

Presence of Diabetes Risk Factors in a Large U.S. Eighth-Grade Cohort

THE STOPP-T2D PREVENTION STUDY GROUP*

OBJECTIVE — The study was conducted in 12 middle schools to determine the prevalence of diabetes, pre-diabetes, and diabetes risk factors in eighth-grade students who were predominantly minority and evaluate the feasibility of collecting physical and laboratory data in schools.

RESEARCH DESIGN AND METHODS — Anthropometric measurements and fasting and 2-h post-glucose load blood draws were obtained from ~1,740 eighth-grade students.

RESULTS — Mean recruitment rate was 50% per school, 49% had BMI \geq 85th percentile, 40.5% had fasting glucose \geq 100 mg/dl, 0.4% had fasting glucose \geq 126 mg/dl, and 2.0% had 2-h glucose \geq 140 mg/dl and 0.1% \geq 200 mg/dl. Mean fasting insulin value was 30.1 μ U/ml, 36.2% had fasting insulin \geq 30 μ U/ml, and 2-h mean insulin was 102.1 μ U/ml. Fasting and 2-h glucose and insulin values increased across BMI percentiles, and fasting glucose was highest in Hispanic and Native American students.

CONCLUSIONS — There was a high prevalence of risk factors for diabetes, including impaired fasting glucose (\geq 100 mg/dl), hyperinsulinism suggestive of insulin resistance (fasting insulin \geq 30 μ U/ml), and BMI \geq 85th percentile. These data suggest that middle schools are appropriate targets for population-based efforts to decrease overweight and diabetes risk.

Diabetes Care 29:212–217, 2006

The prevalence of childhood obesity has increased dramatically over the past 30 years. Among youth 12–19 years of age, the percent with BMI $>$ 95th percentile for age and sex rose from 6% in the early 1970s to $>$ 16% in the 1999–2000 National Health and Nutrition Examination Survey (NHANES) (1). This increase was even more pronounced among African-American and Hispanic youth. Concomitant with this rise, there has been a corresponding increase in the incidence of type 2 diabetes among American children. Before the 1990s, this condition was rare. By 1994, type 2 diabetic patients represented up to 16% of new cases of diabetes in children in urban areas (2), and by 1999, from 8 to 45% (3). Type 2 diabetes is more common in African-American, Mexican-American, and

Native-American youth, during or after puberty (3).

Although several clinical trials have assessed the efficacy of primary and secondary type 2 diabetes prevention programs for adults (4,5), there have been few similar efforts in children and youth. As a result, in 2002, the National Institute of Diabetes and Digestive and Kidney Diseases funded a multicenter collaborative group to conduct a primary prevention trial of type 2 diabetes in children and adolescents. The collaborative group, Studies to Treat Or Prevent Pediatric Type 2 Diabetes (STOPP-T2D), has been developing a school-based multi-component intervention trial that will be conducted in middle schools. In preparation for the trial, in the fall of 2003, a pilot study was conducted to determine the prevalence of

diabetes, pre-diabetes, and diabetes risk factors in eighth-grade students who were predominantly minority, to explore the role of race and ethnicity, and to evaluate the feasibility of collecting physical and laboratory data in the school setting.

RESEARCH DESIGN AND METHODS

Four middle schools with at least 50% minority students were recruited by each participating center in Southern California (University of California at Irvine and the Keck School of Medicine at University of Southern California), Texas (Baylor College of Medicine), and North Carolina (University of North Carolina at Chapel Hill). All eighth-grade students were invited to participate in a health screening. Students with diabetes (as reported by parent or guardian) were excluded. Informed consent from parents and assent from students were obtained. Students were given a \$50 incentive for participation. Before recruitment, data were collected from all eighth-grade students for height, weight, age, ethnicity, and sex. This study was approved by the institutional review boards at each center.

Data collection

Children were directed to wear loose light clothing and to arrive fasting (i.e., having nothing to eat or drink except water after midnight). Students were assembled before school and during the first periods of the day. With shoes, jewelry, and outer layers of clothing removed, height and weight were measured using the Prospective Enterprises PE-AIM-101 stadiometer and the SECA Alpha 882 electronic scale. Tanner stage was determined from a sex-specific self-administered form using the Pubertal Development Scale (6–8). Study staff received training and certification.

