Ethnic differences in the relationship of prediabetes with the presence of target-organ disease


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Abstract

Background. – Cardiovascular risk is associated with prediabetes states. Ethnic differences in risks related to prediabetes have not been well studied. The purpose of this study was to examine the relationship between prediabetes and the presence of target-organ disease in terms of ethnic differences.

Methods. – Cross-sectional analysis of the Multi-Ethnic Study of Atherosclerosis (MESA) involved a prospective cohort of 6814 participants aged 45–84 years in the US, including Black, white and hispanic subjects from an initial examination in 2000 with no known history of heart attack, stroke or diabetes. Main outcomes were comparisons of markers for coronary artery calcification (CAC), carotid stenosis more than 25%, Ankle–Brachial Index (ABI) less than 1.0 and presence of protein in urine (> 30 mg/g) between participants with normal fasting glucose (NFG) and impaired fasting glucose (IFG), and between ethnic groups with prediabetes/IFG.

Results. – There were 2457 white, 1548 black and 1229 Hispanic participants. After adjustments, there were no differences for each outcome between normal and prediabetes black and Hispanic subjects, whereas white participants with prediabetes had significantly higher odds of carotid stenosis (OR: 1.50), low ABI (OR: 1.77) and impaired fasting glucose (IFG), and between ethnic groups with prediabetes/IFG. When comparing those with IFG/prediabetes by ethnicity, blacks and Hispanics had less CAC and carotid stenosis. In addition, Hispanics had lower reduced ABIs (OR: 0.35, 95% CI 0.19–0.65) compared with whites with IFG.

Conclusion. – Prediabetes is related to the presence of several indicators of end-organ damage in white subjects, but not in blacks or Hispanics. Further longitudinal investigations into disease risks related to prediabetes in different ethnic groups are also needed.

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Keywords: Prediabetes; Atherosclerosis; Coronary artery calcification; Ethnic differences

Résumé


Contexte. – Le prédiabète est associé à une augmentation du risque cardiovasculaire. Le rôle de l’ethnicité est mal connu dans cette relation. Le but de cette étude était d’examiner les relations entre le prédiabète et l’atteinte des organes-cible selon l’ethnicité.

Méthodes. – Analyse transversale de la Multi-Ethnic Study of Atherosclerosis (MESA), cohorte prospective de 6814 participants âgés de 45–84 ans réalisée aux États-Unis. Ont été inclus les participants caucasiens, afro-américains et hispaniques, indemnes d’antécédent d’accident coronaire, d’accident vasculaire cérébral et de diabète lors de l’examen d’inclusion réalisé en 2000. Les principaux objectifs comportaient la comparaison de marqueurs [calcifications coronaires, sténose carotidienne plus de 25 %, index cheville-bras inférieur à 1,0, augmentation de l’élimination urinaire d’albumine (> 30 mg/g de créatinine)] entre participants avec glycémie à jeun normale et participants atteints de prédiabète, défini par une hyperglycémie modérée à jeun (IFG), ainsi qu’une comparaison selon l’ethnicité chez les participants atteints de prédiabète.

Résultats. – Ont été inclus dans cette étude 2457 participants caucasiens, 1548 afro-américains et 1229 hispaniques. Après ajustements multiples, il n’y avait pas de différence pour les différents marqueurs entre les participants afro-américains et hispaniques avec glycémie à jeun normale et prédiabète. En revanche, les caucasiens atteints de prédiabète avaient une probabilité significativement plus élevée de sténose carotidienne (OR 1,50), de diminution de l’index bras-cheville (OR 1,77) et d’augmentation de l’albuminurie (OR 1,66) que les caucasiens dont la glycémie à jeun était normale. En outre, les hispaniques avaient un risque plus faible de diminution de l’index bras-cheville (OR 0,35 ; IC à 95% 0,19–0,65) par rapport aux caucasiens prédiabétiques.

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Conclusions. – Le prédiabète est associé à la présence de différents marqueurs de lésions des organes-cible chez les caucasiens, mais non chez les afro-américains et les hispaniques. Cette étude transversale doit être confirmée par une étude longitudinale selon l’origine ethnique.

