Relationship Between Prevalence of Impaired Glucose Tolerance and NIDDM in a Population

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Objective: To study the relationship between the prevalence of impaired glucose tolerance (IGT) and non-insulin-dependent diabetes mellitus (NIDDM) across populations of the Pacific Ocean region to assess whether variability in those two proportions followed some predictable pattern related to modernization of life-style and risk factor levels. Research Design and Methods: Prevalence estimates from studies with 75-g oral glucose loads and World Health Organization criteria were age standardized. Results: The linear correlation between IGT and NIDDM prevalence was poor (0.22 in men and 0.24 in women), although it was improved when the outlying data of Micronesian Nauruans and Australian Aborigines were excluded ($r = 0.65$, $P < 0.01$ in men and $r = 0.54$, $P < 0.01$ in women). However, an epidemicity index calculated as the percentage of total glucose intolerance (TGI) made up by IGT (i.e., IGT/TGI) had the strongest correlation with NIDDM prevalence ($r = -0.81$, $P < 0.001$ in men and $r = -0.77$, $P < 0.001$ in women) and also explained more of its variance, with no population having undue influence on the relationship. When IGT/TGI was plotted against NIDDM prevalence for the genetically homogeneous rural (relatively traditional) and urban (modernized) segments of five Pacific island populations, there was a clear tendency for IGT/TGI to decline as the prevalence of NIDDM increased in association with worsening risk factor levels. However, longitudinal data for the high prevalence population of Nauru demonstrated that at least in a stabilizing epidemic, changes in the prevalence of IGT and NIDDM may not be easily predictable. Conclusions: The epidemicity index may be useful as an indicator of the potential for higher future NIDDM prevalence in whole populations. Populations will probably equilibrate at a certain NIDDM prevalence dependent on the strength of their genetic susceptibility to the disease and their degree of exposure to adverse environmental risk factors, including modern diet, physical inactivity, and obesity. ***Diabetes Care*** **14:968–74, 1991**

The prevalence of both impaired glucose tolerance (IGT) and non-insulin-dependent diabetes mellitus (NIDDM) vary widely between populations (1,2). Although it might be expected that there should be a close relationship between the magnitude of IGT and NIDDM prevalence, this does not appear to be the case. For instance, a community survey of urbanized Australian Aborigines found a high prevalence of NIDDM (16.7 and 14.6% in men and women, respectively) but a comparatively low IGT prevalence (0.7 and 4.2% in men and women, respectively) (3). By contrast, a survey in a rural Polynesian population in Tuvalu found that the prevalence of IGT was 3.4 times that of NIDDM (4).

Not surprisingly, given its definition based on 2-h blood glucose levels intermediate between normal and NIDDM, IGT is a particularly strong risk factor for NIDDM. Longitudinal studies in numerous populations have shown that subjects with IGT have a risk of developing NIDDM over periods varying from 2.5 to 10 yr ~2–8 times that of subjects with normal glucose tol-

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erance (5–11). This relative risk does not appear to correlate with the population prevalence of IGT or NIDDM (Table 1).

However, the summarized data of Table 1 indicates that the actual rate of conversion from IGT to NIDDM corresponds more closely to the prevalence than does the relative risk, with higher rates (~5–7%/yr) in populations with a high background frequency of NIDDM and IGT, i.e., Pima Indians (7,12), Nauruans (6,11), middle-aged and elderly Japanese (5,10), and Maltese (8). The Whitehall (13) and Bedford (14) studies suggest lower rates in relatively low-prevalence white populations (~1–3%/yr), although methodological differences make it difficult to be entirely sure that the group of subjects labeled as IGT in these latter studies can be compared to those described above.

Nevertheless, it seems reasonable to impute that in populations with a relatively high conversion rate from IGT to NIDDM (contributing to a high total incidence) and provided that mortality in those with diabetes is not excessive that the prevalence of NIDDM will rise. At the same time, the relative contribution of IGT to the total amount of glucose intolerance (IGT and NIDDM combined) in a population might be expected to decline. Such a phenomenon has recently been documented in Nauru, where the prevalence of IGT has decreased by 50% over 5 yr. However, here, the prevalence of NIDDM has stabilized in the face of a high mortality rate (11).

