Changes in risk variables of metabolic syndrome since childhood in prediabetic and type 2 diabetic subjects: the Bogalusa Heart Study

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Objective: That type 2 diabetes mellitus is associated with the metabolic syndrome is known. However, information is lacking regarding the long-term and adverse changes of metabolic syndrome variables in the development of type 2 diabetes from childhood to adulthood.

Research Design and Methods: Observations were examined, retrospectively, in a community-based cohort of normoglycemic (n=1838), prediabetic (n=90), and type 2 diabetic (n=60) subjects followed serially for cardiovascular risk factors during childhood (4–11 years), adolescence (12–18 years), and adulthood (19–44 years).

Results: Diabetic subjects versus normoglycemic subjects had significantly higher levels of subscapular skinfold, body mass index (BMI), triglycerides, glucose, insulin, and homeostasis model assessment of insulin resistance (HOMA-IR), and lower levels of high-density lipoprotein cholesterol (HDL-C) beginning in childhood; and higher levels of mean arterial pressure (MAP) in adolescence and adulthood. In a multivariate model including BMI, MAP, HDL-C, low-density lipoprotein cholesterol (LDL-C), triglycerides, glucose, and insulin, adjusted for age, age$^2$, race, sex, and race by sex interaction, adverse changes in glucose and LDL-C were independently associated with prediabetic subjects while adverse changes in BMI, glucose, and HDL-C with diabetic subjects. As young adults, prediabetic and diabetic groups displayed significantly higher prevalence of obesity, hypertension, dyslipidemia, hyperinsulinemia, and metabolic syndrome.

Conclusions: These findings indicate that adverse levels of risk variables of metabolic syndrome, adiposity and measures of glucose homeostasis accelerating since childhood characterize the early natural history of type 2 diabetes, and underscore the importance of early prevention and intervention on risk factors beginning in childhood.
More than 19 million people have type 2 diabetes mellitus and another 54 million individuals show impaired fasting glucose as adults, which may represent a prediabetic state (1). This carbohydrate-insulin imbalance becomes one of the most common causes of death in the United States (2). It is also widely recognized that type 2 diabetes increases the risk for cardiovascular (CV) morbidity and premature death (3). Based on the progressive global epidemic of obesity, it is expected that the worldwide prevalence of type 2 diabetes will rise by 50% to more than 360 million people over the next 30 years (4). Since a much larger population can be classified as prediabetic, the additional risk of CV mortality becomes enormous (5).

As in type 2 diabetes, the pathogenesis of prediabetes is linked to a relative insulin deficiency and/or tissue insulin resistance associated with elevated blood glucose levels, despite secondary hyperinsulinemia (6). According to the classification of the American Diabetes Association (7), both prediabetes and type 2 diabetes represent the two categories of the impaired glucose regulation and are associated with a constellation of disorders characteristic of the metabolic syndrome (8-9). However, most studies have been performed on single, baseline measurements at middle and older ages (10-11). Information is lacking on long-term, longitudinal, and progressive changes of the risk variables of the metabolic syndrome from childhood to younger adulthood. This study observed changes over a 21 years period beginning in childhood.

RESEARCH DESIGN AND METHODS

Study population
The Bogalusa Heart Study is being conducted in a semirural, biracial (65% white and 35% black) community of Bogalusa, LA, USA. Between 1976 and 1994, 6 cross-sectional studies of school-aged children were conducted. In addition, 8 cross-sectional surveys were conducted between 1978 and 2002 with young adults who have been examined previously as children. The detailed description of the study design, participation, and protocols was described elsewhere (12). This panel design, based on repeated cross-sectional examinations conducted approximately every 3 to 4 years, resulted in serial observations from childhood to young adulthood, allowing longitudinal analyses. The participant rate was > 80% for the children and ≈60% for the adult cohort.