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Mots clés : Prédiabète ; Athérosclérose ; Calcification des artères coronaires ; Ethnicité ; Albuminurie ; Index bras-cheville

1. Introduction

Considerable evidence shows a rise in diabetes prevalence in the US, with estimates for even further increases in diabetes burden to come [1,2]. The prevalence of diabetes has become so large that it has been termed an epidemic [3]. In particular, diabetes is increasing in prevalence among adolescents and younger adults [4–6]. Mean age at the time of diagnosis decreased from 52.0 to 46.0 years between 1988 and 2000 [4]. However, diabetes may be prevented or delayed with early intervention [7–10].

Prediabetes, defined as impaired fasting glucose (IFG) and/or impaired glucose tolerance (IGT) by the American Diabetes Association (ADA) [11], refers to the altered metabolic state (increased insulin resistance and decreased insulin secretion) that occurs prior to the development of type 2 diabetes. Prevalence estimates for prediabetes include almost 12 million overweight adults in the US aged 45–74 years by the year 2000, using data from the Third National Health and Nutrition Examination Survey (NHANES III) [12]. Considering those aged 21 years or over, some 54 million Americans have prediabetes [13]. Individuals with prediabetes are at increased risk of developing diabetes [14], with trials showing an 11% annual progression in those who do not lose weight [15,16]. Individuals with prediabetes have increased macrovascular and microvascular pathology, as reflected by increased retinopathy [13], cardiovascular risk [17–19] and chronic kidney disease [20].

Blacks have a higher prevalence of diabetes and have more related complications, including peripheral arterial disease [21], nephropathy [22] and retinopathy [23], compared with whites. In addition, ethnic differences have been shown in the prevalence of coronary artery calcification (CAC), with whites being consistently more affected than blacks and Hispanics [24–27]. Ethnic differences in risks related to prediabetes have not, however, been well studied. For this reason, the purpose of the present study was to examine the relationship between prediabetes and the presence of target-organ disease in a multiethnic sample by evaluating ethnic differences for macrovascular and microvascular endpoints.

2. Materials and methods

An analysis was conducted with public-use data from the Multi-Ethnic Study of Atherosclerosis (MESA), which was designed to study the progression of subclinical cardiovascular disease (CVD). The MESA included 6814 participants, aged 45–84 years, who were recruited at six US field centres. Approximately 38% of the cohort was white, 28% was black, 23% was Hispanic and 11% was Chinese [28]. Subjects included in the MESA were free of clinically apparent CVD at baseline. However, because of ethnic differences shown in previous studies, the present analysis focused on black, white and Hispanic participants without diabetes, using data collected at an initial examination carried out between 2000 and 2002.

2.1. Variables

For the present study, prediabetes was characterized by fasting serum glucose measures. Participants were defined as having prediabetes if their fasting glucose was 100–125 mg/dL, with no diagnosis of diabetes or use of diabetic control medications. Normal fasting glucose (NFG) was defined as fasting glucose less than 100 mg/dL in a participant without diagnosed diabetes or not using diabetic control medications. This definition is consistent with parameters provided by the ADA [11]. Ethnic categories were defined as black, Hispanic and white based on participant self-identification in the MESA data.

2.2. Outcomes

2.2.1. Coronary artery calcium

Coronary artery calcium (CAC) was used as a measure of subclinical coronary atherosclerosis. CAC is a validated indicator of coronary atherosclerosis that can predict risk for coronary heart disease (CHD) events [29–31]. The presence of CAC is associated with an increased risk of CHD events, while a CAC score of 0 predicts an excellent short-term prognosis, even among patients with conventional risk factors [32,33]. CAC was assessed by chest computed tomography (CT), using either a cardiac-gated electron-beam CT scanner or a multidetector CT system. Certified technologists scanned all participants twice. A radiologist or cardiologist read all CT scans at a central reading centre (Los Angeles Biomedical Research Institute at Harbor-UCLA Medical Center in Torrance, CA, USA), using an interactive scoring system similar to that used by Yaghoubi et al. [34]. The Agatston score, a pseudo-continuous variable derived from plaque densities and their areas in all coronary arteries, was computed [35]. CAC was classified as an Agatston score of 0 or greater or equal to 1 and treated as a binary categorical variable in the model comparing subjects with and without any evidence of CAC.

