# A Case-Control Study of Diet in Newly Diagnosed NIDDM in the Wanigela People of Papua New Guinea

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**OBJECTIVE** — To study the association between diet and newly diagnosed NIDDM in the Wanigela people of Papua New Guinea, a population with an extraordinary susceptibility for NIDDM.

**RESEARCH DESIGN AND METHODS** — We performed a case-control study of Wanigela people from an urban settlement (Koki). Case patients ($n$ = 145) were asymptomatic subjects in whom NIDDM was newly diagnosed using a 2-h 75-g oral glucose tolerance test. Control subjects with normal glucose tolerance ($n$ = 140) were group-matched on the basis of age and sex. A detailed food frequency questionnaire was used to determine energy and nutrient intakes. Nutrient intakes were compared directly and after calculation of residuals to correct for energy intake. Odds ratios for NIDDM were computed in relation to total energy and specific nutrient intakes, adjusting for age, sex, BMI, waist-to-hip ratio, and physical activity.

**RESULTS** — There were no differences between case patients and control subjects in mean values of total energy or energy-adjusted nutrient intakes. In logistic regression models, neither total energy nor any of the specific nutrients were associated with increased risk of NIDDM. When models were repeated with nutrients categorized by tertiles, there were marginally significant associations with intakes of fiber (positive) and cholesterol, protein, and sugar (negative).

**CONCLUSIONS** — This study does not support the hypothesis that saturated fat is an independent risk factor for NIDDM. The weak associations of intakes of fiber and cholesterol with newly diagnosed NIDDM were in the opposite directions to those expected and are probably due to chance. Relative homogeneity of diet within a community, such as that found in Koki, makes it difficult to demonstrate risk factor–disease associations. However, changes in diet and reduced levels of physical activity accompanying urbanization undoubtedly contribute to the high prevalence of obesity observed in this community, and hence diet is likely to contribute to NIDDM risk at least by indirect means.

The transition to a more westernized lifestyle in developing populations is associated with an increased risk of NIDDM (1–4). Urbanization leads to diets that are relatively high in fat and low in complex carbohydrates and fiber, and these changes have been implicated in the increased prevalence of NIDDM in modernizing Pacific island and other populations (3,4). However, while there is clear evidence for obesity, fat distribution, and physical inactivity as risk factors for NIDDM (1,5), the role of diet is less established.

Experimental data from animals (6–8) and limited human data (9,10) suggest that high-fat diets may cause insulin resistance. Epidemiological evidence for a direct relationship between fat consumption or other specific components of diet and risk of NIDDM is mixed. In support, Feskens and Kromhout (11) found current intakes or increases over the last 10 years in intake of saturated fat to be detrimental to glucose tolerance, independent of obesity, in men aged 50–70 years. Similarly, intakes of animal fat and protein among Japanese-American men in whom NIDDM was newly diagnosed were higher than in those who had normal glucose tolerance (12). Cross-sectional and longitudinal data from the San Luis Valley Study also show an association between fat intake and risk of NIDDM (13,14).

However, other epidemiological studies, including those in high-risk Pacific populations, have been unable to show specific associations between dietary components and NIDDM (4,15–19). Among the possible reasons for the inconsistency in results of studies of diet and NIDDM are the imprecision of diet questionnaires, inadequate sample size, and, in cross-sectional studies, the inclusion of patients who have modified their diets.

The Melanesian Wanigela people of the Koki settlement in coastal Papua New Guinea have an extremely high prevalence of NIDDM, which appears to have emerged only recently in association with the adoption of a westernized lifestyle (20). A case-control design and a comprehensive diet questionnaire were used to examine the association of dietary factors, especially fat, fiber, and complex carbohydrate, with previously undiagnosed, asymptomatic NIDDM in this population.

