Liver Fat Has a Stronger Association With Risk Factors for Type 2 Diabetes in African-American Compared With Hispanic Adolescents

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Context: Although overweight and obese African-Americans (AAs) have less visceral adipose tissue (VAT) and liver fat (LF) than Hispanics, they have a similar risk for type 2 diabetes.

Objective: We examined ethnic differences in the association between VAT and LF with risk factors for type 2 diabetes to help explain this paradox.

Design: We conducted a cross-sectional study in an academic pediatric care facility.

Subjects: Subjects were overweight and obese AA (n = 131; 15.5 ± 3.3 years old) and Hispanic adolescents (n = 227; 14.7 ± 3.0 years old).

Main Outcome Measures: Outcome measures included insulin sensitivity (SI), acute insulin response (AIR), and disposition index (DI) by frequently sampled iv glucose tolerance test and minimal modeling.

Results: LF, not VAT, was inversely associated with SI, and the effect of high LF compared with low was more pronounced in AAs ($P_{interaction} < .05$). In Hispanics, high LF was associated with a 24% lower SI ($P < .01$) and a 31% increase in AIR ($P < .01$) and was not associated with DI ($P = .35$). In AAs, high LF was associated with a 49% lower SI ($P < .001$), was not associated with an increase in AIR ($P = .25$), and was associated with a 42% lower DI ($P < .01$), indicating failure of compensatory insulin secretion/clearance in response to insulin resistance. Prediabetes changed the relationship between high/low LF and DI in Hispanics ($P_{interaction} = .002$) but not AAs such that prediabetic Hispanics with high LF had a 43% lower DI ($P = .03$) with no difference in those without prediabetes ($P = .06$).

Conclusions: LF has a stronger effect on SI compared with VAT. Our results suggest that the impact of high LF on poor β-cell compensation is more pronounced in AAs. In Hispanics, the combination of high LF and prediabetes contributes to poor β-cell compensation. **(*J Clin Endocrinol Metab* 98: 3748–3754, 2013)**

Studies show that for the same degree of overall adiposity, African-Americans (AAs) have higher levels of sc abdominal adipose tissue (SAAT) and lower levels of visceral adipose tissue (VAT) and liver fat (LF) compared with Hispanics (1, 2). However, despite having lower levels of VAT and LF, AAs are more insulin resistant than Hispanics (1) and have similar or increased risk for type 2 diabetes (T2D) (1–3). These observations conflict with the prevailing hypothesis that higher VAT (4–6) and/or LF (7, 8) contribute to obesity-associated metabolic disease risk. Given this, it is possible that VAT and LF contribute differently to metabolic risk in AAs and Hispanics. Differences in the impact of either fat depot across ethnicity may explain why AAs exhibit similar risk factors for

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Abbreviations: AA, African-American; AIR, acute insulin response; BMI, body mass index; DI, disposition index; FSIVGTT, frequently sampled iv glucose tolerance test; LF, liver fat; MRI, magnetic resonance imaging; NGT, normal glucose tolerant; OGTT, oral glucose tolerance test; PF, pancreatic fat; SAAT, sc abdominal adipose tissue; SI, insulin sensitivity; T2D, type 2 diabetes; TFM, total fat mass; VAT, visceral adipose tissue.

T2D despite having less VAT and LF compared with Hispanics.

Although AAs have a more protective fat profile when compared with Hispanics, they have a comparable risk for T2D, establishing what we have termed the AA versus Hispanic paradox (2). These ethnic differences are even more intriguing given that previous studies have shown that increased VAT (2, 3, 5, 9) and LF (4, 10) are associated with increased T2D risk. Additionally, recent work by our group has shown that prediabetic minority adolescents have higher levels of VAT and LF than those who are normal glucose tolerant, suggesting prediabetes may modify the association between these fat depots and risk for T2D (11). A major limitation of these studies is the high correlation between VAT and LF (12, 13), making it difficult to determine which compartment of abdominal fat drives metabolic risk. Elegant studies, mostly in Caucasian adults, provide evidence that LF is the predominant compartment associated with metabolic disease in obese adults (7, 8). Therefore, the two primary connected goals of this study were to examine 1) the separate and combined effects of VAT and LF on risk factors for T2D in overweight and obese minorities and 2) to determine whether the association between these fat depots and risk factors for T2D differed in AAs and Hispanics, thereby potentially explaining the paradox of similar diabetes risk in AAs at lower levels of VAT and LF. As a secondary aim, we explored whether prediabetes status altered the associations between these fat depots and risk factors for T2D.