2.2.2. Carotid artery stenosis

Carotid artery stenosis is narrowing of the carotid arteries associated with CVD [36,37]. A carotid Doppler ultrasound imaging method was used in the MESA protocol. The imaging protocol involved obtaining a single longitudinal lateral view of the distal 10 mm of the right and left common carotid arteries, and three longitudinal views in different imaging planes of each bulb of the internal carotid artery [38]. Based on these views, the percentage of stenosis was assessed. For our present analyses, significant stenosis was characterized as greater than 25%.
2.2.3. Ankle–Brachial Index

The Ankle–Brachial Index (ABI) is a measure of peripheral vascular disease. It is the ratio between the systolic blood pressures measured from the brachial artery and posterior tibial artery and/or dorsalis pedis at rest. In our sample population, systolic blood pressure was measured in both the right and left brachial, posterior tibial and dorsalis pedis arteries with a Doppler instrument. The average of these measures was used to calculate the ankle-to-arm ratio for each side. The ABI was then calculated as the minimum ratio of ankle blood pressure to brachial blood pressure. Ratios were calculated separately for the left and right sides, and the minimum was then selected. For the model used here, ABIs were separated dichotomously between those less than 1.0 (diminished) and those greater or equal to 1.0 (healthy).

2.2.4. Albuminuria

Urinary microalbuminuria was calculated from a spot urine measurement of albumin (mg) and creatinine (g). Based on these measurements, participants were classified as either normal [albumin (mg)/creatinine (g) < 30 mg/g], or as having either microalbuminuria (albumin/creatinine = 30–300 mg/g) or macroalbuminuria (albumin/creatinine > 300 mg/g). Having either microalbuminuria or macroalbuminuria was considered albuminuria.

2.3. Covariables

A number of demographic variables were used in the present analyses. These included: age, treated as a continuous variable measured in years; gender, treated as a categorical variable; and body mass index (BMI), defined as weight divided by height squared (kg/m²) and treated as a continuous variable. Measures of cardiovascular risk status also included in the analyses were: total cholesterol (mg/dL), based on laboratory measures and included as a continuous variable; hypertension, characterized as a categorical variable based on participants’ self-reports of a history of hypertension or use of any antihypertensive medications; and smoking status, where smokers were defined as individuals who self-reported having smoked more than 100 cigarettes in a lifetime.

2.4. Analysis

Comparisons were initially performed to compile baseline data comparing proportions and mean values, using Chi-square and two-sample Student’s t tests for covariables stratified by fasting glucose classification and grouped by ethnicity. Outcomes classified categorically (CAC, carotid stenosis, ABI and albuminuria) were analyzed using logistic regression. The unadjusted models treated the dichotomous fasting glucose classification (NFG or IFG) as the independent variable in computing odds ratios (ORs) for the respective binary outcomes. Model 1 included fasting glucose classification adjusted for age (continuous) and gender (categorical). Model 2 included the same variables as Model 1, but was also adjusted for BMI (continuous), total cholesterol (continuous), smoking (categorical) and hypertension (categorical). Logistic regressions were performed using LOGISTIC Procedure SAS software. A P value < 0.05 was considered significant.

3. Results

Demographic characteristics for the sample population are presented in Table 1, according to glycaemic groups, for a total of 2457 white, 1548 African-American and 1229 Hispanic participants. Across all ethnic groups, participants with prediabetes were older, had higher BMIs, a greater percentage of men and more hypertension compared with those with NFG levels. On comparing the demographics of the IFG group alone across ethnicity, whites had lower BMIs compared with blacks and Hispanics, whereas black participants had lower cholesterol levels and more hypertension (Table 2). There were no demographic differences across ethnicities for age, gender or smoking status.

Table 3 presents the differences in outcomes between participants with IFG. Whites had more CAC and carotid stenosis, whereas black participants had lower ABI measurements. There were no differences in albuminuria across ethnic groups.

On comparing those with IFG and those with NFG within ethnic groups (Table 4), whites with IFG had significantly higher odds of having carotid stenosis (OR: 1.50), low ABIs (OR: 1.77) and albuminuria (OR: 1.66) compared with whites with NFG in fully adjusted models (Model 2). In contrast, black and Hispanic participants with IFG showed no differences for each of
Table 2
Demographics of participants with impaired fasting glucose by ethnicity.