## RESEARCH DESIGN AND METHODS

### Background and subjects

Koki was first established in Port Moresby, the capital of Papua New Guinea, in the 1950s as a canoe settlement by the Wanigela people from the Marshall Lagoon area, some 200 km southeast of Port Moresby. Most of the men from Koki are

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FFQ, food frequency questionnaire; IGT, impaired glucose tolerance; OGTT, oral glucose tolerance test; OR, odds ratio; WHO, World Health Organization; WHR, waist-to-hip ratio.

employed in skilled or semiskilled occupations, and the community is relatively affluent (20). There are close links between the people of Koki and their families in Wanigela. Traditional garden crops and fish are transported from Wanigela or can be purchased at the adjacent Koki market. Apart from this, the main sources of food in Koki are local supermarkets and trade stores.

The dietary study was part of a larger study of prevalence and risk factors for NIDDM and impaired glucose tolerance (IGT) in Wanigelas. All Wanigela adults aged 25 years and older who usually resided in Koki were eligible. All subjects except those with a valid history of diabetes underwent a fasting 2-h oral glucose tolerance test (OGTT), using a 250-ml solution of 75 g glucose monohydrate in water. Fasting and 2-h plasma glucose values were determined immediately on-site using a YSI glucose analyzer (YSI, Yellow Springs, OH). Classification of glucose tolerance was based on World Health Organization (WHO) criteria (20,21) and required a 2-h plasma glucose level ≥11.1 mmol/l for diagnosis. Height and weight were measured without shoes and in light clothing. BMI was calculated as weight (kilograms)/height (meters) squared. Waist circumference was taken at the horizontal level between the xiphisternum and the umbilicus, yielding the smallest measure. Hip circumference was the horizontal level around the buttocks posteriorly, which yielded the maximum measurement. The means of duplicate measurements were used to calculate the waist-to-hip ratio (WHR). Habitual physical activity was graded as sedentary (1), light (2), moderate (3), or heavy (4) as previously described (20).

Case patients were those subjects in whom NIDDM was diagnosed during the survey ($n$ = 126) or who had higher levels of IGT, with a 2-h plasma glucose >10 mmol/l ($n$ = 19). Control subjects ($n$ = 140) were group-matched for age (within 5 years) and sex with the case patients, and all had normal glucose tolerance. These 285 subjects represented 89.6% of the 318 eligible Wanigelas identified to participate on the basis of the OGTT. The 126 participating case patients represented 89.4% of all patients with newly diagnosed NIDDM identified in the survey. Control subjects were not related to case patients nor could they live in the same house. Subjects were not informed of their diagnosis until completion of the study, and interviewers were also unaware of case-control status.

### Diet questionnaire

Subjects were interviewed either in their homes or in an adjacent community center by the research dietitian (J.M.) or a local nutritionist (T.W.), assisted by two trained interpreters from the settlement. A quantitative food frequency questionnaire (FFQ) was chosen as the most appropriate method for dietary assessment to allow for variations in intake associated with season and pay period. The questionnaire used in this study was designed to assess food and macronutrient intake over the preceding 12 months and included ethnic and regional food items. Before the survey, the questionnaire was pilot tested in a neighboring village with a diet similar to that of Koki, enabling standardization of the interview technique.

Eighty-seven foods and beverages were included on the questionnaire based on information from previous studies and modified according to the pilot study and observations in local stores and markets and in the village itself. Questions regarding alcohol intake were not included because the Wanigela people are generally Seventh Day Adventists and do not drink alcohol. Quantities relative to standard servings were estimated using portion-sized samples of staple foods and food models, along with household measuring spoons and utensils. For each item, the interviewer recorded the subject's usual serving size relative to the standard. Further details concerning food preparation, cooking methods, types and amounts of fats used for cooking and as spreads, use of sugar, and consumption of takeaway or snack foods were also elicited. In open-ended questions, the respondents were asked to recall their intake of any other frequently consumed foods not on the questionnaire. At the beginning of each interview, a 24-h recall was performed to act as a check on the results of the FFQ.

