol/L. The follow up RCF test was measured using the Folate BA assay on the Centaur analyser, and the normal reference range for this assay was 634 nmol/L or higher. For each test, haematocrit was measured to calculate the RCF concentration. To account for the different tests, the RCF data were standardised (to a mean of zero and standard deviation of one for each of the baseline and follow up tests separately), allowing comparison of the baseline and follow up RCF in analyses.

Analysis

A descriptive analysis of demographic factors and risk factors of the selected baseline RCF cohort sample and the whole WPHC baseline sample was undertaken, with differences tested with a t test or chi square test. A chi square test was used to determine if there was a difference in the proportion deficient in folate between the two surveys. The association between demographic variables (only ethnicity categories Aboriginal or Torres Strait Islander were analysed due to small numbers in the other categories) and risk factors and follow-up standardised RCF level were analysed using a linear regression model, controlling for baseline standardised RCF level. These models were then repeated with adjustment for gender, age and ethnicity (Aboriginal, Torres Strait Islander, Aboriginal and Torres Strait Islander, non-Indigenous). Finally, an analysis was conducted to examine associations between demographic variables and any potential protective factors, and being in the normal RCF range at follow up (combining those who remained normal at follow up

and those who became normal at follow up but were deficient at baseline), compared with being deficient at follow up (combining those who remained deficient and those who became deficient after being normal at baseline). This was undertaken with a generalized linear model using a Poisson distribution with robust variance estimators, with estimation of a relative risk (RR). All analyses were conducted using Stata statistical software v 10.0.[22]

RESULTS

Table 1 compares the baseline characteristics of those followed-up (hereafter the 'folate cohort') with those who participated at baseline but not at follow up. The folate cohort were mostly Aboriginal, Torres Strait Islander or both Aboriginal and Torres Strait Islander (98.6%). There was a high prevalence of smoking, risky alcohol drinking and a high mean body mass index, and a correspondingly low intake of fruit and vegetables. Those followed up were more likely to be older, heavier and Torres Strait Islanders and less likely to be smokers or to drink alcohol than those who were not followed up. The distribution of standardised RCF levels at baseline and follow up are presented in Figure 2. According to the reference range for each of the baseline and follow up RCF tests, 15.9% were deficient at baseline, and 36.9% were deficient at follow up ($p<0.001$). Among women of child bearing age (15 to 45 years) 12.8% were deficient at baseline and 33.6% at follow up ($p=0.06$, data not shown). Although

Table 1. Baseline participant characteristics of those who were followed-up (folate cohort), compared with those who participated in the baseline but not the follow-up survey, Well Person's Health Check, 1998-2007, n=2583

Baseline characteristics	Selected sample (n=567) % unless otherwise indicated (95% CI)	Baseline not in follow up survey (n=2016) % unless otherwise indicated (95% CI)
Age, mean (SD)	39.6 (14.9)	36.0 (15.6)*
Female	49.7 (45.6-53.9)	50.7 (48.6-52.9)
Aboriginal	31.7 (27.9-35.6)	49.7 (47.5-51.8)*
Torres Strait Islander	54.9 (50.7-59.0)	28.7 (26.7-30.6)*
Aboriginal and Torres Strait Islander	12.0 (9.3-14.7)	4.9 (4.0-5.9)*
Non-indigenous	1.4 (0.4-2.4)	16.8 (15.1-18.4)*
Smoker	49.7 (45.6-53.9)	55.1 (52.9-57.3)*
Alcohol drinker	63.3 (59.2-67.3)	71.8 (69.8-73.7)*
Risky alcohol drinker	39.6 (35.5-43.6)	91.7 (90.0-93.4)*
Waist circumference men, mean (SD)	99.3 (16.6)	93.2 (15.3)*
Waist circumference women, mean (SD)	99.7 (17.8)	94.1 (17.6)*
Body mass index, mean (SD)	29.2 (7.2)	27.1 (6.8)*
Fruit intake, mean serves (SD)	1.0 (1.4)	0.9 (1.3)
Vegetable intake, mean serves (SD)	1.2 (1.4)	1.4 (1.5)*

