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Dietary intake, dietary quality, and prevalence of obesity among HIV-infected and HIV-uninfected adolescents and young adults in the REACH study

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Dietary intake, dietary quality, and prevalence of obesity among HIV-infected and HIV-uninfected adolescents and young adults in the REACH study

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ABSTRACT

Objective: To examine dietary quality and weight status among HIV-infected and HIV-uninfected youth.

Methods: A cross-sectional dietary intake study was conducted with 264 HIV-infected and 127 HIV-uninfected youth from the REACH cohort study (67% black/non-Hispanic; 75% female). Dietary intake was collected using the Block Food Frequency Questionnaire (98.2). The REACH study provided additional clinical, biochemical, and demographic data. Logistic and linear regression models were used for the analyses.

Results: Dietary quality: HIV was associated with a mixed effect on dietary intake; however, differences in macro- and micronutrient intakes were noted only among males. HIV-infected males had higher energy, fat, saturated fat, and cholesterol intakes than HIV-uninfected males. Although HIV-infected males had higher intakes of vitamin E, almost 40% of all participants had vitamin E intakes below the Estimated Average Requirements (EAR). A modified USDA's Healthy Eating Index (HEI) was used to look at overall dietary quality. The HEI was lower (indicating poor dietary quality) among HIV-infected compared to HIV-uninfected participants. Increased television watching was also associated with a lower HEI. Being female and having a higher self-perceived health was associated with a higher HEI. Obesity: Half of the HIV-infected (50.4%) and HIV-uninfected (54.3%) were overweight or obese. Prevalence of obesity decreased once CD4+ T-cells were <500 cells/μL. HIV did not modulate the effect of factors that increased the risk of obesity (female, living independently from parents/family, watching television ≥3 hours/d, previous dieting, and being from the Northeast or South).

Summary: These results demonstrate two important areas of health concern among HIV-infected adolescents. First, the dietary quality is poor, intakes of energy and dietary fat components are excessive, and the prevalence of obesity is high. Overweight and obese individuals with HIV infection may be at greater risk of developing metabolic abnormalities associated with HIV and antiretroviral therapy, such as hyperlipidemia, lipodystrophy, and insulin resistance. Second, the high prevalence of inadequacy in vitamin E intake may place individuals at increased oxidative stress associated with HIV infection. Nutrition educators

should focus on developing individualized behavioral goals emphasizing improved dietary quality and physical activity to improve health and quality of life for these adolescents.

CHAPTER 1. GENERAL INTRODUCTION

Thesis organization

This thesis contains a general introduction, a review of literature, two manuscripts prepared for submission to scientific journals, general conclusions, and appendices. The references cited in each chapter are listed at the end of the chapter using numeric citation style. References for the appendices are listed within each appendix. The conceptual framework and study hypothesis are described at the end of the literature review.

Introduction

This thesis focuses on the relationship between dietary intake and two public health concerns facing adolescents and young adults in the U.S.: obesity and human immunodeficiency virus (HIV) infection. Nutrition plays an integral role in both the prevention and treatment of obesity as well as HIV disease progression. Adolescents have increased demands for nutrients needed to support growth and development at the same time that they experience changes in life-style and eating habits that affect their ability to meet their nutritional needs.

The prevalence of overweight and obesity has increased among adults as well as adolescents. Numerous health consequences have been associated with increased weight. While the physiological consequences are most often seen among overweight adults, more adolescents are developing health consequences including type 2 diabetes and hyperlipidemia. Psychosocial issues can have negative consequences on an individual's mental health and social adjustment. In addition, overweight adolescents are more likely to be overweight as adults. The prevalence of HIV infection has also increased in the past decade among adolescents and young adults. Nutrition plays a vital role in slowing the progression of HIV infection. Adolescents with HIV infection may be at increased risk nutritionally due to the energy and nutrient demands necessary to support growth as well as compensate for infection.

Although significant research has been done in these areas, there is still much yet to be discovered. Little is known about the relationship between dietary intake and HIV infection among adolescents and young adults. As more is understood about this relationship, health-care professionals will be able to better serve this unique population.

CHAPTER 2. LITERATURE REVIEW

Dietary intake during adolescence

Adolescence is a defined as the period of life beginning with the appearance of secondary sex characteristics and ending with the cessation of somatic growth (1). Food choices and dietary intake during adolescence can have a profound effect on long-term health benefits. Numerous nutritional concerns have been identified among adolescents. Adolescents with high fat and saturated fat intake have increased risk for coronary heart disease (2); inadequate calcium intake and lack of physical activity can result in decreased bone density and osteoporosis (3); and being overweight as an adolescent has been associated with adult obesity (4-8), which can lead to other co-morbid conditions such as type 2 diabetes. In addition, adolescents are more susceptible to weight-related eating disorders such as anorexia nervosa, bulimia nervosa, binge eating disorder and other abnormal dieting behaviors, which have been related to the physiological, psychological and social changes that occur during adolescents (9).