Subjects and Methods

Participants

We combined participants from 5 studies in our laboratory that used a common protocol for assessment of body fat distribution and risk factors for T2D. Participants included 358 overweight and obese (body mass index [BMI] ≥85th percentile for ages <18 y or BMI >30 kg/m^2 for those ≥18 y) AAs and Hispanics (131 AAs and 227 Hispanics) aged 8 to 25 years who had complete measures of SAAT, VAT, and LF. Ethnicity was defined as self, parents, and grandparents being all of AA or Hispanic decent (by parental/self-report). Data from some of these studies have been reported (1, 11, 14–16); however, this is the first combined analysis to examine the independent effect of LF and VAT on metabolic outcomes in AAs and Hispanics who differ in their LF and VAT profiles. Before any testing, informed written consent/assent was obtained from the participant or parents. All studies were approved by the University of Southern California Institutional Review Board.

Procedures

Procedures used in these studies have been previously reported (1, 11, 14–16). Total body fat mass was assessed by dual-energy x-ray absorptiometry (Hologic QDR 4500W; Hologic). Depending on machine availability, a 30-slice whole abdominal magnetic resonance imaging (MRI) scan was performed on a General Electric 1.5- or 3-T magnet to measure abdominal fat distribution and ectopic fat accumulation (17, 18) as previously described (19). SAAT and VAT images were segmented using SliceOmatic (TomoVision). LF was measured during the same MRI test using a modified Dixon 3-point technique. For a subset of participants (n = 37), we performed consecutive MRI scans using both 1.5- and 3-T magnets and found that these two methods were highly correlated for measures of SAAT, VAT, and LF ($r = 0.90$, $r = 0.95$, and $r = 0.96$, respectively). Therefore, we used these data to developed conversion equations to standardize data relative to the 1.5-T magnet for each participant.

Metabolic parameters

All but 34 participants had an overnight stay at the Clinical Trials Unit and were fed a standardized dinner prior to the frequently sampled iv glucose tolerance test (FSIVGTT) the next morning. The remaining 34 participants arrived at the Clinical Trials Unit for their FSIVGTT after an overnight fast. After the FSIVGTT, all participants were fed a standardized lunch (1, 11, 14–16). Plasma was analyzed for glucose and insulin and values entered into Minmod Millenium 2003 (versions 5.16 and 6.02, Richard N. Bergman, University of Southern California) to generate values for insulin sensitivity (SI), acute insulin response (AIR) to glucose, and disposition index (DI) (the product of SI and AIR). Previous studies have shown that decreases in DI is predictive of the development of T2D in Pima Indians as well as women with gestational diabetes (20, 21). In addition to the FSIVGTT, a subset of our participants (n = 299) underwent an oral glucose tolerance test (OGTT) as previously described (11, 14, 22).

Statistical analysis

Definition of high/low LF, high/low VAT, and normal glucose tolerant/prediabetic

Participants were classified into 1 of 4 groups based on high/low LF and high/low VAT. High LF was defined as an LF fraction >5.5%, whereas low LF was defined as an LF fraction <5.5%. We used this cutoff point because it has been shown to be likely indicative of fatty liver disease (23). High or low VAT, relative to SAAT, was defined by positive or negative residuals from the regression between VAT and SAAT in each ethnic group. This approach is supported by previous findings that found that in obese adolescents, high VAT, in the context of low SAAT, was associated with a greater disease risk (24). Participants who underwent an OGTT were classified as normal glucose tolerant (NGT) (fasting glucose <100 mg/dL and 2-hour glucose <140 mg/L) or prediabetic (fasting glucose ≥100–125 mg/dL and/or 2-hour glucose of 140–199 mg/dL).

