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Dietary Fat Intake and Relationship to Serum Lipid Levels Among HIV-Infected Subjects with Metabolic Abnormalities in the Era of HAART

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Abstract

Objective—To evaluate dietary intake and its relationship to lipid parameters in HIV-infected patients with metabolic abnormalities.

Design, Setting, and Participants—We prospectively determined dietary intake (4-day food records or 24-hour recall) in 356 HIV-infected subjects and 162 community-derived HIV-negative controls evaluated for metabolic studies between 1998–2005.

Main Outcome Measures—Differences in dietary intake between HIV-infected subjects and non-HIV-infected controls, in relation to the established 2005 USDA (United States Department of Agriculture) Recommended Dietary Guidelines, were determined. The relationship between dietary fat intake and serum lipid levels among HIV-infected subjects was also evaluated.

Results—Assessment of dietary intake in this group of HIV-infected subjects demonstrated increased intake of total dietary fat ($p<0.05$), saturated fat ($p=0.006$), and cholesterol ($p=0.006$) as well as a greater percentage of calories from saturated fat ($p=0.002$) and from trans fat ($p=0.02$), despite similar caloric intake to the control subjects. A significantly higher percentage of HIV-infected subjects were above the 2005 USDA Recommended Dietary Guidelines for saturated fat ($>10\%$ /day) (76.0% HIV vs. 60.9% controls, $p=0.003$), and cholesterol (>300 mg/day) (49.7% HIV vs. 37.9% controls, $p=0.04$). Saturated fat intake was strongly associated with triglyceride level [triglyceride level increased 8.7 mg/dL (parameter estimate) per gram of increased saturated fat intake, $p=0.005$] while total fat was inversely associated with triglyceride level [triglyceride

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level decreased 3.0 mg/dL (parameter estimate) per gram of increased total fat intake, $p=0.02$] among HIV-infected subjects.

Conclusions—Increased intake of saturated fat is seen and contributes to hypertriglyceridemia among HIV-infected patients who have developed metabolic abnormalities. Increased saturated fat intake should be targeted for dietary modification in this population.

Keywords

HIV; nutrition; saturated fat; hypertriglyceridemia; metabolic abnormalities

INTRODUCTION

Highly active antiretroviral therapy (HAART) has resulted in improvement in the long-term survival of HIV-infected individuals.[1] However, the use of HAART is associated with metabolic complications such as dyslipidemia, insulin resistance, and altered fat distribution. [2, 3] Furthermore, these metabolic complications associated with HAART have been linked to an increased risk of myocardial infarction and cardiovascular disease.[4–7]

In non-HIV-infected individuals, the impact of dietary saturated fat and cholesterol intake on serum lipids and subsequent development of cardiovascular disease is well-established.[8, 9] In contrast, the degree to which HIV-infected individuals consume dietary fat and cholesterol, the composition of the fat intake, and the relationship to serum lipid levels are relatively unknown. Several prior small-scale studies in HIV-infected individuals have not demonstrated any significant relationship between saturated fat intake and serum lipid levels.[10–12] Furthermore, these studies did not compare the dietary intake of HIV-infected subjects with metabolic abnormalities in the era of HAART to that of community-sampled controls in the context of established dietary guidelines. We therefore compared dietary intake in this population of HIV-infected subjects to that of control subjects, using the 2005 United States Department of Agriculture (USDA) Recommended Dietary Guidelines, and assessed the relationship between dietary fat intake and HIV-related dyslipidemia.

METHODS

PATIENTS AND CONTROLS

Data on macronutrient intake were prospectively collected from 1998–2005 in 356 HIV-infected patients participating in metabolic studies at the Massachusetts General Hospital (MGH) and 162 HIV-negative subjects simultaneously recruited from the community as controls for these studies.[13–27] HIV-infected subjects with known wasting or evaluated for studies of AIDS wasting were not included in the analysis. HIV-infected subjects aged 18 – 60 years were recruited from newspaper advertisement, community and referral-based practices. Patients could not be directly referred into the study by providers. For subjects receiving antiretroviral (ARV) therapy, a stable regimen for a minimum of 6 weeks prior to evaluation was required. Subjects were excluded if they had a history of diabetes mellitus; were receiving concurrent therapy with insulin, antidiabetic agents, glucocorticoids, growth hormone, supraphysiologic testosterone replacement, or anabolic steroids; were current substance abusers; had a major opportunistic infection within the 6 weeks prior to the study;

or were pregnant or breast-feeding within the past year. The HIV-negative controls were recruited through hospital and local advertisements using many of the same community newspapers used to recruit HIV-infected subjects. The HIV-negative controls were required to have no history of diabetes mellitus. Other criteria, including age, medication use and reproductive status were similar between the HIV and non-HIV groups. For both HIV-infected and control groups, baseline data were obtained before any intervention. If subjects participated in more than one study, only data from the initial study were used. Collection of nutrient intake was approved by the Institutional Review Board at MGH as well as at MIT (Massachusetts Institute of Technology), and all participants provided informed consent.

