Mortality, All-Cause and Cardiovascular Disease, Over 15 Years in Multiethnic Mauritius

Impact of diabetes and intermediate forms of glucose tolerance

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OBJECTIVE — Little information is available on the impact of abnormal glucose tolerance on mortality in South Asian and African populations in the developing world. We explored this issue in a large, multiethnic cohort from the developing nation of Mauritius.

RESEARCH DESIGN AND METHODS — Population-based surveys were undertaken in 1987, 1992, and 1998. The 9,559 participants (20–82 years old) comprised 66% South Asian (Indian), 27% Creole (African), and 7% Chinese descent. Mortality was ascertained in 2007.

RESULTS — Over a median 15.1-year follow-up, 1,557 participants died. Compared with those with normal glucose tolerance, the all-cause mortality hazard ratios (HR) for known diabetes, newly diagnosed diabetes, and impaired glucose tolerance were 3.35 (95% CI 2.77–4.04), 2.11 (1.73–2.57), and 1.53 (1.26–1.87) in South Asians and 2.14 (1.65–2.79), 1.41 (1.06–1.88), and 1.08 (0.83–1.40) in Africans, respectively. Those with impaired fasting glucose were not at increased risk in either ethnicity. In the Chinese, only those with known diabetes were at increased risk of mortality with HR 3.68 (1.87–7.25).

CONCLUSIONS — This is the first study in a developing country of the impact of glucose intolerance on mortality in an African population, and one of the first studies of a South Asian population. It shows that the impact on mortality in these populations in Mauritius is comparable to that seen in developed countries. These results are important in a global context for future health policy in light of the impact of the rapid increase in prevalence of diabetes, especially in developing nations.

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Seventy percent of adults with diabetes live in the developing world, with India being the country with the largest number of people with diabetes (1). However, although data showing that diabetes is associated with a two to threefold increased risk of all-cause and cardiovascular (CVD) mortality in Europid populations are abundant (2,3), there are few or no such data for Africans and only scant data for South Asian populations living in the developing world. In South Asians, data are limited to pooled analyses of diabetes and mortality from the Diabetes Epidemiology Collaborative Analysis of Diagnostic Criteria in Asia (DECODA) collaboration (4). Within this collaboration, South Asians from two studies, a 5-year follow-up of mortality from the same cohort of Mauritians used in the current analysis and a smaller study of Fijian Indians followed over 8 years, were included. We are not aware of any prospective data on effects of type 2 diabetes on mortality in Africans living in the developing world.

The developing world imposes a very different environment from that of the developed world. There are also differences in health care systems and delivery, and there is a greater burden of communicable disease. Given these differences, data on the association between diabetes and mortality (total and CVD) collected from developed countries cannot readily be extrapolated to developing countries. Understanding the relationship of diabetes and mortality in the context of development is of paramount importance to inform decision makers of developing countries about the likely socioeconomic impact of diabetes in the future.

In this study we used national, population-based prospective data from Mauritius, a country that includes people of South Asian (Indians), African (Creoles), and Chinese ancestry to investigate the relationship of diabetes and abnormal glucose tolerance with all-cause and CVD mortality.

RESEARCH DESIGN AND METHODS

— Mauritius is a subtropical island located in the Southwestern Indian Ocean with a population of ~1.3 million. The population is 68% South Asian (Indian) origin, 3% Chinese origin, 2% Franco Mauritians, and 27% Creole (people predominantly of African origin from Madagascar, Mozambique, Malawi, Tanzania, and Zambia). In 1987, 1992, and 1998, population-based surveys were conducted using similar standardized protocols. Details of the survey methodology and data collection have been published previously (5,6). In 1987, 11 randomly selected (with probability proportional to size) population clusters were surveyed. In 1992 and 1998, all original participants, plus any new residents of the original clusters were invited for further surveys. Three additional clusters were added in 1992 and resurveyed in 1998. These extra clusters were included to increase representation of the African population and to assess whether trends in disease and risk factor contribution observed in the original study cohort also occurred in these three new clusters (6). A total of 9,559 individuals were recruited over the three surveys, and 60% participated in more than one survey (28% in all three surveys). The response rates for these surveys were all >85% (5). In 2007, a mortality follow-up study of all participants used an interviewer-administered survey at the household level. Where contact with the participant was not possible, the next of kin or other household members were interviewed. For those who could not be traced, a thorough search was conducted by interview with neighbors and relatives and tracing within the national death registry to obtain vital status, cause of death, or migration status. The correct identity of each participant was validated using previously known information. Informed and written consent was obtained from all participants. The follow-up protocol was reviewed and approved by the ethics committee of the Ministry of Health and Quality of Life, Mauritius.