Unfortunately, serial estimates of the prevalence of IGT and NIDDM are not available for other populations. However, this study investigated the generality of this finding by comparing the relationship between IGT and NIDDM prevalence in the relatively traditional rural and more modernized segments of several developing Pacific island populations, in which a rise in NIDDM prevalence after life-style change has previously been demonstrated. We also extended the analysis to include other populations of the Pacific region to assess whether the variability in IGT and NIDDM prevalence might have some association with the degree of modernization and extent of life-style–related NIDDM.

RESEARCH DESIGN AND METHODS

Prevalence estimates were age standardized with the direct method (15), and weights were derived from Segi's world population for the age-groups 20–24, 25–34, 35–44, 45–54, and ≥65 yr (16). The standardized prevalence information for Pacific island populations came from our own series of studies, as summarized in a recent review article by Zimmet et al. (2), except that those studies with <300 subjects (predominantly small community studies in Papua New Guinea) were not considered because of unstable estimates. Prevalence for Australian whites (17) and Aborigines (3) was derived from original sources with minor extrapolation of age-groupings for purposes of standardization. Age-specific data for United States blacks, whites, and Mexican Americans were derived from the Second National Health and Nutrition Examination Survey (18) and the Hispanic Health and Nutrition Examination Survey (unpublished observations, M. Harris, Natl. Inst. of Health, Bethesda, MD).

All prevalence estimates came from studies that used 75-g oral glucose loads and World Health Organization criteria for classification (19). The proportion of total glucose intolerance (TGI = IGT and NIDDM combined) made up by IGT (IGT/TGI) was calculated for men and women of each population. The relationship between the prevalence of IGT, NIDDM, TGI, and other indices such as IGT/TGI and IGT/NIDDM was assessed visually by the plotting and calculation of linear and quadratic regression formulas and correlation coefficients. Weighting for different sample sizes and precision of prevalence estimates was not performed because it was the general nature, rather than the specific mathematics of any relationship, that was sought.

Prevalence estimates were available for Nauru, which has an extremely high prevalence of both IGT and NIDDM, at three points in time—1975–1976, 1982, and 1987 (11). All three estimates were shown in the figures and tables, but only the most recent (1987) was used in calculations. Because the Nauru data were relative outliers, analyses were performed with and without their inclusion. Only the relationship of NIDDM prevalence with IGT prevalence per se and with IGT/TGI appeared to give useful information and were reported here.

Obesity was defined as a body mass index ≥27 kg/m^2 in men and ≥25 kg/m^2 in women. Physical activity (occupational and leisure combined) was graded from 1 to 4, corresponding to sedentary, light, moderate, and heavy, by trained interviewers. For these analyses, it was dichotomized as active versus inactive as previously described (20). Differences between variables were assessed by χ^2 or t tests as appropriate.

RESULTS

The populations, their respective prevalence values, and IGT/TGI are shown in Table 2. In Fig. 1, IGT/TGI is plotted against NIDDM prevalence for the rural (relatively traditional) and urban (modernized) segments of the populations of Kiribati (Micronesian), Western Samoa (Polynesian), Wallis Island (Polynesian), and Fiji (Melanesian and migrant Indians). There is a clear tendency for IGT/TGI to decline as the prevalence of NIDDM rises in association with modernization. Modernization in the indigenous Pacific populations is associated with a clear increase in the prevalence of obesity and a decline in levels of physical activity, and both are regarded as important risk factors for glucose intolerance (Table 3). Differences in risk factor levels (at least for obesity) between the rural and urban Fiji Indians

TABLE 1
Estimated conversion rate from impaired glucose tolerance (IGT) to non-insulin-dependent diabetes mellitus (NIDDM) in various populations