The standardized measures of metabolic indices, SAAT, VAT, and LF values, for 358 participants are shown in Table 1. Analyses were done in SPSS Statistics version 18.0 and a priori significance level was set at $P < .05$. A full-factorial 3-way analysis of covariance was used to examine interactions between our main effect variables (high/low LF, high/low VAT, ethnicity, and prediabetes status where appropriate) and metabolic outcomes (SI, AIR, and DI). All 3-way interactions were nonsignificant; therefore, we examined 2-way interactions between high/low LF, high/low VAT, ethnicity, and prediabetes status (where ap-

Table 1. Baseline Descriptive Statistics[a]

Variable	AAs (n = 131)	Hispanics (n = 227)	*P* Value
General characteristics			
Age, y[b]	15.5 ± 3.3	14.7 ± 3.0	.07
Pubertal stage, n[c]			
1–3	23	69	<.001
4	14	44	
5	94	114	
Sex (males/females), n[c]	61/70	131/96	.04
Height, cm	164.2 ± 12.2	161.4 ± 11.6	.04
Weight, kg	89.7 ± 26.7	84.6 ± 22.8	.07
Body composition			
BMI, kg/m^2[b]	32.7 ± 7.7	32.1 ± 6.5	.74
Total fat mass, kg[b,d]	31.6 ± 13.2	31.0 ± 11.1	.55
Total lean tissue mass, kg[d]	52.8 ± 14.4	49.0 ± 12.3	.01
sc fat, L[b]	14.6 ± 6.3	13.7 ± 6.2	.29
Visceral fat, L[b]	1.5 ± 1.0	2.0 ± 1.3	<.01
Liver fat fraction, %[b]	4.2 ± 3.6	9.0 ± 8.5	<.001
Metabolic parameters			
Fasting insulin (μU/mL)[e,f]	15.7 ± 10.0	26.9 ± 24.0	<.001
Fasting glucose (mg/dL)[b,f]	87.3 ± 6.7	83.0 ± 24.1	<.01
SI [($\times 10^{-4}$ min^{-1})/(μU/mL)][e,g]	1.8 ± 1.4	1.9 ± 1.4	.14
AIR (μU/mL × 10 min)[e,g]	1944.2 ± 1326.7	1326.7 ± 823.2	<.001
DI[b,g]	2626.9 ± 1716.1	1972.9 ± 1,044.4	.03
Prediabetes status[c]			
NGT, n	90	146	.03
Prediabetic, n	15	48	

[a] Unless indicated otherwise, results are shown as mean ± SD.

[b] Nonparametric test.

[c] χ^2 test.

[d] AAs, n = 128; Hispanics, n = 217.

[e] Variables were not normally distributed so statistical tests were run on log-transformed data.

[f] AAs, n = 125; Hispanics, n = 169.

[g] AAs, n = 128; Hispanics, n = 190.

propriate). If an interaction term, which included ethnicity, was found to be significant, we stratified our sample and reported the separate adjusted means for AAs and Hispanics. A priori covariates include age, sex, and total fat mass (TFM) as well as ethnicity, high/low LF, and high/low VAT. Controlling for total percent fat mass instead of TFM or including Tanner stage as a covariate did not change our results (data not shown). Repeating these analyses in the subset of participants who underwent an OGTT (n = 299) yielded nearly identical results (data not shown). We also examined the relationships between LF and VAT with SI, AIR, and DI using multivariate linear regression analysis. Each model controlled for age, sex, TFM, and SAAT. Ethnicity was included in each model, and we examined interactions between LF, VAT, and ethnicity. For all of the analyses, SI, AIR, and DI were log transformed to meet assumptions. In some cases, results were back-transformed for presentation and reported as mean ± SE or adjusted mean (95% confidence interval).