<table>
<thead>
<tr>
<th></th>
<th>White</th>
<th>Black</th>
<th>Hispanic</th>
<th>P \text{a}</th>
<th>P \text{b}</th>
<th>P \text{c}</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>293</td>
<td>278</td>
<td>231</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>64.9 (9.8)</td>
<td>63.8 (9.6)</td>
<td>63.9 (9.9)</td>
<td>0.19</td>
<td>0.24</td>
<td>0.95</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>30.1 (5.2)</td>
<td>31.8 (5.7)</td>
<td>31.1 (5.1)</td>
<td>&lt;0.01</td>
<td>0.02</td>
<td>0.12</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>196.7 (36.1)</td>
<td>187.5 (35.3)</td>
<td>199.8 (35.2)</td>
<td>&lt;0.01</td>
<td>0.31</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Male (%)</td>
<td>58.4</td>
<td>52.5</td>
<td>54.6</td>
<td>0.16</td>
<td>0.38</td>
<td>0.65</td>
</tr>
<tr>
<td>Smoker (%)</td>
<td>55.8</td>
<td>53.1</td>
<td>51.1</td>
<td>0.51</td>
<td>0.28</td>
<td>0.65</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>40.1</td>
<td>55.2</td>
<td>33.3</td>
<td>&lt;0.01</td>
<td>0.11</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Data are presented as means (standard deviation) unless otherwise specified; BMI: body mass index.

\text{a White vs Black.}
\text{b White vs Hispanic.}
\text{c Black vs Hispanic.}

Table 3
Comparison of outcomes in the impaired fasting glucose group stratified by ethnicity.

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>White</th>
<th>Black</th>
<th>Hispanic</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery calcification &gt; 0 (%)</td>
<td>56</td>
<td>69</td>
<td>45</td>
<td>55</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Carotid stenosis &gt; 25% (%)</td>
<td>16</td>
<td>25</td>
<td>10</td>
<td>13</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Ankle–brachial index &lt; 1.0 (%)</td>
<td>16</td>
<td>18</td>
<td>21</td>
<td>7</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Albuminuria (%)</td>
<td>12</td>
<td>10</td>
<td>12</td>
<td>13</td>
<td>0.63</td>
</tr>
</tbody>
</table>

these measurements compared with those with NFG within their respective ethnic groups.

Significant interactions between ethnicity and glycaemic categories with \( P \) values <0.05—in particular, for CAC, ABI and carotid stenosis—were revealed by the outcomes. Table 5 presents comparisons between ethnic groups with IFG for each outcome. Blacks and Hispanics with IFG were significantly less likely to have CAC and carotid stenosis compared with whites with IFG. In the fully adjusted models (Model 2), black participants had almost 70% lower odds of CAC and carotid stenosis compared with whites, whereas Hispanics had approximately half the odds of CAC (OR: 0.57, 95% CI: 0.38–0.85) compared with whites. Similar odds were shown for carotid stenosis. Furthermore, Hispanic participants had considerably lower odds of having clinically significant ABI measurements compared with whites (OR: 0.35, 95% CI: 0.19–0.65). However, there were no significant interactions between ethnicity and glycaemic groups for the outcome of albuminuria, nor were there any differences between ethnic groups with IFG and albuminuria in the regression analysis (Table 5). Also, as there were no significant interactions between gender and glycaemic categories for any of the predefined

Table 4
Odds ratios (ORs) and 95% confidence intervals (CI) for participants with impaired fasting glucose vs those with normal fasting glucose for each outcome according to ethnicity.