PROTOCOL

All subjects were studied after an overnight fast of 12 hours. Each individual had a complete medical history, including documentation of all prior antiretrovirals and starting date for current antiretroviral regimen. Height and weight were measured as well as circumference of the hip and waist at the level of the umbilicus to calculate the waist-to-hip ratio (WHR). Subjects were categorized as having lipodystrophy based on WHR and evidence of fat redistribution as previously described. [14]

Individuals underwent total-body dual-energy x-ray absorptiometry (DEXA) with the use of the Hologic QDR-4500A scanner (Hologic Inc., Waltham, MA) to determine total body fat as well as regional body fat measurements.[28] Cross-sectional abdominal computed tomography (CT) scans were performed as described by Borkan et al to assess distribution of subcutaneous and visceral abdominal fat (SAT and VAT, respectively).[29] Resting energy expenditure (REE) and respiratory quotient (RQ) were measured for 20 minutes by indirect calorimetry (Deltatrac or Vmax29, Sensormedics, Yorba Linda, CA, USA) after subjects had been resting for 30 minutes. Subjects also received a standard 75-g oral glucose tolerance test (OGTT).

Collection of nutrient intake data (including alcohol intake) was obtained via 4-day food records (3 weekdays and 1 weekend day) in 320 HIV-infected subjects and 108 controls and via 24-hour food recall in an additional 36 HIV-infected subjects and 54 controls. For the 4-day food records, participants were instructed by trained dietitians to record completely all food and drink consumed. Incomplete records were then addressed by direct questioning. Subjects who were assessed using 24-hour recall were asked both open-ended and closed-ended questions to determine dietary intake. Food models, cups, and measuring tools were used to aid in visualization and quantification of portion sizes. Nutrient calculations for energy intake, macronutrient, and alcohol consumption were performed with the Minnesota Nutrition Data System software (Nutrition Coordinating Center of the University of Minnesota, Minneapolis).

Subjects provided data regarding their home address, including zip code. Zip code information was compared with median income from the 2000 US Census data to provide an estimate of socioeconomic status.[30] Subjects were then stratified into quartiles based on median income. Insurance status (private, public, or other) was also determined to assess socioeconomic status.

LABORATORY METHODS

Complete blood count, CD4 count, HIV viral load, and fasting concentrations of glucose, insulin, cholesterol, HDL and triglycerides were determined by methods described elsewhere.[14]

STATISTICAL ANALYSIS

Data are expressed as mean $\pm$ standard deviation except where indicated. P values were derived from a mixed effects ANOVA model to determine differences between HIV and control subjects adjusting for potential random effects of individual studies into which patients were recruited. We then further adjusted these p values for age, race, gender, Body Mass Index (BMI), insurance status, and income quartile to determine statistically significant differences between HIV-infected subjects and controls in the mixed effects ANOVA model, and performed a sensitivity analysis, determining the effects of insurance status on the modeling. The effects of time (expressed as calendar year of enrollment) on fat intake were also assessed in the modeling. BMI was not included in the adjustment of body composition parameters. Method of dietary assessment (24-hour food recall vs. 4-day food record) was also added to the model as a covariate when analyzing macronutrient data. Use of lipid-lowering medications was adjusted for when analyzing serum lipid levels as well as macronutrient data. The analysis was also repeated including metabolic syndrome as a covariate in the model. The gender effects on fat intake in HIV-infected and non-HIV-infected subjects were examined using the multiple logistic regression models which were adjusted for age, race, gender, BMI, insurance status, use of lipid-lowering agents, method of dietary assessment, and income quartile. The event of interest in each logistic regression model was the fat intake above fat dietary guidelines.

Among HIV-infected subjects, the change in dietary consumption among HIV-infected subjects over time (expressed as calendar year of enrollment) was assessed using a bivariate linear fit model with P values derived from univariate regression analyses. The effects of insurance status, lipodystrophy characterization, or protease inhibitor use on dietary fat intake among HIV-infected subjects were determined by ANOVA.