Subjects from 6 cross-sectional studies of children who participated at least 1 of the 14 cross-sectional surveys of children and adults were eligible for this retrospective cohort study. Of these, a total of 1988 fasting subjects (68% white, 43% male) were selected from the last three surveys of adults from 1995 to 2002. At the baseline examination, the children with a history of the treatment of diabetes mellitus or who had a fasting glucose level >= 7 mmol/l were excluded. At the initial screening, the mean ± SD of age was 10.9 ± 4.0, range 4 to 18 years. At the most recent screening, the mean ± SD of age was 32.0 ± 6.5, range 19 to 44 years. The mean follow-up interval was 21 years. The number of screenings and observations between childhood and adulthood ranged from 2 to 9 times. In all, 91% of subjects were screened >= 3 times and 63% 4 to 6 times, with a total of 9232 set of observations.
Based on the data at the last survey, adult subjects were classified as normoglycemic, prediabetic, and diabetic according to the American Diabetes Association criteria (7). Individuals were considered normoglycemic (n = 1838) if they had a fasting glucose level < 5.6 mmol/l; prediabetic (n = 90) if fasting glucose level between 5.6 to 6.9 mmol/l; diabetic (n = 60) if (1) fasting glucose level >= 7 mmol/l or (2) had a history of the treatment of diabetes mellitus. The Institutional Review Board approved consent forms used for these surveys, and informed consent was obtained from study participants in adulthood and from parents or guardians in childhood.

**General examination:** Identical protocols were used by trained examiners, nurses, and technicians across all surveys; procedures for general examination were reported previously (13). Briefly, subjects were instructed to fast for 12 hours before the screening, with compliance ascertained by an interview on the day of examination. Serum samples were obtained from antecubital venous blood and kept at 4° C until analysis the following day. Each screening day, on a random 10% subsample, a second blood sample was collected, labeled, and analyzed in a blind fashion to estimate the measurement error. Information on personal health and medication history were obtained by questionnaires. All measurements were made in replicate and mean values were used. In the longitudinal analysis, body mass index (BMI (kg/m²) = weight in kilograms divided by the square of height in meters) was used as a measure of overall adiposity and subscapular skinfold measured with Lange skinfold calipers for truncal fatness. Young adults were considered obese if their BMI was >= 30. Blood pressure measurements were obtained on the right arm of the subjects in a relaxed, sitting position. Systolic and diastolic blood pressures were recorded at the first and fifth Korotkoff phases using a mercury sphygmomanometer. Blood pressure levels were reported as the mean of 6 replicate readings, three taken by each of two randomly assigned and trained observers. Mean arterial pressure (MAP), calculated as diastolic blood pressure plus one third pulse pressure, was used in the analysis.

**Laboratory analyses:** From 1973 to 1986, cholesterol and triglycerides levels were measured using chemical procedures on a Technicon Autoanalyzer II (Technicon Instrument) according to the Laboratory Manual of the Lipid Research Clinics Program. These variables were determined by enzymatic procedures on the Abbott VP instrument (Abbott Laboratories) between 1987 and 1996 and on the Hitachi 902 Automatic Analyzer (Roche Diagnostics) afterward. Both chemical and enzymatic procedures met the performance requirements of the Lipid Standardization Program of the Centers for Disease Control and Prevention, which routinely monitors the precision and accuracy of cholesterol, triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) measurements since the beginning of this study. Measurements on the quality control samples assigned by the agency showed no consistent bias over time within or between surveys. Serum lipoprotein cholesterols were analyzed by using a combination of heparin-calcium precipitation and agar-agarose gel electrophoresis procedures (14). The intraclass correlation coefficients between the blind duplicate (10% random sample) values ranged from 0.86 to 0.98 for HDL-C, 0.86 to 0.98 for low-density lipoprotein cholesterol (LDL-C), and 0.88 to 0.99 for
triglycerides. Based on the National Cholesterol Education Program Adult Treatment Panel III guidelines (15), adult subjects were classified as dyslipidemia if they had high LDL-C (≥ 4.14 mmol/l), high triglycerides (≥ 2.26 mmol/l), or low HDL-C (< 1.03 mmol/l) or if they were on medication for dyslipidemia; and metabolic syndrome if they had at least three risk factors, consistent with that diagnosis.