Food intake among adolescents and young adults

Dietary intake information from national nutrition surveillance surveys including the 1994-1996 Continuing Survey of Food Intake by Individuals (CSFII) and the 1988-1994 National Health and Nutrition Examination Survey (NHANES III) has been compared to the Healthy People 2010 food and nutrient consumption goals (Table 1) (10). Intakes of dark green or orange vegetables and whole grains were low among both males and females aged 12-39 y. Among females aged 9-19 y, few met the recommended calcium intake. More males met the vegetable, grain, and calcium intake recommendations than females; however, more females met the recommendations for total fat, saturated fat, and sodium intake than males. No difference was seen between fruit intake by sex. Ethnic differences among adolescents were not available (10).

Munoz et al. (11) reported similar trends among adolescents aged 12-19 y from the 1989-1991 CSFII. White youth aged 2-19 y were more likely than black/non-Hispanic or Hispanic children to meet grain and dairy recommendations but less likely than black/non-Hispanics to meet vegetable requirements. Among 12-19 year olds, 7.1% of males and 18.4% of females did not meet any of the recommended food group servings; and only 1.6%

of males and 0.6% of females in this age group met all of the recommendations. As expected, youth who met none of the food group recommendations had average intakes well below the RDA for vitamin B_6, calcium, iron, and zinc. On the other hand, those who met all of the recommendations had micronutrient intakes above the RDAs. Youth who met only the grain, vegetable, or fruit recommendation had especially low intakes of calcium and zinc.

Table 1. Prevalence (%) of U.S. adolescents and young adults meeting the Healthy People 2010 food and nutrient consumption goals (10)

Daily dietary intake goal	Target Goal	Females 12-19 y	Females 20-39 y	Males 12-19 y	Females 20-39 y
≥2 fruit servings	75	23	20	22	23
≥3 vegetable servings	50	38	43	55	68
≥1/3 from dark green or orange vegetables	50	7	7	4	4
≥6 grain servings	50	49	40	77	70
≥3 whole grain servings	50	6	5	9	11
<30% energy from total fat	75	36	38	30	29
≤10% energy from saturated fat	75	34	41	27	32
≤2400 mg sodium	65	29	30[a]	4	6[a]
Meet calcium recommendation	75	19[b]	40[c]	52[b]	64[c]

[a] ≥20 years; [b] 9-19 years; [c] 20-49 years

Micronutrient intakes

A large proportion of adolescents have been identified with low micronutrient intakes. The relationship between dietary intake and vitamin/mineral supplement use was examined among adolescents aged 13-18 y from the 1994 CSFII (Table 2) (12). In this study, inadequacy was defined as intake less than 75% of the 1989 RDA. Regardless of supplement use, 25% or more of the adolescents had inadequate dietary intakes of calcium, iron, zinc, and vitamins A and E. More females (>37%) than males (<25%) had inadequate iron intake. The prevalence of inadequacies was highest among non-supplement users compared to supplement users regardless of frequency of supplement use, except for iron. However, more than one-third of daily supplement users still had inadequacies for vitamins A and E, calcium and zinc.

Table 2. Percentage of U.S. adolescents receiving less than 75% of the RDA for selected nutrients, by category of supplement use (12)

(n=423)	Nonuse (n=280)	Less-frequent (n=77)	Daily use (n=66)
Vitamin B_6	37.5	31.2	21.2
Folic acid	27.1	18.2	16.7
Vitamin A	55.0	42.9	36.4
Vitamin C	35.0	23.4	16.7
Vitamin E	55.0	42.9	36.4
Calcium	62.9	53.2	47.0
Iron	28.6	32.5	25.8
Zinc	48.9	49.4	51.5

Adolescents who used vitamin/mineral supplements even occasionally had healthier diets than those who do not use supplements. Non-supplement users had a greater proportion of energy intake from total fat and saturated fat and less from carbohydrates than daily supplement users (12). Among 1532 eight graders from the Child and Adolescent Trial for Cardiovascular Health (CATCH) study, higher micronutrient intakes from foods were reported among vitamin/mineral supplement users than non-supplement users (13).

Factors influencing eating behaviors

Various sociodemographic characteristics and psychosocial factors have been associated with inadequate food intake patterns and overweight status among 7th through 12th grade adolescents in the Minnesota Adolescent Health Survey (Table 3) (14). The majority (86%) of the participants were white while 8% were black/non-Hispanic. Socioeconomic status (SES), determined by parental education and employment status, was grouped into three levels: 14% were low, 56% medium, and 30% high. Low socioeconomic status (SES) was associated with low fruit, vegetable, and dairy intake and overweight status. The relationship between SES and overweight status was stronger for females than males. While sex was not associated with inadequate fruit or vegetable intake, being female was associated with inadequate dairy intake. As expected, weight dissatisfaction was associated with inadequate fruit, vegetable, and dairy intake as well as overweight status.

Table 3. Key findings from the Minnesota Adolescent Health Survey regarding food intake patterns and overweight status (n=36,284) (14).

