Mortality, all-cause and CVD, over 15 years in multi-ethnic Mauritius: the impact of diabetes and intermediate forms of glucose tolerance.

DIANNA J MAGLIANO, PHD
STEFAN SÖDERBERG, PHD
PAUL Z ZIMMET, PHD
BENDIX CARTENSEN, MSC(MATH.STAT)
BEVERLY BALKAU, PHD
VASSEN PAUVADAY, FRCP
SUDHIR KOWLESSUR, DIP PUB HEALTH ADMIN
JAAKKO TUOMILEHTO, MD
K GEORGE MM ALBERTI, FRCP
JONATHAN E SHAW, FRACP

D. J. M. And S. S. contributed equally.

1. Baker IDI Heart and Diabetes Institute, Melbourne, Australia.
2. Department of Public Health and Clinical Medicine, Umeå University, and Heart Center, Umeå, Sweden.
3. Steno Diabetes Center Gentofte, Denmark & Department of Biostatistics, University of Copenhagen, Denmark.
4. Inserm CESP Centre for Research in Epidemiology and Public Health U1018, Epidemiology of diabetes, obesity and chronic kidney disease over the lifecourse, Villejuif, France.
5. Université Paris Sud 11, UMRS 1018, Villejuif, France.
7. Hjelt Institute, Department of Public Health, University of Helsinki, Helsinki, Finland.
8. South Ostrobothnia Central Hospital, Seinajoki, Finland.
9. Department of Clinical and Preventive Medicine, Danube-University Krems, Krems, Austria.
10. Department of Endocrinology and Metabolism, St Mary's Hospital and Imperial College, London.

Corresponding author:
Dr Dianna Magliano
Email: dianna.magliano@bakeridi.edu.au

Submitted 18 February 2010 and accepted 2 June 2010.

This is an uncopyedited electronic version of an article accepted for publication in Diabetes Care. The American Diabetes Association, publisher of Diabetes Care, is not responsible for any errors or omissions in this version of the manuscript or any version derived from it by third parties. The definitive publisher-authenticated version will be available in a future issue of Diabetes Care in print and online at http://care.diabetesjournals.org.
Background—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, multi-ethnic cohort from the developing nation of Mauritius.

Methods—Population-based surveys were undertaken in 1987, 1992 and 1998. The 9559 participants (20-82 years old) comprised 66% of South Asian (Indian), 27% of Creole (African), and 7% of Chinese descent. Mortality was ascertained in 2007.

Results—Over a median 15.1 years follow-up, 1557 participants died. Compared to those with normal glucose tolerance, the all-cause mortality HRs (95% confidence intervals) for known diabetes mellitus (KDM), newly diagnosed diabetes mellitus and impaired glucose tolerance were 3.35 (2.77-4.04), 2.11 (1.73-2.57), 1.53 (1.26-1.87) in South Asians, and 2.14 (1.65-2.79), 1.41 (1.06-1.88), 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 KDM were at increased risk of mortality [HR: 3.68 (1.87-7.25)].

Conclusion—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 the light of the impact of the rapid increase in prevalence of diabetes, especially in developing nations.

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, while there is an abundance of data showing that diabetes is associated with a 2 to 3 fold increased risk of all-cause and cardiovascular (CVD) mortality in Europid populations (2; 3), there are few or no such data in Africans, and only scant data on South Asian populations living in the developing world. In South Asians, data are limited to pooled analyses of diabetes and mortality from the DECODA collaboration (4). Within this collaboration, South Asians from two studies were included - a 5 year follow-up of mortality from the same cohort of Mauritians used in the current analysis, and a smaller study of Fijians Indians followed over 8 years. 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 to 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 socio-economic impact of diabetes in the future.

In this study we use national, population-based prospective data from Mauritius, a country, which 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.

