High-fiber diet in HIV-positive men is associated with lower risk of developing fat deposition[1–3]

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ABSTRACT

Background: Lipodystrophy has been described with increasing frequency in patients infected with HIV. This study focused on the identification of dietary components that may predispose HIV-positive patients to the development of fat deposition.

Objective: We evaluated differences in past dietary intake between men with HIV who developed fat deposition and those who did not.

Design: This nested case-control study consisted of 47 cases and 47 controls from the Nutrition for Healthy Living cohort. Food records from 6 to 24 mo before development of fat deposition in cases were analyzed and compared with food records from controls by using *t* tests for normally distributed nutrients and Wilcoxon rank-sum tests for nutrients with skewed distributions.

Results: HIV-positive patients without fat deposition had greater overall energy intakes (kcal/kg; $P = 0.03$) and greater intakes of total protein ($P = 0.01$), total dietary fiber ($P = 0.01$), soluble dietary fiber ($P = 0.01$), insoluble dietary fiber ($P = 0.03$), and pectin ($P = 0.02$) than did HIV-positive patients with fat deposition. Those without fat deposition also tended to currently perform more resistance training ($P = 0.05$) and to not be current smokers ($P = 0.05$).

Conclusion: Our results indicate that an overall high-quality diet, rich in fiber and adequate in energy and protein, may be beneficial in preventing the development of fat deposition in persons infected with HIV. The results of this study further emphasize that a healthy lifestyle, including exercise and avoidance of unhealthy behaviors such as smoking, may also be similarly beneficial. *Am J Clin Nutr* 2003;78:790–5.

KEY WORDS Lipodystrophy, HIV, fat redistribution syndrome, fiber, nutrition, diet

INTRODUCTION

Initial reports of nutritional status in persons living with HIV infection showed strong associations between the loss of lean body mass and death (1). Although weight loss and wasting continue to be a problem (2), lipodystrophy has been described with greater frequency in persons living with HIV infection (3–6). Lipodystrophy is a syndrome that includes changes in body composition, known as fat redistribution syndrome (FRS), and metabolic disorders (3–12). Whereas FRS is likely multifactorial, some investigators theorize that it occurs in response to the use of highly active antiretroviral therapy (HAART)—particularly, the protease inhibitors (3, 6, 9, 12). The syndrome may also occur, however, in the absence of HAART, and other host and viral factors—such as the duration of HIV infection, age, greater body mass index (BMI; in kg/m^2; 13, 14), change in viral load, sex, and ethnicity—may play a role in the development of FRS (15).

FRS consists of fat deposition, fat atrophy, or both. In HIV-positive persons, fat deposition commonly occurs in the visceral fat depot and less commonly in the dorsocervical fat pad; it is similar to the fat deposition that occurs in the metabolic syndrome in HIV-negative persons. In HIV-associated lypodystrophy, fat atrophy occurs in the subcutaneous fat depot, especially in the face, buttocks, and extremities. The metabolic abnormalities include dyslipidemia, characterized by serum triacylglycerol and non-HDL-cholesterol concentrations above established disease risk norms and serum HDL-cholesterol concentrations below such norms, with or without insulin resistance. It is often difficult to diagnose patients who have fat redistribution because there is no clinical definition and the manifestations vary from patient to patient (16). Each part of the syndrome may occur independently of the other parts (11, 15).

Currently, encouraging weight reduction if needed (with a combination of resistance and aerobic exercises), altering antiretroviral therapy regimens, and using growth hormone are methods of treatment for fat deposition that are under investigation. At this time, diet modification has not been investigated thoroughly enough to allow specific recommendations to be made.

