Diet does not predict incidence or prevalence of non-insulin-dependent diabetes in Nauruans

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Cross-sectional and longitudinal relationships between diet and non-insulin-dependent diabetes (NIDDM) were assessed in Nauruan adults to determine if a particular component of the diet contributed to the high prevalence of NIDDM in this population. In 1982, 24-h dietary recall data were colleccted from 430 Nauruans over the age of 20, who were participating in a non-communicable disease (NCD) survey. In 1987 a follow-up survey was performed which included 350 of the subjects from whom dietary data was obtained. Neither cross-sectional nor longitudinal analyses showed any statistically significant associations between any of the specific dietary components studied and NIDDM prevalence or incidence. However, when nutrient intakes were adjusted for energy intake it appeared that the age- and body-mass-index (BMI)-corrected mean intakes of total fat, total carbohydrates, alcohol, sugar and monounsaturated fat were slightly higher in the seven incident cases than in those who remained healthy, while intakes of protein, fibre and cholesterol were lower. Despite the inability to demonstrate an association between NIDDM risk and nutrient intake at the individual level, Nauruans as a population have total energy intakes 115–135% greater than recommended for maintenance of healthy weight, protein intakes about 250% of that required, sugar intakes about twice the recommended, fibre intakes only about 30% of current recommended levels and in men a mean alcohol intake more than three times the recommended level. This adverse diet undoubtedly contributes to the high prevalence of obesity in the population and hence, even if there are no direct dietary effects, to the risk of NIDDM and other diet-related diseases.

Introduction

Diet in general and specific dietary components, notably fat and sugar, have long been suspected of having a role in causing NIDDM[1]. Studies in animals[2,3] have shown high fat diets to be related to insulin resistance but the results of epidemiologic studies in humans have generally been inconclusive[1].

However, several recent studies have suggested that an increased intake of fat, especially saturated fat, is associated with an increased risk of NIDDM or impaired glucose tolerance[4-7]. Less directly, Raheia et al[8] found that the diet of a high NIDDM risk group of urban Indians contained more fat and refined carbohydrate than a lower risk group from a rural area.

Fat intake is generally inversely correlated with carbohydrate intake and it is not clear which is more important in relation to the aetiology of NIDDM. Marshall et al[4] found that the odds ratio (OR) associated with a 40 g increase in fat intake was higher than for an energy equivalent 90 g fall in carbohydrate intake. In contrast, Feskens et al[9] have found a positive association between carbohydrate intake and incidence of glucose intolerance in a four-year study. The type of carbohydrate was important, the positive relationship only being evident with refined carbohydrate from pastries, and not with carbohydrate from legumes. In a study with 25 years follow-up there was no relationship between diet and NIDDM incidence[10].

Many formerly traditional populations subject to rapid modernization, such as Australian Aborigines[11], Pacific Islanders[12,13], Asian Indians[14,15], and American Indians[16,17], have been subject to rapid increases in the frequency of NIDDM. The prevalence of NIDDM and obesity are known to be high in the Pacific island population of Nauru. This has been related to rapid acculturation, characterized by a change to a predominantly imported food diet and mechanised transport reducing physical activity[18]. Even in 1957, Kirk[18] found little use of local food, while rice, bread, tinned meat and milk constituted the bulk of the diet.

In this study we have examined cross-sectional and longitudinal relationships between diet and diabetes in Nauruans, relating 1982 diet to the incidence of diabetes between 1982 and 1987 in subjects who had normal glucose tolerance at baseline.

Materials and methods

Data for this study were collected during surveys performed on population-based samples of adults on the Pacific island of Nauru. Nauru is a small isolated island just south of the Equator with an indigenous population of Micronesian ancestry. In 1982 a survey of all Nauruans

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aged ⩾20 years was performed with an 83% response rate. From the 1583 subjects, a randomly selected subsample of 430 underwent a 24-h diet recall questionnaire[19]. In 1987, a follow-up study of Nauruans attending earlier surveys was performed, with an overall response rate of 86%[20]. Amongst these 1290 individuals were 350 subjects who had participated in the 1982 diet questionnaire. The present study examines cross-sectional dietary and glucose tolerance data, from 423 subjects in 1982, and longitudinal data on diabetes incidence for the five-year period 1982–87 using dietary data from 177 subjects with normal glucose tolerance gathered in 1982, and subsequent follow-up data from 1987.