After each form had been reviewed for completeness, the data for seasonally consumed foods were adjusted to give a mean daily frequency and details of any additional intake revealed by open-ended questions were included. Daily macronutrient and energy intakes were calculated with the Diet 1 package (22), which uses the NUTTAB 91–92 nutrient database (23). Additional information on local food items was provided by the Australian Centre for International Agriculture Research, the South Pacific Commission, and the Papua New Guinea Institute of Medical Research. When published assays were not available, estimates were based on the most similar foods for which nutrient data were available or, in the case of one staple (kabelah, the mangrove bean), nutrient analysis was performed (State Chemistry Laboratory, Melbourne, Australia).

### Data analysis

All analysis was performed using SPSS/PC+ software (24). Differences between case patients and control subjects in diet and general characteristics were assessed using analysis of variance. Actual nutrient intakes and nutrient intakes adjusted for energy intake by the method of Willett and Stampfer (25) were compared with men and women combined (analyses stratified by sex gave similar results and are not shown). As an indication of the range of intakes among subjects, the 10th and 90th percentiles for each dietary component were calculated. Estimates of the odds ratios (ORs) for NIDDM were computed using separate logistic regression models for each dietary component. The dependent variable was coded 0 for control subjects and 1 for case patients. All variables were forced into models for energy and each of the energy-adjusted dietary factors, also adjusting for the effects of age (continuous), sex, BMI (continuous), WHR (continuous), and physical activity (low/high). Intakes of protein, carbohydrate, starch, sugar, fat, and cholesterol were modeled so that each increment was equal to 10 U. To examine the possibility of nonlinear relationships, ORs for NIDDM across tertiles of energy-adjusted intakes were also estimated for selected nutrients in multiple logistic regression models, adjusted as described above.

**RESULTS** — The general characteristics of male and female case patients and control subjects are presented in Table 1. Age distribution and mean ages were similar for case patients and control subjects of the same sex. There was a trend toward lower physical activity scores and higher mean BMIs and WHRs in both male and female case patients, but these differences were significant only for WHR in women.

Table 1—*Comparison of case patients and control subjects, Koki, 1991*

| | Control subjects | | Case patients | |
|---|---|---|---|---|
| | Men | Women | Men | Women |
| *n* | 74 | 66 | 74 | 71 |
| Age distribution (%) | | | | |
| 25–34 years | 27.0 | 43.9 | 28.4 | 40.8 |
| 35–44 years | 43.2 | 36.4 | 39.2 | 32.4 |
| 45–54 years | 14.9 | 13.6 | 20.3 | 19.7 |
| 55+ years | 14.9 | 6.0 | 12.2 | 7.0 |
| Mean values | | | | |
| Age (years) | 41.8 (9.9) | 37.5 (10.0) | 41.1 (10.0) | 38.0 (10.1) |
| BMI (kg/m$^2$) | 27.2 (4.1) | 28.6 (5.3) | 28.6 (4.1) | 30.2 (4.1) |
| WHR | 0.886 (0.049) | 0.803 (0.044) | 0.905 (0.042) | 0.832 (0.044)* |
| Fasting glucose (mmol/l) | 5.5 (1.1) | 5.2 (1.1) | 9.6 (1.5)* | 8.4 (1.5)* |
| 2-h glucose (mmol/l) | 5.8 (1.2) | 6.2 (1.2) | 15.8 (1.4)* | 14.8 (1.4)* |
| Urban years | 4.0 (0.9) | 3.5 (1.2) | 4.1 (0.9) | 3.5 (1.1) |
| Height (m) | 1.63 (0.06) | 1.53 (0.04) | 1.60 (0.06) | 1.54 (0.06) |
| Activity score | 3.0 (1.0–4.0) | 2.5 (2.0–4.0) | 2.8 (1.0–4.0) | 2.3 (2.0–4.0) |

Numbers in parentheses are SDs (continuous variables) and range (activity). Data for fasting glucose and 2-h glucose are geometric means. *Significantly different from control subjects for same-sex comparisons ($P < 0.05$).