From 1976 to 1991, plasma glucose was measured by a glucose oxidase method using a Beckman glucose analyzer (Beckman Instruments). Since then, it has been measured enzymatically as part of a multichemistry (SMA20) profile. Plasma immunoreactive insulin levels were measured by a commercial radioimmunoassay kit (Phadebas, Pharmacia Diagnostics). The intraclass correlation coefficients between the blind duplicate values ranged from 0.94 to 0.98 for insulin and 0.86 to 0.98 for glucose. Hyperinsulinemia was arbitrarily defined as fasting value > 108 pmol/l, a value considered indicative of insulin resistance in normoglycemic subjects (16). In addition, an index of insulin resistance was calculated according to the homeostasis model assessment formula: HOMA-IR = [insulin (μU/mL) x glucose (mmol/L)/22.5]. This model was considered useful to assess insulin resistance in epidemiological studies.

**Statistical analysis:** All of the statistical analyses were performed with SAS version 9.1 (SAS Institute). In the analyses, the race-sex groups were combined to increase statistical power and to simplify the presentation. Continuous variables were tested for normality using a Kolmogorov-Smirnov test. Values of triglycerides, glucose, insulin, and HOMA-IR variables used in the analyses were log transformed to improve normality, as necessary. Mean levels of risk variables in 4- to 11-, 12- to 18-, and ≥ 19-year age groups corresponding with the preadolescence, adolescence, and adulthood period were compared by their diabetes status in adulthood (normoglycemic versus prediabetic, normoglycemic versus diabetic, and prediabetic versus diabetic). A single measurement per subject was used to calculate mean levels of risk variable within age groups. General linear models were used to examine impaired glucose regulation differences in risk factor variables, adjusted for age, race, and sex. Using data from the last survey in adulthood, significant differences in the prevalence of prediabetes and diabetes by race and sex and metabolic syndrome and its related CV risk factors by diabetes status were tested by the Pearson’s χ²-test.

Multivariate analyses (generalized estimation equation method) were used to determine which longitudinal changes in risk variables since childhood predicted adult diabetes status. The model included BMI, MAP, HDL-C, LDL-C, TG, glucose, and insulin, adjusted for age, age², race, sex, and sex by race interaction, as applicable. Nonsignificant terms (P > 0.05) were removed from the model by backward stepwise procedure.

**RESULTS**

Mean levels of anthropometric, hemodynamic, and metabolic variables at baseline are presented in Table 1 by diabetes status. The prediabetic group had significantly higher age, LDL-C, and glucose than the normoglycemic group. With the exceptions of diastolic blood pressure and LDL-C, the diabetic group vs normoglycemic group showed significantly higher age, subscapular
skinfold, BMI, systolic blood pressure, MAP, triglycerides, glucose, insulin, and HOMA-IR and lower HDL-C. Compared to the prediabetic group, diabetic subjects had significantly higher subscapular skinfold, BMI and triglycerides, and lower HDL-C.

The prevalence of metabolic syndrome and its related CV risk factors in adulthood at the last survey is shown in Table 1 by diabetes status. Compared with the normoglycemic group, obesity in terms of excess generalized adiposity (BMI), hypertension, dyslipidemia, hyperinsulinemia, indicative of insulin resistance, and metabolic syndrome were significantly more prevalent among the prediabetic (except HDL-C) and diabetic (except LDL-C) groups. Compared with prediabetic group, high risk levels of HDL-C was more prevalent among diabetic group. Overall prevalence of metabolic syndrome was 18.4% in total sample (data not shown).

Mean levels of variables of metabolic syndrome in childhood (4 to 11 years), adolescence (12 to 18 years), and adulthood (19 to 44 years) are shown in Figure 1 by diabetes status in adulthood. A single measurement per subject was used in each age group. Comparisons were made after adjusting for age, race, and sex. The prediabetic group versus normoglycemic individuals showed higher levels of glucose and subscapular skinfold from childhood through adulthood; higher levels of LDL-C, insulin, and HOMA-IR in adolescence and adulthood; and higher levels of BMI, MAP, and triglycerides in adulthood. On the other hand, the diabetic group versus normoglycemic group had higher levels of subscapular skinfold in adolescence and adulthood; higher of BMI in adolescence; lower levels of HDL-C and LDL-C and higher levels of glucose and HOMA-IR in adulthood.

