Objective. To determine whether 24 hr dietary recalls (DR) are a good measure of polyunsaturated fatty acid (PUFA) intake when compared to plasma levels, and whether plasma PUFA is associated with markers of HIV/AIDS progression and cardiovascular disease (CVD) risk. Methods. In a cross-sectional study among 210 antiretroviral therapy-naive HIV-infected adults from Lusaka, Zambia, we collected data on medical history and dietary intake using 24 hr DR. We measured fatty acids and markers of AIDS progression and CVD risk in fasting plasma collected at baseline. Results. PUFA intakes showed modest correlations with corresponding plasma levels; Spearman correlations were 0.36 ($p < 0.01$) for eicosapentaenoic acid and 0.21 ($p = 0.005$) for docosahexaenoic acid. While there were no significant associations ($p > 0.05$) between total plasma PUFA and C-reactive protein (CRP) or lipid levels, plasma arachidonic acid was inversely associated with CRP and triglycerides and positively associated with HDL-C, CD4+ T-cell count, and plasma albumin ($p < 0.05$). Plasma saturated fatty acids (SFA) were positively associated with CRP ($\beta = 0.24$; 95% CI: 0.08 to 0.40, $p = 0.003$) and triglycerides ($\beta = 0.08$; 95% CI: 0.03 to 0.12, $p < 0.01$). Conclusions. Our data suggest that a single DR is inadequate for assessing PUFA intake and that plasma arachidonic acid levels may modulate HIV/AIDS progression and CVD risk.
levels, advanced HIV/AIDS stage, and immune reconstitution inflammatory syndrome [3–5].

In Zambia, our group recently reported a higher risk of early ART mortality among individuals with moderately elevated triglyceride concentrations, a finding which may be related to the interaction of fatty acids and systemic inflammation [6]. The essential fatty acids (n-3 and n-6 polyunsaturated fatty acids (PUFA)) are nutrients of primary importance for health, and many research works in the last decades have shown the role of an adequate intake of n-3 and n-6 PUFA in the prevention of several diseases, in particular of cardiovascular diseases [7–9]. These studies were done mainly in non-HIV populations. Omega 3 fatty acids have been known to modulate biomarkers such as C-reactive protein (CRP) and CD4 count which are important determinants in the progression of HIV disease and other inflammatory conditions [10, 11]. To our knowledge, apart from one small clinical trial that assessed the benefit of combining fenofibrate with n-3 fatty acids in improving HIV-related clinical outcomes [12], studies on effects of fatty acids on metabolic parameters associated with morbidity and mortality in HIV are lacking, especially in resource-limited settings.

Reliable identification of individuals with key nutrient deficiencies that could be improved with nutritional support would inform the design of nutritional rehabilitation programs to reduce morbidity and mortality in HIV/AIDS patients. The Zambian diet is mainly composed of cereals, predominantly maize, starchy roots, and, to a lesser extent, fruits and vegetables. Cereals provide almost two-thirds of the dietary energy supply.

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2.4. Exposure and Dependent Variable Definitions. For the first objective, we focused on pairwise Spearman rank correlations between diet and plasma PUFA measurements. In the second objective, total plasma PUFA and SFA were the main exposure variables. The dependent variables were markers of HIV/AIDS disease progression (i.e., BMI, CD4+ cell count, and serum albumin) and cardiovascular disease risk (i.e., CRP, triglycerides, HDL-C, and LDL-C). In secondary analyses with plasma arachidonic acid as the main independent variable we investigated BMI, CD4+ cell count, plasma albumin, CRP, triglycerides, HDL-C, LDL-C, and plasma albumin as dependent variables.

2.5. Statistical Analysis. Of the 210 participants, 90% had complete data on fatty acids in plasma. Fatty acids in plasma were expressed as a percentage of the total fatty acids analyzed while dietary fatty acids were expressed as a percentage of total energy per day [21]. To validate fatty acid intakes, we computed pairwise Spearman correlation coefficients for each PUFA estimated from 24 hr DR against a corresponding PUFA measured in plasma. The exception was plasma α-linolenic acid in plasma which was tested for correlation with total dietary linolenic acid since α- and γ-linolenic acid could not be separated in our 24 hour DR.

Next we determined whether plasma fatty acids are associated with CRP and lipid profiles using multivariable linear regression models. For each of the dependent variables, that is, CRP, triglycerides, HDL-C, and LDL-C we estimated associations with the exposure variables, namely, total plasma PUFA and SFA adjusted for age, sex, BMI, plasma monounsaturated fatty acids (MUFA), trans fatty acid, alcohol use, and smoking.