This study focused on dietary components that were identified by research into diseases such as metabolic syndrome and type 2 diabetes as being related to the development of abdominal fat deposition (17–21). Fiber has been shown to help normalize blood

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glucose and lower serum cholesterol in persons who do not have HIV infection, and its consumption is generally associated with overall higher dietary quality (20–22). Diets high in fat, primarily saturated fat, have been linked to increased risks of hyperlipidemia and heart disease (18). Thus, fiber and fat intakes were the 2 dietary components emphasized in this study. The purposes of this study were to evaluate the dietary intake of patients with HIV infection 6–24 mo before they met our definition of fat deposition and to compare the differences in intake between those who had fat deposition and those who did not.

SUBJECTS AND METHODS

We conducted a nested case-control study among participants enrolled in the Nutrition for Healthy Living study, which is an ongoing longitudinal study examining the nutritional and metabolic consequences of HIV infection. Details of the study were published elsewhere (23). Briefly, between 1995 and 1999, 678 HIV-positive subjects were enrolled in the study for semiannual visits. Eligible participants included HIV-positive adults (aged ≥ 18 y) living in the greater Boston area or in Rhode Island. Subjects were excluded if they had any of the following conditions at enrollment: pregnancy, diabetes, thyroid disease, or malignancies other than Kaposi's sarcoma. Participant confidentiality was preserved according to the regulations stipulated by the individual facility and approved by the Institutional Review Board of the Tufts–New England Medical Center.

Data collected at each study visit included weight, height, anthropometry (triceps, subscapular, and suprailiac skinfold thicknesses), bioelectrical impedance analysis resistance and reactance (BIA-101A; RJL Systems, Clinton Township, MI), and waist and hip circumferences (beginning in November 2000). In addition, at each visit, participants completed a detailed questionnaire eliciting information on sociodemographic characteristics, clinical status, health-related quality of life, use of recreational drugs, and use of HIV-related medications. Blood was collected and stored at each visit for immunologic, biochemical, and nutritional testing.

Dietary intake

At the baseline Nutrition for Healthy Living study visit, a nutritionist trained in the study protocol and using a standardized procedure instructed each participant on how to keep a food record for 3 d, including one weekend day. Each participant was given a food scale (Sunbeam Corporation, Mississauga, Canada) and a ruler to document portion sizes. At the second baseline visit, which was 7–14 d later, the nutritionist reviewed the food record with the participant to clarify the details of the foods recorded. Subsequently, 1 wk before each 6-mo follow-up appointment, food record forms were mailed to participants. The completed 3-d food records were then reviewed with a trained nutritionist at the next study visit. A 24-h recall of foods consumed was elicited at all visits if a participant did not have a completed food record.

The food records were analyzed with the use of NUTRITION DATA SYSTEM software (version 2.92; Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN; 24). A single, experienced coder entered all the data.

Case ascertainment and control selection

Participants were eligible for analysis if they were male and had a BMI of 23–26, which was set to exclude those who were either obese or wasted. Cases were defined as those with fat deposition, and controls were defined as those without fat deposition.

Cases were identified at the visit when they were first recognized as having fat deposition, which was referred to as the "index visit." For this study, fat deposition was defined as a waist-to-hip ratio (WHR) > 0.95, which is above the recommended normal range of < 0.90 for men (17). Cases were excluded if they did not have ≥ 2 consecutive study visits before their index visit. The approximate time span between the index visit and the 2 prior visits was 6–24 mo. Cases were also excluded if they did not have complete diet records for both of the 2 visits before the index visit.

For each case, we randomly selected one control without fat deposition, matched by age (± 5 y), race (African American or non–African American), HAART use (ever or never), length of follow-up (± 12 mo), and CD4 cell count (ever ≤ 200 or always > 200 cells/μL). Controls were excluded if they had a mid-upper-arm circumference < 28.7 cm, which is equal to or greater than the 10th percentile of men aged ≥ 18 y and was used to eliminate those with wasting or fat atrophy (25). The control match date was referred to as the "index visit" for the controls. After the matching process for cases and controls was completed, 47 cases and 47 controls were identified for this study.