Survey procedure
Details of methodology used in the 1982 and 1987 surveys have been presented elsewhere[12,20]. Fasting and 2-h (after 75 g glucose monohydrate load) blood samples were collected into fluoridated tubes and centrifuged immediately. Plasma glucose levels were measured on site with a YSI glucose analyser (Yellow Springs Instrument Co., Yellow Springs, Ohio, USA) which uses a glucose oxidase method.

Height and weight were measured in all subjects and BMI calculated as weight (kg) divided by the square of height (m^2). The 24-h recall method was used to collect dietary data in 1982. The questionnaires were administered by two Melbourne dietitians, each with a local assistant, while subjects were at the survey centre and before blood glucose results were known. Household measures, food models and food samples were used to assist in determining serving sizes. Dietary data were analysed using Diet 1 software which uses the NUTTAB91–92 food composition database[21], with additional information coming from 'Food composition tables for use in the Pacific Islands'[22]. Diets were analysed for protein, carbohydrate, alcohol, sugar, starch, cholesterol, total fat and saturated, monounsaturated and polyunsaturated fat separately.

Diagnostic criteria for glucose tolerance
Glucose tolerance was classified in accordance with current WHO criteria modified for epidemiological studies[23]. Diabetes mellitus was diagnosed if plasma glucose concentration 2h after a 75 g oral glucose load was ⩾11.1 mmol/l. In the absence of a 2-h sample diabetes was diagnosed by a fasting plasma glucose⩾7.8 mmol/l. Impaired glucose tolerance (IGT) was defined by a 2-h glucose concentration ⩾7.8 mmol/l but <11.1 mmol/l with a fasting glucose <7.8 mmol/l. Subjects were considered normal if fasting and 2-h glucose concentrations were <7.8 mmol/l. Subjects reporting previous diagnosis of diabetes were accepted as such if they were taking oral hypoglycaemic agents or insulin or if they fulfilled diagnostic criteria.

Statistical analysis
In the longitudinal analysis only those people who were normoglycaemic at baseline were included. All analyses were performed using Statistical Package for the Social Sciences software[24,25]. The method of Willett and Stampfer[26] was used to adjust intakes of different dietary components for energy intake by using the residuals from the regression of nutrient intake (y) on energy intake (x). The adjusted intake figures reported are the sum of the residuals and the expected nutrient intake for the mean energy intake. Means of actual and energy-adjusted intakes have been corrected for age and BMI by covariance analysis. Males and femals have been grouped together in all analyses. Logistic regression[25] was used to look at the odds ratios for newly-diagnosed NIDDM (dependent variable 1=newly diagnosed NIDDM, 0=normoglycaemic) associated with intake of energy or other dietary components in cross-sectional and longitudinal data. Separate models were computed for each dietary component controlling for age (continuous), sex (categorical) and BMI (continuous).

Results

Characteristics of the populations included in cross-sectional and longitudinal analyses are shown in Table 1. Although the mean ages of the subjects who were followed-up in 1987 were less than the mean of all those included in the 1982 diet survey, the two groups did not differ greatly in other characteristics.

Table 1. Characteristics of study population. Means (sem).

Viable	Cross-sectional study		Longitudical study (baseline)	
	Men	Women	Men	Women
Number	218	205	92	85
Mean age (yrs)	36 (0.9)	37 (1.0)	29 (0.8)	31 (0.2)
Mean BMI (kg/m^2)	33 (0.4)	35 (0.5)	33 (0.6)	33 (0.6)
Prevalence of IGT%	16	17	10	8
Prevalence of DM%	28	26	4	3
Total energy intake (mJ)	12.8 (0.4)	10.1 (0.4)	13.4 (0.6)	9.9 (0.5)
Fat (g)	89 (4)	86 (4)	100 (7)	78 (6)
Protein (g)	133 (4)	114 (6)	136 (7)	112 (7)
Carbohydrate-total (g)	325 (9)	291 (11)	353 (14)	296 (16)
-sugar (g)	151 (6)	140 (6)	165 (9)	158 (10)
-starch (g)	173 (6)	152 (6)	187 (9)	140 (10)
Alcohol (g)	61 (7)	5 (1)	53 (9)	6 (2)
Fibre (g)	9 (0.4)	8 (0.4)	9 (0.7)	7 (0.6)
Cholesterol (mg)	418 (17)	368 (20)	425 (25)	361 (28)
Sat:poly:mono fats ratio	2:1:2	2:1:2	2:1:3	2:1:2

