The various colours of type 2 diabetes: Pathogenesis and epidemiology in different ethnic groups
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Ethnic disparities in the association of impaired fasting glucose with the 10-year cumulative incidence of type 2 diabetes

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Submitted
Abstract

Aims
Evidence of ethnic disparities in the conversion of prediabetes to type 2 diabetes is scarce. We studied the association of impaired fasting glucose (IFG) and fasting plasma glucose (FPG) with the 10-year cumulative incidence of type 2 diabetes in three ethnic groups.

Methods
We analysed data for 90 South-Asian Surinamese, 190 African-Surinamese, and 176 ethnic Dutch that were collected in the periods 2001-2003 and 2011-2012. We excluded those with type 2 diabetes or missing FPG data. We defined baseline IFG as FPG of 5.7-6.9 mmol/L. We defined type 2 diabetes at follow-up as FPG ≥7.0 mmol/L, HbA1c ≥48 mmol/mol (6.5%), or self-reported type 2 diabetes.

Results
10-year cumulative incidences of type 2 diabetes were: South-Asian Surinamese, 18.9%; African-Surinamese, 13.7%; ethnic Dutch, 4.5% (p<0.05). The adjusted association of baseline IFG and FPG with the 10-year cumulative incidence of type 2 diabetes was stronger for South-Asian Surinamese than for African-Surinamese and ethnic Dutch. The IFG (compared to normoglycaemia) ORs were 11.1 [3.0-40.8] for South-Asian Surinamese, 5.1 [2.0-13.3] for African-Surinamese, and 2.2 [0.5-10.1] for ethnic Dutch.

Conclusions
The 10-year cumulative incidence of type 2 diabetes was higher and associations with baseline IFG and FPG were stronger among South-Asian Surinamese and African-Surinamese than among ethnic Dutch. Our findings confirm the high risk of type 2 diabetes in South-Asians and suggest more rapid conversion in populations of South-Asian origin and (to a lesser extent) African origin than European origin.
Introduction

The prevalence of type 2 diabetes has grown to epidemic proportions in the last few decades [1;2]. The burden of type 2 diabetes is expected to increase even further due to factors as ageing, urbanization, and the increasing prevalence of physical inactivity and obesity [2].

Several studies have shown that the prevalence and incidence of type 2 diabetes differ between ethnic groups; in particular, people of South-Asian origin are disproportionally affected [3-9]. Not only is the prevalence higher, but type 2 diabetes also seems to develop at an earlier age among South Asians than among populations of European origin [4;6]. Moreover, a high risk of type 2 diabetes and its related morbidity and mortality have been reported among populations of African origin [9].

Overt type 2 diabetes is often preceded by a condition known as prediabetes, which is characterized by increased glucose levels that are not high enough to justify the diagnosis of diabetes, yet hallmark the development of insulin resistance [10;11]. According to the ADA guidelines, prediabetes can be classified as having impaired fasting glucose (IFG), impaired glucose tolerance (IGT), and/or a glycosylated hemoglobin A1c (HbA1c) level of 43-48 mmol/mol (5.7%-6.5%) [11]. Although it has been stated that IFG and IGT should not be viewed as clinical entities in their own right, prediabetes is now widely recognized as a risk factor for type 2 diabetes and cardiovascular disease [11]. According to the ADA guidelines, patients with known prediabetes should be screened for type 2 diabetes every 1-2 years [11].

Despite the ethnic differences in the prevalence and incidence of type 2 diabetes, the knowledge about ethnic disparities in conversion from prediabetes to type 2 diabetes is limited to one study that has described a stronger association between prediabetes and incident type 2 diabetes among Hispanics than among non-Hispanic whites [12]. It is unknown whether there are disparities in conversion from prediabetes to type 2 diabetes between those of South-Asian, African, and European origins. Knowledge about such disparities may help to determine whether more frequent diabetes testing of those with prediabetes is warranted. It will also determine whether early interventions should be recommended in order to reduce the incidence of type 2 diabetes among these disproportionately affected groups. Therefore, we aimed to compare the associations of IFG and fasting plasma glucose (FPG) with the 10-year cumulative incidence of type 2 diabetes among Hindustani Surinamese (South-Asian origin), African Surinamese (African origin), and ethnic Dutch (European origin) living in the Netherlands.
Materials and Methods