In HIV-infected subjects, a least squares multiple regression model was used to evaluate the relationship between dietary intake and metabolic parameters, with adjustment for age, gender, BMI, race, protease inhibitor (PI) use, alcohol consumption, total fiber intake, and impaired glucose tolerance (IGT)/diabetes in the model. We included time (expressed as calendar year of enrollment) in this model to assess whether time altered the relationship between dietary fat intake and lipid parameters. We also examined whether use of anti-lipid lowering drugs altered the relationship between dietary fat intake and lipid parameters by including use of anti-lipid lowering therapy in the model. The study had 80% power to detect a difference of 4 g/day in saturated fat between the HIV and control groups, based on a two-sided t test, $\alpha=0.05$ and SD of 15 g/day. The minimum detectable statistical effect size was 26.7% of the SD. All statistical analyses were performed using SAS JMP software, version 5.0.1 (SAS Institute).

RESULTS

Three hundred and fifty-six HIV-infected subjects (197 men, 159 women) and 162 non-HIV-infected controls (73 men, 89 women) were evaluated. There were no statistically significant differences between the two groups in terms of age, gender, race and income quartile. The mean duration of HIV illness was 8.5 ± 4.8 years. 88.8% of the HIV-infected subjects were receiving ARVs, of whom 66.8% were receiving PIs and 93.2% were receiving nucleoside reverse transcriptase inhibitors (NRTIs). The majority (72.5%) of HIV-infected individuals were categorized as having lipodystrophy (Table 1).

DIETARY INTAKE

Dietary macronutrient intake for HIV-infected subjects and non-HIV-infected controls is presented in Table 2A and Figures 1A and 1B. There was no statistically significant difference in total caloric intake between the two groups. Compared to controls, HIV-infected subjects consumed a greater amount of total dietary fat ($p < 0.05$), saturated fat ($p = 0.006$) and cholesterol ($p = 0.006$) as well as a greater percentage of calories from saturated fat ($p = 0.002$) and from trans fat ($p = 0.02$).

Using the 2005 USDA Recommended Dietary Guidelines for total fat, saturated fat ($< 10\%$ of total caloric intake), and cholesterol (< 300 mg/day), we determined the percentage of HIV-infected subjects and non-HIV-infected controls exceeding these recommendations, as an indicator of poor dietary habits.[31] Although these guidelines recommended total fat intake to be 20 – 35% of total caloric intake, we chose a value of $< 35\%$ for consistency. A significantly higher percentage of HIV-infected subjects compared to controls were above these guidelines for saturated fat (76.0% vs. 60.9%, $p = 0.003$) and cholesterol intakes (49.7% vs. 37.9%, $p = 0.04$) (Figure 1C).

The effects of HIV status on dietary fat intake parameters were not substantially altered by the addition of insurance status into the models. Similarly, no significant differences in fat consumption were seen between HIV-infected subjects with private insurance vs. those with public insurance, indicating that insurance status did not have a significant effect on dietary fat consumption among HIV-infected subjects in this study (data not shown).

There were no statistically significant differences in dietary carbohydrate, protein, monounsaturated fat, and polyunsaturated fat intakes between the two groups (Figures 1A and 1B). Fiber intake ($p = 0.03$) and alcohol consumption ($p = 0.005$) were significantly lower in HIV-infected subjects compared to controls (Table 2A).

Since dietary intake could be influenced by changes over time, a time trend analysis was undertaken, in which dietary macronutrient intake was compared to time (expressed as calendar year), using a bivariate linear fit model. Among HIV-infected patients, the percentage of calories derived from total fat ($r^2 = 0.01$, $p = 0.03$) as well as from polyunsaturated fat ($r^2 = 0.02$, $p = 0.006$) increased linearly from 1997–2005, whereas the percentage intake from saturated fat, trans fat and cholesterol did not change with time. No statistically significant changes were seen in the primary endpoints when time was included as a covariate in our ANOVA mixed model. The usage of lipid-lowering drugs did not

change significantly among HIV-infected individuals with time, and was included as a covariate in the ANOVA model used to examine dietary differences between HIV-infected and HIV-uninfected subjects.

The effect of gender on the percentage of subjects exceeding the USDA Recommended Dietary Guidelines was also examined. Only cholesterol intake was significantly affected by gender since a greater percentage of males than females in the HIV-infected population exceeded guidelines for cholesterol intake (Table 2B).

METABOLIC, BODY COMPOSITION, AND ENERGY PARAMETERS

HIV-infected subjects demonstrated higher total cholesterol ($p=0.003$), higher triglyceride ($p<0.0001$), and lower HDL ($p<0.0001$) levels than controls (Table 3). BMI and waist circumference were not different between HIV-infected and control groups, but hip circumference was lower and waist-to-hip ratio higher among HIV-infected than control subjects. Significant differences in VAT and SAT were not seen in the HIV-infected compared to control subjects, but HIV-infected patients demonstrated an increase in VAT/SAT, trunk to extremity ratio, and a decrease in total extremity fat. Fasting glucose levels were not different between the HIV-infected and control groups, but glucose area-under-the-curve based on OGTT and fasting insulin levels were higher in HIV-infected compared to control subjects (Table 3). Thirty-two percent of HIV-infected subjects and 22% of controls met criteria for metabolic syndrome ($p=0.02$), based on the third report of the National Cholesterol Education Program/Adult Treatment Panel (NCEP/ATP III).[32] Differences in dietary fat, saturated fat and cholesterol remained highly significant between HIV-infected and control subjects controlling for metabolic syndrome as a covariate in the analysis (data not shown).