Table 2 shows the mean nutrient intakes for all the Nauruans surveyed in 1982 in comparison with currently recommended nutrient intakes for Australians, both as absolute values and percentage of the total energy intake. Energy intakes were similarly elevated in both Nauruan men (118–135% of recommended) and women (115–131% of recommended), associated with absolute intakes of protein, sugars and, in men alcohol, well in excess of the recommended levels. Mean intakes of fat and total carbohydrate in men were similar to recommended intakes but in women were somewhat higher. Intakes of starch and fibre were low in both men and women, reflecting the high intake of refined carbohydrate. Cholesterol intakes were similar to the upper limit of the 'safe' range for men and women. Fatty acid composition results show a low percentage of polyunsaturated fats. On the basis of their contributions to total energy, in men intakes of protein, sugar and alcohol, were higher than desirable, fat was within the desirable range and total carbohydrate was relatively low despite being adequate on an absolute basis.

Cross-sectional analysis showed no significant vari-

Table 2. Comparison of mean daily intakes for Nauruans (1982 survey) with recommended intakes for Australians (RIA).

Variable	Nauru		RIA			Nauru as % of RIA	
	Men	Women	Men	(%)	Women	Men	Women
Total energy intake (MJ)[a]	12.8	10.1	9.5–10.8		7.7–8.8	118%–135%	115%–131%
Fat (g)[b]	89 (26)	86 (32)	85–96	(33)	69–78	93%–105%	110%–125%
Protein (g)[b]	133 (18)	114 (19)	55	(10)	45	242%	253%
Carbohydrate-total (g)[b]	325 (41)	291 (46)	309–351	(52)	251–286	93%–105%	102%–116%
-sugar (g)	151 (19)	140 (22)	71–81	(12)	58–66	186%–213%	212%–241%
-starch (g)	173 (22)	152 (24)	238–270	(40)	193–220	64%–73%	69%–79%
Alcohol (g)[b]	61 (14)	5 (1)	16–19	(5)	13–15	321%–381%	33%–38%
Dietary fibre (g)	9	8		30		30%	27%
Cholesterol (mg)	418	368	200–400			104%	92%
Sat:poly:mono fats ratio	2:1:2	2:1:2	1:1:1				

[a] Energy intakes based on mean heights (men 166 cm, women 155 cm) and ages (men 36 yrs, women 35 yrs) for all Nauruans in 1982 using Australian RDI[30], for maintenance of 'ideal' weight BMI = 22.5.
[b] Numbers in parentheses are percentage contributions to total energy intakes.
Protein intakes from RDI[30], based on 0.75 g/kg 'ideal' body weight. Recommended proportions of total energy intake coming from specific nutrients (fat 33%, sugar 12%, alcohol 5%) are from Health for all Australians[34].
The percentage contribution of starch to total energy is calculated by difference after accounting for fat, protein, sugar and alcohol = 100%–33%–10%–12%–5% = 40%.
Total carbohydrate is calculated as the sum of starch and sugar.
Recommended fatty acid ratio and 'safe' cholesterol intake from[38].

ation in intakes of any dietary component across glucose tolerance categories in 1982, whether adjusted for energy intake or not (Table 3). Unadjusted intakes suggested that known diabetics avoided sugar in comparison with other groups and had a lower energy intake accompanied by smaller intakes of protein, carbohydrate, alcohol and fibre. Newly diagnosed diabetics consumed more energy, fat, protein, carbohydrate, fibre, starch and cholesterol and less sugar than people with normal glucose tolerance and intermediate levels were generally seen for subjects with IGT. However, when intakes were adjusted for total energy (Table 3) the differences in intakes of fat, protein and carbohydrate were reduced. Intakes of alcohol and sugar were lower in new diabetics than in normoglycaemic people, while intakes of starch, cholesterol and fibre remained higher. Among known diabetics, intakes of protein and fat were higher, and of total carbohydrate and sugar, lower, than in the normoglycaemic subjects.

Longitudinal analysis also did not show significant differences in mean intakes, whether adjusted for energy or not (Table 4), but trends were apparent. Newly

Table 3. Cross-sectional analysis of diet in 1982 (corrected for BMI and age) by diabetes status in 1982, actual and energy adjusted figures. New = newly diagnosed DM. Known = known DM.