Population	Period*	Baseline			Conversion rate (%/yr)	Relative risk†	Diagnostic criteria and notes‡	Ref.
		Age (yr)	NIDDM (%)	IGT (%)				
British men (Whitehall)	1968–1970 (5 yr)	>40	NA	NA	3.0	NA	Screening test + 50-g OGTT with 2-h capillary glucose 6.7–11.0 mM	Jarrett et al. (13)
British (Bedford)	1962 (10 yr)	Adult	NA	NA	1.5	NA	Glycosuria + 50-g OGTT with 2-h capillary glucose 6.7–11.1 mM	Keen et al. (14)
Japanese	1964–1965 (7 yr)	All ages (bias to older)	15.9	6.3	5.5	9.0	Glycosuria + 50-g OGTT; baseline = 13 IGT only	Sasaki et al. (5)
Pima Indians	1972–1985 (mean 3.3 yr)	≥5	24.8§	NA	6.1	6.3	Estimated 10-yr cumulative incidence	Saad et al. (7)
Maltese	1981 (6 yr)	≥29	6.8	5.3	5.1	7.2		Schranz (8)
Mexican Americans	1979–1982 (8 yr)	25–64	10.2	11.6	2.7	6.2	Numbers calculated indirectly from paper	Haffner et al. (9)
Japanese-American men	1983–1985 (mean 2.5 yr)	44–73	34.1	32.3	6.8	4.2		Bergstrom et al. (10)
Nauruans	1975–1976 (6.2 yr)	≥20	27.9	21.1	3.5	2.1	Age standardized for comparison	Dowse et al. (11)
Nauruans	1982 (5 yr)	≥20	24.7	17.4	5.6	7.6	Age standardized for comparison	Dowse et al. (11)

NA, not available. OGTT, oral glucose tolerance test.
*Baseline investigations + duration of follow-up.
†Relative risk of conversion to NIDDM: IGT relative to normal.
‡Data based on ref. 19, unless otherwise indicated.
§Knowler et al. (12).

were less marked; however, this was in accord with the fact that their NIDDM prevalence and IGT/TGI proportion remained relatively stable.

Data for all the populations are plotted in Fig. 2. Note that the linear relationship between IGT/TGI and NIDDM prevalence corresponds to the general direction and slope of the lines seen for the intrapopulation rural-urban comparisons of Fig. 1 (Fig. 2, *bottom*).

The linear correlation between the prevalence of IGT and NIDDM was 0.22 in men (NS) and 0.24 in women (NS) when Nauru (1987) and the Australian Aboriginal data were included (Fig. 2, *top*). When these two populations were excluded on the basis that they were outliers from the rest of the data, the correlation coefficients were 0.65 in men ($P < 0.01$) and 0.54 in women ($P < 0.01$). The relationship between the IGT/TGI proportion and NIDDM prevalence was stronger than that between IGT and NIDDM, regardless of whether the Nauruan and Aboriginal data were included (Fig. 2, *bottom*). With all populations included, the correlation coefficient was -0.81 in men ($P < 0.001$) and -0.77 in women ($P < 0.001$). Excluding Nauru (1987), which was the data point most likely to exert undue influence, made little difference to these results ($r = -0.84$ in men and -0.79 in women).

The linear relationship between IGT/TGI and NIDDM prevalence fit the data quite well, with adjusted R^2 values of 0.64 in men and 0.58 in women when all populations were included (and 0.70 in men and 0.60 in women excluding Nauru [1987]). By contrast, adjusted R^2 values for the simple linear association between IGT and NIDDM prevalence were only 0.003 and 0.01, respectively, when all populations were included and 0.39 and 0.26 when the outlying Nauruan and Aboriginal groups were not considered.

A quadratic equation in fact fit the IGT and NIDDM prevalence data as well as or better than a linear equation, with adjusted R^2 values of 0.11 in men and 0.12

TABLE 2
Age-standardized prevalence (%) of impaired glucose tolerance (IGT) and non-insulin-dependent diabetes mellitus (NIDDM) in adults aged ≥20 yr from 22 populations of the Pacific region