Food Intake Patterns and overweight status	Associations with sociodemographic characteristics	Associations with psychological factors and weight concerns	Patterns of covariation with other behaviors
Low fruit and vegetable intake: 28% and 36%, respectively	• Low SES [a] • Black/non-Hispanic	• Weight dissatisfaction • Poor school achievement • Low family connectedness	• Modest assoc. with binge eating, substance use, & suicide attempts
Low dairy intake: 7% M [b], 13% F[c]	• F • Non-white • Low SES	• Weight dissatisfaction • Poor school achievement • Low family connectedness	• Strongly assoc. with chronic dieting • Modest assoc. with binge eating, substance use, & suicide attempts
Overweight status >85th percentile: 18% M, 12% F >95th percentile: 4% M, 3% F.	• Low SES (stronger association for F than M)	• Weak association with global psychological concerns • Strong assoc. with weight-specific concerns	• More frequent dieting and binge eating behaviors

[1] Male; [2] Female; [3] Socioeconomic status

Factors influencing food choices among adolescents were identified through focus group discussions (15). The most important factors identified were hunger/food cravings, appeal or taste of the food, time, and convenience. Other factors mentioned less frequently include: food availability, parental influence, perceived benefits, situational factors, mood, body image, habit, cost, media, and vegetarianism. Perceived barriers to healthful eating identified during these focus group discussions included: 1) lack of priority; 2) fruits, vegetables, and dairy products are less appealing than other options; 3) healthy options are not promoted or as appealing than other options when eating out; 4) fruits and vegetables are less convenient than other foods; and 5) healthful foods are more expensive. Of these listed, the first two were identified as the most important barriers since they were discussed more frequently or with greater intensity.

Adolescents, in general, have poor dietary quality as evidenced by inadequate micronutrient intake and excessive intake of fat and saturated fat. To better understand how eating behaviors impact weight status and other health conditions associated with poor diet quality, an understanding of growth and body composition changes that occur during adolescence is necessary.

Adolescent growth and body composition changes

Growth during adolescence is related to both growth hormones and sex hormones, testosterone in males and estrogen and progesterone in females. Several physiological changes occur during adolescence. Adolescents gain about 20% of their adult height and 50% of their weight during this period (1). The adolescent growth spurt is a period of rapid growth usually lasting 18-24 months. Peak height velocity (PHV) is the fastest rate of growth during the growth spurt and occurs at approximately 12 and 14.5 years for girls and boys, respectively.

Sexual maturation, growth, and body composition changes during puberty

Since adolescents begin puberty at different ages and mature at different rates, accounting for sexual maturation when assessing physical growth is important. Sexual maturity ratings (SMR), also called Tanner stages, have been widely used when evaluating growth during adolescence (16). Males have three components in the SMR: the size of the testes, the length of the penis, and the development of pubic hair; females have two components: breast and pubic hair development. Stage 1 is associated with early sexual development while stage 5 is related to completion of the sexual maturation process. The growth spurt occurs typically two years earlier in females than males (17). In males, the growth spurt occurs relatively late in relationship to the development of genitalia. In females, menarche usually occurs 1.3 years after PHV. Most females gain no more than 2-3 inches in height following menarche.

As the body matures, changes in body composition occur as well. In prepubertal period, the adipose and muscle composition between males and females tend to be fairly similar with body fat averaging about 15% and 19%, respectively (1). During puberty, females gain more adipose tissue than males while males gain twice as much lean tissue as females. In adulthood, females have about 22% to 26% body fat, compared to 15% to 18% in males. The rate of maturation affects body composition. A faster maturing child has significantly larger total body fat, percent body fat, and fat-free mass than a slower-maturing child at the same age (18).

Overweight in adolescents and later consequences in adulthood

Body mass index (BMI) has become widely accepted as a screening tool for obesity in the adult population and recently BMI-for age growth charts have been developed by the Centers for Disease Control and Prevention (CDC) (19) for use in assessing weight status in children and adolescents from 2 to 20 years old (Appendix A). Recent discussions about how to describe excessive body weight among adolescents have lead to new terminology. "At risk of overweight" (85th to <95th percentile BMI-for-age) and "overweight" (≥95th percentile BMI-for-age) are the preferred terms due to the negative connotations associated with the term "obesity" (20). Additional information on classification of adolescent overweight will be discussed in a later section.

The increase in overweight and obesity has become a growing public health concern not only among adults but also among adolescents. Results from the 1999 National Health and Nutrition Examination Survey (NHANES IV) revealed approximately 61% of adults over age 20 were overweight (BMI ≥25) or obese (BMI ≥30), a 5% increase from NHANES III (1988-1994); 14% of adolescents ages 12-19 years were overweight, about a 3% increase from NHANES III and a 9% increase from earlier surveys (1963-1970) (21,22). Results from NHANES III demonstrated that obesity (BMI ≥30) was highest among ethnic minorities; 38% of black/non-Hispanic and 35% of Hispanic women were obese compared to 24% of white women (10). The effect of ethnicity appears to be less pronounced among adolescents; 13-14% of Hispanics and black/non-Hispanics were overweight (≥95th BMI-for-age percentile) compared to 11% of white adolescents (10). The revised growth charts were developed for use with all children and adolescents in the United States regardless of their race or ethnicity based on the rational that "the most important influences on growth potential appear to be economic, nutritional, and environmental" (20).