Race/ethnicity information was collected by student self-report. A parent or guardian completed a questionnaire about family and medical history. The child was considered to have a first-degree family history if the respondent indicated that the natural mother, the natural father, or any full sister or brother had diabetes. Students underwent two blood draws with the option

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Received for publication 7 June 2005 and accepted in revised form 23 October 2005.

*A complete listing of STOPP-T2D Prevention Study Group members can be found in the APPENDIX.

Abbreviations: IFG, impaired fasting glucose; IGT, impaired glucose tolerance; NHANES, National Health and Nutrition Examination Survey; STOPP-T2D, Studies to Treat Or Prevent Pediatric Type 2 Diabetes.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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of using local anesthetic cream. Students were questioned about fasting, and those who indicated they had consumed food or beverage were rescheduled and still received their incentive. Fasting blood was obtained to determine glucose, insulin, and HbA_{1c} (A1C) levels. Students were then given oral glucose (glucose solution 1.75 g/kg, to a maximal dose of 75 g). Two hours post-glucose load, blood was obtained to determine glucose and insulin levels. A fasting glucose value ≥ 100 mg/dl was defined using American Diabetes Association criteria (9) as impaired fasting glucose (IFG), and a 2-h post-glucose load value ≥ 140 mg/dl was defined as impaired glucose tolerance (IGT).

Blood for glucose analysis was collected in 2-ml BD-vacutainers (Becton Dickinson, Franklin Lakes, NJ) containing Na-fluoride, while blood for insulin analysis was collected in 2-ml BD-vacutainers containing Na-heparin. Vacutainers were inverted immediately to ensure proper mixing, iced or refrigerated for 30–60 min, and centrifuged at 1,300g for 15 min. Plasma was transferred into cryovials, frozen, and shipped on dry ice to Northwest Lipid Metabolism and Diabetes Research Laboratories, University of Washington, Seattle. Analyses of glucose were performed on a Hitachi 917 auto-analyzer by the hexokinase method using reagent from Roche Diagnostics. Analyses of total immunoreactive insulin was performed by a double-antibody radioimmunoassay (10). The assay was a 48-h, PEG-accelerated assay in which a limiting amount of guinea pig anti-insulin antibody was incubated with iodinated insulin tracer and plasma samples. The sensitivity limit of the assay was 3 μ U/ml. The among assay coefficients of variation (CVs) of the two low- and high-insulin quality control samples were 6.9 and 4.6%, respectively. The CV on blind split duplicates was consistently $< 8.0\%$. Results of the child's health screening were mailed to parents explaining the values.

In addition, waist circumference, assessment for the presence of acanthosis, blood pressure, skin folds, plasma lipid profile (total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides), C-peptide, and proinsulin levels were determined but are not reported here.

Statistical analysis

Descriptive statistics are expressed as means, SDs, frequencies, and percentages. Because this was a feasibility study,

sample size was determined by resource availability rather than by power calculations. *P* values represent exploratory findings and are presented without adjustment for multiple comparisons.

BMI percentile by age and sex was calculated using the SAS program provided by the CDC referencing year 2000 (11, 12). Youth with BMI ≥ 85 th but < 95 th percentile were classified as at risk for overweight and those ≥ 95 th percentile were classified as overweight.

P values are given from analyses of linear models that included a random effect for school to adjust for clustering of children within schools. Continuous outcomes were analyzed using linear mixed models and categorical outcomes using generalized estimating equations. Fasting insulin and 2-h insulin had skewed distributions and were log transformed. SAS statistical software version 8.2 (SAS Institute, Cary, NC) was used for all statistical analyses.

RESULTS — A total of 1,740 eighth-grade students provided informed consent; however, complete data were not collected on all students. The mean number of participants per school was 144 (range 85–199). The mean recruitment rate (participants/total number of available eighth-grade students) was 50% per school with a range of 33–67%. Recruitment in larger schools was closed even though more students were willing to participate. Participants who entered the study in each school were representative with regard to the percent of students with BMI ≥ 85 th percentile, percent by sex, and percent minority compared with the entire eighth-grade student population of each school. For fasting glucose and insulin, 1,643 and 1,633 subjects, respectively, had data. For 2-h post-glucose load data, the number of students with glucose and insulin values was 1,128 and 1,124, respectively. The decreased number of specimens was due to an inappropriate dose of oral glucose solution given at one site to some students without untoward effects. This invalidated their postload results.