METHODS

Background and population: Mauritius is a subtropical island located in the South-western Indian Ocean with a population of about 1.3 million. The population is 68% of South Asian (Indian) origin, 3% Chinese origin, 2%, Franco Mauritian 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 previously been published (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. The inclusion of these extra clusters was to increase representation of the African population and to assess if trends in disease and risk factor contribution observed in the original study cohort also occurred in these three new clusters (6). A total of 9559 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 over 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 neighbours, 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, waist and hip circumferences were measured. In 1987, waist circumference (WC) was measured at the narrowest point between the umbilicus and xiphoid process, and in 1992 and 1998, at the mid-point between the iliac crest and lower margin of the ribs, and thus the 1987 WC measurement was adjusted by adding 1.5 cm and 2.7 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: i) primary school/never attended school, or ii) 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 (WHO) criteria (8). Diabetes was classified on the basis of
fasting plasma glucose (FPG) ≥ 7.0 mmol/l or 2 hour plasma glucose (2h-PG) ≥ 11.1 mmol/l or current treatment with insulin or oral hypoglycaemic agents. Participants reporting a history of diabetes and taking hypoglycaemic medication or with fasting and/or 2h-PG in the diabetes range were labeled as known diabetes (KDM). Other cases of diabetes were labeled newly diagnosed diabetes (NDM). Cases of diabetes were almost exclusively type 2 (9). For others, FPG < 7.0 mmol/l and 2h-PG ≥ 7.8 mmol/l but < 11.1 mmol/l indicated impaired glucose tolerance (IGT), FPG 6.1–6.9 mmol/l and 2h-PG < 7.8 mmol/l indicated impaired fasting glucose (IFG), and both FPG < 6.1 mmol/l and 2h-PG < 7.8 mmol/l indicated normal glucose tolerance (NGT).

Causes of death: Among the 1557 deaths, death certificates were available for 1228 participants, relatives gave information for 1319 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. As International Classification of Diseases (ICD) coding was not available for deaths prior to 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 categorised 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 to that adjudicated by study physicians using hospital records.

Statistical analyses: Characteristics of the participants are described by the mean (standard deviation), 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 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 modelling for the exposures of diabetes were met.

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

\[
PAR_i = \frac{p_i (RR_i - 1)}{1 + \sum_{j=1}^{5} p_j (RR_j - 1)}
\]

where \( p_i \) is the proportion of individuals in the \( i \)th of 5 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 to those with normal glycaemia, so RR\(_1\)=1. Analyses used Stata Statistical Software version 10.0 (StataCorp, College Station, Texas, Texas).
USA). The authors had full access to and take full responsibility for the integrity of the data.

RESULTS
At follow-up, 7182 (75%) participants were alive, 1557 (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, while 467 who attended only one survey were excluded, leaving a total study population of 9092. The median follow-up time was 15.1 (0.12 to 20.5) years, and the crude death rate was 11.5 (95% CI 11.0 to 12.1) per 1000 person-years. The baseline characteristics of the cohort by vital status at follow-up are shown in Table 1.

The proportion of all deaths contributed by diabetes (KDM and NDM) was 15% in men and 17% in women.

Cause of death was available for 1527 of the 1557 deaths. The CVD mortality rate was 6.7 (95% CI, 6.3 to 7.2) per 1000 person-years. A total of 906 (58%) deaths were due to CVD (65% coronary heart disease, 25% cerebrovascular disease, 1% hypertension, 9% renal failure). Sixty-two percent (562/906) of all CVD deaths occurred in people with abnormal glucose metabolism at baseline. The percentage agreement of cause of death ascribed on the death certificate to that adjudicated by study physicians using hospital records was 63%, with no significant difference between those with and without diabetes.

Fig. 1 shows the HRs (95% CI) by glucose tolerance categories compared to 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 people 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 in comparison to 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 with other groups.

In both sexes, compared to being of South Asian descent, Africans had a similar risk of all-cause mortality while the Chinese had a significantly lower all-cause mortality rate, after adjustment for other risk factors (Table 2). Compared to 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 of all-cause mortality.