	Men			Women		
	IGT	NIDDM	IGT/TGI	IGT	NIDDM	IGT/TGI
Micronesian						
Nauru						
1975–1976 (1)	18.8	37.4	33.5	26.8	32.4	45.3
1982 (2)	20.2	33.4	37.7	21.5	32.1	40.1
1987 (3)	9.2	33.0	21.8	11.3	34.8	24.5
Kiribati						
Rural (A)	12.7	3.7	77.4	15.1	3.9	79.5
Urban (B)	19.2	11.7	62.1	18.9	11.1	63.0
Polynesian						
Western Samoa						
Rural (C)	4.7	1.7	73.4	6.5	4.2	60.7
Urban (D)	7.6	8.2	48.1	9.1	8.5	51.7
Wallis Island						
Rural (E)	4.7	2.0	70.1	7.7	4.1	65.2
Urban (F)	10.4	10.0	51.0	8.1	14.0	36.7
Rarotonga (G)	10.2	5.5	65.0	11.3	8.5	57.1
Niue (H)	6.9	5.6	55.2	7.1	8.6	45.2
Tuvalu (I)	8.8	0.8	91.7	19.7	8.4	70.1
Ouvea (J)	5.1	7.3	41.1	9.9	6.5	60.4
Melanesian						
Papua New Guinea, highlands (K)	4.7	0	100.0	1.1	0	100.0
Ouvea (L)	3.7	0	100.0	6.5	4.2	60.7
Fiji						
Rural (M)	6.9	2.1	76.7	11.5	2.1	84.6
Urban (N)	8.9	5.9	60.1	15.1	10.3	59.4
Asian Indian						
Fiji						
Rural (O)	12.3	15.1	44.9	11.8	13.6	46.5
Urban (P)	10.4	17.5	37.3	13.7	16.3	45.7
Australia						
Aborigine (Q)	0.5	20.5	2.4	4.8	19.2	20.0
White (R)	2.6	3.2	44.8	2.1	2.2	48.8
United States						
White (S)	9.4	5.0	65.3	10.5	6.5	61.8
Black (T)	11.0	8.3	57.0	14.2	10.7	57.0
Mexican American (U)	13.8	11.6	54.3	13.8	13.0	51.5

Letter (A–U) or number (1–3) corresponds to points plotted in Fig. 2. TGI, total glucose intolerance = IGT + NIDDM.

in women when all populations were included and 0.39 in men and 0.34 in women when the Nauruans and Australian Aborigines were excluded.

CONCLUSIONS

These cross-population data demonstrated that there is no simple relationship between IGT and NIDDM prevalence in populations at different stages of modernization. Nonetheless, the proportion of total glucose intolerance made up by IGT has a strong negative correlation with the population prevalence of NIDDM, and evidence from those populations for which data is available at different stages of modernization (rural vs. urbanized communities) suggests that IGT/TGI may have some predictive significance for the epidemicity or potential for NIDDM increase in the event of increased risk factor levels. This epidemicity index (IGT/TGI) also has a more direct relationship with the prevalence of NIDDM than does the prevalence of IGT per se. Although this is not surprising, given that NIDDM prevalence appears on both sides of the equation in a plot of IGT/TGI versus NIDDM, the fact remains that this relationship removes much of the "noise" observed in a simple plot of IGT and NIDDM prevalence and seems to offer conceptual advantages, at least for illustrative purposes.