The sample had an age of 13.6 ± 0.6 years (mean \pm SD) and was 43.5% male, 52.7% Hispanic, 23.2% African American, 15.1% Caucasian, 2.2% Native American, and 6.3% other race/ethnicity. BMI was 24.3 ± 5.9 kg/m². Girls were more advanced in puberty than boys; 93% of girls were Tanner stages 4 and 5, whereas 80.8% of boys were Tanner

stages 3 and 4. A total of 13% reported having a first-degree relative with diabetes.

Of the students, 49% had a BMI ≥ 85 th percentile. As shown in Table 1, the mean fasting glucose was 98.2 mg/dl, and 40.5% had fasting glucose values ≥ 100 mg/dl, the cut point for IFG (9). Few subjects had an elevated 2-h glucose level ≥ 140 mg/dl, or IGT. The mean fasting insulin value was 30.1 μ U/ml, and 36.2% had values ≥ 30 μ U/ml, which has been previously described as the cutoff for hyperinsulinism for midpubertal youth (13). Two-hour mean insulin was 102.1 μ U/ml. The mean A1C (not shown) was $5.4 \pm 0.3\%$; 3.1% ($n = 51$) had A1C $\geq 6.0\%$ (nondiabetes range for A1C from 3 to 6%).

Glucose and insulin values rose across increasing BMI percentiles (Table 1). The increase was more pronounced for insulin than for glucose; 36.5% of students with a BMI < 85 th percentile had a fasting glucose ≥ 100 mg/dl, and 47% of those with a BMI ≥ 95 th percentile had a fasting glucose ≥ 100 mg/dl. The percent of students with a fasting glucose ≥ 110 mg/dl doubled from 4.4 to 8.9% from the lowest to the highest BMI category. With regard to insulin, among youth with BMI < 85 th percentile, 16.0% had values ≥ 30 μ U/ml, whereas 72.3% of those with BMI ≥ 95 th percentile had elevated values.

There was a nonlinear relationship between fasting insulin and BMI percentile, with the greatest increase in the curve starting in the 90–95th BMI percentile. There were significant differences across racial/ethnic groups for BMI, fasting glucose, fasting insulin, and 2-h insulin (Table 1). Native Americans had the highest BMI and Caucasians had the lowest. There was no statistically significant difference between Hispanics and African Americans for BMI, but these two groups were significantly lower than Native Americans and significantly higher than Caucasians. Native Americans and Hispanics had the highest fasting glucose, and African Americans had the lowest. Despite having similar BMI, Hispanic youth had significantly higher fasting glucose levels than African Americans. The difference in mean fasting glucose was greater between Native Americans and African Americans than between Hispanics and African Americans, but the former difference was not statistically significant.

Table 2 shows mean fasting and 2-h glucose and insulin values by race/ethnicity and BMI percentile. Linear mixed models were used to explore

Table 1—Glucose, insulin, and BMI overall and by subgroup

	Overall	Race/ethnicity*				BMI percentile		
		Hispanic	African American	Caucasian	Native American	<85th	85th to <95th	≥95th
n		862	379	247	44	876	341	501
BMI (kg/m ²)	24.3 ± 5.9	24.8 ± 5.9	24.4 ± 6.3	22.3 ± 4.6	27.6 ± 6.9			
			P < 0.0001					
BMI percentile								
<85th	51.0%	45.1%	52.0%	65.6%	37.2%			
85th to <95th	19.8%	20.6%	20.9%	21.0%	16.3%			
≥95th	29.2%	34.3%	27.1%	13.4%	46.5%			
Fasting glucose (mg/dl)	98.2 ± 8.5	99.1 ± 9.2	96.5 ± 7.9	97.8 ± 7.5	99.6 ± 5.7	97.3 ± 7.0	98.3 ± 7.9	99.9 ± 10.8
			P = 0.0092				P = 0.0172†	
Fasting glucose ≥100 mg/dl	40.5%	44.5%	32.4%	38.8%	45.5%	36.5%	42.1%	47.0%
Fasting glucose ≥110 mg/dl	6.2%	6.5%	6.2%	5.3%	6.8%	4.4%	6.7%	8.9%
Fasting insulin (μU/ml)	30.1 ± 19.1	33.5 ± 21.6	27.0 ± 15.5	24.4 ± 12.4	28.6 ± 14.0	22.5 ± 11.5	28.9 ± 16.4	44.8 ± 22.9
			P < 0.0001‡				P < 0.0001‡†	
Fasting insulin ≥30 μU/ml	36.2%	44.3%	29.3%	20.5%	36.4%	16.0%	36.2%	72.3%
2-h glucose (mg/dl)	97.9 ± 21.9	96.9 ± 24.8	100.8 ± 17.4	95.4 ± 19.6	108.1 ± 22.5	93.2 ± 18.9	98.5 ± 18.6	105.8 ± 26.4
			P = 0.2126				P < 0.0001†	
2-h glucose ≥140 mg/dl	2.3%	3.2%	1.3%	0.9%	7.3%	0.9%	3.4%	4.1%
2-h insulin (μU/ml)	102.1 ± 105.7	121.3 ± 131.4	90.6 ± 67.3	68.6 ± 41.2	98.1 ± 89.4	76.1 ± 48.7	102.5 ± 138.9	148.6 ± 133.4
			P < 0.0001‡				P < 0.0001‡†	