In terms of energy metabolism, HIV-infected subjects demonstrated higher absolute REE values ($p<0.0001$), higher REE adjusted for fat-free mass ($p<0.0001$), and lower RQ ($p=0.02$) when compared to controls. (Table 3).

RELATIONSHIP OF DIETARY INTAKE TO SERUM LIPID LEVELS IN HIV-INFECTED SUBJECTS

A multiple regression model was used to assess the contribution of dietary fat (g/d), saturated fat (g/d), trans fat (g/d), and cholesterol (mg/d) intakes to serum lipid levels, controlling for age, race, gender, BMI, PI use, alcohol consumption, total fiber intake, and IGT/diabetes among the HIV-infected subjects. Saturated fat was significantly associated with triglyceride level [triglyceride level increased 8.7 mg/dL (parameter estimate) per gram of increased saturated fat intake, $p=0.005$], while total fat intake was inversely associated with triglyceride level [triglyceride level decreased 3.0 mg/dL (parameter estimate) per gram of increased total fat intake, $p=0.02$]. These relationships between saturated fat or total fat and triglyceride levels were not significantly altered when use of anti-lipid lowering medications was entered into the model. There were no significant associations between dietary saturated fat, total fat, trans fat, or cholesterol intake and total cholesterol or HDL. (Table 4).

Saturated fat and total fat still remained significantly associated with triglyceride level, even when time was entered into the model, indicating that although there were slight differences in dietary intake (specifically, in percentage of calories derived from total fat and polyunsaturated fat) over time, there was no significant effect of time on the association between saturated fat or total fat and triglyceride levels among HIV-infected individuals (data not shown).

Use of PI was not shown to be significantly associated with hypertriglyceridemia among our HIV-infected population. Importantly, no significant differences in fat intake were seen between HIV-infected subjects receiving and not receiving PIs as well as between those with lipodystrophy and those without (data not shown).

DISCUSSION

Metabolic complications, including dyslipidemia occur commonly among HIV-infected patients, and may contribute to increased CVD in this population. [3, 4] Given the potential significance of these metabolic complications, and the limited information regarding the relationship to dietary composition in the HIV-infected population, we prospectively evaluated the composition of dietary fat intake of HIV-infected subjects with metabolic abnormalities and compared these data to that of community-derived controls, using the 2005 USDA Recommended Dietary Guidelines. We also determined the relationship between dietary fat intake and dyslipidemia in this group of HIV-infected subjects.

HIV-infected subjects in our study consumed a greater amount of total dietary fat, saturated fat, and cholesterol as well as a greater percentage of calories from trans fat and from saturated fat. More importantly, a higher percentage of HIV-infected subjects compared to controls exceeded the established 2005 USDA Recommended Dietary Guidelines for saturated fat and cholesterol. In particular, among HIV-infected subjects, saturated fat intake directly correlated with hypertriglyceridemia whereas total fat intake inversely correlated with hypertriglyceridemia. These highly significant associations between saturated fat or total fat and serum lipid levels in HIV-infected subjects in our study were seen even after controlling for PI use and major factors known to influence lipid levels, including gender, alcohol use, fiber intake, diabetes/IGT, BMI, age, and race.

The reasons for increased dietary fat consumption among our HIV-infected population were not formally assessed in this study and may be multifactorial. Our group as well as others have demonstrated an increased REE adjusted for fat-free mass among HIV-infected individuals compared to controls.[33] Increased dietary fat consumption among HIV-infected individuals with metabolic abnormalities may be a form of calorie-dense compensation for increased energy expenditure. The dietary fat consumption could also be centrally mediated through effects of HIV itself or HAART on satiety/hunger centers or peripherally mediated through alterations in taste perception. Or, possibly, dietary assessment and advice may have been overlooked in this population.