Physiological and psychosocial consequences of overweight

Obesity in adults has been associated with numerous adverse health consequences. Several cardiovascular risk factors have been associated with obesity including glucose intolerance and diabetes, hypertension, and dyslipidemia (23,24). Obesity has also been associated with increased risk of gallbladder disease, especially in women, osteoarthritis, and breast, prostrate, and colon cancers. In addition, upper gastrointestinal problems, such as

gastroesophageal reflux and hiatal hernia, and sleep disorders, such as sleep apnea, have been associated with adult obesity (2,23,24). Among adults, a U-shaped relationship has been described between BMI and mortality with increased mortality seen with low BMI (<20 kg/m^2) as well as high BMI (>30 kg/m^2) (24). While health consequences associated with obesity are commonly seen in adults, there are an increasing number of overweight-related problems seen also in adolescents. These increased health complications during adolescence are precursors to adult disease (2). Numerous conditions, such as hypertension, hepatic steatosis, sleep apnea, cholelithiasis and orthopedic problems, have been documented among obese adolescents. Those conditions pertinent to this project will be discussed.

Hyperlipidemia

Abnormal blood lipid concentrations, namely elevated serum low-density lipoprotein (LDL)-cholesterol and triacyl-glycerol (TAG) (also referred to as triglycerides) and lowered high-density lipoprotein (HDL)-cholesterol, have been found in overweight adolescents (2). These patterns are similar to those commonly seen in adults. Hyperlipidemia has been related to central fat distribution. Increased free fatty acids produced by increased lipolysis relating to visceral adipocytes and hyperinsulinemia, promoting increased TAG and LDL cholesterol synthesis (25-28).

Glucose intolerance and type 2 diabetes

Impaired glucose tolerance has been reported in 25% of obese children (4-10 y) and 21% of obese adolescents (11-18 y) with type 2 diabetes identified in 4% of the obese adolescents (29). After adjusting for BMI, insulin resistance was the best predictor of impaired glucose intolerance. As with hyperlipidemia, the mechanism for development of type 2 diabetes among adolescents is similar to that with adults. Visceral fat is positively associated with basal insulin secretion, stimulated insulin secretion, and insulin resistance (27).

Psychosocial consequences

In the U.S. culture, strong messages encouraging thinness, especially among women prevail. Negative attitudes resulting in social stigmatization towards obese individuals often translate into discrimination in employment opportunities, college acceptance, job earnings, rental availabilities, and opportunities for marriage (2,24). These societal pressures can

result in a preoccupation with weight and the development of eating disorders and other unhealthy eating patterns. Binge eating disorder, characterized by eating larger amounts of food with a lack of control during these episodes (30). Most obese binge eaters do not engage in purging behaviors. In addition to being more susceptible to binge eating disorder, obese individuals are more likely to have other psychological illnesses such as depression (24). Binge eating has been associated with higher prevalence of depression and anxiety (31).

As mentioned earlier, more black/non-Hispanic women are overweight and obese than white/non-Hispanic women. Ethnic differences in body image and self-esteem have been studied. Among college female students, body image and self-esteem were more positive among black/non-Hispanic than white/non-Hispanic women (32). Among overweight adolescent females, both black/non-Hispanic and white/non-Hispanic females expressed a desire to be thinner, felt dissatisfied with their body shape and size, and felt self-conscious about their weight; however, black/non-Hispanic adolescents were more likely to discuss the positive aspects of their bodies than white/non-Hispanics (33). This ethnic difference in body image may relate to differences in cultural perceptions and social stigmatizations of obesity (34). Black/non-Hispanic women described themselves with more masculine or androgynous traits and believed that black/non-Hispanic men prefer larger women (32). Other cultural influences have been related to positive body image seen among black/non-Hispanic women (34).

Adolescent BMI as a predictor of adult BMI

Investigators have studied the relationship between adolescent BMI and adult BMI (4-8). Must et al. (4) studied overweight adolescents (13-18 y) who participated in the Harvard Growth Study from 1922 to1935 with subsequent morbidity and mortality. Of the surviving subjects who were overweight (>75th percentile), 52% were overweight as adults in 1988. Being overweight as an adolescent was a more powerful predictor of adulthood morbidity and mortality than adulthood overweight.

Scrinivasan et al. (5) examined the impact of adolescent overweight on adult overweight and related cardiovascular risk factors in a biracial cohort from the Bogalusa Heart Study. The risk of overweight adolescents (>75th percentile) remaining overweight as

adults was 58%; black males had the lowest risk (52%) and black females had the highest risk (62%). As young adults, the overweight adolescent cohort had increased abdominal fat distribution, hypertension, hyperlipidemia, hyperglycemia, and hyperinsulinemia compared to the lean adolescent cohort (25^{th} to 75^{th} percentile).