Data are means ± SD unless otherwise indicated. *n = 103 "other" race/ethnicity dropped from analysis. †P value based on testing both variables as continuous measures. ‡Insulin log transformed for testing.

whether the difference in BMI explained the difference in fasting glucose, fasting insulin, and 2-h insulin across race/ethnicity. The BMI-by-race interaction term was significant for fasting insulin (P = 0.0002) and 2-h insulin (P = 0.0253) but not for fasting glucose (P = 0.8596). Fasting insulin increased more in Hispanics and Caucasians from BMI percentiles <85th to 85–95th; at ≥95th percentile, the increase continued linearly in Caucasians and accelerated in the other three groups. For 2-h insulin, whereas the values in Hispanics increased markedly from BMI <85th percentile to 85–95th, in those ≥95th percentile, African Americans and Native Americans increased more than Caucasians, but Hispanics increased more than other racial groups.

More than half (57.6%) of our students had both normal fasting and 2-h glucose levels, compared with 41.1% who had elevated fasting but normal 2-h glucose values. Very few students (2%) had both elevated fasting and 2-h glucose levels in the pre-diabetes range, and <1% had diabetes by any criteria (sample excluded students already reportedly diagnosed with diabetes).

Three risk factors for diabetes were identified (Table 3): 1) BMI ≥85th percentile, 2) fasting glucose ≥100 mg/dl, and 3) fasting insulin ≥30 μU/ml. All three risk factors were elevated in 14.8% of the sample. There was a higher than expected proportion of males (19.4%), Hispanics (19.8%), Native Americans (20.9%), and students with a positive family history of diabetes (24.7%) in this high-risk group. In contrast, 28.1% of students had BMI, fasting insulin, and fasting glucose below the cut points.

CONCLUSIONS— The purpose of this pilot study was to determine the prevalence of diabetes, pre-diabetes, and diabetes risk factors in eighth-grade students who were predominantly minority, to explore the role of race and ethnicity, and to evaluate the feasibility of collecting physical and laboratory data in the school setting.

This study showed that half of eighth-grade students and their families were willing to participate in a school-based health screening to determine risk factors for diabetes. The students who chose to participate in this study were representative of their schools' general populations with regard to race/ethnicity, sex breakdown, and percent with BMI ≥85th percentile. A small percentage (13%) of

Table 3—Distribution of risk indicators for type 2 diabetes for the total sample

	BMI <85th percentile		BMI ≥85th percentile	
	Fasting insulin <30 μU/ml	Fasting insulin ≥30 μU/ml	Fasting insulin <30 μU/ml	Fasting insulin ≥30 μU/ml
Fasting glucose <100 mg/dl	28.1	4.1	13.6	13.5
Fasting glucose ≥100 mg/dl	14.5	4.0	7.4	14.8

Data are percent.

participants reported having a first-degree relative with diabetes. With the rise of diabetes in adults of Hispanic, African-American, and Native-American descent approaching 10% (14), it had been anticipated that more students would have had a family history of diabetes.

Of the students who participated, 49% had a BMI ≥85th percentile for sex and age, 19.8% were at risk for overweight (BMI ≥85th and <95th percentile), and 29.2% were overweight (BMI ≥95th percentile). The 85th percentile, which is approximately equivalent to a BMI of 25 kg/m² in adults, has been reported as the level above which youth develop type 2 diabetes (3). Our BMI data are similar to what has been recently reported by Elkins et al. (15), who showed that in low-income inner-city public school students with a mean age of 16 years, 44.4% of boys and 50.4% of girls had a BMI ≥85th percentile. However, our data showed higher rates of elevated BMI than reported in NHANES 1999–2000 (1). In NHANES, 34% of children 6–19 years of age had a BMI ≥85th percentile, 20% were at risk for overweight, and 14% were overweight, indicating the difference is accounted for almost exclusively by the percent of subjects in the highest overweight category. Racial/ethnic differences were greater for individuals with a BMI ≥95th percentile than ≥85th percentile. There was a nearly 2.5-fold greater percentage of Hispanics and 3.5-fold greater percentage of Native American students compared with Caucasians in the overweight category.