Statistical analysis

Diet records were analyzed and averaged for the 2 visits before the index visits of cases and controls. That is, dietary intake comparisons between cases and controls were the average intake from two 3-d food records ≈6–24 mo before the index visit. Specific macronutrients and micronutrients were compared between cases and controls by using *t* tests for normally distributed nutrients and Wilcoxon rank-sum tests for nutrients with skewed distributions. Conditional logistic regression analysis was used to estimate the odds ratio of fat deposition associated with each nutrient. Potential confounders examined included the number of AIDS-defining conditions, whether a person was trying to change his weight (either gain or lose), individual food insecurity, use of protease inhibitors, use of vitamin and mineral supplements, smoking, and resistance training. All of these potential confounders were assessed at the index visit. Statistical analyses were carried out with the use of SAS statistical software (version 9.0; SAS Institute Inc, Cary, NC).

RESULTS

Seventy-five cases with fat deposition were identified, and appropriate controls were identified for 47 of the 75. The analysis was thus based on 47 cases and 47 controls.

Demographic data and other characteristics of the cases and controls at their index visits are shown in **Table 1**. Both groups consisted of 6 African American and 41 non–African American subjects. Mean age and BMI at the index visit did not differ significantly between cases and controls. Eighty-one percent of cases and controls were taking HAART. Thirty-eight percent of both groups had CD4 cell counts > 200 cells/μL.

Cases had WHRs and waist measurements that were significantly greater than those of controls. Hip circumference, mid-upper-arm circumference, tricep skinfold thickness, and suprailiac skinfold thickness did not differ significantly between cases and controls. However, cases had significantly greater subscapular skinfold thickness than did controls.

TABLE 1
Characteristics of the cases and controls at the index visit[1]

	Cases	Controls
Race [*n* (%)]		
African American	6 (12.8)	6 (12.8)
Latino or Hispanic	1 (2.1)	2 (4.2)
White	39 (83.0)	36 (76.6)
Other	1 (2.1)	3 (6.4)
Age (y)	43.2 ± 5.3[2]	41.9 ± 5.1
BMI (kg/m^2)	24.7 ± 0.9	24.5 ± 0.8
Waist circumference (cm)	92.5 ± 4.5[3]	85.6 ± 4.4
Hip circumference (cm)	94.1 ± 4.1	94.4 ± 3.4
Waist-to-hip ratio	0.98 ± .03[3]	0.91 ± .04
Midupper-arm circumference (cm)	31.1 ± 2.1	33.1 ± 8.7
Subscapular skinfold thickness (mm)	18.6 ± 7.6[4]	15.6 ± 5.9
Triceps skinfold thickness (mm)	8.6 ± 5.1	7.5 ± 4.4
CD4 cell count (cells/μL)	478 ± 301	377 ± 269
Viral load (copies/mL)	21 506 ± 48 241	31 290 ± 81 933
$\log_{10}$ Viral load	3.08 (2.30, 4.24)[5]	3.20 (2.30, 4.46)
Lowest viral load ever	200 (200, 1100)	200 (200, 3640)
Highest viral load ever	17 369 (2853, 84 808)	38 945 (1597, 138 201)
Resting energy expenditure	2055 ± 213	2070 ± 213

[1] $n = 47$ in each group.
[2] $\bar{x}$ ± SD.
[3,4] Significantly different from controls (chi-square test for frequencies, *t* test for means, and Wilcoxon rank-sum test for medians): [3] $P < 0.0001$, [4] $P = 0.03$.
[5] Median; 25th percentile and 75th percentile in parentheses.

Additional laboratory values available for comparison in this study were fasting serum triacylglycerol and hemoglobin concentrations and viral load. Serum triacylglycerol concentrations did not differ significantly between cases and controls ($\bar{x}$± SD: 197 ± 169 mg/dL for cases and 256 ± 247 mg/dL for controls; $P = 0.20$). Hemoglobin concentrations did not differ significantly between the 2 groups. Mean, lowest ever, and highest ever viral loads did not differ significantly between cases and controls. Other biochemical measures of metabolism, such as glucose and insulin concentrations, were not available for comparison.