<table>
<thead>
<tr>
<th></th>
<th>White</th>
<th></th>
<th></th>
<th>Black</th>
<th></th>
<th></th>
<th>Hispanic</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery calcification ≥ 1</td>
<td>ORs</td>
<td>95% CI</td>
<td></td>
<td>ORs</td>
<td>95% CI</td>
<td></td>
<td>ORs</td>
<td>95% CI</td>
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</tr>
<tr>
<td>Unadjusted</td>
<td>1.88</td>
<td>1.45–2.44</td>
<td>1.23</td>
<td>0.94–1.59</td>
<td>1.90</td>
<td>1.43–2.54</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Model 1</td>
<td>1.40</td>
<td>1.03–1.90</td>
<td>0.92</td>
<td>0.69–1.23</td>
<td>1.35</td>
<td>0.97–1.87</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 2</td>
<td>1.19</td>
<td>0.87–1.63</td>
<td>0.89</td>
<td>0.66–1.19</td>
<td>1.10</td>
<td>0.78–1.56</td>
<td></td>
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</tr>
<tr>
<td>Carotid stenosis &gt; 25% (%)</td>
<td>ORs</td>
<td></td>
<td></td>
<td>ORs</td>
<td></td>
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<td>ORs</td>
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<tr>
<td>Unadjusted</td>
<td>1.99</td>
<td>1.48–2.67</td>
<td>0.85</td>
<td>0.55–1.31</td>
<td>1.51</td>
<td>0.97–2.35</td>
<td></td>
<td></td>
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<tr>
<td>Model 1</td>
<td>1.67</td>
<td>1.24–2.26</td>
<td>0.71</td>
<td>0.45–1.10</td>
<td>1.11</td>
<td>0.70–1.74</td>
<td></td>
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<tr>
<td>Model 2</td>
<td>1.50</td>
<td>1.09–2.06</td>
<td>0.71</td>
<td>0.45–1.12</td>
<td>1.06</td>
<td>0.66–1.72</td>
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<tr>
<td>Ankle–brachial index &lt; 1.0</td>
<td>ORs</td>
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<td></td>
<td>ORs</td>
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<td>ORs</td>
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</tr>
<tr>
<td>Unadjusted</td>
<td>1.68</td>
<td>1.21–2.34</td>
<td>1.21</td>
<td>0.87–1.67</td>
<td>0.94</td>
<td>0.53–1.68</td>
<td></td>
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<tr>
<td>Model 1</td>
<td>1.75</td>
<td>1.25–2.47</td>
<td>1.15</td>
<td>0.82–1.61</td>
<td>0.80</td>
<td>0.45–1.44</td>
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<tr>
<td>Model 2</td>
<td>1.77</td>
<td>1.25–2.53</td>
<td>1.20</td>
<td>0.85–1.70</td>
<td>0.81</td>
<td>0.45–1.48</td>
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<tr>
<td>Albuminuria (%)</td>
<td>ORs</td>
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<td>ORs</td>
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<td>ORs</td>
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<tr>
<td>Unadjusted</td>
<td>2.32</td>
<td>1.51–3.55</td>
<td>1.86</td>
<td>1.22–2.85</td>
<td>2.10</td>
<td>1.33–3.32</td>
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<td></td>
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<tr>
<td>Model 1</td>
<td>2.00</td>
<td>1.28–3.13</td>
<td>1.68</td>
<td>1.07–2.62</td>
<td>1.82</td>
<td>1.15–2.88</td>
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<tr>
<td>Model 2</td>
<td>1.66</td>
<td>1.05–2.62</td>
<td>1.41</td>
<td>0.89–2.24</td>
<td>1.42</td>
<td>0.86–2.33</td>
<td></td>
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</tr>
</tbody>
</table>

Model 1: adjusted for age and gender; Model 2: adjusted for age, gender, body mass index (BMI), total cholesterol, smoking and hypertension; reference groups: participants with normal fasting glucose in each ethnic group.
Table 5
Odds ratios (ORs) and 95% confidence intervals (CI) for participants with impaired fasting glucose stratified by ethnicity.

<table>
<thead>
<tr>
<th></th>
<th>Unadjusted</th>
<th></th>
<th></th>
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<th></th>
<th></th>
<th>Model 2</th>
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<td></td>
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<tr>
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<td></td>
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<tr>
<td>White</td>
<td>1.00 –</td>
<td>1.00 –</td>
<td>1.00 –</td>
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<tr>
<td>Black</td>
<td>0.37 0.27–0.53</td>
<td>0.35 0.24–0.51</td>
<td>0.31 0.21–0.47</td>
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<tr>
<td>Hispanic</td>
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<td>0.56 0.38–0.83</td>
<td>0.57 0.38–0.85</td>
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<tr>
<td>Carotid stenosis &gt; 25%</td>
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<tr>
<td>White</td>
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<td>1.00 –</td>
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<tr>
<td>Black</td>
<td>0.34 0.21–0.55</td>
<td>0.35 0.21–0.58</td>
<td>0.34 0.20–0.58</td>
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<tr>
<td>Hispanic</td>
<td>0.47 0.29–0.75</td>
<td>0.47 0.28–0.77</td>
<td>0.48 0.29–0.79</td>
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<tr>
<td>Ankle–brachial index &lt; 1.0</td>
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<td>White</td>
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</tr>
<tr>
<td>Black</td>
<td>1.26 0.83–1.92</td>
<td>1.31 0.85–2.01</td>
<td>1.24 0.79–1.94</td>
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<tr>
<td>Hispanic</td>
<td>0.33 0.18–0.60</td>
<td>0.32 0.17–0.59</td>
<td>0.35 0.19–0.65</td>
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<td>Albuminuria</td>
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<tr>
<td>White</td>
<td>1.00 –</td>
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<tr>
<td>Black</td>
<td>1.17 0.69–1.98</td>
<td>1.20 0.70–2.05</td>
<td>0.98 0.57–1.71</td>
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<tr>
<td>Hispanic</td>
<td>1.30 0.76–2.23</td>
<td>1.33 0.77–2.28</td>
<td>1.30 0.75–2.25</td>
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Model 1: adjusted for age and gender; Model 2: adjusted for age, gender, body mass index (BMI), total cholesterol, smoking and hypertension; reference group: white ethnicity.