Study population
The study population consisted of participants in the SUNSET study (*Surinamese in the Netherlands: study on health and ethnicity*). The SUNSET study was a cross-sectional study based on a random sample of 2975 Surinamese and ethnic Dutch individuals, aged 35-60, drawn from the population register of two neighborhoods in southeast Amsterdam, as previously described [3]. Almost half the population of the former Dutch colony of Surinam migrated to the Netherlands in 1975. Approximately 80% of these Surinamese immigrants are Hindustani (but “South Asian”, originally from the Indian sub-continent) or of African origin.

At baseline (2001-2003), 339 South-Asian Surinamese, 597 African Surinamese and 508 ethnic Dutch participants completed an interview and underwent a medical examination. In 2011 and 2012, all participants who still resided in Amsterdam were invited to take part in a follow-up examination, which was organized within the framework of the study *Healthy life in an urban setting* (HELIUS; www.heliusstudie.nl). Residency in Amsterdam was determined on the basis of the last known address of each participant in 2007, the year in which the informed consent declarations for updates of the addresses via municipal registers expired.

For our present study, we included participants who completed the interview and underwent a medical examination both at baseline and follow-up (Supplementary Figure S1). We excluded individuals with missing FPG data either at baseline or at follow-up. In addition, we excluded those with known or newly diagnosed type 2 diabetes at baseline, leaving data for 456 participants (176 ethnic Dutch, 90 South-Asian Surinamese, and 190 African Surinamese) available for analysis.

The Medical Ethical Committee of the Academic Medical Centre in Amsterdam approved the study protocols. All participants provided written informed consent. New informed consent declarations were obtained for the follow-up measurements.

Measurements
Ethnicity was classified on the basis of self-identification in the baseline questionnaire. Having a first-degree relative with type 2 diabetes was determined by self-report. The educational level was determined by the highest education achieved at baseline. The levels were grouped as: secondary school and less, i.e., ≤12 years of education (low), and vocational school and more, i.e., ≥13 years of education (high). The participants were weighed in light clothing at baseline on a SECA mechanical scale (SECA, Hamburg, Germany) to the nearest
200 g; height and waist circumference were measured to the nearest 0.01 m. We used an OMRON-M4 semi-automatic sphygmomanometer (Omron Healthcare Europe, Hoofddorp, the Netherlands) for determining blood pressure. We defined hypertension as systolic blood pressure of ≥140 mmHg, or diastolic blood pressure of ≥90 mmHg, or receiving antihypertensive therapy. All anthropometric measurements and blood pressure measurements were obtained twice, and the means were used for analysis.

We defined baseline type 2 diabetes as an FPG ≥ 7.0 mmol/L (HK/glucose-6-P-dehydrogenase test; Roche Diagnostics) and/or self-reported type 2 diabetes, excluding gestational diabetes. We defined baseline IFG as an FPG of 5.7 mmol/L through 6.9 mmol/L.

At follow-up, type 2 diabetes was defined as an FPG of ≥7.0 mmol/L (HK/glucose-6-P-dehydrogenase test; Roche Diagnostics), and/or an HbA1c level of ≥ 48 mmol/mol (6.5%), and/or self-reported type 2 diabetes. Weight and height at follow-up were measured according to a protocol similar to baseline measurement.

**Statistical analysis**

Baseline data were expressed as percentages, means and standard deviations (SDs), or medians and interquartile ranges (IQRs). We used Chi-squared Mann-Whitney U or independent t-tests to evaluate potentially selective drop-out, comparing the baseline characteristics of those with follow-up data available after 10 years with characteristics of those who were lost to follow-up. We then used Chi-squared, one-way-ANOVA, or Kruskal-Wallis tests to determine the ethnic group differences in baseline characteristics of participants present at follow-up.