Additional analyses were conducted to understand whether individual PUFA was associated with markers of CVD risk and HIV disease progression (e.g., CD4+ counts and plasma albumin). In this analysis, Spearman rank correlations between plasma AA and each of the markers of CVD risk and HIV disease progression (e.g., BMI, CD4+ cell count, and serum albumin) were determined. We then distributed plasma AA concentrations into quartiles and used ANOVA with robust variance estimator to determine whether markers of CVD risk and HIV/AIDS disease progression significantly varied by quartiles of AA before and after adjustment for age, sex, smoking, and alcohol consumption.

Data were analyzed using SAS version 9.4 (Cary, NC) and STATA version 12.1 (College Station, TX).

3. Results

3.1. Description of the Population. The characteristics of the study population are shown in Table 1. Most participants were women (54%) and relatively young, with a median age of 32 years for women and 35 years for men. The proportion of participants with BMI <18.5 kg/m² was 32%. Although the median CD4+ cell count was somewhat higher in women (143 cells/μL) compared to men (129 cells/μL), this difference did not reach statistical significance (p > 0.05). Concentrations of CRP also tended to be higher in women than in men but the differences were not statistically significant (p > 0.05). The frequency of smoking was quite low in our study population with only one (0.9%) current smoker among women and 10 (11%) among men (p < 0.05).

3.2. Validation of the Fatty Acid Estimates from 24 hr DR. The proportional distributions of fatty acids in the diet expressed as percent energy intake per day and in the plasma as percent of total fat are shown in Figure 1. The highest median percent energy was from linoleic acid (LA) (11.5%) and the lowest was from EPA (0.007%). The highest median percent of total fat in the plasma was from LA (16.1%) and the lowest was from α-linolenic acid (ALA) (0.15%).

Table 2 shows the Spearman correlation coefficients between plasma and corresponding dietary PUFA. The correlations are for the major n-3 and n-6 fatty acids. Two of the six fatty acids examined showed statistically significant correlations. The correlations ranged from very low to moderately low. The highest significant correlation was for EPA (r = 0.36, p < 0.01) and the second highest was for DHA (r = 0.21, p = 0.005). The correlations for ALA, DPA, LA, and AA were not statistically significant.

In multivariable linear regression analyses, total plasma PUFA concentrations were not significantly associated with CRP on a log scale (β = −0.10; 95% CI: −0.22 to 0.02, p = 0.09) in analyses adjusting for age, sex, BMI, MUFA, trans fatty acids, current smoking, and alcohol status (data not
### Table 1: Baseline characteristics of the study population.

| Variable                  | Women \( n = 113 \) | Men \( n = 97 \) | \( p \) |
|---------------------------|----------------------|------------------|--------|
| Age, years                | 32 (27, 37)          | 35 (31, 40)      | 0.003  |
| BMI, kg/m²                | 19.6 (18.0, 21.7)    | 19.6 (18.0, 21.3)| 0.62   |
| Current smoker, n (%)     | 1 (0.93)             | 10 (10.5)        | 0.003  |
| Current drinker, n (%)    | 10 (9.26)            | 12 (12.6)        | 0.41   |
| CD4, count, cell/µL       | 143 (108, 175)       | 129 (88, 168)    | 0.07   |
| C-reactive protein, mg/L  | 12.6 (2.01, 32.2)    | 5.71 (1.51, 32.1)| 0.24   |
| Albumin, g/dL             | 3.00 (2.60, 3.40)    | 3.20 (2.60, 3.70)| 0.09   |
| Total cholesterol, mmol/L | 3.56 (2.94, 4.11)    | 3.21 (2.68, 3.75)| 0.01   |
| Triglycerides, mmol/L     | 1.10 (0.89, 1.51)    | 1.05 (0.81, 1.43)| 0.39   |
| LDL-cholesterol, mmol/L   | 2.13 (1.60, 2.50)    | 1.77 (1.34, 2.38)| 0.02   |
| HDL-cholesterol, mmol/L   | 0.81 (0.47, 1.09)    | 0.71 (0.52, 1.04)| 0.42   |
| TC: HDL-C, ratio          | 4.49 (3.44, 7.00)    | 4.73 (3.32, 6.12)| 0.47   |
| Total energy intake, kcal/day | 1588 (1179, 1956) | 1777 (1356, 2305) | 0.03   |
| Total fat, % energy/day   | 34.1 (27.8, 42.2)    | 28.9 (20.5, 35.5)| <0.001 |
| Total trans fatty acids, % energy/day | 0.27 (0.09, 0.51) | 0.30 (0.12, 0.63) | 0.37   |
| Total saturated fatty acids, % energy/day | 6.52 (5.12, 8.00) | 5.52 (3.74, 7.64) | 0.02   |
| Total MUFA, % energy/day  | 9.47 (7.84, 11.8)    | 8.05 (5.72, 10.9)| 0.01   |
| Total dPUFA, % energy/day | 15.1 (11.4, 19.2)    | 11.1 (8.0, 14.8) | <0.001 |
| n-3 dPUFAs, % energy/day  | 1.89 (1.30, 2.60)    | 1.40 (0.92, 1.99)| <0.001 |
| Total PUFA, % total fat   | 32.1 (29.9, 33.1)    | 31.7 (29.6, 33.3)| 0.38   |