The mean fasting glucose (98.2 mg/dl) was higher than values previously reported in population-based and clinical studies (1,16–18). Ford et al. (17) reported that fasting glucose levels have decreased by 2.5 mg/dl over the last decade from NHANES III (1988–1994) to NHANES 2000 (1999–2000). They reported mean values in 13-year-old subjects of 91.9 mg/dl in 1994 and 85.3

mg/dl in 2000; the lower value reported in NHANES may reflect the smaller percent of overweight and ethnic minority youth in the NHANES cohort compared with ours. Our mean fasting glucose was higher than values found in recent clinical studies. These studies either included a large percentage of prepubertal subjects (18), who are known to have lower fasting glucose values, or a higher percentage of not only young children but also Caucasians and African Americans (16), groups that also have lower mean values. Our mean fasting glucose data are similar to those described by Goran and Gower (19) in subjects across the weight spectrum. For their subjects in Tanner stages 3–4, like our students, mean fasting glucose was 95.6 mg/dl for Caucasians and 96.2 mg/dl for African Americans—an identical mean value to that found in our African-American subjects.

Using the new American Diabetes Association cutoff of 100 mg/dl, a surprisingly high percentage (40.5%) of youth in our study had IFG. Because there were a large number of students with fasting glucose values between 100 and 110 mg/dl, the percentage with IFG would have been much lower (6.2%) if we used the previous cutoff for fasting glucose of 110 mg/dl. Very few studies have described the percentage of adolescents with IFG using the 100-mg/dl cutoff. Using the NHANES 2000 data, Duncan et al. (20) found that 7.6% of adolescents had fasting glucose values ≥100 mg/dl, although nearly double the percent of Hispanic youth (13.5%) met this cutoff. Dolan et al. (21) reported on a cohort of 2,501 primarily African-American and non-Hispanic white students in grades 5–12. They found 175 (7%) had IFG on initial screening in the school setting. The lower percentage with IFG might be due to the difference in racial/ethnic distribution (low percentage of Hispanic and Native American, high percentage of African American and Caucasian) and to there being only 40 youth in puberty.

Table 2—Glucose and insulin by BMI percentile and race/ethnicity

	Fasting glucose by BMI			2-h glucose by BMI			Fasting insulin by BMI			2-h insulin by BMI		
	<85th	85th to <95th	≥95th	<85th	85th to <95th	≥95th	<85th	85th to ≥95th	≥95th	<85th	85th to <95th	≥95th
Hispanic	97.9 ± 6.9	99.5 ± 7.3	100.4 ± 12.3	90.0 ± 18.6	99.1 ± 20.6	104.8 ± 31.0	23.6 ± 13.3	31.3 ± 19.2	48.2 ± 23.9	82.8 ± 55.4	131.4 ± 191.1	166.6 ± 143.0
African American	96.2 ± 7.7	94.9 ± 8.7	98.1 ± 7.6	97.8 ± 16.4	99.3 ± 18.1	107.2 ± 17.1	21.8 ± 10.3	24.7 ± 12.7	38.9 ± 19.2	77.2 ± 40.4	78.1 ± 51.9	124.6 ± 98.5
Caucasian	97.0 ± 6.8	98.7 ± 7.8	100.5 ± 9.6	93.0 ± 20.8	97.7 ± 14.6	102.8 ± 18.5	20.6 ± 8.4	28.4 ± 10.4	36.8 ± 19.8	61.7 ± 32.7	77.3 ± 48.2	88.0 ± 55.7
Native American*	97.4 ± 5.2	99.1 ± 6.4	101.5 ± 5.7	112.3 ± 19.5	82.6 ± 16.0	115.6 ± 20.5	21.5 ± 8.4	24.4 ± 13.1	36.0 ± 14.9	90.8 ± 50.8	42.5 ± 17.4	126.3 ± 116.6

Data are means ± SD. n = 103 "other" race/ethnicity dropped from analysis. *Fasting values based on sample sizes 16, 7, and 20 and 2-h values based on sample sizes 14, 7, and 19 across the three BMI percentile categories.