Among HIV-infected individuals, there was no significant effect of time on percentage of calories derived from saturated fat, but instead a small increase in percentage of calories

derived from polyunsaturated fat (PUFA), leading to an increase in percentage of calories from total fat with time. Increased consumption of “good fats” such as PUFA has been advocated by the NCEP/ATP III guidelines for non-HIV-infected individuals with metabolic syndrome and hyperlipidemia. [32] But, those dietary guidelines do also recommend total fat and saturated fat restriction. Thus, although recognition of metabolic syndrome and lipodystrophy among HIV-infected individuals is increasing, application of dietary guidelines to this population is only partially heeded. The need for greater emphasis on saturated fat reduction in addition to increases in “good fat” consumption is required.

Only a few prior studies have specifically examined dietary fat intake and its relation to serum lipid levels in HIV-infected subjects.[10–12] Moreover, the inverse relationship between total fat intake and serum triglyceride levels has been previously reported in non-HIV-infected subjects but not in HIV-infected patients.[8, 34] In contrast, the percentage of energy from trans fat was significantly higher in HIV-infected subjects, but did not correlate with serum lipid levels in our study. Nonetheless, it is important to recognize that increased trans fat intake may increase LDL: HDL ratios and Lp(a) levels, and increase the risk of atherosclerosis.[35, 36] Total fiber intake was lower in HIV-infected subjects compared to controls, suggesting a lack of dietary variety in the form of fruits, vegetables, and legumes. Increased fiber intake (particularly soluble fiber) has been associated with a lower risk of cardiovascular disease.[37, 38]

In non-HIV-infected subjects, dietary intervention focusing on saturated fat restriction results in a decrease of 2 – 20% in triglyceride and 1–15% in total cholesterol levels.[39–41] In small clinical trials of dietary intervention in HIV-infected subjects, similar reductions in total cholesterol (4–11%) and triglyceride levels (12–23%) have been achieved.[42–44] Based on our data, an increment of 1 gram/day in saturated fat intake is associated with a statistically and clinically significant 8.7 mg/dL increase in triglyceride levels in HIV-infected subjects with metabolic abnormalities. Thus, if the mean saturated fat intake of our HIV-infected subjects could be decreased from the observed level of 31 grams/day shown in this study (representing 12% of total caloric intake) to 25 grams/day (<10% total caloric intake), this 6 gram reduction in daily saturated fat intake would potentially result in a significant 52 mg/dL decrease in serum triglyceride levels. The mean serum triglyceride level among the HIV-infected subjects in our study was 230 mg/dL, with a prevalence of hypertriglyceridemia similar to that of the DAD study.[4] Therefore a 6 gram reduction in saturated fat would correspond to a 23% decrease in serum triglyceride levels, similar to published effects of dietary intervention trials in HIV-infected subjects.[42–44]

Since we were evaluating dietary intake in HIV-infected individuals with metabolic abnormalities, we simultaneously recruited community-derived, non-HIV-infected controls with metabolic abnormalities as the appropriate comparator group. This control group was similar to the American population in terms of percentage meeting metabolic syndrome based on NCEP/ATP criteria (22% vs. 24%, our controls vs. American population) as well as percentage of calories derived from saturated fat intake. [45–48] The control population in our study often failed to meet the 2005 USDA Recommended Dietary Guidelines for total fat, saturated fat, and cholesterol, but demonstrated less saturated fat, total fat and cholesterol intake than the HIV group.

HIV-infected subjects in our study demonstrated higher REE adjusted for fat-free mass and lower RQs, the latter implying a higher oxidation of lipids relative to that of carbohydrates. This occurred in the context of increased intake of dietary fats. Increased fat oxidation has been shown to occur as an adaptation to high fat diets in non-HIV-infected subjects.[49, 50] Our findings could possibly be explained by futile triglyceride/fatty acid cycling, in which there is a primary defect in adipocyte hormone-sensitive lipase, causing accelerated lipolysis, excess free fatty acid (FFA) flux, and increased hepatic production of VLDL (very-low density lipoproteins) in the fasted state. The increased FFA transit and VLDL formation are thought to result in increased energy expenditure while the increased FFA levels may promote lipid oxidation by mass action.[51, 52] These mechanisms may contribute to hypertriglyceridemia in HIV-infected subjects.

There are limitations to our study. This convenience sample was chosen specifically to assess dietary intake among HIV-infected patients with metabolic abnormalities and a significant proportion was classified as having lipodystrophy. These results cannot, therefore, be generalized to the larger group of HIV-infected patients without metabolic abnormalities nor to HIV-infected patients with wasting or undernutrition. Patients were not followed longitudinally over time to determine the relationship between changes in dietary intake and dyslipidemia. The method of dietary food assessment was primarily by prospective 4-day food record, but 17% of subjects were assessed using 24-hour food recall. However, we did control for the method of dietary assessment in our analysis and still demonstrated significant findings. Despite these limitations, our findings of increased saturated fat intake in HIV-infected subjects with metabolic abnormalities using a standardized reference guideline, e.g. the 2005 USDA Recommended Dietary Guidelines, and the independent strong association of saturated fat intake to hypertriglyceridemia point to a critical role for dietary intervention in this important subset of HIV-infected individuals with increased cardiovascular risk.