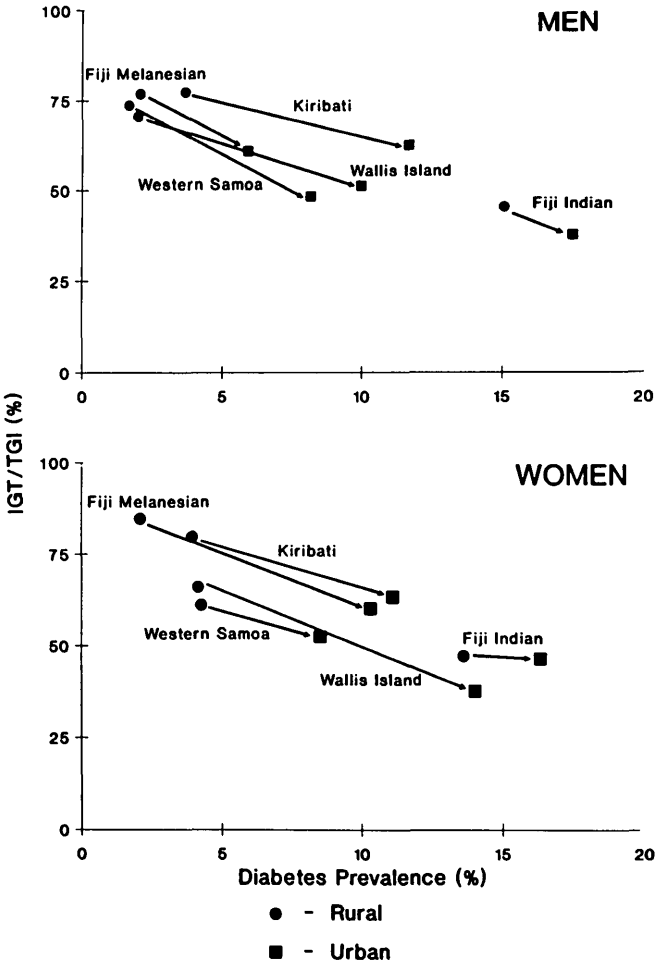

FIG. 1. Epidemicity index (impaired glucose tolerance/total glucose intolerance [IGT/TGI]) vs. non-insulin-dependent diabetes mellitus (NIDDM) prevalence in rural and urban populations from Kiribati (Micronesian), Western Samoa (Polynesian), Wallis Island (Polynesian), and Fiji (Melanesian and Indian).

It is probable that populations will equilibrate at a certain point along the line of descent shown in Fig. 2, depending on *1*) the strength of their genetic susceptibility to the disease, *2*) their degree of exposure to adverse environmental risk factors, i.e., modern diet, physical inactivity, and obesity, and *3*) the impact of their medical care and preventive health services. The relative stabilization in IGT/TGI in the rural and urban migrant Indian populations, despite the fact that there are still significant differences in activity levels, may indicate that other undetermined risk factors (such as dietary habits) are preeminent and of similar magnitude in these populations (20). The longitudinal data from Nauru, however, where the prevalence of NIDDM and risk factor levels appear to have stabilized simultaneously with a dramatic reduction in IGT prevalence (and IGT/TGI), highlight the fact that any simplistic association between IGT and NIDDM prevalence is unlikely (11).

That a high IGT/TGI or absolute prevalence of IGT should predict a higher future prevalence of NIDDM is not surprising given the evidence from several ethnic groups, including British whites (13), Maltese (8), Japanese (5,10), Micronesian Nauruans (6,11), and Pima Indians of Arizona (7), that, over 5 yr ~15–30% of individuals with IGT are likely to progress to NIDDM. Although the rate of this deterioration may vary somewhat between populations, it is unclear to what extent this variability reflects differences in levels of associated risk factors, i.e., obesity and physical inactivity, rather than innate differences between populations. That is, although genetic factors might be of primary importance in influencing the original deterioration to IGT, further decompensation to NIDDM might be modulated principally by the above-mentioned environmental factors interacting with the pathogenetic sequence of β-cell decompensation already set in motion as a result of peripheral insulin resistance, for example (21).

The epidemicity index may be of use as an illustrative indicator, for public health purposes, of the potential for higher future NIDDM prevalence in populations. Nauruans (11) and the Australian Aboriginal community of Bourke (3) may have reached close to their maximum potential for NIDDM, presumably because risk factor

TABLE 3
Changes in non-insulin-dependent diabetes mellitus risk factor levels associated with urbanization in 5 Pacific populations

Population	Men		Women	
	Rural	Urban	Rural	Urban
Kiribati				
n	469	906	556	971
Mean age (yr)	42.1	35.7*	40.6	35.2*
Obesity (%)	25	55*	40	72*
Inactive (%)	25	48*	19	51*
Western Samoa				
n	358	325	387	419
Mean age (yr)	41.7	42.0	41.5	43.1
Obesity (%)	33	53*	66	83*
Inactive (%)				
Wallis Island				
n	261	257	288	314
Mean age (yr)	40.8	41.7	39.6	39.1
Obesity (%)	39	73*	78	92*
Inactive (%)	7	16*	27	94*
Fiji Indian				
n	212	381	239	454
Mean age (yr)	37.8	38.9	38.4	37.4
Obesity (%)	8	13	34	40
Inactive (%)	17	73*	76	97*
Fiji Melanesian				
n	239	396	236	460
Mean age (yr)	40.0	40.0	40.5	39.0
Obesity (%)	25	36	56	68*
Inactive (%)	9	91*	47	94*

*$P < 0.01$ for rural-urban difference within populations.