*$p< 0.05$, †SD: standard deviation, CI: confidence interval

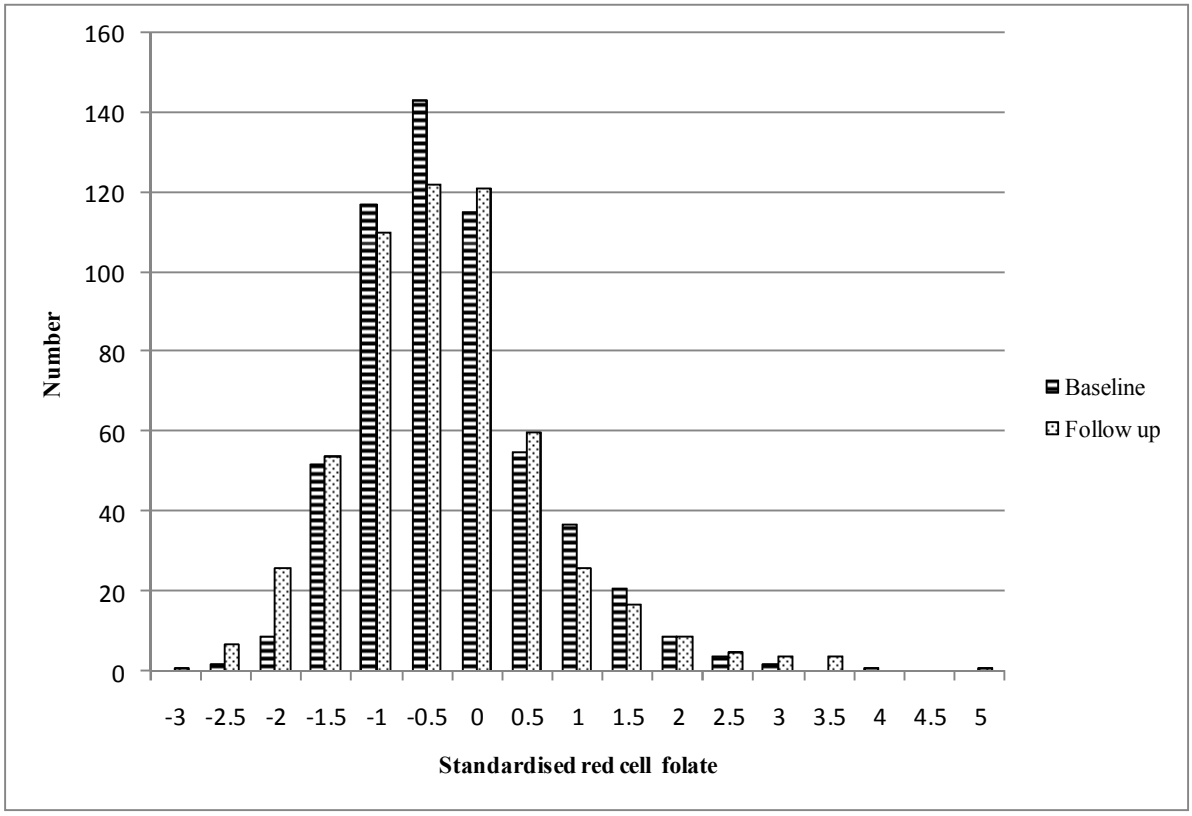

Figure 2. Red cell folate distribution at baseline (1998-2000) and follow up (2007), Well Person's Health Check, n=567

there was an overall increase in RCF deficiency in the cohort, 40 people who were deficient at baseline were in the normal range at follow up.