As shown in Supplementary Figure 1 of the online appendix (which is available at http://care.diabetesjournals.org), white males had significantly higher prevalence of prediabetes than white females. With respect to diabetes, white males vs white females and black females vs white females had a significantly greater prevalence. Males vs females in prediabetes and blacks vs whites in diabetes had a significantly higher prevalence; overall prevalence of prediabetes and diabetes was 4.5% and 3.0% in total sample, respectively (data not shown).

Independent relationships between adverse longitudinal changes in risk variables since childhood and adult prediabetes and diabetes conditions were determined in the multivariate models, presented in Table 2. Adverse changes in LDL-C and glucose were independently associated with prediabetes status and adverse changes in BMI, HDL-C, and
Longitudinal traits of younger adults for diabetes

When HOMA index, instead of glucose and insulin, were included to the models, HOMA index, LDL-C, and glucose were significant independent predictors in prediabetic group; and HOMA-IR, BMI, HDL-C in diabetic group; further, alternate multivariate analyses using subscapular skinfold, instead of BMI, gave essential identical results (data not shown).

CONCLUSIONS

These observations explore the natural history of impaired glucose regulation and diabetes status in a community-based population of children free from a selection bias monitored longitudinally over a period of 21 years. Data linked the conditions of prediabetes and type 2 diabetes in young adults with concurrent longitudinal changes in some metabolic syndrome risk variables from childhood to young adulthood. The present population study shows that, among the metabolic syndrome risk variables, compared to normoglycemic subjects, glucose was consistently higher from childhood through adulthood in both prediabetic and diabetic subjects; LDL-C, insulin, HOMA-IR higher in prediabetic subjects since adolescence; and obesity, triglycerides, insulin, HOMA-IR higher and HDL-C lower in diabetic subjects beginning in childhood. In terms of adverse longitudinal changes from childhood to adulthood, LDL-C and glucose were independently related with prediabetic status while obesity, HDL-C, and glucose with diabetic status.

In the current study, the prevalence of both prediabetes, which may represent an impaired fasting glucose state, and diabetes was lower than that in previous findings (1, 8, 10). This difference can be explained by the lower average age of our cohort. The observed differences of sex (males > females) in prediabetic group (1, 8, 10) and race (blacks > whites) in diabetic group (1) in the study cohort are in agreement with the earlier reports. Further, compared to other race-sex groups, prediabetic (10) and diabetic groups (1) were less prevalent among white females.

It is of interest that levels of obesity were higher beginning in childhood, changed adversely through adulthood, and related independently with diabetes in adulthood. This observation is consistent with the known tracking of risk factor variables over time, and especially of childhood obesity in predicting adulthood obesity (17). The persistent elevation of obesity has influenced the onset of type 2 diabetes occurring at a younger age (17, 18). With respect to the longitudinal changes in this present study cohort, obesity was the most consistent predictor of adverse changes leading to diabetes, regardless age, race or gender. A number of studies have shown baseline obesity as an independent and modifiable risk factor for type 2 diabetes (18, 19). Although of different populations, studies showed obesity in young children and adolescents as a strong predictor of subsequent diabetes (17, 18). Such observations suggest the molecular mechanisms by which obesity plays a part to glucose intolerance are complex and include a combination of genetic factors and mechanism in which skeletal myocytes and central adipocytes play a role (19).

This study demonstrates that both prediabetic and diabetic groups displayed excess of basal glucose, insulin, and HOMA-IR index at least by adolescence. Of note, the diabetic group displayed a persistent elevation of glucose from childhood through adulthood. Further,
glucose, but not insulin, along with lipid and obesity (in diabetes group) variables were the independent predictors of adverse longitudinal changes of impaired glucose regulation. Of interest, blood pressure was not independently associated in the models which is consistent with an earlier study in childhood and adolescence (18). Although blood pressure and insulin were individually predictors of diabetes, they were not independently correlated once obesity, glucose, and lipid variables were introduced in the multivariate analyses (18).