Risk factors at baseline

In brief, in each survey, eligible adults attended a survey site after an overnight fast. Weight, height, and waist and hip circumferences were measured. In 1987, waist circumference was measured at the narrowest point between the umbilicus and xiphoid process and in 1992 and 1998 at the midpoint between the iliac crest and lower margin of the ribs, and, thus, the 1987 waist circumference measurement was adjusted by adding 1.5 and 2.7 cm for men and women, respectively (7). Data on education, smoking, ethnicity, and leisure time physical activity were collected by trained interviewers. Education was classified as primary school/never attended school or high school education or higher. Fasting serum samples for lipids were collected, and an oral glucose tolerance test was undertaken. Glucose assays and adjustments have been described previously (5). Glucose tolerance status was determined according to 1999 World Health Organization criteria (8). Diabetes was classified on the basis of fasting plasma glucose (FPG) ≥7.0 mmol/l or 2-h plasma glucose ≥11.1 mmol/l or current treatment with insulin or oral hypoglycemic agents. Participants reporting a history of diabetes and taking hypoglycemic medication or those with fasting and/or 2-h plasma glucose in the diabetes range were labeled as known diabetes (KDM). Participants with other cases of diabetes were labeled as newly diagnosed diabetes (NDM). Cases of diabetes were almost exclusively type 2 (9). For others, FPG <7.0 mmol/l and 2-h plasma glucose ≥7.8 mmol/l but <11.1 mmol/l indicated impaired glucose tolerance (IGT), FPG 6.1–6.9 mmol/l and 2-h plasma glucose <7.8 mmol/l indicated impaired fasting glucose (IFG), and both FPG <6.1 mmol/l and 2-h plasma glucose <7.8 mmol/l indicated normal glucose tolerance (NGT).

Causes of death

Among the 1,557 deaths, death certificates were available for 1,228 participants, relatives gave information for 1,319 participants, and hospital files were retrieved and adjudicated for 460 randomly chosen participants. No information was available for 30 deaths, other than notification of death by the next of kin. Because ICD coding was not available for deaths before 2005, causes of death were classified by study physicians into 11 groups: cardiac (n = 586, 38%), cerebrovascular (n = 228, 15%), cancer (n = 199, 13%), trauma (n = 62, 4%), diabetes (n = 25, 2%), respiratory disease (n = 104, 7%), hypertension (n = 7, 1%), renal failure (n = 85, 6%), gastrointestinal/hepatic/alcohol (n = 98, 6%), other (n = 81, 5%), and not known (n = 82, 5%). We defined CVD mortality to include deaths categorized as cardiac, cerebrovascular, hypertension, and renal failure as the primary cause of death. The accuracy of cause of death ascribed on the death certificate in the three groups (cardiovascular, cancer, and other) was compared with that adjudicated by study physicians using hospital records.

Statistical analyses

Characteristics of the participants are described by the mean ± SD, median (25th and 75th percentile), and percentages. The censoring date for all-cause mortality was the date that the participant or next of kin was interviewed or the date of death, whichever occurred first. Ascertainment of mortality or other exit status (i.e., censored/lost to follow-up) of all participants was ascertained between 2 April 2007 and 31 October 2007. Participants who attended only one survey and then were lost to follow-up (vital status at follow-up was missing) were excluded (n = 467).

For both all-cause and CVD mortality, we used the proportional hazards model (Cox model) with age as the time scale and with glucose tolerance status and all covariates updated at each survey for those present at more than one survey. The proportionality assumptions required for proportional hazards modeling for the exposures of diabetes were met.

The population attributable fraction for diabetes and all-cause mortality was calculated for each sex by the following formula (10):

$$\mathrm{PAR}_i = \frac{p_i(\mathrm{RR}_i - 1)}{1 + \sum_{j=1}^{5} p_j(\mathrm{RR}_j - 1)}$$

where p_i is the proportion of individuals in the ith of five groups (1 = NGT, 2 = IFG, 3 = IGT, 4 = NDM, and 5 = KDM) and RR_i is the mortality rate ratio in each of these groups compared with that of those with normal glycemia, so $\mathrm{RR}_1 = 1$. Analyses were performed with Stata statistical software (version 10.0; StataCorp, College Station, TX).