Results

Table 1 displays the mean physical characteristics and metabolic parameters of the 131 AA and 227 Hispanic males and females. Consistent with our previous studies, AAs had lower LF and VAT compared with Hispanics ($P < .01$). Although the unadjusted values of SI were not different by ethnicity, after controlling for age, sex, and TFM, AAs had a lower SI (1.82 ± 0.11 vs 2.01 ± 0.09 [($\times 10^{-4}$ min^{-1})/(μU/mL)]; $P = .01$). LF was correlated with VAT in AAs and Hispanics after controlling for TFM (AA, $r = 0.27$, $P < .01$; Hispanics, $r = 0.44$, $P < .001$).

High/low LF and high/low VAT analysis

In regard to SI, AIR, and DI, the interaction between high/low LF and ethnicity was significant in each model ($P_{\text{LF*Ethnicity}} < .05$), whereas the interaction between high/low LF and high/low VAT was not ($P_{\text{LF*VAT}} \geq .18$). Figure 1 shows these results stratified by ethnicity. Here we report the adjusted means for AAs and Hispanics wherein the models do not include the interaction between high/low LF and high/low VAT. High LF was associated with a 49% lower SI in AAs (high LF, 0.77 [($\times 10^{-4}$ min^{-1})/(μU/mL)] [0.58, 1.03], vs low LF, 1.52 [1.35, 1.71]; $P < .001$) and a 24% lower SI in Hispanics (high LF, 1.39 [($\times 10^{-4}$ min^{-1})/(μU/mL)] [1.23, 1.57], vs low LF, 1.83 [1.63, 2.05]; $P < .01$). In Hispanics, high LF was associated with a 31% higher AIR (high LF, 1264.74 μU/mL × 10 minutes [1106.62, 1445.44],

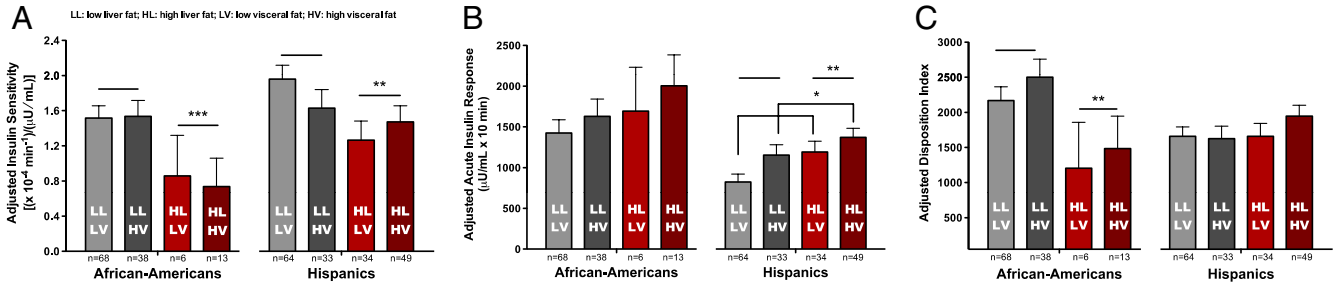

Figure 1. A–C, Adjusted mean ± SE. Model included high/low LF, high/low VAT, age, sex, TFM, and high/low LF*high/low VAT. Although the interaction between high/low LF and high/low VAT was nonsignificant in all models, we included this term to show the adjusted means for each of the 4 groups in AAs and Hispanics: LL, low liver fat; HL, high liver fat; LV, low visceral fat; HV, high visceral fat. ***, $P < .001$; **, $P < .01$; *, $P < .05$. Variables were log transformed to meet analysis of covariance assumptions; adjusted mean values were back transformed for ease of interpretation.

vs low LF, 963.83 [849.18, 1091.44]; $P < .01$), indicating appropriate compensation as also demonstrated by no association of high LF with DI (high LF, 1815.52 [1595.88, 2065.38], vs low LF, 1659.59 [1472.32, 1874.99]; $P = .35$). In AAs, however, there was no association of high LF with AIR, suggesting poor compensation as also demonstrated by the 42% lower DI in those with high LF (high LF, 1352.07; [1000.00, 1828.10], vs low LF, 2328.09 [2051.16, 2648.50]; $P < .01$).