Values are median (25th, 75th percentile) unless otherwise stated. BMI, body mass index; HDL, high density lipoprotein; LDL, low density lipoprotein; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; dPUFA, PUFA from dietary sources; pPUFA, PUFA levels determined from plasma; TC, total cholesterol.

### Table 2: Spearman correlation coefficients between plasma and dietary polyunsaturated fatty acids.

| PUFA type  | Plasma composition (% of total fat) | Dietary composition (% energy/day) | \( r \) | \( p \) |
|------------|-----------------------------------|-----------------------------------|--------|--------|
| ALA (18:3n-3) | 0.15 (0.12, 0.20) | 1.50 (0.96, 1.92) | -0.05 | 0.50 |
| EPA (20:5n-3) | 0.39 (0.28, 0.51) | 0.01 (0.12) | 0.36 | <0.01 |
| DPA (22:5n-3) | 0.95 (0.77, 1.13) | 0.01 (0.03) | -0.01 | 0.90 |
| DHA (22:6n-3) | 3.54 (2.98, 4.27) | 0.03 (0.17) | 0.21 | 0.01 |
| LA (18:2n-6) | 16.1 (14.6, 18.0) | 11.5 (8.10, 14.9) | 0.06 | 0.09 |
| AA (20:4n-6) | 11.1 (9.58, 12.3) | 0.04 (0.01, 0.08) | 0.09 | 0.21 |

Plasma and dietary composition values are median (25th, 75th percentile). AA, arachidonic acid; ALA, α-linolenic acid; DHA, docosahexaenoic acid; DPA, docosapentaenoic acid; EPA, eicosapentaenoic acid; LA, linoleic acid; PUFA, polyunsaturated fatty acid.

A positive, though weak, association was observed between total plasma PUFA and triglycerides on a log scale (\( r = -0.14 \), \( p = 0.05 \)) and positively associated with CD4+ count (\( r = 0.16 \), \( p = 0.02 \)), albumin (\( r = 0.44 \), \( p < 0.001 \)), HDL-C (\( r = 0.26 \), \( p = 0.01 \)), and LDL-C (\( r = 0.29 \), \( p = 0.001 \)). As shown in Table 3, the associations between AA and CRP, serum albumin, triglycerides, HDL-C, and LDL-C remained significant after adjustment for age, sex, smoking, and alcohol consumption in ANOVA models.

In multivariable regression, a positive association was also observed between total plasma SFA and CRP (\( \beta = 0.24 \); 95% CI: 0.08 to 0.40, \( p = 0.003 \)) and between SFA and triglyceride concentrations (\( \beta = 0.08 \); 95% CI: 0.03 to 0.12, \( p < 0.01 \)). We did not detect an association with HDL-C and LDL-C.

### 4. Discussion

We observed modest correlations between EPA and DHA estimated from 24hr DR and corresponding measures in plasma but weak correlations for other PUFAs. We also observed null associations between total plasma PUFA and markers of HIV/AIDS disease progression and CVD-risk, but significant beneficial associations were observed between Plasma AA was inversely correlated with CRP (spearman correlation coefficient, \( r = -0.19 \), \( p = 0.02 \)) and triglycerides (\( r = -0.14 \), \( p = 0.05 \)) and positively associated with CD4+ count (\( r = 0.16 \), \( p = 0.02 \)), albumin (\( r = 0.44 \), \( p < 0.001 \)), HDL-C (\( r = 0.26 \), \( p = 0.01 \)), and LDL-C (\( r = 0.29 \), \( p = 0.001 \)). As shown in Table 3, the associations between AA and CRP, serum albumin, triglycerides, HDL-C, and LDL-C remained significant after adjustment for age, sex, smoking, and alcohol consumption in ANOVA models.