Whitaker et al. (6) studied childhood and adolescent obesity (1-17 y) and parental obesity as a predictor of obesity during young adulthood (21-29 y). Of those who were obese during childhood, the chance of adulthood obesity was lowest (8%) for those aged 1-2 y without obese parents and highest (79%) for those aged 10 -14 y with at least one obese parent. After adjusting for parental obesity, the odds ratio for adulthood obesity was greatest for overweight children aged 10-14 y (22.3, 95% CI: 10.5-47.1) and those aged 15-17 y (17.5, 95% CI: 7.7-39.5).

Guo and Chumlea (7) investigated the predictive value of childhood BMI for overweight adults, defined as BMI >28 and >26 for men and women, respectively. Data from 555 white children indicated that overweight at age 35 y could be predicted from BMI at younger ages. The strongest prediction was BMI at 18 y followed by BMI at 13 y. BMI at ages <13 y was only a moderate predictor of adult BMI. For 18 year olds with BMI >60^{th} percentile, the probability of being overweight at age 35 y was 34% for men and 37% for women.

Guo et al. (8) further examined the BMI-adult overweight relationship with data from the Fels Longitudinal Study. BMI parameters for early childhood, pubescence, and post-pubescence (age 2 to 25 y) were compared to adult BMI values at 35 to 45 y. The BMI rebound period (early childhood) was a significant predictor of adult overweight in females but not in males. BMI patterns during and post adolescence were more important than BMI rebound for adult total body fat and percent body fat. The BMI at approximately 20 y was a strong predictor of adult BMI. In addition, the pattern of BMI changes from 2 to 25 y had stronger effects on subsequent adult overweight than birth weight and adult lifestyle factors.

Lifestyle habits among adolescents including eating behaviors and physical activity can have lasting health implications. Overweight adolescents are more likely to be overweight as adults as well as develop immediate and long-term health consequences

associated with increased weight. In this study, adolescents and young adults had an additional health concern related to HIV infection.

HIV/AIDS

Definition of disease

Acquired Immune Deficiency Syndrome (AIDS) is characterized by a decreased immune function and opportunistic infections caused by the human immunodeficiency virus (HIV) (35). HIV binds to the CD4+ T-lymphocyte cell surface receptor. The viral DNA is integrated into the host cell's DNA. As a retrovirus, HIV replicates using the enzyme reverse transcriptase to copy RNA into DNA. The disease is primarily characterized by a gradual deterioration in immune function due to the destruction of CD4+ T-cells resulting in immunodeficiency, neurological complications, wasting, opportunistic infections, and neoplasms (35).

Prevalence of HIV infection in the U.S.

The number of AIDS cases reported in the 1980s to the CDC dramatically increased after the first cases of AIDS were reported in the U.S. in 1981 (36). In 1993, reported cases peaked corresponding to the expansion of the CDC classification to include CD4+ T-cells (37). The most dramatic decline in reported cases and deaths has been since 1996, corresponding to the increased use of antiretroviral therapy (ART). As of December 2000, 774,467 cases of AIDS have been reported in the U.S. of which 58% have died with AIDS (37). Since people with AIDS are surviving longer, the number of people with HIV infection or AIDS has steadily increased. Through the end of 2000, 450,151 cases of individuals living with HIV infection or AIDS had been reported to the CDC. However, the CDC has estimated that 800,000 to 900,000 individuals in the U.S. in 1999 were living with HIV infection or AIDS. This discrepancy may be due to the fact that reporting of those diagnosed with HIV infection has not been implemented in all states and territories, anonymous tests are excluded from case reports, and many individuals are unaware of their HIV status.

The prevalence of HIV infection is growing among the adolescent and young adult population in the U.S. Of the 31,293 AIDS cases reported in adolescents and young adults under age 25 y through December 2000, approximately 70% were male and 65% were

black/non-Hispanic or Hispanic. More females (78%) than males (59%) were from a minority background; about 60% of the females were black/non-Hispanic (36).

Classification system for HIV infection

To account for the relationship between decreased CD4+ T-cells and impaired immune function, the CDC revised the classification system for HIV infection to include CD4+ T-cells in addition to clinical conditions associated with HIV infection (37). As the CD4+ T-cell count decreases, the risk and severity of opportunistic illnesses increase. Three ranges of CD4+ T-cells have been defined: ≥500, 200-499, and <200 cells/μL. Three clinical categories were defined as asymptomatic HIV infection, symptomatic HIV infection, and AIDS-indicator conditions. A detailed description of the classification system has been provided (Appendix B).

The first stage of HIV infection, called acute HIV infection, occurs 4-7 weeks after primary infection when there is rapid viral replication. Symptoms seen during this period include fever, malaise, lymphademopathy syndrome (swollen lymph nodes), pharyngitis, headache, myalgia (widespread muscle pain), and occasionally a rash (38). The first stage of infection is not included in the CDC classification system since individuals may not test positive for HIV infection yet. The time between initial infection and seroconversion, or the development of HIV antibodies, varies from one week to several months (39). Once HIV antibodies appear in the blood, an individual will test positive for HIV infection.