Case patients and control subjects had lived in an urban environment for similar periods and had similar heights.

Daily intakes of energy and nutrients, expressed either as absolute amounts or adjusted for energy, did not differ between the control subjects and case patients, and estimates were in fact strikingly similar (Table 2). Adjusting for energy intake had a minimal effect on the estimated intakes and *P* values. Adjusted values have been used in subsequent analyses. The ranges for dietary intakes were similar for case patients and control subjects and indicate considerable variation in intakes within the study population.

ORs from logistic regression models for total energy and energy-adjusted intakes of nutrients were all close to unity (Table 3). In each model, sex (higher in women), BMI, and WHR were positively and significantly associated with risk of NIDDM.

Logistic regression models were recomputed using tertiles of energy-adjusted intakes to assess nonlinear associations between selected dietary factors and NIDDM. Generally, ORs did not differ significantly from unity, and consistent trends across intake tertiles were observed only for starch and protein (negative) and monounsaturated fat and fiber (positive). The middle tertile of cholesterol intake was associated with a significantly reduced OR for NIDDM (0.48 [95% CI 0.26–0.91]); and the OR for the top tertile (0.56 [0.30–1.06]) was also indicative of a reduced risk, as were the upper tertile of protein (0.51 [0.27–0.97]), the middle (0.48 [0.26–0.89]) and upper (0.55 [0.30–1.03]) tertiles of sugar intake, and the upper tertile of fiber intake (1.85 [1.00–3.42]). These patterns were consistent with the small differences observed in univariate analysis and the results of multivariate models using continuous dietary variables.

**CONCLUSIONS**— These data do not support the hypothesis that specific components of diet have an important role in the etiology of NIDDM in this high-risk population. The marginally significant relationships observed with intakes of cholesterol (negative) and fiber (positive) are contrary to the results of other studies (3,11–13,26,27) and appear unlikely to be real effects. The trend for reduced NIDDM risk with increased sugar intake is consistent with results of

Table 2—*Actual and energy-adjusted mean daily intakes of macronutrients by glucose tolerance status, Koki, 1991*

| | Glucose tolerance status | | |
|---|---|---|---|
| | Control subjects with normal glucose tolerance | Case patients with newly diagnosed NIDDM | *P* value |
| *n* | 140 | 145 | |
| Actual intakes | | | |
| Energy (mJ) | 11.1 (7.0–16.2) | 11.0 (6.1–16.1) | 0.826 |
| Protein (g) | 95.5 (57.3–145.2) | 93.4 (57.4–137.7) | 0.583 |
| Carbohydrate (g) | 407.7 (255.7–610.0) | 405.9 (221.0–635.7) | 0.927 |
| Starch (g) | 348.2 (204.1–520.5) | 349.3 (181.7–545.3) | 0.948 |
| Sugar (g) | 58.6 (25.3–105.8) | 55.7 (22.5–99.5) | 0.498 |
| Fat (g) | 68.6 (33.8–110.4) | 67.5 (34.2–106.6) | 0.752 |
| Saturated (g) | 32.4 (14.6–52.9) | 31.1 (14.6–48.7) | 0.393 |
| Monounsaturated (g) | 21.6 (9.8–35.5) | 21.6 (9.8–34.8) | 0.971 |
| Polyunsaturated (g) | 8.1 (4.1–12.5) | 8.2 (4.1–13.2) | 0.722 |
| Cholesterol (mg) | 271.7 (148.6–447.0) | 257.0 (127.7–440.1) | 0.307 |
| Fiber (g) | 21.7 (13.3–32.2) | 22.1 (11.8–31.7) | 0.719 |
| Energy-adjusted intakes | | | |
| Protein (g) | 95.2 (76.3–116.7) | 93.8 (73.6–117.6) | 0.517 |
| Carbohydrate (g) | 405.9 (347.1–451.7) | 408.1 (348.8–478.1) | 0.688 |
| Starch (g) | 346.8 (270.3–406.2) | 351.2 (274.7–428.2) | 0.518 |
| Sugar (g) | 58.2 (35.1–86.4) | 56.0 (28.2–85.1) | 0.466 |
| Fat (g) | 68.4 (51.3–87.6) | 67.9 (43.0–91.0) | 0.814 |
| Saturated (g) | 32.4 (22.8–42.4) | 31.2 (17.9–41.8) | 0.278 |
| Monounsaturated (g) | 21.5 (15.4–30.3) | 21.7 (13.5–29.6) | 0.744 |
| Polyunsaturated (g) | 8.1 (5.8–11.8) | 8.3 (5.7–11.9) | 0.494 |
| Cholesterol (mg) | 270.8 (185.2–373.7) | 258.1 (142.4–383.5) | 0.252 |
| Fiber (g) | 21.6 (17.5–26.3) | 22.2 (17.9–26.8) | 0.195 |