The deterioration in glucose levels to prediabetes or diabetes that, following a relatively stable period, occurs as a rapid, incremental increase accompanied by a decline in insulin sensitivity has been noted (20). In the current cohort study, both showed progressively increased glucose levels beginning in early life, prior to the onset of impaired glucose regulation status, suggesting even small changes of glucose levels may be a marker of altered carbohydrate-insulin imbalance.

In the present study, adverse changes in LDL-C and HDL-C were independently correlated among the prediabetic and diabetic groups. Of relevance to this present finding, Pima Indians demonstrated HDL-C as an independent modifiable predictor of diabetes in childhood and adolescence (18). Individuals with diabetes usually have increased triglycerides and decreased HDL-C resulting from the release of fatty acids from adipose tissue, the elevation of delivery of free fatty acids to the liver, and the hepatic synthesis of very low-density lipoproteins (21). This abnormal lipid profile is characterized by modestly elevated LDL-C and high triglycerides levels with a markedly increased cardiovascular risk among diabetic patients (22).

Perhaps most important is the observations that young adults with prediabetes and diabetes showed an increased prevalence of metabolic syndrome and its multiple risk factors. The observed overall prevalence of metabolic syndrome is in agreement with an earlier report (23). Also, the metabolic syndrome status was more prevalent in prediabetic and diabetic groups, as might be expected and is consistent with previous findings (11). A prediabetic status reflects an atherogenic profile of metabolic syndrome risk variables long before an overt clinical macrovascular events (24). Further, these multiple risk factors related to autopsy findings of coronary atherosclerosis we studied in our young individuals, evidence of "silent", subclinical disease that evolves from childhood.

As limitations, this study lacks postchallenge glucose and in vivo insulin action and secretion. Instead, an established simple surrogate measure HOMA index applicable to population studies was used. Also, due to lack of dietary intake data, this study did not address the role of diet in the regulation of glucose homeostasis and related risk of obesity, CV disease, and type 2 diabetes (25). The fasting status was based on self-report.

In summary, these findings indicate that adverse levels of risk variables of metabolic syndrome, adiposity and measures of glucose homeostasis in particular, and their accelerated rates of change since childhood characterizes the early natural history of carbohydrate-insulin imbalance. The current findings reinforce a primary role for early prevention and intervention
of these risk factors beginning in childhood, especially of obesity.

**ACKNOWLEDGEMENTS**

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The Bogalusa Heart Study is a joint effort of many investigators and staff members, whose contributions are gratefully acknowledged. We especially thank the study participants.