We used chi-squared tests to compare the 10-year cumulative incidences of type 2 diabetes in the ethnic groups (both in the total group and after stratification by presence of IFG). Subsequently, we calculated the ORs with corresponding 95% CIs for the association between baseline IFG and the cumulative incidence of type 2 diabetes. We adjusted for age, sex, BMI and change in BMI after 10 years. In these unstratified analyses, we added ethnicity and interaction terms of IFG with ethnicity to the model to investigate whether the association between IFG and type 2 diabetes differed in the ethnic groups. Then, we repeated the analyses using FPG instead of IFG as a central determinant of type 2 diabetes. Furthermore, we repeated all analyses using waist circumference as an indicator of weight status instead of BMI. We considered p≤0.05 statistically significant. We used SPSS 18.0 (Chicago, Ill.) for all analyses.
Results

Those who were lost to follow-up were younger, had a higher BMI and greater waist circumference, a higher mean FPG, and more often had baseline IFG than those with follow-up data available after 10 years (Supplementary Table S2). The patterns of loss to follow-up were similar across the ethnic groups. The proportions of people with a family history of type 2 diabetes were similar for those with and without follow-up data.

Table 1 presents the baseline characteristics of the study population stratified by ethnicity. The ethnic Dutch were older, more frequently had a high level of education and less frequently had a family member with type 2 diabetes than the African and South-Asian Surinamese. The African Surinamese had a higher BMI and more often had hypertension than the South-Asian Surinamese and ethnic Dutch. The baseline prevalence of IFG was highest among the South-Asian Surinamese (34.4%), followed by the ethnic Dutch (22.7%) and the African Surinamese (21.1%; p<0.05).

The 10-year cumulative incidence of type 2 diabetes was highest among the South-Asian Surinamese, followed by the African Surinamese and ethnic Dutch participants (18.9%, 13.7%, and 4.5% respectively, p<0.05; Figure 1). For those with IFG at baseline, the cumulative incidences of type 2 diabetes were 41.9% for the South-Asian Surinamese, 35.5% for the African Surinamese, and 7.5% for the ethnic Dutch (p<0.01). These ethnic differences were much smaller and non-significant for those without IFG at baseline.

The association between baseline IFG and incident type 2 diabetes was stronger for the South-Asian Surinamese and, to a lesser extent, for the African Surinamese than for the ethnic Dutch (Table 2). This difference in association across the ethnic groups remained after adjustment for sex, age, BMI, and change in BMI after 10 years. The OR for IFG was 11.1 [3.0–40.8] for the South-Asian Surinamese, 5.1 [2.0–13.3] for the African Surinamese, and 2.2 [0.5–10.2] for the ethnic Dutch when compared to those without IFG at baseline (South-Asian Surinamese versus ethnic Dutch: p=0.11, African Surinamese versus ethnic Dutch: p=0.24, African Surinamese versus South-Asian Surinamese p=0.35). Furthermore, we observed a similar difference across the ethnic groups for the association of baseline FPG with incident type 2 diabetes (ORs: 18.0 [3.7–87.8] for South-Asian Surinamese, 6.8 [2.6–17.8] for African Surinamese, and 1.8 [0.5–7.0] for ethnic Dutch per mmol/L increase of FPG (South-Asian Surinamese versus ethnic Dutch, p=0.03; African Surinamese versus ethnic Dutch, p=0.09; African Surinamese versus South-Asian Surinamese p=0.29; Table 3). We obtained similar results when we adjusted for waist circumference instead of BMI (data not shown).
Discussion

We found a higher 10-year cumulative incidence of type 2 diabetes among South-Asian Surinamese and African Surinamese than among ethnic Dutch. This ethnic difference was even more striking for those with IFG than for those with normoglycemia at baseline. In line with this finding, we observed that the associations of both baseline IFG and FPG with incident type 2 diabetes were stronger among the South-Asian Surinamese and, to a lesser extent, African Surinamese than among the ethnic Dutch. This difference in associations between the ethnic groups persisted after adjustment for other well-known risk factors for type 2 diabetes.