Dietary intake	1982 DM status				*P*
	Normal (236)[a]	IGT (71)	New (57)	Known (61)	
Unadjusted intakes					
Energy (kJ)	11 221	11 666	11 596	10 502	0.611
Total fat (g)	82.4	92.4	84.0	83.4	0.627
Protein (g)	122.8	125.0	129.3	119.1	0.912
Carbohydrates (g)	304.4	304.3	314.5	277.4	0.521
Sugar (g)	147.5	141.8	139.0	119.1	0.266
Starch (g)	158.5	162.3	174.2	157.3	0.710
Fibre (g)	8.7	9.5	9.7	7.8	0.270
Alcohol (g)	34.1	35.6	34.2	25.8	0.904
Cholesterol (mg)	391.9	378.6	425.8	377.8	0.740
Saturated fat (g)	31.5	34.8	30.3	31.3	0.719
Monounsat. fat (g)	30.4	34.8	33.2	31.4	0.568
Polyunsat. fat (g)	13.2	16.5	14.0	13.4	0.253
% saturated fat	38.7	37.8	36.7	38.6	0.730
% monounsat. fat	36.5	37.8	39.2	37.8	0.129
% polyunsat. fat	15.7	16.5	16.0	14.4	0.716
Intakes adjusted for energy intake (kJ)					
Total fat (g)	84.4	91.0	83.1	90.8	0.470
Protein (g)	125.3	123.0	128.3	128.2	0.931
Carbohydrate (g)	309.9	300.5	312.2	298.0	0.931
Sugar (g)	150.1	139.9	137.9	129.0	0.296
Starch (g)	161.4	160.3	173.0	168.1	0.710
Fibre (g)	8.8	9.4	9.7	8.2	0.462
Alcohol (g)	35.9	34.3	33.3	32.7	0.991
Cholesteroll (mg)	399.3	373.5	422.6	405.5	0.652
Saturated fat (g)	32.3	34.3	30.0	34.1	0.571
Monounsat. fat (g)	31.2	34.2	32.9	34.3	0.520
Polyunsat. fat (g)	13.6	16.3	13.9	14.6	0.279

[a] (*n*): number of subjects

Table 4. Longitudinal analysis of diet in 1982 (corrected for BMI and age) by diabetes status in 1987. Actual and energy-adjusted figures.

Dietary intake	1987 DM status			*P*
	Normal (155)[a]	IGT (17)	New DM (7)	
Unadjusted intakes				
Energy (kJ)	11 948	12 539	11 168	0.837
Total fat (g)	91.2	93.0	87.6	0.981
Protein (g)	130.6	142.3	90.8	0.186
Carbohydrate (g)	330.9	337.0	340.1	0.974
Sugar (g)	162.6	179.4	176.0	0.721
Starch (g)	170.4	158.1	162.7	0.855
Fibre (g)	8.9	7.4	6.6	0.454
Alcohol (g)	27.5	36.8	33.1	0.867
Cholesterol (g)	410.4	448.0	280.5	0.333
Saturated fat (g)	35.4	34.0	33.2	0.968
Monounsat. fat (g)	33.8	37.9	35.0	0.820
Polyunsat. fat (g)	14.0	13.5	13.0	0.960
% saturated fat	38.4	37.0	38.5	0.886
% monounsat. fat	37.1	38.9	37.0	0.623
% polyunsat. fat	15.7	15.8	16.4	0.973
Intakes adjusted for energy intake (kJ)				
Total fat (g)	87.6	85.0	89.9	0.955
Protein (g)	126.3	132.5	93.7	0.137
Carbohydrate (g)	321.2	314.8	346.8	0.707
Sugar (g)	157.8	168.7	179.1	0.636
Starch (g)	165.4	148.6	166.2	0.567
Fibre (g)	8.7	6.9	6.7	0.364
Alcohol (g)	24.2	29.3	35.3	0.868
Cholerestrol (mg)	397.3	418.3	289.4	0.365
Saturated fat (g)	34.1	31.1	34.2	0.868
Monounsat. fat (g)	32.4	34.8	36.0	0.775
Polyunsat. fat (g)	13.5	12.3	13.5	0.872