Figure 3 displays the change in consumption of fruit and vegetables from baseline to follow up in the folate cohort. There was an increase in consumption of fruit and vegetables over time, however 45% of people reported not eating any fruit and 38% any vegetables in the 24 hrs

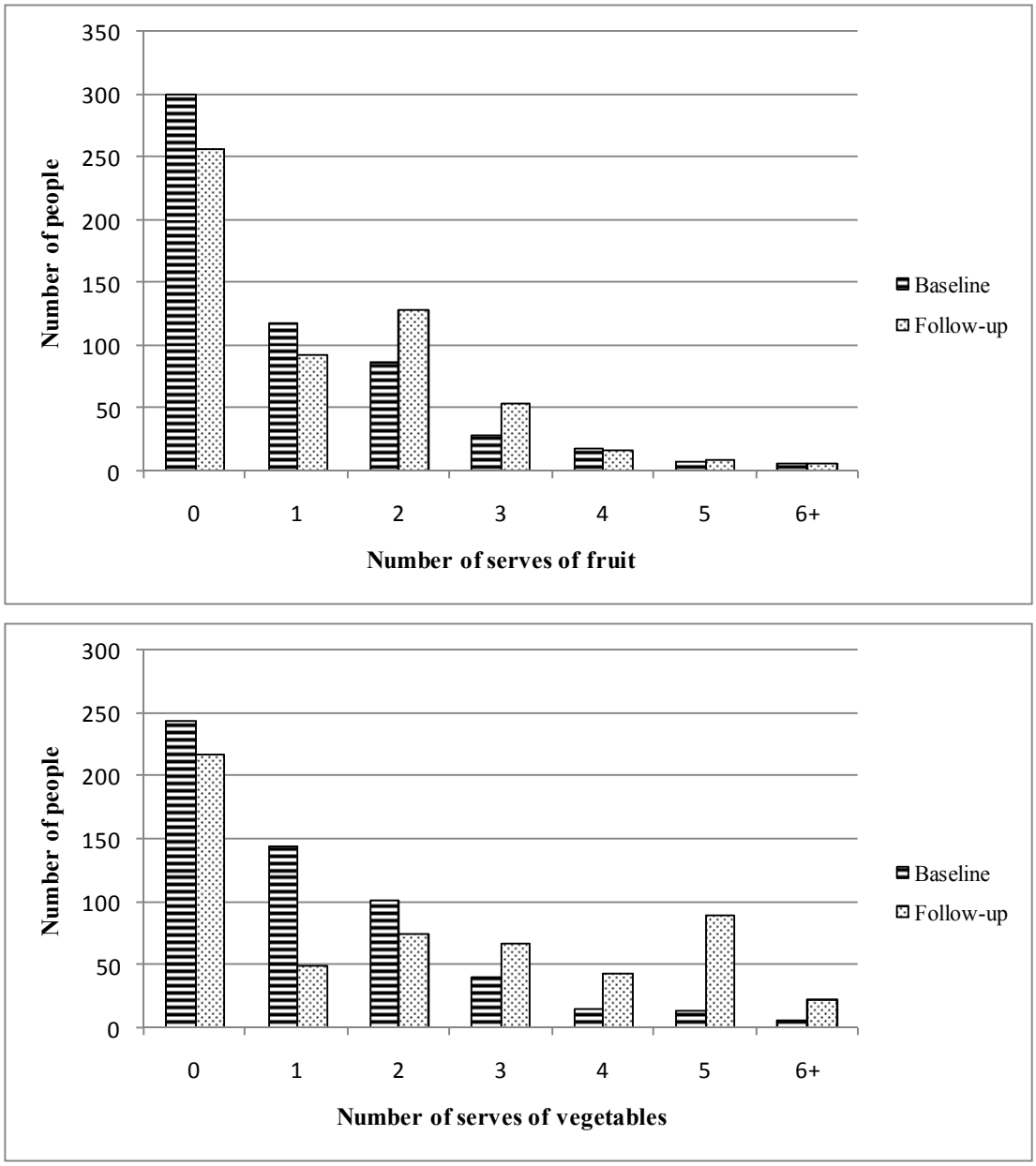

Figure 3. Fruit and vegetable consumption at baseline and follow up, Well Person's Health Check, n=567

Table 2. The association between demographic/risk factors at baseline and follow up standardized red cell folate, adjusted for standardized baseline folate in standard deviation units, Well Person's Health Check, 1998-2007, n=567