Data are means (10th-90th percentile). The case-patient group includes 19 subjects with IGT.

**Table 3—*ORs for newly diagnosed NIDDM associated with energy and energy-adjusted daily nutrient intakes, from multiple logistic regression models controlling for age, sex, BMI, WHR, and physical activity, Koki, 1991***

| Nutrient | ORs | 95% CI |
|---|---|---|
| Energy (mJ) | 0.99 | 0.93–1.05 |
| Protein (10 g) | 0.90 | 0.78–1.03 |
| Carbohydrate (10 g) | 1.02 | 0.97–1.08 |
| Starch (10 g) | 1.03 | 0.98–1.07 |
| Sugar (10 g) | 0.94 | 0.85–1.04 |
| Fat (10 g) | 0.98 | 0.84–1.14 |
| Saturated (g) | 0.99 | 0.96–1.02 |
| Monounsaturated (g) | 1.00 | 0.96–1.04 |
| Polyunsaturated (g) | 1.04 | 0.95–1.14 |
| Cholesterol (10 mg) | 0.97 | 0.94–1.00 |
| Fiber (g) | 1.06 | 0.99–1.13 |

analytical studies (11,13,14), but in contrast with expectation based on ecological studies (3,4).

Dietary questionnaires are subject to a number of potential shortcomings in disease-association studies (15). The FFQ used in this study was designed to provide representative data for the previous year, and considerable effort was used to compile a food list relevant to the study population and locate the most appropriate food composition data. The questionnaire was pretested in a similar community to identify additional foodstuffs for inclusion, and for each individual was cross-checked with the 24-h recall and other background information. While there are no more objective data available to validate the results of this study, the mean energy intakes of Koki subjects are in general agreement with data from similar populations (28,29). Qualitative results also indicated food choices similar to those for other studies in coastal Papua New Guinea (30).

The response rate (126 of 141, 89.4%) among eligible case patients in this study was high, minimizing the risk of selection bias, and characteristics of responders and nonresponders were similar. Because diagnosis was unknown at the time of interview and the majority of subjects were relatively young, it is unlikely that case patients would have made systematic changes in diet because of either diabetes or comorbidities, including cardiovascular disease, which is unusual in this population. Thus, the sample studied can be assumed to be representative of asymptomatic subjects with newly diagnosed diabetes in Koki.

Significant misclassification of diabetes status is also unlikely given the strict laboratory quality control measures and generally high carbohydrate intake before the OGTT in this population. It is known from longitudinal studies in other Pacific populations that subjects with 2-h glucose levels diagnostic of NIDDM on a single OGTT are almost invariably confirmed to have NIDDM when OGTTs are repeated 5 or more years later. Inclusion of 19 subjects with high IGT (2-h glucose >10 mmol/l) as case patients is justified on the basis that ~50% of such subjects have NIDDM in follow-up studies (G.K.D., unpublished observations) and that risk factors for IGT are the same as those for NIDDM (5). The high IGT group also had clearly elevated mean fasting glucose levels, BMIs, and WHRs relative to the control group. Finally, the strong associations observed with nondiet risk factors (BMI, WHR, and physical activity) suggests that misclassification of disease was unlikely to explain the lack of association with dietary factors.