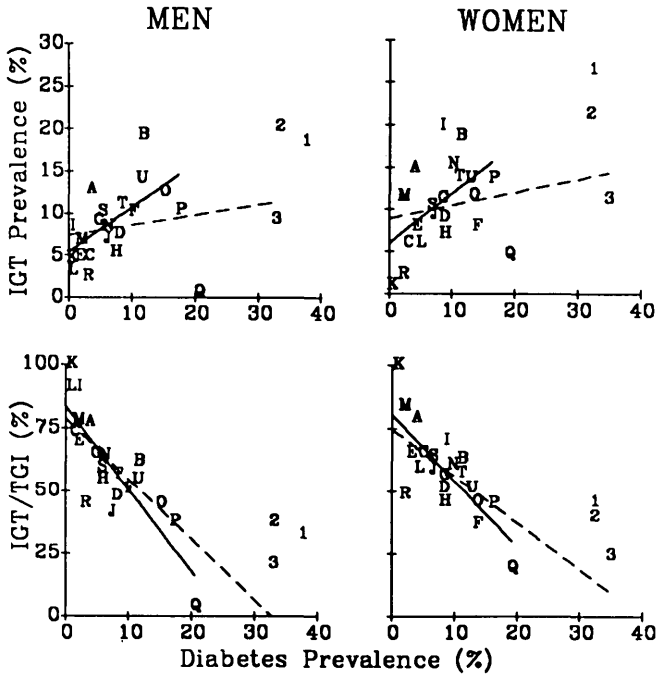

FIG. 2. Relationship of impaired glucose tolerance (IGT) prevalence and epidemicity index (IGT/glucose intolerance [TGI]) with non-insulin-dependent diabetes mellitus (NIDDM) prevalence in 22 Pacific region populations. Letters (A–U) and numbers (1–3) correspond to populations in Table 2. *Dashed lines*, linear regression calculated including all alphabetical points plus Nauru 1987 (*3*) but not 1975–1976 (*1*) or 1982 (*2*). *Solid lines*, regressions that also exclude Nauru 1987 (*3*) (*bottom*) and both Nauru 1987 (*3*) and Australian Aborigines (*Q*) (*top*).

levels are sufficiently high to produce disease in those with the susceptibility gene(s). Even in populations considered to have low or moderate susceptibility to NIDDM (e.g., whites), adverse changes in risk factor levels are likely to drive them downward along the line illustrated in Fig. 2 as NIDDM emerges from the underlying reservoir of IGT. Conversely, with improvement in risk factor levels, they may move upward to the left.

However, we do not mean to imply that by simply plotting a population's current IGT/NIDDM relationship it would be possible to predict the future prevalence of NIDDM with certainty. In general terms, it seems reasonable to suggest that if the prevalence of IGT is high relative to that of NIDDM, then the future NIDDM prevalence will be higher, presuming that people with IGT progress to NIDDM at a greater rate than people with NIDDM die. Eventually some steady state will be reached for each population depending on *1*) the frequency of susceptible genotype(s), *2*) the distribution of risk factors in the population, i.e., age, nutrition, body mass, fat distribution, and physical activity, *3*) innate incidence of IGT and NIDDM in that population or ethnic group (itself a product of *1* and *2*), and *4*) mortality rates in people with IGT and NIDDM.

If the above parameters were known, then it might be feasible to develop a more informative predictive model. The development of such models could be facilitated and validated by the documentation of trends in the prevalence and incidence of glucose intolerance and other parameters in well-characterized populations.

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