**CONFLICT OF INTEREST**

The authors have no conflict of interest.
REFERENCES


Table 1. Mean levels at baseline in childhood and prevalence at last survey in adulthood of risk variables related to metabolic syndrome by adult diabetes status: the Bogalusa Heart Study.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normoglycemia (n = 1838)</th>
<th>Prediabetes (n = 90)</th>
<th>Diabetes (n = 60)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>At baseline (mean ± SE)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)a</td>
<td>10.8 ± 0.1</td>
<td>12.4 ± 0.4‡</td>
<td>12.3 ± 0.5†</td>
</tr>
<tr>
<td>Subscapular skinfold (mm)b</td>
<td>12.6 ± 0.2</td>
<td>14.2 ± 0.7</td>
<td>17.0 ± 1.1§£</td>
</tr>
<tr>
<td>BMI (kg/m²)b</td>
<td>18.3 ± 0.1</td>
<td>19.5 ± 0.4</td>
<td>22.2 ± 0.8§‖</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)b</td>
<td>100.8 ± 0.3</td>
<td>105.0 ± 1.2</td>
<td>107.5 ± 1.7‡</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)b</td>
<td>61.8 ± 0.2</td>
<td>64.5 ± 1.2</td>
<td>65.0 ± 1.3</td>
</tr>
<tr>
<td>MAP (mm Hg)b</td>
<td>74.8 ± 0.2</td>
<td>78.0 ± 1.1</td>
<td>79.2 ± 1.3</td>
</tr>
<tr>
<td>LDL-C (mmol/l)b</td>
<td>2.27 ± 0.02</td>
<td>2.36 ± 0.07*</td>
<td>2.32 ± 0.10</td>
</tr>
<tr>
<td>HDL-C (mmol/l)b</td>
<td>1.60 ± 0.01</td>
<td>1.62 ± 0.06</td>
<td>1.38 ± 0.06†#</td>
</tr>
<tr>
<td>TG (mmol/l)b</td>
<td>0.73 ± 0.01</td>
<td>0.78 ± 0.04</td>
<td>0.90 ± 0.05§£</td>
</tr>
<tr>
<td>Glucose (mmol/l)b</td>
<td>4.7 ± 0.01</td>
<td>5.0 ± 0.1‡</td>
<td>5.1 ± 0.1§</td>
</tr>
<tr>
<td>Insulin (pmol/l)b</td>
<td>53.6 ± 1.6</td>
<td>76.7 ± 15.9</td>
<td>98.1 ± 17.4†</td>
</tr>
<tr>
<td>HOMA-IRb</td>
<td>1.8 ± 0.1</td>
<td>2.8 ± 0.6</td>
<td>3.8 ± 0.8‡</td>
</tr>
<tr>
<td><strong>At last survey (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI &gt;= 30 kg/m²</td>
<td>32.9c</td>
<td>60.0§</td>
<td>75.0§</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;= 140/90 or Rx</td>
<td>10.6</td>
<td>23.3‡</td>
<td>38.3§</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL-C &lt; 1.03 mmol/l</td>
<td>24.8</td>
<td>31.1</td>
<td>47.5§£</td>
</tr>
<tr>
<td>LDL-C &gt;= 4.14 mmol/l</td>
<td>11.9</td>
<td>24.4‡</td>
<td>18.6</td>
</tr>
<tr>
<td>Triglycerides &gt;= 2.26 mmol/l</td>
<td>10.1</td>
<td>25.6§</td>
<td>32.2§</td>
</tr>
<tr>
<td>Hyperinsulinemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 108 pmol/l</td>
<td>13.6</td>
<td>55.1§</td>
<td>51.7§</td>
</tr>
<tr>
<td>Metabolic syndromed</td>
<td>14.1</td>
<td>68.9§</td>
<td>78.0§</td>
</tr>
</tbody>
</table>

BMI: body mass index; BP: blood pressure; HDL-C: high-density lipoprotein cholesterol; HOMA-IR: homeostasis model assessment of insulin resistance; LDL-C: low-density lipoprotein cholesterol; MAP: mean arterial pressure; TG: triglycerides.

aP-values were adjusted for race and sex.
bP-values were adjusted for age, race, and sex.
cPrevalence (%) at last survey.
dDefined by the National Cholesterol Education Program Adult Treatment Panel III.

Different from normoglycemia: *P < 0.05; †P < 0.01; ‡P < 0.001; §P < 0.0001.
Different from prediabetes: †P < 0.05; ‡P < 0.01; †P < 0.0001.
Table 2. Adverse longitudinal changes in risk variables since childhood as independent correlates of adult diabetes status in the study cohort: the Bogalusa Heart Study.

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Prediabetes vs normoglycemia</th>
<th>Diabetes vs normoglycemia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β*</td>
<td>95% CI</td>
</tr>
<tr>
<td>BMI</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>HDL-C</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>LDL-C</td>
<td>0.37</td>
<td>0.18 – 0.55</td>
</tr>
<tr>
<td>Glucose</td>
<td>1.87</td>
<td>1.64 – 2.11</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 1.
*Generalized equation estimation method regression coefficient adjusted for age, age², race, and sex, and the race by sex interaction, as applicable. Model includes BMI, MAP, LDL-C, HDL-C, TG, glucose, and insulin.
Legends

Figure 1. Mean levels of BMI, subscapular skinfold, mean arterial pressure, HDL-C, LDL-C, triglycerides, fasting glucose and insulin and insulin resistance index (HOMA-IR) from childhood to adulthood by adult diabetes status. The Bogalusa Heart Study.
Longitudinal traits of younger adults for diabetes

Differences (P < 0.05, adjusted for age, race, and sex)

- a Normoglycemia vs. prediabetes
- b Normoglycemia vs. diabetes
- c Prediabetes vs. diabetes