LF and VAT regression analysis

As shown in Figure 2, we observed that there was a stronger negative association between LF and SI in AAs compared with Hispanics ($P_{LF*Ethnicity} < .01$; $\beta_{AA} = -0.53$; $P = .001$ vs $\beta_{Hisp} = -0.21$; $P = .003$). In AAs and Hispanics, there was no relationship between LF and AIR, whereas we did observe an ethnic difference in the relationship between VAT and AIR ($P_{VAT*Ethnicity} < .001$). Specifically, in AAs, there was no relationship between VAT and AIR, whereas in Hispanics, there was a significant positive relationship between VAT and AIR ($\beta_{AA} = 0.03$; $P = .55$ vs $\beta_{Hisp} = 0.06$; $P = .01$). In AAs, with increasing LF, there was a significantly lower DI, whereas this relationship was not observed in Hispanics ($P_{VAT*Ethnicity} = 0.04$; $\beta_{AA} = -0.38$; $P = .007$ vs $\beta_{Hisp} = -0.02$; $P = .80$). There was no relationship between VAT and SI or VAT and DI in AAs or Hispanics.

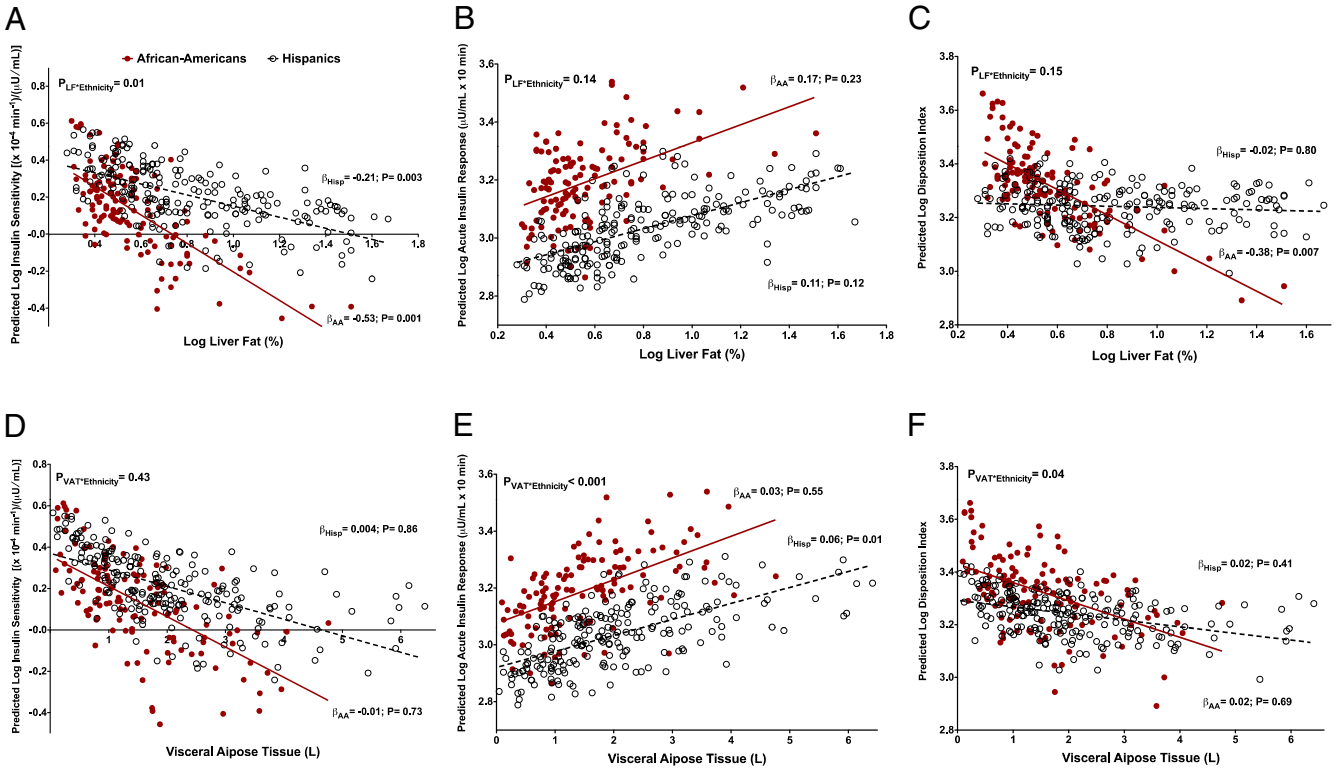

Figure 2. Results from the multivariate linear regression analysis. Models included LF, VAT, age, sex, TFM, and SAAT. Results are shown as predicated values of log SI, log AIR, and log DI versus log LF (A–C) or VAT (D–F). AAs are represented by solid red circles and red regression line. Hispanics are represented by open black circles and dashed black regression line. Variables were log transformed to meet assumptions.

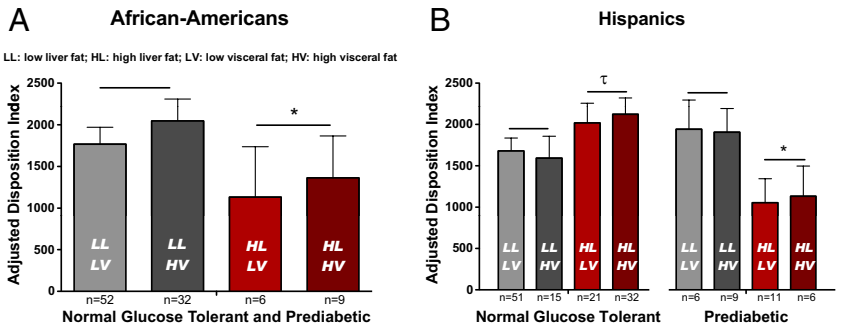