The comparison of average dietary intake between the cases and controls 6–24 mo before the index visit is shown in **Table 2**. Intakes of total dietary fiber, soluble dietary fiber, insoluble dietary fiber, and pectin were all significantly higher in persons without fat deposition than in those with fat deposition. There was no significant difference between cases and controls in crude energy intake. However, when energy intakes were adjusted for weight (kcal/kg), subjects with fat deposition had significantly lower energy intakes than did those without fat deposition. Participants with fat deposition also had significantly lower total protein intakes than did those without fat deposition.

There were no significant differences between groups with regard to intakes of carbohydrates, fats, cholesterol, alcohol, or micronutrients. The median intake of calories from fat was 36% (interquartile range: 33–39) in cases and 35% (interquartile range: 32–40) in controls, and the median intake of calories from saturated fat was ≈12% (interquartile range: 10–14) in both groups. The ratio of polyunsaturated fat to saturated fat (P:S) also did not differ significantly between cases and controls. Although the difference was not significant, cases tended to have a lower intake of n−3 fatty acids than did controls.

The results from the final conditional logistic regression models for the nutrients that significantly differed between the cases and controls are shown in Table 2. An increase in the intake of all types of fiber was significantly associated with a decreased risk of development of fat deposition. The magnitude of risk reduction ranged from 2% for each 1-g increase in total protein intake to ≈30% for each 1-g increase in pectin intake.

When we examined other health status and lifestyle factors as potential confounders, we found some differences between cases and controls in resistance training and smoking status. Resistance training, defined as weight-bearing exercise, was performed significantly ($P = 0.05$) more often by those without fat deposition ($n = 24$) than by those with fat deposition ($n = 15$). Smoking, defined as being a current smoker, occurred significantly ($P = 0.05$) more often in those with fat deposition ($n = 20$) than in those without fat deposition ($n = 10$). However, neither of these variables was associated with any of the dietary intake variables. Therefore, although their inclusion in the multivariate analyses affected the significance of some of the nutrients, it did not substantially change any of the risk estimates for these nutrients. In the end, given our small sample size and the borderline significance of these two variables, we decided to leave them out of the final models to increase our power.

DISCUSSION

Studies investigating the role of diet in the development of fat deposition in patients with HIV infections are few (7–9, 13). In the current study we found that, in normal-weight HIV-positive persons with fat deposition, dietary intakes 6–24 mo before the development of FRS were lower in calories per kilogram, protein, and all dietary fiber components than were these same intakes in matched controls.

We believe that the cases and controls in this study were accurately identified and appropriately matched. In this study, WHR was used to indicate abdominal fat deposition, because waist circumference, not hip circumference, increases with visceral fat accumulation (5). Waist circumference has also been found to be superior to other body-composition measurements in predicting hyperinsulinemia and insulin area under the curve in HIV-positive men (26). In addition, we included only participants with normal BMIs of 23–26 to exclude obese or wasted patients. We did not obtain abdominal computed tomography to document the localization of fat, but cases had greater waist circumferences and WHRs than did controls, which strongly suggested abdominal fat deposition in the cases. Furthermore, there were no significant differences in hip circumference between those with and without fat deposition, which further indicated that the higher WHRs in the cases were not due to wasting in the buttocks. Tricep skinfold-thickness measurements did not differ between cases and controls, which also indicated that controls did not have fat atrophy. Greater subscapular skinfold thickness and more kilograms of body fat as measured by BIA ($\bar{x}$± SD: 17.65 ± 5.30 for cases, 15.00 ± 4.86 for controls; $P = 0.01$), and lower percentage lean body mass by BIA in the cases further indicate fat deposition in these patients. However, it is important to note that BIA does not accurately depict where the differences in body fat or lean body mass are located (27–30).