Our finding that the 10-year cumulative incidence of type 2 diabetes was highest among the South-Asian Surinamese, followed by the African Surinamese and then the ethnic Dutch, is in line with previously reported (cross-sectional) ethnic differences in the baseline prevalence of type 2 diabetes in the SUNSET study [3]. At baseline, the prevalences of type 2 diabetes were 16.7%, 8.1%, and 4.2% for the South-Asian Surinamese, African Surinamese, and ethnic Dutch, respectively [3]. Furthermore, the cumulative incidences in our study are in line with type 2 diabetes incidences reported among ethnically diverse populations in other studies [8;13]. For example, Tillin et al. found that the 20-year cumulative incidences of type 2 diabetes were 14% for those of European origin, 33% for Indian Asians, and 30% for African Caribbeans [8]. In another study, the 10-year cumulative incidences of type 2 diabetes were somewhat higher than those in our study, namely, 8.6% for the white Americans and 17.0% for the black Americans [13]. However, Tillin et al.’s participants were approximately 15 years older than our participants of European and African origin.

In our study, baseline IFG was strongly associated with incident type 2 diabetes for both the South-Asian and African Surinamese. This is in line with results of previous studies that show that IFG is a risk factor for type 2 diabetes among populations of South-Asian and African origin [14;15]. Baseline IFG was also associated with incident type 2 diabetes for our ethnic Dutch participants, although this association was weaker and not statistically significant. Our association for the ethnic Dutch is in line with the association that some studies report among populations of European origin [12], but smaller than the associations in other studies [16;17]. The lack of statistical significance in our study is likely the result of a lack of power, due to the low number of cases of incident type 2 diabetes among the ethnic Dutch.

We found that the association of IFG with type 2 diabetes was stronger among the South-Asian Surinamese and, to a lesser extent, African Surinamese than among the ethnic Dutch. In other words, the South-Asian and African Surinamese with IFG had a higher cumulative
risk of developing type 2 diabetes than the ethnic Dutch with IFG. As mentioned, the current ADA guidelines state that people with prediabetes should be screened every 1-2 years (11). Future studies should investigate whether even more frequent testing for type 2 diabetes, particularly among South Asians with prediabetes, in various settings is warranted. Likewise, the value of early targeted interventions aimed at primary prevention of prediabetes or secondary prevention for those with prediabetes should be investigated in order to reduce the incidence of type 2 diabetes for these high-risk individuals.

Our findings suggest not only a higher absolute or cumulative risk, but also a possible ethnic difference in the conversion rate from IFG to type 2 diabetes. To our knowledge, there is only one other study that has addressed ethnic differences in the conversion from prediabetes to type 2 diabetes over time [12]. Lorenzo et al. report that, after adjustment for sex and age, Hispanics with isolated IGT or isolated IFG had higher odds of developing type 2 diabetes than their non-Hispanic white counterparts. However, when Lorenzo et al. adjusted for BMI in a multivariate analysis, the excess risk of type 2 diabetes for the Mexican Americans was partially attenuated [12]. In our study, the differential association between ethnic groups remained after adjustment for both BMI and 10-year change in BMI. As it is well known that insulin resistance and beta-cell dysfunction are the two principal components involved in the pathophysiology of type 2 diabetes [18;19], this ethnic difference may be the result of either an ethnic disparity in progression of insulin resistance, or an ethnic disparity in progression of beta-cell failure, or a combination of the two. Previous studies have found that weight gain is one of the most important causes in the development of insulin resistance [18;19]. Although we could not verify whether the effect of BMI change was potentially different between the ethnic groups because of the limited sample size in our study, the ethnic differences remained after adjusting for change in BMI over time in our study. Therefore, we speculate that a faster progression of beta-cell failure is more likely a predominant factor in the rapid conversion rate of IFG to type 2 diabetes in the South-Asian and African Surinamese than a faster progression of insulin resistance. Other studies are needed to confirm our findings and to explain the ethnic differences in the conversion of prediabetes to type 2 diabetes.