The sample size of our study was limited by the number of subjects with previously undiagnosed diabetes identified, but because of the high background prevalence of NIDDM in the urban Wanigelas, the number of case patients available for study was still high relative to other studies. Moreover, all data were collected over a short period of 6 weeks, minimizing the risk of drift in methods that might apply to studies completed over longer periods in low-prevalence populations. In any case, the magnitude of the ORs observed suggests that a larger sample size is unlikely to have altered the results.

The weak associations with cholesterol and fiber intakes, in directions opposite to those that might have been expected, are unlikely to be biologically important and are probably due to chance. It is difficult to reconcile these findings with the results of ecological studies (1–4) or our current understanding of the role of diet in the etiology of NIDDM.

The lack of association between diet and NIDDM prevalence in Koki is consistent with both cross-sectional and longitudinal data for Nauruans (15). As with Wanigelas, Nauruans are extremely susceptible to development of obesity and NIDDM in association with westernization (20,31). Lifestyle changes are relatively homogeneous within these modernizing populations, as suggested by the similarity of dietary intakes in the case patients and control subjects. Expression of NIDDM may therefore be more dependent on variation in individual genetic susceptibility than on environmental risk factors such as diet and physical activity, and specific effects of diet are difficult to demonstrate (32).

Metabolic changes associated with glucose intolerance occur long before the diagnosis of NIDDM (33–35). On this basis it could be argued that diet at some time in the past, rather than the previous 12 months, may be more strongly associated with diabetes risk. Nevertheless, in the San Luis Valley Diabetes Study, intake was measured at the time of diagnosis of NIDDM and a significant effect of fat intake was found (13). Diet was also significant in predicting NIDDM once IGT had developed (14), supporting the use of intakes measured later in the natural history of NIDDM. Indeed, given the relatively rapid pace of modernization in the Koki Wanigelas, a longitudinal study may have failed to detect important baseline risk factors: Case patients had moved to the urban area an average of 3.8 years before the study, and most lifestyle change had taken place during that period.

Traditional diets in coastal Papua New Guinea were typically based on fish, taro, yams, coconuts, bananas, and sago, characteristically low in fat and high in complex carbohydrates. In this regard, the current Koki diet, although not adverse in comparison with Western diets, represents a significant departure from that enjoyed by the previous generation. Early dietary data for comparable coastal populations are not available, but as an example, an early study in a highland Papua New Guinea population found that carbohydrate contributed 95%, protein 3%, and fat only 2% of total energy (36). In comparison, the 1991 Koki diet included 15% protein, 23% fat, and 61% carbohydrate.

There are no data concerning maternal or infant nutritional status in the study participants. While some might invoke the thrifty phenotype hypothesis (37) (that is, poor nutrition during fetal development and infancy) to explain the susceptibility of this population to NIDDM, rather than genetic factors, the

former explanation seems unlikely (38). First, there is a massive difference in NIDDM prevalence relative to other nearby communities thought to have experienced similar diets and nutrition historically and currently (20). Second, within the close-knit subsistence communities of traditional times, it is unlikely that there would have been marked variations in maternal and infant nutrition between family units. Third, although an imperfect indicator of early nutrition, case patients and control subjects had very similar adult heights.

These results do not support the hypothesis that specific dietary factors, including high-fat intake and/or low-fiber intake, are associated with the risk of NIDDM. However, the high level of obesity observed in Koki, itself associated with changes in diet and activity (39), contributes strongly to the increased risk of NIDDM, and efforts to avoid obesity should therefore remain an integral component of prevention programs. Studies promoting traditional diets in other high-risk communities, with or without changes in physical activity, have shown impressive results in terms of weight loss and normalization of metabolic parameters (40,41).

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