In conclusion, we investigated dietary intake in and relationship to lipid parameters in HIV-infected subjects. The anthropometric and biochemical features of the HIV-infected subjects in our study are consistent with the altered metabolic phenotype seen in a high proportion of HIV-infected individuals in the era of HAART. Our study demonstrates that this specific subset of HIV-infected individuals consumes a more “atherogenesis-promoting” diet compared to community-sampled controls and a significantly greater percentage of these HIV-infected subjects than community-sampled controls exceeded the 2005 USDA Recommended Dietary Guidelines for saturated fat and cholesterol. Importantly, saturated fat intake correlated directly and total fat correlated inversely with hypertriglyceridemia in these HIV-infected subjects. Thus, based on these findings, the primary goal of dietary intervention in HIV-infected subjects with metabolic abnormalities should be reduction in saturated fat, rather than an excessive reduction in total fat. Careful assessment of dietary intake and more aggressive dietary intervention may prove to be beneficial to the prevention of cardiovascular disease in this population of patients.

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Figure 1A. A: Percentage of Total Calories From Macronutrients

Results are expressed as mean $\pm$ standard deviation.

The white bar denotes HIV-infected subjects while the dark gray bar denotes non-HIV-infected controls.

* p values for the differences between HIV-infected and control subjects derived from a mixed effects ANOVA model with adjustment for age, race, gender, BMI, insurance status, use of lipid-lowering agents, method of dietary assessment, and income quartile, with each study assigned a random effect

B: Percentage of Total Calories from Fat Components

Results are expressed as mean $\pm$ standard deviation.

The white bar denotes HIV-infected subjects while the dark gray bar denotes non-HIV-infected controls.

* p values for the differences between HIV-infected and control subjects derived from a mixed effects ANOVA model with adjustment for age, race, gender, BMI, insurance status, use of lipid-lowering agents, method of dietary assessment, and income quartile, with each study assigned a random effect

Abbreviations: SFA, Saturated Fatty Acids; MUFA, Monounsaturated Fatty Acids; PUFA, Polyunsaturated Fatty Acids

C: Percentage of Subjects Exceeding Recommended Dietary Guidelines $\ddagger$

The white bar denotes HIV-infected subjects while the dark gray bar denotes non-HIV-infected controls.

* p values for the differences between HIV-infected and control subjects derived from a mixed effects ANOVA model with adjustment for age, race, gender, BMI, insurance status, use of lipid-lowering agents, method of dietary assessment, and income quartile, with each study assigned a random effect

$\ddagger$ based on the USDA Dietary Guidelines for Americans 2005[31]

Table 1**Demographics in HIV-Infected and Non-HIV-Infected Subjects**

Variable	Means and Standard Deviations		P- value [†]	P Value adjusted [‡]
	HIV+ (N=356)	Control (N=162)		
Demographics				
Age (y)	42±7	41±10	1.0	-
Gender (%)			1.0	-
Male	55.3	45.1		
Female	44.7	54.9		
Race (%)			0.53	-
Caucasian	56.3	61.1		
African American	28.4	25.3		
Hispanic	9.9	7.4		
Other	5.4	6.2		
% Individuals in Each Income Quartile based on Zipcode				
1 st	49.1	31.6		-
2 nd	22.8	30.3		
3 rd	20.4	23.9		
4 th	7.7	14.2		
% Having Private Insurance	20.7	31.9	0.0001	-
% Taking Lipid Lowering Drugs	12.1	9.9	0.18	-
Enrollment Date (year)	2001± 2	2003±2	<0.0001	0.04
HIV Parameters				
CD4 (#/mm ³)	444±254	871±286	<0.0001	<0.0001
Viral Load (copies/mL) ¶	400 (50,5744)	-	-	-
Duration HIV (years)	8.5±4.8	-	-	-
Currently taking PI (%)	66.8	-	-	-
Currently taking NRTI (%)	93.2	-	-	-
Currently taking NNRTI (%)	37.5	-	-	-

Variable	Means and Standard Deviations		P-value [†]	P Value adjusted [‡]
	HIV+ (N=356)	Control (N=162)		
% Currently not taking Antiretrovirals	11.2	-	-	-
% Categorized with Lipodystrophy	72.5	-	-	-

Results expressed as mean ± standard deviation

[†] p values for the differences between HIV-infected and control subjects derived from a mixed effects ANOVA model with each study assigned a random effect