Our study has its limitations. First, there was a high loss to follow-up, so we cannot rule out selection bias. Given that those lost to follow-up had a higher BMI, a greater waist circumference, and a higher prevalence of baseline IFG than those with follow-up data available, it is likely that the loss to follow-up resulted in an underestimation of the cumulative incidence of type 2 diabetes. However, the patterns of loss to follow-up were similar across the ethnic groups. Therefore, we find it unlikely that the loss to follow-up affected the reported differences between the ethnic groups.
Second, we could only determine the presence of IFG at baseline because an oral glucose tolerance test and an HbA1c measurement were not performed at baseline. Although IFG is one of the components of prediabetes, we could not determine whether similar patterns of ethnic differences in the incidence of type 2 diabetes exist for the other components (IGT and/or an HbA1c of 43-48 mmol/mol [5.7%-6.5%]).

Third, our definition of incident diabetes may have been incomplete, as it was based on a single glucose measurement. Although this is common practice in epidemiological studies, a confirmation of the diagnosis is required in clinical practice. Moreover, we could not include the oral glucose tolerance test in our classification of type 2 diabetes because it was not performed at the follow-up visit after 10 years. Ethnic differences in the overlap of diagnoses of type 2 diabetes based on the OGTT, FPG level, and HbA1c level have been reported [20], which may have influenced the ethnic differences in the 10-year cumulative incidences of type 2 diabetes reported in our study.

All in all, we found a striking difference in the 10-year cumulative incidences of type 2 diabetes for the ethnic groups in our study and a clear difference in the risks of type 2 diabetes associated with IFG for the ethnic groups. Our findings do not only confirm the high risk of type 2 diabetes in South Asians, but also suggest a more rapid conversion from IFG to type 2 diabetes in South Asians and, to a lesser extent, in those of African origin than in those of European origin.

**Acknowledgements**

We are most grateful to the participants of the SUNSET and HELIUS study and the management team, research nurses, interviewers, research assistants and other staff who have taken part in gathering the data of this study.

The SUNSET study was funded by The Netherlands Organisation for Health Research and Development (ZonMw) and the Academic Medical Centre (AMC). The HELIUS study is conducted by the Academic Medical Center Amsterdam and the Public Health Service of Amsterdam. Both organisations provided core support for HELIUS. The HELIUS study is also funded by the Dutch Heart Foundation, the Netherlands Organization for Health Research and Development (ZonMW), and the European Union (FP-7).
Reference List

Table 1. Baseline characteristics of the study population stratified by ethnicity

<table>
<thead>
<tr>
<th></th>
<th>Total Population (N=456)</th>
<th>South-Asian Surinamese (n=90)</th>
<th>African Surinamese (n=190)</th>
<th>Ethnic Dutch (n=176)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age in years</td>
<td>45.4 ± 6.5</td>
<td>44.1 ± 6.8</td>
<td>44.2 ± 5.9</td>
<td>47.3 ± 6.6*</td>
</tr>
<tr>
<td>High level of education, n (%)b</td>
<td>116 (25.4)</td>
<td>11 (12.2)</td>
<td>40 (21.1)</td>
<td>65 (36.9)*</td>
</tr>
<tr>
<td>First-degree relative with type 2 diabetes, n (%)</td>
<td>249 (54.7)</td>
<td>69 (76.7)</td>
<td>111 (58.7)</td>
<td>67 (38.1)*</td>
</tr>
<tr>
<td>Current smoking, n (%)</td>
<td>173 (37.9)</td>
<td>23 (25.6)</td>
<td>77 (40.7)</td>
<td>72 (40.8)</td>
</tr>
<tr>
<td>Mean BMI in kg/m²</td>
<td>26.4 ± 4.7</td>
<td>25.7 ± 3.9</td>
<td>27.4 ± 5.4</td>
<td>25.6 ± 4.2*</td>
</tr>
<tr>
<td>Mean waist circumference in cm</td>
<td>90 ± 12</td>
<td>90 ± 11</td>
<td>90 ± 13</td>
<td>90 ± 13</td>
</tr>
<tr>
<td>Hypertension, n (%)c</td>
<td>118 (25.8)</td>
<td>23 (25.6)</td>
<td>62 (32.4)</td>
<td>33 (18.8)*</td>
</tr>
<tr>
<td>FPG in mmol/L</td>
<td>5.2 ± 0.5</td>
<td>5.3 ± 0.5</td>
<td>5.2 ± 0.5</td>
<td>5.3 ± 0.5</td>
</tr>
<tr>
<td>Impaired fasting glucose, n (%)d</td>
<td>111 (24.3)</td>
<td>31 (34.4)</td>
<td>40 (21.1)</td>
<td>40 (22.7)*</td>
</tr>
</tbody>
</table>