A number of factors could account for the high percentage of our students with glucose dysregulation. It is likely our percentage with IFG was high, as was the mean fasting glucose, because our sample included a high percentage of overweight youth. IFG increased across BMI percentile categories. Another contributing factor was the high percentage of Hispanics and Native Americans in our cohort (about three-quarters of the students); 8–12% more Hispanic and Native American students had IFG compared with African Americans and Caucasians. Finally, our subjects, particularly the boys, were midpubertal, a time of innate insulin resistance. It is also possible that stress may have influenced our results. The setting in which phlebotomy was performed—a noisy crowded gym or assembly hall with each subject in view of peers and study personnel and without their parents—may have caused stress. Stress activation of the hypothalamic-adrenal axis could elevate cortisol and other counterregulatory hormones and thereby increase glucose values. This stress response may have downregulated by the time of the second blood draw 2 h later because students had become familiar with the procedure and had been kept quiet. We believe we did all we could to mitigate students eating or drinking before the blood draw, falsely elevating the number of subjects with IFG. Students were given ample opportunity to admit they were not fasting, reschedule the blood tests, and still receive the full \$50 incentive.

Very few of our subjects had 2-h post–glucose load values in the IGT range (≥ 140 mg/dl). The 2-h glucose increased with BMI percentile, but there was no association of this value with race/ethnicity. Our findings are in contrast to studies done in overweight youth in clinical settings, where as many as 25% of subjects have been found to have IGT (16,22,23). Children who seek medical attention due to obesity represent a cohort with more significant disease and/or a positive family history, making it much more likely they will have glucose dysregulation and IGT. We found that very few children had undiagnosed diabetes (fasting glucose ≥ 126 mg/dl and 2-h value ≥ 200 mg/dl), similar to multiple studies in overweight youth (16,22,23).

Many subjects had elevated fasting insulin levels (mean value $30 \mu\text{U/ml}$), indicative of insulin resistance (13). There was a twofold increase in mean fasting insulin levels when comparing those

with a BMI <85th percentile ($22.5 \mu\text{U/ml}$) to those with a BMI ≥ 95 th percentile ($44.8 \mu\text{U/ml}$). Similarly, the percent with fasting insulin values $>30 \mu\text{U/ml}$ increased by nearly 4.5-fold across the three BMI categories. Hispanics and Native Americans had the highest mean fasting insulin levels and a 1.5- to 2-fold increase in the percent with values $\geq 30 \mu\text{U/ml}$. Our mean fasting insulin values are similar to the levels reported by Weiss et al. (16) in obese and severely obese subjects (31.3 and $38.6 \mu\text{U/ml}$, respectively). The same doubling across BMI percentiles was seen for the 2-h insulin values in our study.

In conclusion, the purpose of this study was to determine if there was a high enough percentage of students with IGT or diabetes to power a trial targeting these outcomes. Whereas we found a very low prevalence of both IGT and diabetes, our subjects exhibited a high prevalence of risk factors for diabetes. These included IFG (fasting glucose ≥ 100 mg/dl), hyperinsulinemia indicative of insulin resistance (fasting insulin $\geq 30 \mu\text{U/ml}$), and BMI associated with diabetes risk (BMI ≥ 85 th percentile). IFG is an accepted indicator of risk for type 2 diabetes and confers high likelihood that normal insulin secretion is already impaired (24). In addition to defective insulin secretion, insulin resistance is generally present if type 2 diabetes is to develop. A fasting insulin level $\geq 30 \mu\text{U/ml}$ is suggestive of insulin resistance and is a measure that can be performed in the field setting. Almost 15% of the students were found to have all three of these risk factors for diabetes. These data suggest that strategies should be developed to reduce the prevalence of risk factors for diabetes.

Acknowledgments—This work was completed with funding from National Institutes of Diabetes and Digestive and Kidney Diseases/National Institutes of Health Grant U01-DK61230 (George Washington University), U01-DK61249 (University of California at Irvine), U01-DK61231 (Baylor College of Medicine), and U01-DK61223 (University of North Carolina at Chapel Hill).

APPENDIX

The STOPP-T2D Prevention Study Group

The following individuals and institutions contributed to the reported results as members of the STOPP-T2D Prevention Study Group (*writing group).

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