Baseline characteristics	Red cell folate at follow up Standard deviation, (95% CI‡)	Red cell folate at follow up adjusted for ethnicity, age and gender Standard deviation (95% CI)
Female†	0.10 (-0.05 to 0.26)	-
Age 13-<35†	-0.01 (-0.17 to 0.15)	-
Aboriginal†	0.02 (-0.15 to 0.19)	-
Torres Strait Islander†	-0.15 (-0.31 to 0.004)	-
Smoking†	-0.22 (-0.38 to -0.06)	-0.20 (-0.36 to -0.04)
Risky alcohol drinking	-0.09 (-0.26 to 0.07)	-0.09 (-0.26 to 0.08)
Waist circumference	0.002 (-0.003 to 0.006)	0.003 (-0.002 to 0.008)
Fruit serves quantity	-0.01 (-0.06 to 0.05)	-0.02 (-0.08 to 0.03)
Vegetable serves quantity	0.07 (0.01 to 0.12)	0.06 (0.01 to 0.12)

†Females relative to males, Age 13-<35 relative to Age 35+, Aboriginal relative to non-Aboriginal, Torres Strait Islander relative to non-Torres Strait Islander, smoking relative to non-smokers, risky alcohol drinkers relative to non-risky alcohol drinking/no alcohol drinking
‡CI: confidence interval

Table 3. Associations between demographic/risk factors in baseline and follow up, and red cell folate trajectory, Well Person's Health Check, 1998-2007, n=567

	Remained in normal range n=318 (% of n)	Deficient at baseline, normal at follow-up (n=40) (% of n)	Normal at baseline, deficient at follow-up (n=159) (% of n)	Remained deficient (n=50) (% of n)	Normal at follow-up, RR (95% CI)	Normal at follow up, adjusted for age, sex and ethnicity, RR (95% CI)
Female (%)	172 (54.1)	13 (32.5)	75 (47.2)	22 (44.0)	1.0 (reference)	-
Male (%)	146 (45.9)	27 (67.5)	84 (52.8)	28 (56.0)	0.93 (0.82-1.05)	-
Age 13 to <35 (SD)	130 (40.9)	18 (45.0)	59 (37.1)	21 (42.0)	1.04 (0.91-1.20)	-
Age 35+ (SD)	188 (59.1)	22 (55.0)	100 (62.9)	30 (58.0)	0.96 (0.83-1.10)	-
Aboriginal	91 (28.6)	19 (47.5)	42 (26.4)	28 (56.0)	0.95 (0.83-1.10)	-
Torres Strait Islander	174 (54.7)	15 (37.5)	105 (66.0)	17 (34.0)	0.92 (0.81-1.04)	-
Smoker at baseline (%)	130 (40.9)	26 (65.0)	97 (61.0)	28 (56.0)	0.78 (0.69-0.89)	0.79 (0.69-0.90)
Smoker at follow up (%)	119 (37.4)	22 (55.0)	86 (54.1)	26 (52.0)	0.81 (0.71-0.93)	0.81 (0.710.93)
Waist circumference change cm (SD)	4.87 (9.5)	4.14 (9.0)	5.66 (9.7)	6.09 (7.2)	1.0 (0.99-1.00)	0.99 (0.99-1.00)
Alcohol risk baseline (%)	108 (34.0)	22 (55.0)	63 (39.6)	27 (54.0)	0.89 (0.78-1.02)	0.90 (0.78-1.03)
Alcohol risk follow up (%)	106 (33.3)	18 (45.0)	56 (35.2)	13 (26.0)	1.02 (0.90-1.17)	1.06 (0.92-1.23)
Fruit serves baseline (SD)	1.00 (1.3)	1.00 (1.6)	0.93 (1.5)	0.73 (1.2)	1.02 (0.98-1.07)	1.01 (0.97-1.06)
Fruit serves follow up (SD)	1.30 (1.5)	1.08 (1.4)	1.13 (1.4)	0.86 (1.1)	1.04 (1.00-1.08)	1.03 (0.99-1.07)
Vegetable serves baseline (SD)	1.36 (1.5)	1.26 (1.5)	0.96 (1.2)	1.16 (1.3)	1.06 (1.02-1.10)	1.06 (1.02-1.10)
Vegetable serves follow up (SD)	2.22 (2.1)	2.59 (2.1)	1.80 (2.1)	1.58 (1.7)	1.04 (1.01-1.07)	1.04 (1.01-1.07)

RR relative risk, SD standard deviation, CI confidence interval, cm centimetres

prior to follow up.