Data are given as mean (standard deviation) or number (percentage)

a p < 0.05 for one-way ANOVA, Kruskal-Wallis test, and Chi-Square test for comparisons between the ethnic groups

b Definition: systolic blood pressure ≥140 mmHg, or diastolic blood pressure ≥90 mmHg, or receiving antihypertensive therapy
c Definition: vocational school and above, i.e., ≥ 13 years of education (high)
d Impaired fasting glucose was defined as a fasting plasma glucose value of 5.7 mmol/L through 6.9 mmol/L

Table 2. Association between baseline impaired fasting glucose and type 2 diabetes in the total population and by ethnic group (compared to normoglycemia)

<table>
<thead>
<tr>
<th></th>
<th>Total population</th>
<th>South-Asian Surinamese</th>
<th>African Surinamese</th>
<th>Ethnic Dutch</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unadjusted</td>
<td>5.7 (3.1-10.5)</td>
<td>9.9 (2.9-34.3)</td>
<td>6.2 (2.6-14.9)</td>
<td>2.1 (0.5-9.3)</td>
</tr>
<tr>
<td>Adjusted for sex, age</td>
<td>6.3 (3.3-12.0)</td>
<td>10.3 (2.9-36.7)</td>
<td>5.6 (2.3-13.9)</td>
<td>2.14 (0.5-9.7)</td>
</tr>
<tr>
<td>Adjusted for sex, age, BMI</td>
<td>5.6 (2.9-10.8)</td>
<td>9.2 (2.5-33.2)</td>
<td>4.8 (1.9-12.1)</td>
<td>2.10 (0.5-9.6)</td>
</tr>
<tr>
<td>Adjusted for sex, age, BMI, BMI change after 10 years</td>
<td>6.1 (3.1-12.1)</td>
<td>11.1 (3.0-40.8)</td>
<td>5.1 (2.0-13.3)</td>
<td>2.21 (0.5-10.2)</td>
</tr>
</tbody>
</table>

Data are presented as ORs with 95% confidence intervals

All statistical models included formal testing for multiplicative interaction between impaired fasting glucose and ethnicity; the significance for the differences between ethnic groups was: South-Asian Surinamese vs ethnic Dutch: p=0.11, African Surinamese vs ethnic Dutch: p=0.24, African Surinamese vs South-Asian Surinamese p=0.35
Table 3. Association between baseline plasma fasting glucose and type 2 diabetes in the total population and by ethnic group

<table>
<thead>
<tr>
<th></th>
<th>Total population</th>
<th>South-Asian Surinamese</th>
<th>African Surinamese</th>
<th>Ethnic Dutch</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unadjusted</td>
<td>5.6 (3.1–10.3)</td>
<td>16.4 (3.6–74.9)</td>
<td>7.0 (2.9–16.8)</td>
<td>1.8 (0.5–7.1)</td>
</tr>
<tr>
<td>Adjusted for sex, age</td>
<td>6.3 (3.3–12.0)</td>
<td>17.9 (3.6–88.2)</td>
<td>6.8 (2.8–16.9)</td>
<td>1.8 (0.5–6.9)</td>
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<tr>
<td>Adjusted for sex, age, BMI</td>
<td>5.7 (2.9–10.9)</td>
<td>15.3 (3.2–74.4)</td>
<td>6.2 (2.5–15.6)</td>
<td>1.7 (0.5–6.7)</td>
</tr>
<tr>
<td>Adjusted for sex, age, BMI, BMI change after 10 years</td>
<td>6.2 (3.2–12.0)</td>
<td>18.0 (3.7–87.8)</td>
<td>6.8 (2.6–17.8)</td>
<td>1.8 (0.5–7.0)</td>
</tr>
</tbody>
</table>

Data are presented as ORs with 95% confidence intervals per mmol/L increase of fasting plasma glucose. All statistical models included formal testing for multiplicative interaction between fasting plasma glucose and ethnicity; the significance for the differences between ethnic groups was: South-Asian Surinamese vs ethnic Dutch p=0.03, African Surinamese vs ethnic Dutch p=0.09, African Surinamese vs South-Asian Surinamese p=0.29.