[a](*n*): number of subjects

diagnosed diabetics tended to have a lower energy intake associated with reduced intakes of fat, protein, fibre, starch and cholesterol and increased intakes of total carbohydrate, alcohol and sugar, before adjusting for energy intake. After adjusting for energy intake the mean total fat intake in newly diagnosed subjects was higher than in those who remained normal, although protein, fibre and cholesterol intakes were still lower. Newly diagnosed diabetics also consumed more alcohol, total carbohydrate and sugar than people who remained normal or developed IGT. Trends were not consistent across glucose tolerance groups for cross-sectional or longitudinal studies, with the intakes of the IGT subjects varying relative to normal and diabetic subjects. Combining incident IGT and NIDDM groups, in order to increase numbers, tended to diminish differences from normal subjects and the results are not shown. Logistic regression analyses (Tables 5 and 6) adjusting for age, sex and BMI, confirmed the lack of significance of dietary factors in relation to diabetes. In cross-sectional analyses considering each dietary factor separately, total energy intake and energy adjusted intakes of protein, carbohydrate, starch, cholesterol, and monounsaturated and polyunsaturated fats were associated with odds ratios greater than one for NIDDM, while increases in intakes of total fat, sugar, saturated fat and alcohol were associated with reduced risk. By contrast, the longitudinal data suggested that increasing intakes of total carbohydrate, sugar, starch, alcohol, polyunsaturated and monounsaturated fats were associated with odds ratios greater than one for NIDDM over five years of follow-up, while increases in intakes of energy, total fat, protein, fibre, cholesterol and saturated fat were associated with reduced risk of NIDDM, as suggested by the mean intake results.

Table 5. Odds ratios (OR) and 95% confidence intervals (95% CI) for energy-adjusted nutrient intakes in 1982 with diabetes status in 1982 (dependent = 1 newly diagnosed diabetes, 0 normal glucose tolerance), controlling for sex, age and body mass index.

Nutrient	OR	95% CI
Energy (1000kJ)	1.007	0.941–1.076
Fat (20g)	0.985	0.826–1.174
Protein (10g)	1.012	0.953–1.074
Carbohydrate (100g)	1.087	0.746–1.584
Sugar (50g)	0.888	0.687–1.149
Starch (50g)	1.133	0.906–1.417
Fibre (10g)	1.450	0.852–2.467
Alcohol (10g)	0.990	0.943–1.040
Cholesterol (50mg)	1.015	0.945–1.090
Saturated fat (10g)	0.942	0.787–1.126
Monounsaturated fat (10g)	1.060	0.867–1.295
Polyunsaturated fat (10g)	1.035	0.720–1.486

Discussion

With only seven incident cases of NIDDM occurring over the five-year follow-up period in people with dietary intake data who were normoglycemic in 1982, it was difficult to detect significant differences in intakes between glucose tolerance groups in this study. Moreover, the lack of consistency between cross-sectional and longitudinal data, at least in terms of the direction of associations between nutrient intakes and glucose tolerance, makes interpretation of these data difficult. It may be that there are short-term, reversible effects of diet on glucose tolerance such as reported from carbohydrate[27] and alcohol[28], which are distinct from longer term effects associated with true diabetes.

Table 6. Odds ratios (OR) and 95% confidence intervals (95% CI) for energy-adjusted nutrient intakes in 1982 with diabetes status in 1987 (dependent = 1 newly diagnosed diabetes, 0 normal glucose tolerance), controlling for sex, age and body mass index.

Nutrient	OR	95% CI
Energy (1000kJ)	0.948	0.800–1.124
Fat (20g)	0.997	0.676–1.444
Protein (10g)	0.845	0.685–1.042
Carbohydrate (100g)	1.409	0.616–3.221
Sugar (50g)	1.278	0.737–2.214
Starch (50g)	1.027	0.599–1.759
Fibre (10g)	0.685	0.159–2.958
Alcohol (10g)	1.021	0.908–1.148
Cholesterol (50mg)	0.863	0.673–1.105
Saturated fat (10g)	0.975	0.720–1.319
Monounsaturated fat (10g)	1.076	0.723–1.601
Polyunsaturated fat (10g)	1.033	0.451–2.364

However, there were some results that were consistent over different methods of analysis, such as the non-significant association between reduced intakes of protein and fibre, and increased intakes of carbohydrate and sugar with NIDDM incidence in the univariate and multivariate longitudinal analysis. These data provide some support for previously reported results showing an association between increased sugar intakes and decreased fibre intakes with NIDDM[4,6], although none of the results were significant. In the longitudinal analyses, the apparent tendency towards reduced energy intakes in the subjects who develop diabetes may be due to general malaise before any overt signs of diabetes have occurred rather than a direct link between NIDDM and diet. Incident diabetics may have been less physically active[29], in which case they would have had lower energy requirements, but data to confirm this are not available.