Table 2 displays the association between baseline characteristics and standardised follow up RCF, and so the results represent the change in standard deviation units from baseline to follow up RCF. Column 1 results are adjusted for baseline standardised RCF, and column 2 results are also adjusted for age, gender and ethnicity. Females, for example had a RCF that was 0.10 standard deviations of RCF higher at follow up compared with males (95% confidence interval, CI -0.05 to 0.26, column 1 Table 2). For continuous variables the table shows the effect on standardised RCF for each increase in 1 unit of the baseline factor, for example, for every 1cm increase in waist circumference at baseline there was a corresponding increase in RCF at follow up of 0.003 standard deviations (95% CI -0.002 to 0.008). There was no difference in follow up RCF for those with different ethnicity, age or gender, although for women the direction of RCF was positive (an increase) and for men negative (a decrease in RCF level). The only risk factor associated with RCF level was smoking at baseline, which was associated with a decrease in 0.2 standard deviations of RCF compared with not smoking at baseline (95% confidence interval, CI, -0.36 to -0.04). In the follow up RCF test this 0.2 standard deviation change is equivalent to a decrease of 47.8 nmol/L.

The analysis was then stratified by the RCF trajectory, with results presented in Table 3. The people who either remained in the normal range or who had improved from the deficient to the normal range were less likely to be smokers at baseline and follow up than those who were deficient at follow up (including those who remained deficient and those who became deficient at follow up). Each additional serve of vegetables reported both at baseline and follow-up increased the chance of having normal RCF at follow up by 6% (RR 1.06, 95% CI 1.02-1.10). Age, gender and ethnicity were not associated with a normal RCF at follow up. Those who had a normal RCF at follow up gained less in waist circumference than those who were deficient at follow up, however the relative risk (RR) was 0.99 (95% CI 0.99-1.0).There was a suggestion that risky drinking at baseline, but not at follow-up, was associated with a normal RCF at follow up, although this was measured with low precision (RR 0.90, 95% CI 0.78-1.03).

DISCUSSION

This study found that the proportion of adults with low folate more than doubled over a seven year period, from an already high baseline in 1999, in a number of Aboriginal and Torres Strait Islander communities in remote North Queensland, including in women of child bearing age. Although folate deficiency increased overall across the cohort, a group of people remained in the normal range and a smaller group moved from folate deficiency at baseline to normal at follow up. These individuals were less likely to smoke, more likely to eat vegetables and possibly also fruit, and although there was no significant effect of waist circumference change on folate levels, those who became or remained in the healthy folate range gained less in waist circumference than those whose folate levels deteriorated or remained low over time. This demonstrates that some people were able to adopt or maintain healthier behaviours against the trend of the majority of the cohort who had worsening folate status over the period. Further research is needed to better understand the protective factors that contribute to an improved health status over time for some people, particularly where the overall population experienced a deteriorating health status, in order to identify strategies to make such changes achievable for the wider population in remote settings.

This study followed the introduction of voluntary fortification of a range of foods with folate (1995), but preceded the introduction of mandatory folate fortification of wheat flour for bread (2009).[23] While there was some

evidence of effectiveness of the voluntary programs in the general Australian population[11], there was concern that women of childbearing age did not have an adequate folate intake, and so folate fortification of flour was mandated. This mandatory program has not been fully evaluated, although a study that examined 20 592 samples of serum and red cell folate that were sent to a large laboratory for testing both prior to and following the introduction of the mandatory program found a reduction in folate deficiency from 9.3% in 2009 to 2.1% in 2010.[24] There has not however been an evaluation of the mandatory folate fortification program in remote Aboriginal and Torres Strait Islander communities, and so the effect for these communities is unknown. For this study it is possible that the level of folate deficiency in the cohort in 2007 will have improved following the mandatory fortification program. Nevertheless, the level of deficiency reported in this sample in 2007 was at least three times that reported in Sydney in 2009 prior to the introduction of the mandatory program, indicating a large gap in the access to a nutritious diet between the general Australian community and Aboriginal and Torres Strait Islander communities in the remote north of Australia. Furthermore, as opposed to a reduction in deficiency over time, this population had an overall doubling of folate deficiency over a seven year period, suggesting a deteriorating access to a nutritious diet over time.