[‡] p values for the differences between HIV-infected and control subjects derived from a mixed effects ANOVA model with adjustment for age, race, gender, BMI, insurance status, and income quartile, with each study assigned a random effect

[¶] Results expressed as median (interquartile range); Abbreviations: PI: Protease Inhibitor, NRTI: Nucleoside Reverse Transcriptase Inhibitor, NNRTI: Non-Nucleoside Reverse Transcriptase Inhibitor

Table 2

A: Dietary Intake in HIV-infected and Non-HIV-Infected Subjects				
Variable	Means and Standard Deviations	P value [†]	P value Adjusted [‡]	
	HIV + Control (N=356)			
Total Calories (kcal/d)	2235±798	2065±725	0.08	0.29
Carbohydrate (g/d)	273±107	249±93	0.31	0.92
Protein (g/d)	91±37	86±35	0.08	0.27
Fat (g/d)	87±37	79±37	0.02	0.048
Saturated Fat (g/d)	31±15	27±15	0.004	0.006
Monounsaturated Fat (g/d)	33±14	30±16	0.04	0.08
Polyunsaturated Fat (g/d)	17±8	16±8	0.36	0.67
Trans fat (g/d)	5±3	4±3	0.09	0.05
Cholesterol (mg/d)	342±187	294±209	0.004	0.006
Total Fiber (g/d)	17±9	18±9	0.09	0.03
Alcohol (g/d)	3±8	7±18	0.02	0.005

B. Gender Effects on Fat Intake in HIV-Infected and Non-HIV-Infected Subjects												
	Means and Standard Deviations				Main Gender Effect			Gender Effect in HIV Group			Gender Effect in Control Group	
	HIV+ (N=356)		Control (N=162)		OR*	P value [†]	OR*	P value [‡]	OR*	P value [‡]	OR*	P value [‡]
	Male (%)	Female (%)	Male (%)	Female (%)								
Individuals Above Total Fat Dietary Guidelines [‡]	54.1	51.6	42.5	42.7	1.25	0.21	-	-	-	-	-	-
Individuals Above Saturated Fat Dietary Guidelines [‡]	76.9	74.8	55.6	65.2	1.11	0.59	-	-	-	-	-	-
Individuals Above Cholesterol Dietary Guidelines [‡]	62.6	34.0	44.4	32.6	1.64	0.007	1.82	0.02	1.35	0.30	1.35	0.30

Results expressed as mean ± standard deviation

[†] p values for the differences between HIV-infected and control subjects derived from a mixed effects ANOVA model with each study assigned a random effect

[‡] p values for the differences between HIV-infected and control subjects derived from a mixed effects ANOVA model with adjustment for age, race, gender, BMI, insurance status, use of lipid-lowering agents, method of dietary assessment, and income quartile, with each study assigned a random effect

* OR (Odds Ratio) obtained from the logistic regression model with adjustment for age, race, gender, BMI, insurance status, use of lipid-lowering agents, method of dietary assessment, and income quartile

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[†] p value for the gender effect (male vs. female) obtained from the logistic regression model with adjustment for age, race, gender, BMI, insurance status, use of lipid-lowering agents, method of dietary assessment, and income quartile

[‡] based on the USDA Dietary Guidelines for Americans 2005³¹

Table 3
Body Composition, Energy, and Metabolic Parameters in HIV-Infected and Non-HIV-Infected Subjects

Variable	Means and Standard Deviations		P- value [†]	P Value adjusted [‡]
	HIV+ (N=356)	Control (N=162)		
Body Composition and Energy Parameters [§]				
BMI (kg/m ²)	26.8±5.2	28.7±7.1	0.86	0.80
Waist (cm)	94.7±12.8	96.9±18.3	0.15	0.52
Hip (cm)	99.8±11.4	107.4±14.4	0.02	0.02
Waist-to-Hip Ratio	0.95±0.07	0.90±0.08	<0.0001	<0.0001
CT SAT (cm ²) [#]	225.5±149.2	320.1±190.2	0.05	0.07
CT VAT (cm ²) [#]	125.4±69.0	131.0±86.4	0.04	0.24
CT VAT:SAT	0.82±0.84	0.43±0.23	0.001	0.002
Total Fat (kg)	20.4±10.0	26.1±13.5	0.08	0.13
Total Lean (kg)	55.7±11.1	55.0±13.3	0.06	0.40
Total Extremity Fat (kg)	8.3±5.2	12.4±6.2	0.0001	0.0008
Trunk: Total Extremity Fat	1.5±0.6	1.0±0.4	<0.0001	<0.0001
REE (kcal/d) [#]	1730±363	1705±480	<0.0001	<0.0001
REE/Fat Free Mass (kcal/kg/d) [#]	31.8±4.1	29.8±3.6	<0.0001	<0.0001
RQ [#]	0.83±0.08	0.85±0.09	0.21	0.02
Metabolic Parameters ^{‡‡}				
Total Cholesterol (mg/dL) [*]	196±52	178±37	0.07	0.003
Triglycerides (mg/dL) [*]	230±241	130±135	0.001	<0.0001
HDL (mg/dL) [*]	41±13	48±14	<0.0001	<0.0001
Fasting Glucose (mg/dL) [*]	90±13	89±14	0.19	0.44
Glucose AUC (mg/dL x 120 min) ^{*#}	16980±3855	15224±3468	0.05	0.02
Fasting Insulin (μIU/mL) [*]	13±12	12±10	0.03	0.03
% Meeting Criteria for Metabolic Syndrome	32.3	22.1	0.005	0.02