Energy intakes in Nauruans in 1982 were high compared with Australian Recommended Daily Intakes[30] although in comparison with previously reported figures of 30 000 kJ for men and 21 830 kJ for women measured by 24-h recall in 77 subjects in 1976[31], the current energy intakes appear moderate. Ringrose and Zimmet[31] discuss possible reasons for the extremely high intakes they found, suggesting that there was a 'feast or famine' situation occurring, dependent on the arrival of food supplies. It was also the mango season when their survey was conducted and mangoes may have supplied up to 5000 kJ/day, although whether this substituted for other foods or added to the total intake is not clear.

Intakes of dietary fibre are low compared with the current Australian average of about 20 g/day[32] and recommended levels of about 30 g/day[33], and sugar intakes high, consistent with a refined diet. In comparison with Australian figures (37% of daily energy from fat[34]) the proportion of energy from fat in the Nauruan diet (26% in men and 32% in women) appears moderate and is below the recommended 33%[33]. However, due to their high energy intake, the Nauruans absolute fat intakes of 89 g/day in men and 86 g/day in women are respectively, similar to and higher than, the levels recommended for Australians. The mean alcohol intake among Nauruan men was also high, more than three times higher than the amount desirable for their age and height on an absolute basis, and providing more than twice the proportion of energy recommended[33]. Mean alcohol intakes include non-drinkers as well so that the amount of alcohol consumed by drinkers would be considerably higher.

It is now generally accepted that in relation to blood cholesterol concentration and subsequent atherosclerosis, fatty acid composition of dietary fat as well as the absolute amount is important. Evidence from animal studies suggests that different fatty acids also have different effects on the development of insulin resistance. Storlein et al[34] found that insulin resistance was most closely associated with saturated fat intake but that polyunsaturated and monounsaturated fats could also impair insulin action. Fish oils high in w-3 fatty acids reduced insulin resistance when they replaced other sources of fat, consistent with the protective effect of fish against abnormal glucose tolerance observed by Feskens and Kromhout[35]. Unfortunately we were not able to distinguish between w-3 and w-6 fatty acids. We have been unable to show a consistent effect of fat type on diabetes risk in the present study.

There are a number of problems associated with epidemiologic studies of diet-disease associations which may account for inconsistencies in results.

(1) Collecting accurate, representative dietary data is difficult. The 'standard' method is the cross-checked dietary history, which gives a representative description of usual intake but is tedious and time-consuming for subjects[36]. Twenty-four hour recall can give an accurate indication of intake over 24 hours but how representative of usual intake this is, is harder to assess. While 24-h recall is not considered to be appropriate for assessing an individual's actual intake there is considerable evidence to suggest that it is a valuable method for assessing group mean intakes[36]. The inconsistency between energy intakes in the 1976 and 1982 Nauru diet studies, discussed earlier, illustrates the problems that may arise from the use of a single 24-h recall.

(2) The conversion of dietary data to daily nutrient intakes is subject to errors from miscoding of food items and deficiencies in the food composition tables used, as well as inaccuracies in amounts[37]. However, for a comparison of two or more groups these errors should not affect the results unless there is a bias towards greater errors in one group. Nonetheless, the added variability of such measurement error will make real differences more difficult to detect.

(3) Unless the sample size is very large diet studies may lack power to identify small differences. Particularly in a disease prevalence study it is difficult to get enough cases to show significant differences between groups[37].

(4) Unless there is a large range in dietary intakes between subjects in the study any relationships between diet and disease incidence will be more related to individual susceptibility to disease rather than to differences in diet[38]. As most food in Nauru is imported the range offered may be limited by the importers, making variations less likely to occur.

(5) Differences in the way the results are analysed can affect the conclusions drawn. Willett and Stampfer[26] discuss this in detail, showing how comparing actual means of nutrient intakes can give the opposite result to using nutrient intakes 'corrected' for energy by dividing by total energy intake. Both methods are commonly used.

These points should be considered when comparing results of dietary studies and may explain the lack of consistent and significant results. A major deficiency of the current study is the small number of incident cases of NIDDM, making it difficult to detect significant differences. However, the longitudinal results are in general accord with previous work, suggesting that higher carbohydrate and sugar intakes and lower intakes of fibre may be associated with an increased risk of NIDDM. Certainly compared with Australian averages and current dietary recommendations, Nauruans consume extremely high amounts of energy, sugar and alcohol and low amounts of fibre; fat intake is also high in absolute terms. These characteristics are consistent with the current view of an 'unhealthy' diet.