CD4 cell counts and viral loads did not differ significantly between cases and controls at their index visits. However, persons

TABLE 2
Comparison of dietary factors 2 visits before the index visit

Dietary variables	Cases	Controls	P^1	Risk ratio[2]
Energy (kcal)	2771 (2303, 3192)[3]	2898 (2474, 3748)	0.15	
Energy/weight (kcal/kg)	35 (31, 42)	40 (34, 49)	0.03	0.95 (0.91, 0.99)
Total carbohydrate (g)	339 (277, 401)	372 (303, 462)	0.39	
Total protein (g)	106 (92, 116)	119 (101, 145)	0.01	0.98 (0.96, 0.99)
Total fat (g)	112 (86, 128)	113 (89, 149)	0.42	
Cholesterol (mg)	382 (288, 512)	413 (269, 584)	0.84	
Alcohol (g)	0.14 (0.04, 7.25)	0.70 (0.04, 6.22)	0.46	
Percentage of calories (%)				
From carbohydrate	50 (45, 53)	48 (43, 53)	0.49	
From protein	15 (13, 18)	17 (14, 18)	0.10	0.87 (0.74, 1.02)
From fat	36 (33, 39)	35 (32, 40)	0.69	
From saturated fatty acids	12 (10, 14)	12 (10, 14)	0.90	
From monounsaturated fatty acids	14 (11, 15)	13 (12, 15)	0.99	
From polyunsaturated fatty acids	6.7 (4.7, 8.1)	6.6 (5.4, 7.5)	0.78	
Polyunsaturated-to-saturated fat ratio	0.59 (0.42, 0.83)	0.57 (0.42, 0.81)	0.98	
n−3 Fatty acids (g)	1.84 (1.43, 2.77)	2.07 (1.71, 3.07)	0.10	0.74 (0.52, 1.1)
Simple sugars (g)[4]	167 (116, 216)	164 (106, 237)	0.91	
Starch (g)	134 (102, 171)	157 (106, 188)	0.18	
Dietary fiber (g)				
Total	19 (14, 24)	23 (18, 29)	0.01	0.93 (0.88, 0.99)
Soluble	7.0 (5.4, 8.6)	8.8 (7.0, 11.0)	<0.01	0.79 (0.66, 0.95)
Insoluble	11.8 (8.8, 14.5)	13.9 (10.7, 18.4)	0.03	0.91 (0.84, 0.99)
Pectin (g)	2.1 (1.2, 2.6)	2.6 (1.5, 3.5)	0.02	0.71 (0.50, 0.99)

[1] Based on Wilcoxon rank-sum test.
[2] Based on conditional logistic regression models with dietary variable as the exposure and case or control status as the outcome. 95% CIs in parentheses.
[3] Median; 25th and 75th percentiles in parentheses.
[4] Include fructose, galactose, glucose, lactose, maltose, and sucrose.

with fat deposition had slightly higher CD4 cell counts and slightly lower viral loads. These values may indicate that fat deposition occurs in those with optimal response to HIV medication therapy, which is consistent with other reports (4–6).

Our study found fiber intake to be significantly higher among controls. This is in agreement with the findings of Hadigan et al (13), who studied 62 men and 23 women with FRS (15 with atrophy, 14 with deposition, and 56 with mixed syndrome). Dietary assessment by the Burke diet history method found that a 5-g increase in fiber intake was associated with a 14% reduction in insulin area under the curve. In our study, those without fat deposition had greater intakes of total dietary fiber, soluble dietary fiber, insoluble dietary fiber, and pectin. A 1-g increase in total dietary fiber intake was associated with a 7% reduction in the risk of development of fat deposition. These results were not surprising, because fiber has been credited for lowering blood lipids (especially cholesterol), improving glycemic control, and decreasing hyperinsulinemia (20, 22, 31). A recent study by Pereira et al (31) found that the consumption of whole-grain food, which is high in fiber, reduced fasting insulin concentrations in presumably healthy persons. A study by Hadigan et al (32) showed that improvements in insulin sensitivity can lead to decreased visceral fat deposition in FRS. Although the mechanism is unclear, it is possible that the lower fiber intake by cases in our study may have contributed to insulin resistance, which led to abdominal fat accumulation. The American Dietetic Association recommends that persons consume 20–35 g dietary fiber/d from a variety of plant foods (22). Cases in our study did not meet this recommendation.