The association between smoking and folate in this study is consistent with findings elsewhere.[25] There is some debate about the relative role that biological processes may play in this association compared with the association between smoking and eating a less nutritious diet.[12] It is likely that this represents a complex causal pathway. For example, smoking is associated with eating a less nutritious diet which could be accounted for by a range of factors, including reduced food security from the increased costs of smoking in those who have a low SEP,[26] reduced health literacy,[27] and socio-cultural factors in food selection.[28] This study shows that although smoking was a determinant of individual folate level, there was no evidence of it being a population determinant of folate level. The cohort experienced a 4.9% absolute reduction in smoking from baseline to follow up, with a reduction in smoking for all RCF trajectories (although relatively more reduction in those with normal RCF at follow-up) coinciding with an increase in folate deficiency over the same time period. Similarly, drinking alcohol to risky levels reduced over the follow up period. Fruit and vegetable intake, conversely increased for those with a normal RCF at follow up to a greater extent than in those with deficient RCF at follow up, suggesting that increased fruit and vegetable intake (in excess of 1 more serve a day) was the most important determinant of folate levels measured in this cohort.

The finding of an increase in folate deficiency over time is surprising in this cohort given the food supply is thought to have improved over the time between baseline and follow up surveys in Queensland. Since the mid-90s there have been a range of policies and strategies in Queensland that aimed to improve Aboriginal and Torres Strait Islander nutrition, including the Queensland Aboriginal and Torres Strait Islander Food and Nutrition Strategy (1995), the Torres Diabetes Strategy: 'Meriba Zageth for Diabetes' (1996), Eat Well Queensland 2002-2012 and nationally Eat Well Australia incorporating the National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan 2000–2010. While implementation of the national strategy was incomplete[29], in Queensland there was significant resource allocation ($12 million) to upgrade store facilities in the Torres Strait[30] to be better able to store fresh fruit and vegetables in greater quantities and for longer, with a consequent improvement in supply. Further, the Queensland Department of Communities Retail Stores group which includes three community stores on Cape York developed and implemented a nutrition policy, with an increase in sales of fruit by 57% and vegetables by 54% from June 1996 to June 2000 (personal communication Noel Burgess).

The apparent discrepancy between increased fresh food supply and sales and an increase in folate deficiency could be explained by a number of possible factors. It is possible that an increase in fruit and vegetable intake occurred in certain groups of the community only, rather than across the community. The fruit and vegetables consumed may also have been lower in folate, given the evidence that people from low-income households consume vegetables that are lower in folate,[3] and that the long time from harvesting of vegetables to consumption in remote areas may degrade the folate content of vegetables.[31] Additionally, concerns have been raised that improvements in the supply of fresh produce to remote community stores has been accompanied by increased supply of 'junk' foods and soft drinks, with potentially negative net impact on the health of the remote communities overall.[32]

Together this evidence suggests that a broader focus on improved nutrition is required. This may include addressing the balance of nutritious foods to junk foods in remote stores and take-away food outlets, and strategies to increase local food production[29] in conjunction with the community in order to reduce the current long supply route. This broad approach to food policy will become increasingly important, as transport costs are likely to increase and food security may lessen across Australia particularly for economically vulnerable populations due to climate change and related policies, but there is a need to complement work on food supply with increased local level nutrition promotion. In addition, the provision of subsidies to make fruit, vegetables and other healthy foods more accessible to all residents of these remote communities should be trialed.