RESULTS

— At follow-up, 7,182 (75%) participants were alive, 1,557 (16%) were deceased, and 820 (9%) were lost to follow-up. Among those lost to follow-up, 353 attended at least two surveys and were censored, and 467 who attended only one survey were excluded, leaving a total study population of 9,092. The median follow-up time was 15.1 (0.12–20.5) years, and the crude death rate was 11.5 (95% CI 11.0–12.1) per 1,000 person-years. The baseline charac-

Table 1—*Baseline characteristics of the cohort by ethnic group according to vital status and sex*

	South Asians				Africans				Chinese			
	Men		Women		Men		Women		Men		Women	
Baseline risk factor	Alive	Dead	Alive	Dead	Alive	Dead	Alive	Dead	Alive	Dead	Alive	Dead
n	2,237	581	2,815	424	818	256	1,183	209	223	55	258	32
Age (years)	37 (30, 45)	54 (46, 63)	37 (30, 47)	58 (49, 65)	38 (31, 49)	57 (47, 64)	39 (31, 52)	57 (50, 65)	44 (35, 53)	61 (55, 68)	43 (35, 51)	63 (56, 67)
Smoking	50	60	1	4	59	70	15	15	34	36	2	3
Education*	51	18	31	6	40	12	31	11	66	27	61	9
Prior CVD†	2	11	1	5	2	8	1	7	0	5	0	3
Hypertension‡	17	45	15	46	24	52	26	65	26	55	21	59
BMI (kg/m^2)	23 ± 4	23 ± 4	24 ± 5	26 ± 6	23 ± 4	23 ± 5	26 ± 5	26 ± 5	24 ± 3	24 ± 4	23 ± 4	24 ± 5
Waist circumference (cm)	83 ± 10	83 ± 10	79 ± 11	86 ± 13	82 ± 10	83 ± 11	82 ± 11	88 ± 12	83 ± 9	86 ± 8	77 ± 9	80 ± 10
Hip circumference (cm)	91 ± 8	88 ± 8	94 ± 10	95 ± 12	92 ± 8	89 ± 9	97 ± 10	97 ± 11	91 ± 7	90 ± 7	92 ± 7	92 ± 8
Glucose tolerance status (%)												
NGT	72	44	70	38	72	48	66	40	65	25	65	44
IFG	6	4	2	2	7	8	5	3	5	7	5	0
IGT	11	15	18	18	13	17	17	22	15	16	19	22
NDM	7	21	6	17	6	15	8	19	9	24	8	13
KDM	4	17	5	26	3	11	4	16	6	27	4	22

Data are median (25th, 75th percentile), means ± SD, or %. For those who attended more than one survey, baseline refers to the first survey attended. Those who were missing at follow-up (n = 467) were excluded. *Education is defined as completed secondary school or higher. †CVD defined as previously reported angina, coronary heart disease, stroke, or amputation. ‡Hypertension is defined as blood pressure ≥140/90 mmHg or taking antihypertensive medication.

teristics of the cohort by vital status at follow-up are shown in Table 1.

The proportion of all deaths contributed to by diabetes (KDM and NDM) was 15% in men and 17% in women. Cause of death was available for 1,527 of the 1,557 deaths. The CVD mortality rate was 6.7 (95% CI 6.3–7.2) per 1,000 person-years. A total of 906 (58%) deaths were due to CVD (65% coronary heart disease, 25% cerebrovascular disease, 1% hypertension, and 9% renal failure). Of all deaths due to CVD, 62% (562 of 906) occurred in individuals with abnormal glucose metabolism at baseline. The percent agreement of cause of death ascribed on the death certificate with that adjudicated by study physicians using hospital records was 63%, with no significant difference between those with and without diabetes.

Figure 1 shows the hazard ratios (HRs) with 95% CI by glucose tolerance categories compared with NGT for all-cause and for CVD mortality, separately for the three ethnic groups. Within ethnic groups, the relative impact of diabetes and other categories of abnormal glucose tolerance on mortality was similar for both outcomes. South Asians with IGT, NDM, and KDM were at higher risk of all-cause mortality than those with NGT; the increased risk in individuals with IFG was of borderline significance. For those of African descent, only NDM and KDM had a significantly higher all-cause and CVD mortality risk compared with NGT. In the Chinese, only those with KDM were at a significantly increased risk of all-cause or CVD death. There was a significantly greater impact of diabetes (KDM and NDM) on all-cause and CVD mortality among South Asians than among Africans (interaction term: all-cause mortality, $P = 0.003$; CVD, $P = 0.032$). The number of deaths among the Chinese was too small to draw firm conclusions about differences from other groups.