Individuals without fat deposition had greater energy intakes per kilogram of weight and higher total protein intake. The energy differences noted in this study are unique. The study performed by Batterham et al (7) showed that individuals with fat redistribution had higher caloric intakes than those that did not have fat redistribution; however, the diets of these patients were analyzed with the use of food-frequency questionnaires. Our study used 3-d food records, which are considered the gold standard in assessing dietary intake (33). Adequate energy and protein intakes may be associated with a diet that is high in nutrient density and may not play a specific protective role in the development of fat deposition, because overall good nutrition is associated with better immune response (34). The difference in energy intake per kilogram among participants with BMI and resting energy expenditure (REE) levels that do not differ significantly might be attributed to lower levels of physical activity among those with fat deposition than among those without.

Increased total fat intake, especially the intake of saturated fat, leads to hyperlipidemia and increases the risk of heart disease in patients without HIV infection (18, 35). Although we did not find differences in fat intake between cases and controls, both groups had intakes of saturated fat (≈12% of calories) higher than those recommended (36). It is important to encourage diets within the recommended dietary fat guidelines to decrease the risk of heart disease.

The ratio of fatty acids may be a better indicator of the risk of heart disease. Hadigan et al (13) found that a high P:S was a strong independent predictor of hyperinsulinemia in patients with HIV infection and fat redistribution. Our data showed a slightly but not significantly higher median P:S in cases than in controls. Differences between studies may exist: for example, the study of Hadigan et al used food data collected by diet history recall for the past month, and there were also differences in the characteristics of the subjects studied.

Polyunsaturated fatty acids, especially n−3 fatty acids, have been found to have a protective effect against heart disease (18, 35). Fish oils, in particular, have been shown to decrease triacylglycerol synthesis in patients with severe hypertriacylglycerolemia (36). A study by Hellerstein et al (37) showed a reduction in serum triacylglycerol concentrations with fish oil supplementation in hypertriacylglycerolemic patients with HIV infection. In the current study, there was no significant difference in n−3 fatty acid intake between cases and controls.

Other lifestyle factors are important to consider when educating patients about healthy behaviors. In this study, current smoking and lack of resistance training were more frequent among cases. Exercise has been shown to be beneficial in promoting weight management and fat loss, improving HDL and LDL cholesterol concentrations, and aiding in glucose control in patients with HIV infection (11).

This study has both limitations and strengths. We included only men, and thus our findings may not be generalizable to women. The focus of the study was on the development of fat deposition, and our results may not apply to fat atrophy or to the metabolic abnormalities of FRS. Fat deposition and fat atrophy may have different, distinct risk factors for development, and thus we feel that exclusively evaluating fat deposition strengthened our findings. Another strength of our study was its detailed evaluation of diet; however, although cases were matched for other risk factors, we did not thoroughly investigate the role of specific medications and other variables. Whereas all data were collected prospectively, the analysis was essentially retrospective, and thus cause and effect must be viewed with caution.

In conclusion, our study showed that a diet high in fiber may be beneficial in preventing the development of fat deposition in normal-weight HIV-positive persons. The importance of overall good nutrition, especially adequate energy and protein intakes, in patients with HIV infection is emphasized by this study. We suggest that a healthy lifestyle, including diet, resistance training, and avoidance of unhealthy habits such as smoking, may be beneficial in preventing the development of fat deposition. These findings call attention to the importance of targeted nutrition education and follow-up and to the need for well-designed nutrition intervention studies, which carefully evaluate the efficacy of nutrition therapy in this patient population.

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