outcomes—all P values were ≥ 0.50—analyses stratified by gender were not performed.

4. Discussion

The present study has demonstrated that prediabetes may be related to cardiovascular status differently according to ethnicity, particularly in terms of markers of early atherosclerotic disease. Our findings showed that black, white and Hispanic participants differed in the prevalence of these markers—whites had more carotid stenosis, while blacks and Hispanics had more albuminuria—and that a prediabetes state does not appear to carry the same risk of association with these markers in blacks and Hispanics as it does in whites compared with those of concordant ethnicity with NFG. Our present study also showed that white participants had increases in several indicators of end-organ damage that represent both macrovascular and microvascular injury in the presence of prediabetes compared with whites with NFG. These differences persisted despite adjusting for demographics and traditional cardiovascular risk factors. This suggests that the relationship may occur through effects on non-traditional risk factors such as oxidative stress, which would be consistent with the proposed mechanisms of vascular injury in diabetes.

It might be expected that, as blacks and Hispanics have more diabetes and more diabetes-related complications, prediabetes would be related to higher risk markers of atherosclerosis in these ethnic groups. However, such a relationship was not seen in the present sample. On comparing the prediabetes/IFG state across ethnic groups, blacks and Hispanics were no different from whites and, in the case of CAC and carotid stenosis, had lower odds. These results may be a reflection that other disease processes, such as raised blood pressure and obesity, have a greater impact on cardiovascular health than prediabetes in blacks and Hispanics, thus essentially negating the impact of prediabetes. This would be consistent with previous studies showing that hypertension is one of the most important risk factors for stroke in blacks [39], while blood pressure control is more important than glycaemic control for decreasing cardiovascular risk in diabetes [40].

As with other, previous studies, markers such as carotid stenosis and CAC were less prevalent in the present sample of blacks and Hispanics. For these outcomes, a relationship with prediabetes may not have been seen in these participants due to their lower prevalence of disease. Further studies are needed to identify the underlying causes of the ethnic differences seen in the relationship between prediabetes and the various cardiovascular outcomes evaluated in our study.

Furthermore, in our sample, 18% of black and 19% of Hispanic participants had prediabetes, while only 12% of whites did. This suggests that, while whites had higher risks of atherosclerotic markers associated with prediabetes, black and Hispanic participants had more prediabetes, which may lead to more diabetes and other morbidity in the long term. Further investigations into the onset and progression of prediabetes in each ethnic population will be important for elucidating the associated risks and potential interventions to reduce these risks. Current studies are already underway in these areas of prevention [41,42].

There are several limitations to the present study. First, as this was a cross-sectional sample, we could only evaluate associations between factors. The associations found suggest the need for prospective studies to further evaluate the findings. Second, although our outcomes are markers of atherosclerosis, they do not necessarily mean that the disease will result. However, it is well established that the presence of atherosclerosis places individuals at much higher risk for future cardiovascular outcomes. Longitudinal analyses will be necessary to determine whether or not prediabetes-related increases in markers of atherosclerosis do indeed translate into future cardiovascular morbidity and mortality. Finally, although prediabetes was here defined...
according to ADA guidelines, no information on glucose tolerance tests was available and, thus, we were unable to include those data in our definition.

5. Conclusion

The present study findings suggest that prediabetes is associated with the presence of vascular disease. This suggests that more aggressive screening and/or treatment may be worthy of future policy discussions. Moreover, prediabetes-associated risks differ according to ethnicity and are associated with an increased risk of atherosclerosis markers in whites, suggesting that ethnicity should be considered in clinicians’ decision-making when screening for prediabetes.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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References