Results expressed as mean ± standard deviation

[†] p values for the differences between HIV-infected and control subjects derived from a mixed effects ANOVA model with each study assigned a random effect

[‡] p values for the differences between HIV-infected and control subjects derived from a mixed effects ANOVA model with adjustment for age, race, gender, BMI, insurance status, and income quartile, with each study assigned a random effect

^{†‡} Use of lipid lowering agents included as an additional covariate in the determination of lipid levels

[¶] BMI not included as a covariate in the comparison of body composition parameters

[#] Data for CT available in 304 HIV+ and 123 controls, for REE in 283 HIV+ and 146 controls, and for OGTT in 301 HIV+ and 51 controls

^{*} To convert mg/dL to mmol/L for total cholesterol and HDL, multiply by 0.0259; for triglycerides, multiply by 0.0113; for glucose, multiply by 0.0555; and to convert µIU/mL to pmol/L for insulin, multiply by 6.945

Abbreviations: BMI: Body Mass Index, SAT: Abdominal Subcutaneous Adipose Tissue, VAT: Visceral Adipose Tissue, REE: Resting Energy Expenditure, RQ: Respiratory Quotient, HDL: High-Density Lipoprotein, AUC: Area-Under-the-Curve

Table 4

Multiple Regression Modeling For the Relationship of Dietary Fat Intake to Serum Lipid Levels in HIV-Infected Individuals

Variable	Triglyceride (mg/dL)		HDL (mg/dL)		Cholesterol (mg/dL)	
	Estimate [†]	P value [‡]	Estimate [†]	P value [‡]	Estimate [†]	P value [‡]
Saturated Fat (g/d)	8.7	0.005	0.05	0.80	0.98	0.15
Total Fat (g/d)	-3.0	0.02	-0.03	0.67	-0.38	0.18
Trans Fat (g/d)	4.8	0.43	0.50	0.15	0.14	0.92
Cholesterol (mg/d)	-0.13	0.24	-0.002	0.78	0.007	0.79
Total Fiber (g/d)	-1.2	0.59	0.21	0.09	-0.14	0.76
Age	2.0	0.37	0.34	0.008	0.35	0.48
Gender (Male vs. Female)	54.6	0.001	-4.2	<0.0001	7.6	0.04
Race	-	0.15	-	0.02	-	0.20
Caucasian vs. Other	4.2	0.87	-3.1	0.04	2.1	0.72
African American vs. Other	-38.4	0.20	3.2	0.06	-12.0	0.07
Hispanic vs. Other	80.3	0.04	-0.26	0.91	10.8	0.21
BMI	-2.1	0.43	0.14	0.35	1.7	0.007
IGT or Diabetes	-	0.10	-	0.03	-	0.06
None vs. Diabetes	-55.6	0.03	3.3	0.02	-11.6	0.04
IGT vs. Diabetes	-20.1	0.47	-1.0	0.51	3.7	0.55
Alcohol (g/d)	0.24	0.89	0.24	0.02	0.20	0.60
Current PI use	-7.3	0.63	0.87	0.31	-2.5	0.45

[†] Parameter estimate derived from least squares multiple regression modeling assessing the relationship of total fat, saturated fat, cholesterol, and trans-fat to serum lipid levels after controlling for age, gender, BMI, race, protease inhibitor use, alcohol consumption, total fiber intake, and impaired glucose tolerance/diabetes

[‡] P value for parameter estimate derived from least squares multiple regression modeling assessing the relationship of total fat, saturated fat, cholesterol, and trans-fat to serum lipid levels after controlling for age, gender, BMI, race, protease inhibitor use, alcohol consumption, total fiber intake, and impaired glucose tolerance/diabetes

Abbreviations: HDL: High-Density Lipoprotein, BMI: Body Mass Index, IGT: Impaired Glucose Tolerance, PI: Protease Inhibitor

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