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The finding from our study that total plasma PUFA was not associated with lower CRP and lipid profiles may be a result of opposing actions among fatty acids when combined as total PUFA, in contrast with the roles they play individually. For example, supplementation with fish oil rich in EPA and DHA (n-3 PUFAs) has been reported to reduce inflammation in conditions such as colitis in both animal and clinical studies [30, 31]. In contrast, AA (an n-6 PUFA) is known to exert both proinflammatory and anti-inflammatory effects through its metabolites [32, 33].

Our finding that AA was inversely associated with CRP and triglycerides and positively associated with HDL-C, CD4+ cell count and plasma albumin suggests that individual fatty acids may influence clinical outcomes in HIV/AIDS patients. However, this observation needs further investigation.

The observation that SFA was positively associated with CRP (β = 0.24, p = 0.003) is consistent with findings from a previous study in which, after adjusting for other covariates, adults in Northern and Eastern Europe, North America, and Australasia (mean intakes ~0.15–0.25 g/d) [25].
SFA emerged as the single most important nutrient contributing to an increase in serum CRP levels [34]. The finding that SFA was positively associated with serum triglyceride concentration ($\beta = 0.08, p < 0.01$) supports evidence from previous studies that found SFA to be positively associated with coronary heart disease risk [35, 36].

In a clinical trial, daily supplementation with 1 g of n-3 fatty acids did not reduce the rate of cardiovascular events in patients at high risk for cardiovascular events [37]. However, the study reported a significant reduction in triglyceride levels (0.16 mmol/L), more among patients receiving n-3 fatty acids than among those receiving placebo ($p < 0.001$), without a significant effect on other lipids. Similarly, in our secondary analyses total n-3 fatty acids were not associated with reductions in CRP or improved lipid profiles and this may justify the need to explore the role of individual fatty acids in improving the CVD risk profiles.

The cross-sectional nature of our study limits causal inference between the exposure variables and the dependent variables of interest. The study could have been prone to interviewer bias arising from inconsistency in the way the DR was administered by different interviewers. The study could also have been prone to reporting bias which may have arisen from the participants being inclined to report healthier foods more than the less healthy foods. To mitigate these potential biases, the interviewers were specifically trained to elicit complete dietary histories from all participants. Lastly, the study population of adult HIV patients was recruited at a single health facility, which could limit generalizability to HIV-infected individuals in other settings. We also acknowledge that because the study was done among patients not yet on ART, we do not know the associations between fatty acids and markers of HIV/AIDS disease progression or CVD risk among those already on ART. Studies that assess effects of ART before and during ART are warranted so as to determine whether supplementation with fatty acids will modulate outcomes from ART in resource-limited settings.

5. Conclusions

The significant but generally low diet-plasma long-chain PUFA correlations could suggest that a single self-reported 24 hr DR may be inadequate for assessing PUFA intake in HIV/AIDS patients in Zambia. The study also suggests that SFA, which were positively related to markers of CVD risk, could play a role in HIV-related cardiovascular disease. Our study has found no evidence that total PUFA are inversely associated with CVD risk markers in HIV patients. However, there was evidence from secondary analyses that individual fatty acids, particularly AA, may play a role in improving CVD risk profiles and markers of HIV/AIDS disease progression.

Disclaimer

The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health or other funding agencies.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

Authors’ Contribution

Study conception and design were performed by Edmond K. Kabagambe, Christopher K. Nyirenda, and Douglas C. Heimburger. Data acquisition was conducted by Edmond K. Kabagambe, Douglas C. Heimburger, Claire N. Bosire, Christopher K. Nyirenda, Benjamin H. Chi, and Michael Y. Tsai. Data analysis, interpretation, and paper drafting or critical review were accomplished by Christopher K. Nyirenda, Edmond K. Kabagambe, Claire N. Bosire, Patrick Musonda, Meridith Blevins, James N. Kiage, John R. Koethe, Benjamin H. Chi, and Douglas C. Heimburger.

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