In both sexes, compared with those of South Asian descent, Africans had a similar risk of all-cause mortality and Chinese had a significantly lower all-cause mortality rate, after adjustment for other risk factors (Table 2). Compared with men with NGT, men with KDM, NDM, and IGT had a significantly increased risk of all-cause mortality. In women, only those with KDM or NDM were at increased risk for all-cause mortality.

For CVD mortality, in multiple-adjusted analyses, NDM and KDM were risk factors in both sexes. IGT was a risk

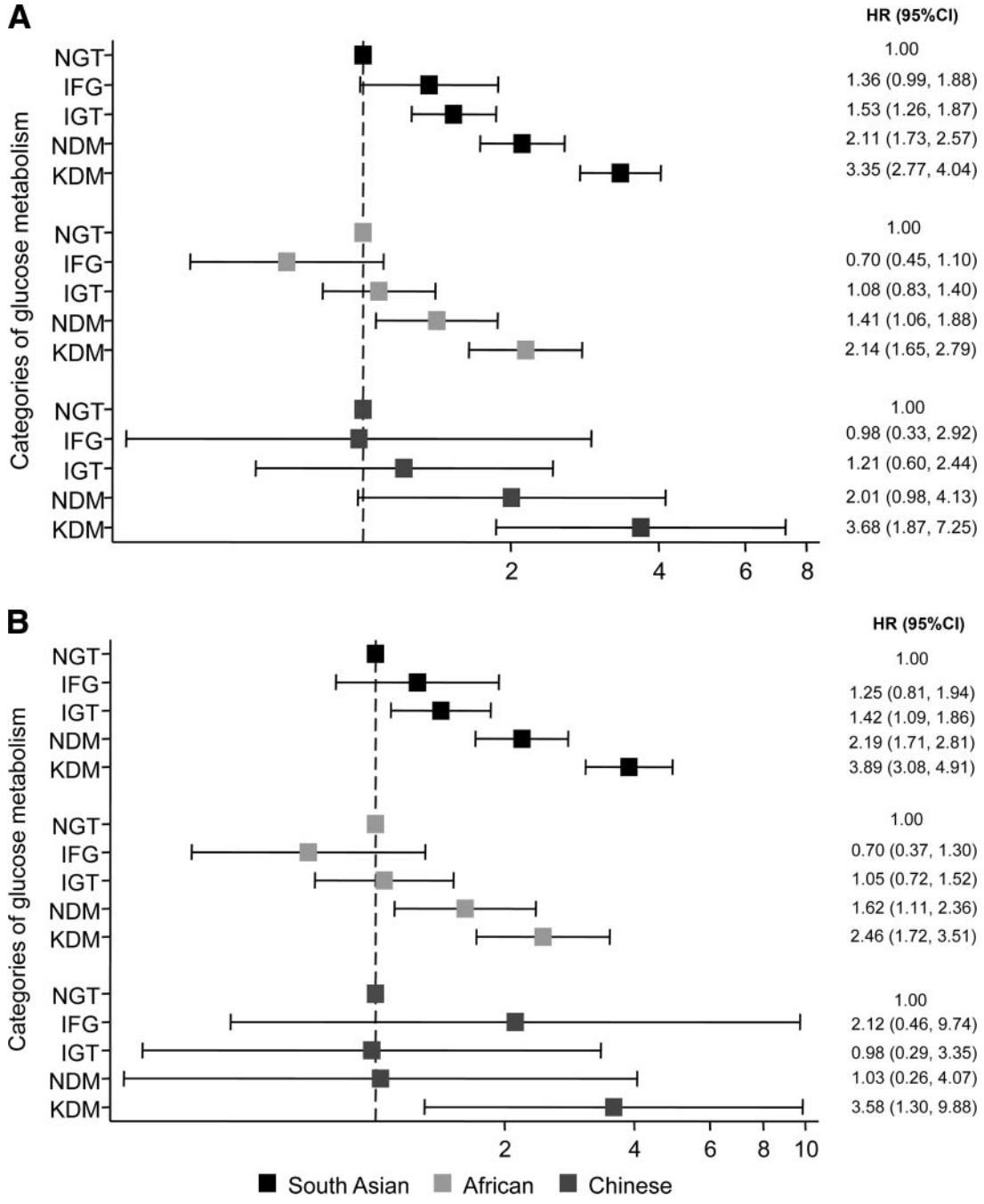

Figure 1—*Adjusted all-cause (*A*) and CVD (*B*) mortality HRs (95% CIs) for IFG, IGT, NDM, and KDM compared with NGT according to ethnic group. HRs are adjusted for prior CVD, education, sex, hypertension, waist and hip circumference, smoking, HDL cholesterol, triglycerides, and total cholesterol.*

factor for CVD mortality in men only (Table 2). When the CVD analysis was repeated using CVD deaths coded from death certification only and not adjudicated deaths, the findings were not materially different.