For CVD mortality, in multiple-adjusted analyses, NDM and KDM were risk factors for CVD mortality in both sexes. IGT was a risk 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 the adjudicated deaths, the findings are not materially different.

In a sensitivity analysis which assumed that the 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 to 1.52), 1.39 (1.21 to
1.71), 2.03 (1.65 to 2.49), and 3.12 (2.53 to 3.86). For women, the corresponding HRs were: 0.94 (0.58 to 1.51), 1.22 (0.97 to 1.53), 1.51 (1.18 to 1.95), and 2.33 (1.88 to 2.90).

CONCLUSIONS
With the current high prevalence of diabetes and the predicted dramatic increase in the number of people 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 known diabetes, compared to NGT was approximately doubled in Africans and increased three and a half times in Asian Indians and Chinese. Furthermore, there was approximately a 40-50% increased mortality from CVD and all-causes for South Asians with IGT while in Africans with IGT mortality was not significantly increased. Mortality in people with IFG was not increased in any ethnic group. We also showed that 62% of all CVD deaths occurred in people with abnormal glucose regulation (KDM, NDM, IFG or IGT) at baseline. Approximately 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; 12; 13). There are also studies of people of African origin living in developed countries. The Chicago Heart Association Detection Project in Industry Study showed that among 666 black American men, that both asymptomatic and clinical diabetes were associated with an increased risk of death with RR (95% CI): 1.37 (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 (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 the 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 which includes studies from south-east 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 DECODE and DECODA studies have demonstrated that when 2h-PG elevation is controlled for, fasting glucose within the non-diabetic 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 non-fatal CVD events, but were unable to separate IFG from diabetes and IGT based on 2h-PG.

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 between 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 by diabetes (KDM and NDM) of 15% and 17% in men and women, respectively in this study is higher than has been reported in other studies. In the Asia Pacific Cohort Collaboration, the overall PAF of diabetes ranged from 2.3%-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 or, especially in the light of recent evidence of the efficacy of lifestyle intervention for diabetes (18; 19; 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 (21; 22; 3). 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 suggests that the public health benefits of targeting CVD prevention for those with pre-diabetes and the early stages of type 2 diabetes would likely be of great benefit. Such strategies are underpinned by evidence from trials (18; 23; 20).

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 spectrum 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 were not ICD 9 or ICD10 coded, and were not available electronically prior to 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 to 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 death certificate or information on the certified extract of death. Finally, the small number of Chinese participants limit our ability to provide any certainty around the HR estimates for this ethnic group.

In conclusion, 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 in developing countries need to recognise and plan for the rapid emergence and escalation of non-communicable diseases, especially diabetes and CVD, with the latter likely to be as important a component of the disease spectrum in developing as it is in developed countries.
Author Contributions —DJM and SS wrote the manuscript and the cleaned data. DJM and SS contributed equally to manuscript. PZ designed and supervised the cross-sectional studies and reviewed/edited the manuscript. BC and BB advised on statistical methods, VP and SK contributed to data collection, JT reviewed/edited the manuscript, KGGMA reviewed/edited the manuscript, JES reviewed/edited the manuscript.

ACKNOWLEDGEMENTS
We are most grateful to staff at the Ministry of Health and Quality of Life in Mauritius for conducting both the baseline and the follow-up study. We would also like to thank the participants for volunteering their time to be involved in the study. We would like to acknowledge, in particular, the work of Gary Dowse, Department of Health, Western Australia and Max De Courten, University of Copenhagen, Denmark, Ray Sparks, Department of Pathology, Monash University, Australia and Pierrot Chitson, Ministry of Health, Mauritius for their work on the cross-sectional surveys.

Role of funding source: The baseline surveys were funded by the World Health Organisation, Baker IDI Heart and Diabetes Institute, the University of Newcastle upon Tyne (UK), and the National Public Health Institute, Finland and by a National Institutes of Health Grant DK-25446. The mortality follow-up was funded by Baker IDI Heart and Diabetes Institute. S.S. is supported by grants from the Västerbotten County Council (ALF), and Swedish Heart and Lung Foundation. J.E.S is supported by a NHMRC Fellowship (586623).