During asymptomatic HIV infection, few if any noticeable symptoms occur. While the progression of HIV infection has large individual variability based on treatment as well as health-related factors, this stage averages about eight years (40). Symptomatic HIV infection occurs when symptoms such as fevers, sweats, skin problems, fatigue, or other symptoms that are not considered AIDS defining conditions (Category B in Appendix B).

The term AIDS is used for individuals who have at least one well-defined life-threatening condition linked to immunosuppression. Individuals with CD4+ T-cells <200 cells/μL are classified with AIDS. A detailed list of AIDS-defining conditions is listed in Category C (Appendix B). Classic conditions include AIDS wasting syndrome (AWS), HIV encephalopathy, HIV nephropathy, and AIDS enteropathy. AWS is defined as involuntary weight loss of >10% plus chronic diarrhea or chronic weakness and fever for >30 days

(CDC, 1993). A 5% weight loss over four months has been associated with an increased risk of opportunistic infections and death (Odds ratio: 2.22, $p<0.001$) (41). AIDS enteropathy has been defined as chronic diarrhea in absence of identifiable enteric pathogens. Kotler (42) has suggested that intestinal injury is related to complications from other conditions rather than immunodeficiency related to HIV infection.

Nutrition and HIV infection

The relationship between nutritional status and immune function has been studied for decades. It is well known that infectious illnesses influence nutritional status, which increases susceptibility to additional infection (43,44). Many immune function changes seen in AIDS are similar to those of protein-energy malnutrition (PEM) (45). With PEM, the body lacks the necessary protein synthesis required for every immune function. Without new protein synthesis, the body draws upon lean tissue stores to meet the basic energy and protein needs for survival. Immune changes similar to both HIV infection and PEM include decreased CD4+ T-cells, reduced immunoglobulin A (IgA), and impaired primary and secondary delayed cutaneous hypersensitivity responses.

Antioxidants and oxidative stress

Highly reactive oxidative species (ROS) are produced during normal metabolic processes. Some common ROS include singlet oxygen, superoxide radical anion, hydroxyl radical, hydrogen peroxide and lipid peroxide (46). Most cells have enzymes or other molecules with antioxidant properties that detoxify the ROS or free radicals in the body. Oxidative stress can result from decreased intake of antioxidants, excessive production of free radicals, or a combination of both (47). The immune system is one of the main sources of oxidative stress in the body (48). Both neurophils and activated macrophages release hydrogen peroxide and other ROS as part of their immune fighting properties.

In HIV infection, the oxidative stress cycle may be activated by depletion of serum antioxidants or through overproduction of free radicals (Figure 1) (49). In addition, increased oxidative stress has been associated with increased disease progression. Oxidative stress leads to an impaired immune function and increased apoptosis (cell death) resulting in destruction of CD4+ T-cells. In addition, oxidative stress has been related to increased HIV replication. Antiretroviral therapy (ART) has been associated with decreased oxidative stress

by decreasing viral load and HIV progression. Several mechanisms have been suggested in relationship to antioxidant depletion in HIV infection: 1) low dietary intake as a result of decreased appetite, 2) changes in intestinal mucosa resulting in malabsorption, excessive production of free radicals, and 2) loss of electron-transport capacity resulting in decreased recovery of reduced forms of antioxidants (49). Some of the micronutrients important to immune function and HIV infection will be discussed briefly.

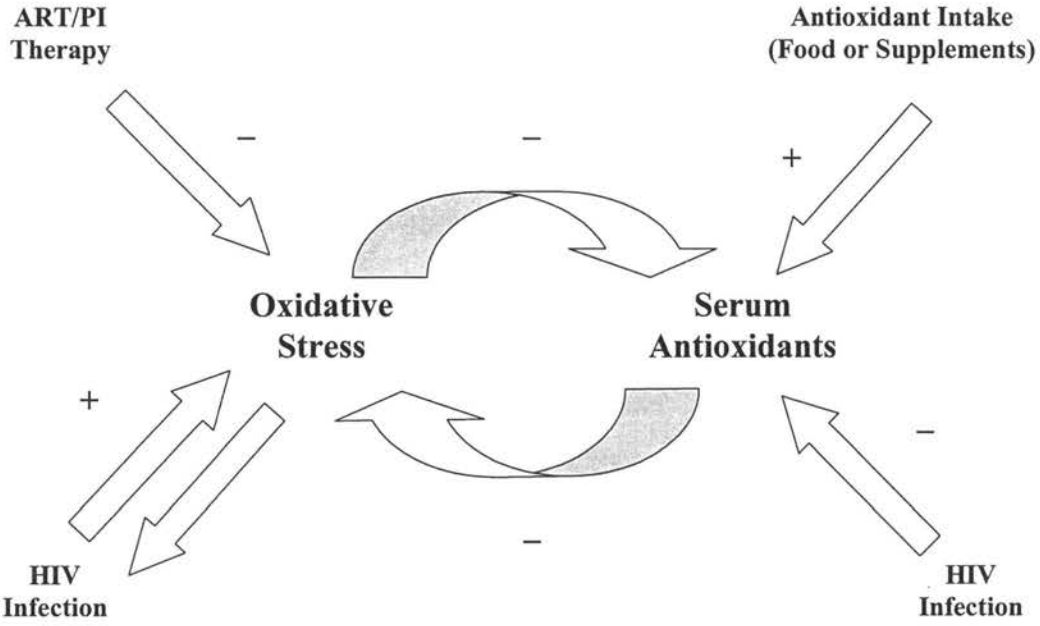

Figure 1. Mechanisms of oxidative stress in HIV infection.
Adapted from: Tang and Smith (49).