In a sensitivity analysis that assumed participants with missing follow-up information were alive at the last day of follow-up: the HRs (95% CI) for IFG, IGT, NDM, and KDM for men were 1.10 (0.80–1.52), 1.39 (1.21–1.71), 2.03 (1.65–2.49), and 3.12 (2.53–3.86). For women, the corresponding values were 0.94 (0.58–1.51), 1.22 (0.97–1.53), 1.51 (1.18–1.95), and 2.33 (1.88–2.90).

CONCLUSIONS — With the current high prevalence of diabetes and the predicted dramatic increase in the number of individuals with diabetes in many developing nations, studies of outcomes of diabetes, particularly morbidity and mortality become of prime importance. This study is one of the first to examine the impact of diabetes on all-cause and CVD mortality over the long term in a developing nation. We showed that there was a greater impact of diabetes on all-cause and CVD mortality among South Asians than among Africans. All-cause mortality risk in those with KDM, compared with that in those with NGT, was approximately doubled in Africans and in-

Table 2—*Adjusted HRs (95% CI) for all-cause and CVD mortality in the total population according to sex*

	HRs (95% CI)			
	All-cause mortality		CVD mortality	
Baseline characteristic	Men	Women	Men	Women
Number of deaths (*n*/*N*)	892/4,171	665/4,921	517/4,171	389/4,921
Ethnicity				
South Asian	1.00	1.0	1.0	1.0
African	1.07 (0.91–1.26)	0.86 (0.71–1.03)	0.86 (0.70–1.07)	0.74 (0.57–0.95)
Chinese	0.60 (0.47–0.78)	0.62 (0.41–0.92)	0.35 (0.23–0.54)	0.48 (0.26–0.87)
Glucose tolerance status				
NGT	1.00	1.00	1.00	1.00
IFG	1.13 (0.82–1.56)	0.90 (0.56–1.45)	1.24 (0.82–1.87)	0.66 (0.32–1.38)
IGT	1.39 (1.12–1.71)	1.21 (0.96–1.53)	1.37 (1.02–1.83)	1.16 (0.84 –1.59)
NDM	2.00 (1.63–2.46)	1.56 (1.22–2.01)	2.04 (1.56–2.67)	1.78 (1.29–2.46)
KDM	3.29 (2.68–4.03)	2.51 (2.03–3.11)	3.66 (2.82 –4.73)	3.11 (2.37–4.08)

Data were analyzed using a proportional hazards model (Cox-model) with age as the time scale and with glucose tolerance and all covariates updated at each survey for those present at more than one survey. Those who were missing at follow-up and did not contribute any follow-up information ($n = 467$) were excluded, whereas those who attended more than one survey but whose vital status could not be ascertained were censored ($n = 353$). HRs are adjusted for waist and hip circumference, smoking, hypertension, ethnicity, prior CVD, education, HDL cholesterol, triglycerides, and total cholesterol.

creased 3½ times in Asian Indians and Chinese. Furthermore, there was an ~40–50% increased mortality from CVD and all causes for South Asians with IGT, whereas in Africans with IGT mortality was not significantly increased. Mortality in individuals with IFG was not increased in any ethnic group. We also showed that 62% of all CVD deaths occurred in individuals with abnormal glucose regulation (KDM, NDM, IFG, or IGT) at baseline. Of all deaths, ~15–17% of all deaths were attributed to diabetes (KDM and NDM).