A limitation of this study was the low follow-up rate from baseline including a follow-up bias towards an older, healthier group and where younger Aboriginal adults who were smokers and alcohol drinkers at baseline were less likely to participate in the follow-up survey. It would be expected that this would bias the results at follow up towards a normal level of folate in the population, but this study found an increase in deficiency over time, suggesting that the increase in folate deficiency was a true finding. Further, there was no difference in the mean RCF level at baseline between those that were and were not followed up (p=0.12). A sensitivity analysis was performed to determine how different the findings for the sample who had an RCF level taken at baseline but were

not followed up (n=1926) would need to be in order that there was a halving in the prevalence of RCF deficiency (8% from around 16%) as was reported in another Australian jurisdiction[11]. The sample not followed up would need to have had no RCF deficiency at follow up (from a baseline of 16.6%), a highly unlikely scenario. Further, if the RCF is assumed to have remained at 16% at follow up, the sample not followed up would have needed a 9.9% prevalence of deficiency, an absolute difference of 27% from the sample followed up.

Given the different social, environmental and cultural context of north Queensland Aboriginal and Torres Strait Islander communities compared with other Indigenous communities across Australia, the findings here have uncertain generalisability beyond Queensland. The high attrition of the sample also may have reduced the generalisability of the findings within Queensland, although it is difficult to quantify this risk.

The study was unable to make direct comparisons between the RCF findings at baseline and at follow-up as a different assay was used for each, requiring the tests to be standardised before analysis. There was a risk of measurement error particularly with the self-reported variables (smoking, fruit and vegetable intake), and it is likely that using fruit and vegetable intake as a marker of a generally nutritious diet was inadequate. As with any cohort study, there is a risk of residual and unmeasured confounding which limits the ability to draw firm causal conclusions from the data presented.

There are a number of factors that can influence folate status beyond dietary factors. These include increased physiologic demands for folate (such as pregnancy and malignancy), impaired absorption of folate (such as with certain anti-convulsant medications, coeliac disease), and increased excretion or loss (such as renal dialysis)[33]. These factors could not be accounted for in this study and have an unknown potential effect on the results presented here, although it is likely these factors are relatively rare in the population.

In conclusion, there was an overall increase in folate deficiency in this small cohort of remote-living Indigenous adults although a sub-group in the cohort maintained or achieved normal folate over the same time period. Fruit and vegetable intake was the most plausible overall determinant of normal folate levels for this population, although smoking was associated with individual folate levels. Improved nutrition should continue to be a focus of efforts to reduce the life expectancy gap between Aboriginal and Torres Strait Islander people and non-indigenous Australians, including an evaluation of the impact of the 2009 folate fortification program.

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AUTHOR DISCLOSURES

There is no conflict of interest.

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Original Article

Lack of folate improvement in high risk indigenous Australian adults over an average of 6.5 years: a cohort study

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葉酸缺乏高風險的澳洲原住民成人狀況並未改善：追蹤平均 6.5 年的世代研究

攝取高能量密度且營養素含量低的飲食，使得已開發國家中社經狀況較差的弱勢族群面臨過多的慢性疾病。低葉酸可視為低飲食品質的標記，它也受到抽菸及長期飲酒影響，這些因子與較低的社經地位成聚集風險。一個 4.5-9 年的追蹤研究，自 1998 至 2007 年共追蹤 567 名在澳洲北昆士蘭偏遠社區的原住民成人。分析人口學變項、抽菸、酗酒、蔬果攝取及腰圍，對於紅血球葉酸狀況的改變影響。經過 7 年，這個世代的低紅血球葉酸盛行率增加為基線的兩倍：2007 年葉酸缺乏者有 36.9%，基線為 15.9% (p<0.001)。抽菸與較低葉酸量有相關。比起追蹤時為葉酸缺乏者，有正常紅血球葉酸的人較少抽菸，並攝取較多份數的蔬菜(RR: 1.06，95%CI: 1.02-1.10)。自 1995 年引進的自主葉酸強化對世代中那些葉酸狀況原本就差的成人，並沒有顯現任何的效果。儘管食物供應有改善，低葉酸盛行率反倒增加，顯示有必要在偏遠地區施行營養推廣及健康的食物補助。自 2009 年麵粉強制葉酸強化對高風險族群的影響應該被評估。

關鍵字：葉酸、原住民族群、政策、營養政策、世代研究