Figure 3. A and B, Adjusted mean ± SE. Model included high/low LF, high/low VAT, age, sex, TFM, prediabetes status, and high/low LF*high/low VAT. Although the interaction between high/low LF and high/low VAT was nonsignificant in all models, we included this term to show the adjusted means for each of the 4 groups in AAs and Hispanics: LL, low liver fat; HL, high liver fat; LV, low visceral fat; HV, high visceral fat. *, $P < .05$; $\tau = 0.06$. Variables were log transformed to meet analysis of covariance assumptions; adjusted mean values were back transformed for ease of interpretation.

High/low LF, high/low VAT, and prediabetes status analysis

Eighty-four percent of the participants had an OGTT, which we used to identify 15 AAs and 48 Hispanics as being prediabetic. Controlling for prediabetes status did not change our findings regarding SI and AIR (data not shown). As shown in Figure 3, when examining DI, we found that prediabetes status significantly changed the relationship between high/low LF and DI in Hispanics ($P_{LF*Prediabetes} = .002$) but not AAs ($P_{LF*Prediabetes} = .24$). NGT Hispanics with high LF showed a trend for a 26% higher DI (high LF, 2084.49; [1778.28, 2454.71], vs low LF, 1655.77 [1412.54, 1949.84]; $P = .06$), whereas prediabetic Hispanics with high LF had a 43% lower DI (high LF, 1088.93 [790.68, 1496.24], vs low LF, 1909.85 [1361.44, 2673.00]; $P = .03$). In NGT and prediabetic AAs, high LF was associated with a 35% lower DI (high LF, 1244.51 [895.36, 1729.82], vs low LF, 1905.46 [1548.82, 2338.84]; $P = .02$).