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References

1 West KM. Epidemiology of diabetes and its vascular lesions. Holland: Elsevier North, 1978.
2 Storlein LH, James DE, Burleigh KM, Chisholm DJ, Kraegen EW. Fat feeding causes widespread insulin resistance, decreased energy expenditure and obesity in rats. Am J Physiol 1986; 251:E576-E583.
3 Kraegen EW, Clark PW, Jenkins AB, Daley EA, Chisholm DJ, Storlein LH. Development of muscle insulin resistance in high-fat-fed rats. Diabetes 1991; 40:1397–403.
4 Marshall JA, Hamman RF, Baxter J. High-fat, low-carbohydrate diet and the etiology of non-insulin-dependent diabetes mellitus: The San Luis Valley Diabetes Study. Am J Epidemiol 1991; 134:590–603.
5 Tsunehara CH, Leonetti DL, Fujimoto WY. Animal fat and cholesterol intake is high in men with IGT progressing to NIDDM. Diabetes 1991;40 Suppl 1:427A.
6 Feskens EJM, Kromhout D. Habitual dietary intake and glucose tolerance in euglycaemic men: The Zutphen study. Int J Epidemiol 1990; 19:953–9.
7 Swinburn BA, Boyce VL. High-fat diet causes deterioration in glucose tolerance, insulin secretion and insulin action. Diabetes 1989; 38 Suppl 1:376A.
8 Raheia BS, Pathak RB, Vispute KV, Koppikar GD. Dietary habits and prevalence of type 2 (non-insulin dependent) diabetes in Indian subjects. J Diab Assoc India 1989; 29:3–8.
9 Feskens EJM, Bowles CH, Kromhout D. Carbohydrate intake and body mass index in relation to the risk of

glucose intolerance in an elderly population. Am J Clin Nutr 1991; 54:136–40.

10 Feskens EJM, Kromhout D. Cardiovascular risk factors and the 25-year incidence of diabetes mellitus in middle-aged men: The Zutphen Study. Am J Epidemiol 1989; 130:1101–8.

11 Cameron WI, Moffit PS, Williams DRR. Diabetes mellitus in the Australian Aborigines of Bourke, New South Wales. Diabetes Res Clin Pract 1986; 2:307–14.

12 Zimmet P, King H, Taylor R, Raper LR, Balkau B, Borger J, Heriot W, Thoma K. The high prevalence of diabetes mellitus, impaired glucose tolerance and diabetic retinopathy in Nauru-the 1982 survey. Diabetes Res 1984; 1:13–18.

13 Zimmet P, Dowse G, Finch C, Serjeantson S, King H. The epidemiology and natural history of NIDDM-lessons from the South Pacific. Diabetes Metab Rev 1990; 6:91–124.

14 Ramachandran A, Jali MV, Mohan V, Snehalatha C, Viswanathan M. High prevalence of diabetes in an urban population in south India. Brit Med J 1988; 297:587–90.

15 Dowse GK, Gareèboo H, Zimmet PZ, Alberti KGMM, Toumilheto J, Fareed D, Brissonette LG, Finch CF. High prevalence of NIDDM and impaired glucose tolerance in Indian, Creole and Chinese Mauritians. Diabetes 1990; 39:390–6.

16 West KM, Diabetes in American Indians and other native populations in the New World. Diabetes 1974; 23:841–55.

17 Knowler WC, Bennett PH, Hamman RF, Miller M. Diabetes incidence and prevalence in Pima Indians: a 19-fold greater incidence than in Rochester, Minnesota. Am J Epidemiol 1978; 108:497–505.

18 Kirk N. Dietary survey of the Trust Territory of Nauru, Central Pacific 1957.

19 Ringrose H, Pargeter K, Taylor R, Thoma K, King H, Zimmet P. Nutrient intakes of a Pacific Population with a high diabetes prevalence-rate and marked obesity. Proc Nutr Soc Aus. 1982; 7:170.

20 Dowse GK, Zimmet PZ, Finch CF, Collins VR. Decline in incidence of epidemic glucose intolerance in Nauruans: Implications for the "Thrifty genotype". Am J Epidemiol 1991; 133:1093–104.