Financial disclosure: There is no information to disclose.

REFERENCES


incidence of type 2 diabetes with lifestyle intervention or metformin. [see comment]. N Engl J Med 346:393-403, 2002


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Notes: For those who attended more than one survey, baseline refers to the first survey attended. Those who are missing at follow-up (n= 467) are excluded. Data are means ±SD, % or medians (25th, 75th percentile). *Education is defined as completed secondary school or higher. †CVD defined as previously reported angina, coronary heart disease, stroke or amputation; ‡Hypertension defined as blood pressure ≥140/90mmHg or taking anti-hypertensive medication. Abbreviations: CVD – cardiovascular disease (angina, coronary heart disease, stroke or amputation); Hip – hip circumference; HT-hypertension; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; KDM – known diabetes mellitus; NDM – newly diagnosed diabetes mellitus; NGT – normal glucose tolerance; WC– waist circumference.
Table 2—Adjusted hazard ratios (95% CI) for all-cause and CVD mortality in the total population according to sex.

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<td>1.0</td>
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<td>African</td>
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<tr>
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<td>3.29 (2.67 – 4.04)</td>
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**Notes:** Data were analysed using 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 are missing at follow-up and do not contribute any follow up information (n= 467) are excluded while those who attended more than one survey but whose vital status could not be ascertained were censored (n=353). HR are adjusted for waist and hip circumference, smoking, hypertension, ethnicity, prior CVD, education, HDL-C, triglycerides and total cholesterol. **Abbreviations:** CI – confidence interval; HR – hazards ratio; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; KDM – known diabetes mellitus; NDM – newly diagnosed diabetes mellitus; NGT – normal glucose tolerance.

**Figure Legends**

**Figure 1A and B**—Adjusted all-cause (A) and CVD (B) mortality hazard ratios (95% confidence intervals) for IFG, IGT, NDM and KDM compared to NGT according to ethnic group. HRs are adjusted for prior CVD, education, sex, hypertension, waist and hip circumference, smoking, HDLC, triglycerides and total cholesterol. **Abbreviations:** AF- African; CVD- cardiovascular disease; IFG – impaired fasting glucose; IGT – impaired glucose tolerance; KDM – known diabetes mellitus; NDM – newly diagnosed diabetes mellitus; NGT – normal glucose tolerance; SA- South Asians.
A

Categories of glucose metabolism

<table>
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<th>African</th>
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<td></td>
<td>3.08 (1.87, 7.23)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

HR (95%CI) for all-cause mortality

B

Categories of glucose metabolism

<table>
<thead>
<tr>
<th>Type</th>
<th>South Asian</th>
<th>African</th>
<th>Chinese</th>
</tr>
</thead>
<tbody>
<tr>
<td>NGT</td>
<td>1.00</td>
<td>1.25 (0.81, 1.94)</td>
<td>1.42 (1.05, 1.86)</td>
</tr>
<tr>
<td>IFG</td>
<td>1.05 (0.72, 1.52)</td>
<td>2.19 (1.71, 2.81)</td>
<td>3.89 (3.06, 4.91)</td>
</tr>
<tr>
<td>IGT</td>
<td>1.00</td>
<td>0.76 (0.37, 1.30)</td>
<td>1.62 (1.11, 2.36)</td>
</tr>
<tr>
<td>NDM</td>
<td>1.00</td>
<td>1.21 (0.90, 1.64)</td>
<td>2.46 (1.72, 3.51)</td>
</tr>
<tr>
<td>KDM</td>
<td>1.00</td>
<td>2.12 (0.46, 9.74)</td>
<td>0.98 (0.20, 3.35)</td>
</tr>
<tr>
<td></td>
<td>1.03 (0.20, 4.07)</td>
<td>3.58 (1.30, 9.88)</td>
<td></td>
</tr>
</tbody>
</table>