Many studies have demonstrated that diabetes is an important risk factor for both all-cause and CVD mortality in Europid populations (11–13). There are also studies of individuals of African origin living in developed countries. The Chicago Heart Association Detection Project in Industry Study showed that among 666 African American men, both asymptomatic and clinical diabetes were associated with an increased risk of death with relative risks of 1.37 (95% CI 0.85–2.20) and 1.78 (0.97–3.25) after adjustment for conventional risk factors over a period of 22 years (11). In another study of Africans from Barbados, those with diabetes had a HR for all-cause and CVD mortality of 1.80 (95% CI 1.53–2.11) and 2.10 (1.69–2.59), respectively, over a follow-up period of 9 years (14). There are, however, no data about diabetes and mortality in Africans living in developing countries. In the DECODA study, the HR for KDM for Asians of 3.22 (2.50–4.14) was similar to our estimate for South Asians and Chinese (4). DECODA includes data from a shorter follow–up (5 years) of Mauritians from our study population. In the Asia Pacific Cohort Collaboration, a pooling project that includes studies from Southeast Asia (Japan, Hong Kong, Taiwan, Korea, and China), New Zealand, and Australia, the HRs associated with diabetes for all-cause mortality for south-east Asians and non Asians were 1.62 and 1.76, lower than our estimates (15).

Our findings that IGT but not IFG is associated with an increased risk of mortality are consistent with several studies (2,4,16). The Diabetes Epidemiology: Collaborative Analysis of Diagnostic Criteria in Europe (DECODE) and DECODA studies have demonstrated that when 2-h plasma glucose elevation is controlled for, fasting glucose within the nondiabetic range is not associated with increased mortality (2,4). Contrasting findings include those of Coutinho et al. (16), who showed that IFG was significantly associated with fatal and nonfatal CVD events but were unable to separate IFG from diabetes and IGT based on 2-h plasma glucose. Barr et al. (3) showed that IGT was associated with all-cause but not CVD mortality and that IFG predicted all-cause and CVD mortality, but the follow-up was only 5 years. We found no evidence of an impact of IFG on all-cause or CVD mortality in any ethnic group. The reasons for the discrepancy among study findings is not known but could be due to differences in the populations studied, the study design, or the length of follow-up.

The proportion of all deaths contributed to by diabetes (KDM and NDM) of 15% in men and 17% in women, in this study is higher than has been reported in other studies. In the Asia Pacific Cohort Collaboration, the overall population attributable fraction of diabetes ranged from 2.3 to 12.2% for coronary heart disease and was reported to be 6% for all-cause mortality in Thailand (17). Our figure of 15–17% highlights the possible benefit of initiating lifestyle-based or pharmaceutical intervention, especially in the light of recent evidence of the efficacy of lifestyle intervention for diabetes (18–20).

Several studies have shown that abnormal glucose metabolism is present in approximately two-thirds of patients with acute myocardial infarction or coronary artery disease (3,21,22). In the current analysis, we have shown that 62% of all CVD deaths occurred in those with either diabetes, IFG, or IGT at baseline. This finding suggests that the public health benefits of targeting CVD prevention for those with pre-diabetes and the early stages of type 2 diabetes would probably be of great benefit. Such strategies are underpinned by evidence from trials (18,20,23).

The strengths of this study include a large national, population-based sample with excellent response rates and an extremely low loss to follow-up of 8.6%. Sensitivity analyses, which assumed that all participants who were lost to follow-up were alive at the last day of mortality ascertainment, showed that the HR for all-cause mortality across the spec-

trum of categories of glucose intolerance differed little from the point estimates of the primary analyses, indicating the robustness of the findings of this study.

This study is not without limitations. Cause of death was ascertained by death certificates, hospital records, and next of kin. Further, in Mauritius, information on death certificates was not ICD9 or ICD10 coded and was not available electronically before 2005; thus, ICD codes were unavailable for this study. Instead, causes of death were coded into broad categories based on the text written on death certificates. It is possible that some misclassification has occurred. It is important to note that this limitation only affects the CVD mortality data and not the all-cause mortality data. However, adjudication of cause of death of 19% of deaths using hospital records compared with text on the death certificate showed good agreement. Furthermore, in sensitivity analyses, the CVD findings are not materially changed when cause of death coding was based on the death certificate or information on the certified extract of death. Finally, the small number of Chinese participants limits our ability to provide any certainty regarding the HR estimates for this ethnic group.

In summary, the impact of diabetes on all-cause and CVD mortality in South Asians and Africans living in a developing country is just as large as it is in the developed world. Health policy and planning agencies in developing countries need to recognize and plan for the rapid emergence and escalation of noncommunicable diseases, especially diabetes and CVD, with the latter likely to be as important a component of the disease spectrum in developing countries as it is in developed countries.

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D.J.M. and S.S. wrote the manuscript and cleaned the data. P.Z.Z. designed and supervised the cross-sectional studies and reviewed/edited the manuscript. B.C. and B.B. provided advice on statistical methods, V.P. and S.K. contributed to data collection. J.T., K.G.G.M.A., and J.E.S. reviewed/edited the manuscript.

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