Discussion

Elevated levels of VAT and LF have been shown to be associated with insulin resistance (1, 4, 7, 8, 25), whereas some previous studies have demonstrated the importance of LF over VAT in regard to increased risk for T2D (7, 8). Given this, overweight and obese AAs with lower LF and VAT should be protected from T2D risk when compared with Hispanics. Despite this, previous work by our group has shown AAs have higher or similar risk factors for T2D (1–3). Therefore, the objective of this analysis was to determine the separate effects of LF and VAT on risk factors for T2D and to see whether these relationships differed between AAs and Hispanics. To accomplish this, we examined LF and VAT by identifying subgroups that were contrasted for high versus low LF and high versus low VAT. Using this method, we demonstrated that high LF was associated with increased insulin resistance, whereas high VAT was not. This association was more prominent in AAs compared with Hispanics, suggesting that even though LF tends to be lower in AAs, its relationship with insulin resistance is more pronounced.

By examining those with high and low LF and VAT, we found that there was a stronger association between high LF and low SI in AAs compared with Hispanics. In AAs, we observed that high LF was associated with a 49% lower SI when compared with those with low LF. Therefore, based on the hyperbolic relationship between SI and AIR, we anticipated that AAs with high LF would have to compensate with an AIR that was double that of AAs with low LF. Despite this, we found that AAs with high LF had only a 22% higher AIR. Conversely, Hispanics with high compared with low LF had a 31% higher AIR, which was in line with the 24% lower SI that we observed among those with high LF. These findings suggest that in response to a lower SI, Hispanics but not AAs are able to compensate for LF-induced insulin resistance by adequately increasing their AIR. Taken together, we found that in AAs, high LF was associated with a 49% lower SI and a small and nonsignificant increase in their AIR, which translated into high LF being associated with a 42% lower DI. In Hispanics, high LF was associated with a 24% lower SI and an appropriate increase in AIR, which resulted in no relationship between high/low LF, high/low VAT, and DI.

To further explore the relationship between LF and VAT with risk factors for T2D, we also used regression analyses to complement the approach of classifying participants based on high/low LF and VAT. Overall, our results were similar using the 2 approaches, and we found that in AAs, there was a stronger inverse relationship between LF and SI when compared with Hispanics. We also found that VAT, and not LF, was positively related to AIR in Hispanics but not AAs. In AAs, LF was inversely associated with a lower DI, whereas this relationship was not observed in Hispanics. When examining our 4 groups, we found that high LF and high VAT were associated with a higher AIR in Hispanics, whereas when we examined LF and VAT continuously, we observed that only VAT was related to a higher AIR. This difference is likely due to the fact that these 2 analyses differed in the manner in which they examined LF, VAT, and SAAT. A previous study has shown that VAT relative to SAAT is important when con-

sidering metabolic disease risk (24). Therefore, it is possible that our high/low classification method for VAT relative to SAAT is better suited to examine the relative importance of VAT compared with LF. Furthermore, it is important to note that our regression analysis largely supports our findings regarding the differential effects of high LF on risk factors for T2D in AAs and Hispanics.

Recent work by our group has shown that minority adolescents with prediabetes differ in their ectopic fat distribution and risk for T2D when compared with those without prediabetes (11). Additionally, another study in overweight adults demonstrated that LF, more than VAT, increases when glucose tolerance moves from normal to impaired fasting glucose/impaired glucose tolerance (26). For this reason, we explored prediabetes status in the current study and found that it changed the results for DI but not SI or AIR. Specifically, NGT Hispanics with high LF showed a trend for a 26% higher DI, whereas prediabetic Hispanics with high LF had a 43% lower DI. In AAs, including prediabetes status in the model resulted in high LF being associated with a 35% lower DI. These findings suggest that the combination of high LF with prediabetes is particularly problematic for β-cell function. Specifically, high LF was associated with poor β-cell function only in Hispanics with prediabetes. It is important to note that this secondary analysis is greatly limited by the small number of participants with prediabetes in this study (15 AAs and 48 Hispanics).