Vitamin A and carotenoids

Vitamin A affects many aspects of the immune function. Retinoids are involved in the immune cell differentiation and proliferation in response to immune stimuli. Vitamin A deficiency has been associated with reduced leukocytes, impaired T-cell function, and decreased growth, differentiation, and activation of B-lymphocytes (50). Carotenoids have been shown to enhance immune cell activity via their pro-vitamin A activity as well as their antioxidant characteristics. β-carotene has been associated with increased total white blood cells and T-cells in HIV-infected individuals (51). Intakes of vitamin A and beta-carotene during early HIV infection had a U-shaped relationship with risk of developing AIDS; both

deficiencies and excessive intakes had a subsequent negative association with immune function and disease progression (52,53).

Vitamin E

Vitamin E, specifically α-tocopherol, is a potent fat-soluble antioxidant blocking the chain reaction of lipid peroxidation by scavenging intermediate peroxyl radicals (54). Vitamin E is also important for normal immune function particularly with T-lymphocytes and natural killer cell activity. Vitamin E supplementation has been associated with increased resistance to infections among the elderly (50). Vitamin E intake during early HIV infection has also been associated with slower HIV progression (52,53,55,56).

Vitamin C

Vitamin C, or ascorbic acid, is another well-known antioxidant that works as a scavenger of free radicals, especially environmental pollutants of the respiratory system. Vitamin C assists vitamin E in lipid peroxidation by recycling the α-tocopherol free radicals (50). Vitamin C has been shown to improve immune function. The high intracellular content of vitamin C in leukocytes provides cellular protection against oxidative damage associated with respiratory burst. While vitamin C has known antioxidant properties, in the presence of excessive iron or copper, vitamin C can act as a prooxidant generating free radicals (47). Vitamin C intake during early HIV infection has also been positively associated with CD4+ T-cells and survival after a 6-8 y follow-up period (52,53,55).

Iron

The functions of iron relate to its ability to participate in oxidation and reduction reaction (57). Iron is highly reactive and can react with oxygen to form intermediates that can cause cellular or DNA damage. To prevent these oxidative effects, iron is tightly bound to proteins. Iron deficiency has been associated with reduced T-cell numbers, antibody production, as well as other immune changes (50). The role of iron in immune function relates to its involvement with folate metabolism, mitochondrial energy production, respiratory burst, and metalloenzymes. Iron intake during early HIV infection has also been positively associated with CD4+ T-cells and survival after a 6-8 y follow-up period (52,53,55).

Zinc

Adequate zinc status is essential for many immune functions including division, maturation and differentiation of T-cells, lymphocyte response to mitogens, gene transcription, and biomembrane function (58). Zinc-dependent thymulin is essential for the formation of T-lymphocytes. Copper-zinc superoxide dismutase (Cu-Zn SOD) is important in the antioxidant defense system. Zinc deficiency may compromise the antioxidant response to oxidative stress from free radicals and lipid peroxides, which has been shown to stimulate replication of HIV virus (59).

Individuals with AIDS have exhibited symptoms of zinc deficiency including immune deficiencies, impaired taste and appetite, decreased food intake, diarrhea, hair loss, skin lesions, hypogonadism and hypospermia (58,60). Thus zinc deficiency may be a compounding factor in maintaining adequate oral intake. While zinc deficiency is known to decrease immune function (43,61), excessive intake of zinc (300 mg/day) has been associated with impaired immune function among healthy adult men (62). Low plasma zinc concentrations have been associated with a three-fold increase in risk of HIV-related death in HIV-infected drug users (63). Among HIV-infected men, increased zinc intake was associated with increased disease progression and death (52,53). This suggests a similar U-shaped relationship between zinc intake and immune function, which was described earlier with vitamin A.

No general consensus has been reached about the use of zinc supplementation in HIV-infected individuals (64). Short-term zinc supplementation of one month has been shown to be beneficial in reduction, prevention or elimination of certain opportunistic infections in HIV-infected individuals (65). Supplementation may be helpful to prevent disease progression and opportunistic infections in those individuals with low dietary intake (64). However, due to the adverse effects of excessive zinc on the immune function, supplementation should be used with caution. Additional research is needed to determine the mechanisms and interactions of zinc on the immune systems of HIV-infected individuals.

Dietary intake studies and HIV infection

Numerous studies have been looked at the association between dietary intake and HIV infection (Table 4). Intakes of thiamin, riboflavin, niacin, vitamins A, B_6, C, and E, and iron during early HIV infection have been positively associated with CD4+ T-cells and survival after a 6-8 y follow-up period (52,53,55). Other studies have found a positive association between macro- and micronutrient intakes and CD4+ T-cells (66,67).