Figure 1. Cumulative incidence of type 2 diabetes after 10 years among the South-Asian Surinamese, African Surinamese, and ethnic Dutch.

Type 2 diabetes was defined as fasting glucose level ≥ 7.0 mmol/L and/or an HbA1c level ≥ 48 mmol/mol (6.5%) and/or self-reported type 2 diabetes.

P-values are given for chi-square tests for comparison between the ethnic groups. IFG = impaired fasting glucose; NS = not statistically significant.
Ethnic disparities in the association of impaired fasting glucose with the 10-year cumulative incidence of type 2 diabetes

Supplementary Figure S1. Flow diagram of the SUNSET study

Supplementary Figure S1. Flow diagram of the SUNSET study

* From all participants in the baseline (2001-2003) measurement of the SUNSET study, approximately 19% had moved outside of Amsterdam, 32% had actively declined to participate in the follow-up measurement, 2% had died and 13% was lost as a result of non-response at the time of the follow-up measurement.
Supplementary Table S2. Baseline characteristics of those included and those lost to follow up without baseline diabetes mellitus in the total population and stratified by ethnicity.

<table>
<thead>
<tr>
<th></th>
<th>Total Population</th>
<th>South Asian Surinamese</th>
<th>African Surinamese</th>
<th>Ethnic Dutch</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Included in analyses</td>
<td>Not included in analyses</td>
<td>Included in analyses</td>
<td>Not included in analyses</td>
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<tr>
<td>Mean age in years</td>
<td>45.4 ± 6.5</td>
<td>44.7 ± 6.5</td>
<td>44.1 ± 6.8</td>
<td>43.5 ± 6.0</td>
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<tr>
<td>Male (%)</td>
<td>41.0</td>
<td>41.5</td>
<td>43.3</td>
<td>45.4</td>
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<tr>
<td>First degree relative with DM (%)</td>
<td>54.7</td>
<td>57.4</td>
<td>76.7</td>
<td>78.5</td>
</tr>
<tr>
<td>Current smoking (%)</td>
<td>37.9</td>
<td>42.4</td>
<td>25.6</td>
<td>39.9</td>
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<tr>
<td>Mean BMI in kg/m²</td>
<td>26.4 ± 4.7 (a)</td>
<td>27.1 ± 5.3</td>
<td>25.7 ± 3.9</td>
<td>26.9 ± 5.8</td>
</tr>
<tr>
<td>Mean waist circumference in cm</td>
<td>90 ± 12 (b)</td>
<td>92 ± 14</td>
<td>90 ± 11</td>
<td>93 ± 13</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>25.8</td>
<td>30.4</td>
<td>25.6</td>
<td>30.1</td>
</tr>
<tr>
<td>Mean FPG in mmol/L</td>
<td>5.2 ± 0.5 (c)</td>
<td>5.3 ± 0.6</td>
<td>5.3 ± 0.5</td>
<td>5.4 ± 0.6</td>
</tr>
<tr>
<td>Impaired fasting glucose (%)</td>
<td>24.3 (d)</td>
<td>32.0</td>
<td>34.4</td>
<td>40.0</td>
</tr>
</tbody>
</table>

Those with baseline diabetes mellitus were excluded from the analyses.

Data are given as mean (standard deviation) or number (percentage).

- \(a\) \(p<0.05\) in independent t-test / One Way ANOVA / Kruskal Wallis test / Chi-Square for comparison between those included and not included or between the ethnic groups.
- \(b\) Definition: systolic blood pressure ≥ 140 mmHg, or diastolic blood pressure ≥ 90 mmHg, or being on antihypertensive therapy.
- \(c\) Impaired fasting glucose was defined as a fasting plasma glucose value of 5.7 mmol/L through 6.9 mmol/L.

FPG= fasting plasma glucose