21 Diet 1 Nutrient Calculation Software User's Guide. Queensland: Xyris Software, 1991.

22 South Pacific Commission, Fiji National Food and Nutrition Committee and Fiji School of Medicine. Food composition tables for use in the Pacific Islands. Noumea, New Caledonia: South Pacific Commission, 1983.

23 World Health Organization: Diabetes Mellitus: Report of a WHO Study Group. Geneva, World Health Org., 1985 (Tech. Rep. Ser., no. 727).

24 SPSS Inc. SPSS/pc+ Statistics 4.0. Chicago, 1990.

25 SPSS Inc.: SPSS/pc+ Advanced Statistics 4.0. Chicago, 1990.

26 Willett W, Stampfer M. Total energy intake: Implications for epidemiological analyses. Am J Epidemiol 1986; 124:17–27.

27 Anderson JW, Herman RH, Zakim D. Effect of high glucose and sucrose diets on glucose tolerance of normal men. Am J Clin Nutr. 1973; 26:600–07.

28 Nikkila AE, Taskinen MR. Ethanol-induced alterations of glucose tolerance, post glucose hypoglycemia and insulin secretion in normal, obese and diabetic subjects. Diabetes 1975; 24:933–43.

29 Dowse GK, Zimmet PZ, Gareeboo H, Alberti KGMM, Tuomilheto J, Finch CF, Chitson P, Tulsidas H. Abdominal obesity and physical inactivity as risk factors for NIDDM and impaired glucose tolerance in Indian, Creole, and Chinese Mauritians. Diabetes Care 1991;14:271–282.

30 National Health and Medical Research Council. Recommended Dietary Intakes for use in Australia. Canberra: Aust Govt Publishing Service, 1987.

31 Ringrose H, Zimmet P. Nutrient intakes in an urbanized Micronesian population with a high diabetes prevalence. Am J Clin Nutr 1979; 32:1334–41.

32 Dept of Community Services and Health. National dietary survey of adults: 1983. No.2 Nutrient intakes. Canberra: Aust Govt Publishing Service, 1987.

33 Health for all Australians, Report of the Health Targets and Implementation (Health for All) Committee to Australian Health Ministers, 1988.

34 Storlein L, Jenkins A, Chisholm D, Pascoe W, Khouri S, Kraegen E. Influence of dietary fat composition on developmnent of insulin resistance in rats. Relationship to muscle triglyceride and w-3 fatty acids in muscle phospholipid. Diabetes 1991; 40:280–9.

35 Feskens E, Bowles C, Kromhout D. Inverse association between fish intake and risk of glucose intolerance in normoglycaemic elderly men and women. Diabetes Care 1991; 14:935–41.

36 Block G. A review of validations of dietary assessment methods. Am J Epidemiol 1982; 115:492–504.

37 Bingham S. Aspects of dietary survey methodology. Nutr Bull 44 1985; 10:90–103.

38 Hegsted AE. Dietary goals-a sceptical view. Am J Clin Nutr 1978; 31:310–21.

Diet does not predict incidence or prevalence of non-insulin-dependent diabetes in Nauruans

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膳食不能預測瑙魯人(NAURUANS)非胰島素依賴性糖尿病的發病率

摘要

作者從縱橫剖面分析了瑙魯人(NAURUANS)膳食與非胰島素依賴性糖尿病(NIDD M)的關系，并試圖找出膳食中某一特別成份是否可以促使NIDDM的高發病率。在１９８２年， 作者從４３０個超過２０歲，正在進行非傳染性疾病調查的瑙魯人(NAURUANS)中收集了２４小時的回憶膳食數據。在１９８７年對３５０個已有膳食數據的對象繼續復查，從縱橫剖面分析，沒有發現任何特別的膳食成份與NIDDM發病率有顯著的統計學差异。但當調整能量進食后，即按年齡、體重指數(BMI)調整總脂肪，總碳水化合物，酒、糖和單不飽和脂肪的進食后，發現７個病例較健康者高，盡管不能證實個體營養素進食與NIDDM的關系，但NAURUANS 人群的總能量進食比維持健康體重的建議超出１１５％－１３５％，蛋白質進食比需要超出約２５０％，糖進食比建議超出約２倍，纖維素進食僅爲建議的３０％，男子飲酒量比建議超出三倍多。這有害的膳食雖然仍未發現與 NIDDM和其它營養性疾病有關，但毫無疑問可促使這個人群多發生肥胖症。