Although it is impossible to determine causality from this study, we would postulate that AAs and Hispanics differ in their β-cell compensation in response to LF-induced insulin resistance. We would hypothesize that Hispanics with high LF are able to compensate for a lower SI by increasing their AIR, thereby showing appropriate β-cell compensation to LF-induced insulin resistance. Conversely, AAs would fail to have an adequate increase in their AIR, thus showing poor β-cell compensation to LF-induced insulin resistance. In light of these findings, we were left with the question of why LF might have a more prominent association with risk factors for T2D in AAs compared with Hispanics. One explanation is that AAs already have a higher AIR when compared with Hispanics. Due to this, their β-cells could have a harder time compensating for the added stress of LF-induced insulin resistance because they are already on the steeper part of the DI curve. It is also conceivable that AAs and Hispanics not only differ in their ectopic fat distribution but also in the amount of adipokines that these fat depots secrete. This is particularly important given that leptin and adiponectin have been shown to affect insulin secretion and β-cell apoptosis (27). Finally, another possible explanation is a high correlation between LF and pancreatic fat (PF) (28), which may impair β-cell compensation to insulin resistance. One of our previous studies found that obese prediabetic AAs have higher levels of PF than those with NGT, whereas this relationship was not observed in Hispanics (11). Given these findings, it is possible that PF interferes with insulin secretion from β-cells and may be why AAs show poor β-cell compensation.

One limitation of this study is that we enrolled only overweight and obese AAs and Hispanics and did not include lean participants or Caucasians. Although we observed that AAs had a lower fasting insulin and higher fasting glucose compared with Hispanics, a previous study by our group has demonstrated that fasting indicators of insulin sensitivity are not always reflective of SI from an FSIVGTT with minimal modeling (29). Also, in the current study, we found that after adjusting for age, sex, and TFM, AAs had a lower SI than Hispanics. Another limitation of this study is that, due to the low prevalence of high LF and VAT in AAs compared with Hispanics (2), we observed a small number of AAs with high LF ($n = 19$). For this reason, we completed a power analysis and found that we were powered to detect up to a 39% higher AIR in AAs with high compared with low LF. As previously mentioned, given the 49% lower SI in AAs with high LF, we expected an AIR that was double that of those with low LF, which we were more than powered to detect. Additionally, despite the small number of AAs with high LF, we were able to confirm these results using regression analyses.

This study is also limited in that we did not have data regarding hepatic insulin sensitivity, hepatic insulin clearance, or C-peptide measures, making it possible that our DI findings reflect changes in hepatic insulin clearance and/or β-cell secretion of insulin in response to decreased SI. We also cannot determine whether our observed SI, primarily based on muscle, is a consequence of chronic hyperinsulinemia induced by hepatic insulin resistance and/or reduced insulin clearance due to high LF. For example, because Hispanics had higher insulin levels and a smaller AIR compared with AAs, it is possible that impaired hepatic insulin clearance could partially explain our findings. Lastly, because race/ethnicity was defined based on self-report, future studies should verify these finding by using a more definitive measure of ethnicity.

In summary, we found that LF, more than VAT, was associated with insulin resistance in AAs and Hispanics and that these effects are more pronounced in AAs. Additionally, we found that there was a greater association of LF with risk factors for T2D in AAs as seen by compromised β-cell compensation to the LF-induced insulin resistance in this group. Additionally, we found that the combination of high LF and prediabetes was associated with decreased β-cell function in Hispanics but not AAs.

This is the first study to demonstrate that high LF has a more profound effect on SI, AIR, and DI among AAs compared with Hispanics. This finding could explain why AAs are at a similar risk for T2D despite having lower levels of LF and VAT (2) and suggest that even though LF tends to be lower in AAs, its association with metabolic risk is more pronounced.

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