Many of the above mentioned studies had micronutrient intakes at or above the RDA for the majority of nutrients with intake for some micronutrients far exceeding the RDA. The prevalence of deficiency, compared to either the 1989 RDAs or the new DRIs, was low for the majority of subjects in these studies. Differences in macro- and micronutrient intakes have also been seen between HIV-infected and HIV-uninfected individuals (Table 5). The majority of these studies reported higher dietary intakes for HIV-infected compared to HIV-uninfected individuals (68,69,70,71,72). A similar trend was seen by Sharkey et al. (66); however, did no reach significance.

Differences in serum micronutrient concentrations have also been seen between HIV-infected and HIV-uninfected individuals (68,69). While intakes at the RDA were associated with normal serum micronutrient concentrations in HIV-uninfected participants, HIV-infected participants required intakes much greater than the RDA to achieve normal serum concentrations (69). These discrepancies between dietary intake and serum concentrations among HIV-infected individuals may be related to a variety of mechanisms including altered intestinal absorption, increased needs related to HIV infection and altered immune function, altered utilization related to metabolic changes, or a combination of factors (73,74).

Vitamin/mineral supplementation is common among HIV-infected individuals (55,67,69,75,76). Among the above studies, 46% to 71% of the individuals used vitamin/mineral supplements. Micronutrient intake from food alone was higher among supplement users compared to non-supplement users (55,69). This was consistent with dietary intake studies on the general population of U.S. adolescents (12,13). This suggests that individuals who take supplements make more micronutrient-dense food choices compared to those who do not take supplements.

Table 4. Studies comparing dietary intake and HIV disease progression

Study	Sample (n)	Dietary intake	Micronutrient intake/ Prevalence of deficiency	Relationship to disease progression
Sharkey et al. 1992 (66)	Homosexual M [a] HIV+ (n=28) HIV- (n=8)	7-day food record	No significant differences by HIV status in any nutrient intakes	Positive relationship between energy intake, weight, & CD4+ T-cells
Abrams et al. 1993 (55)	San Francisco Men's Health Study (n=296) Homo/bisexual HIV+ M (25-50 y)	HHHQ Current supplement use was reported	**Food intake:** *Supplement users > non-supplement users *Intakes > RDA (except zinc, thiamin, & vitamin E) **Food & supplement intake:** Intakes > RDA (except for zinc) *Lowest quartile of most nutrients > RDA (Iron: 200% RDA, vitamin E: 300% RDA, vitamin C: 400% RDA)	*36% (107) incidence of AIDS in 6 y period *Protective relationship against developing AIDS with iron, vitamin E, Riboflavin, ($p<0.05$) vitamin C, thiamin ($p<0.10$) vitamin A ($p<0.12$). *Supplement use associated with 31% ↓ in AIDS risk
Tang et al. 1993 (52)	Multicenter AIDS Cohort study (n=281) Homo/bisexual HIV+ M (>18 y)	Willett FFQ Current supplement use was reported HIV status not known at time of FFQ	**Food Intake:** ≥ RDA except for thiamin, vitamin E, & zinc **Food & supplement intake:** ≥ RDA for all nutrients Vitamin A: >75% of sample consumed >180% RDA & 25% consumed >400% RDA Vitamin C: >75% consumed >250% of RDA for C Zinc: >50% consumed < RDA for zinc & ~25% consumed 130% RDA	*38% developed AIDS during next 6.8 y. *Highest quartiles of vitamin C, niacin, thiamin associated with decreased risk of AIDS; tendencies seen with riboflavin & vitamin B_6. *The 2nd & 3rd quartile of vitamin A associated with ↓ progression to AIDS *↑ zinc associated with ↑ progression to AIDS
Tang et al. 1996 (53) 8 y follow-up from Tang et al. 1993 (52)	See Tang et al. 1993 (52)	See Tang et al. 1993 (52)	See Tang et al. 1993 Thiamin, vitamins B6 & E, & zinc: >20% had intakes < RDA. 50% of subjects had zinc intake < RDA	*58% survived 8 y *91.6% of deaths due to AIDS *Highest quartile of thiamin, riboflavin, niacin, & vitamin B_6 associated with ↑ survival *U-shaped relationship with β-carotene & vitamin A
Woods et al. 2002 (67)	HIV+ cohort M (n=386), F (n=130) Mean age 40 y	3-day food records Current supplement use was reported	*Intake >NHANES III *F: 21-64% consumed <75% DRI for vitamins A, B_6, C, & E, iron, & zinc *M: 26-40% intakes <75% DRI for vitamins A & E, & zinc	*M: CD4+ T-cells ≤500 associated with ↑ intakes for all micronutrients except zinc

[a] Age not available; Abbreviations: DRI=Dietary Reference Intake, F=Female, FFQ=Food frequency questionnaire, HHHQ=Health Habits and History Questionnaire, M=Male, NHANES III=Third National Health and Nutrition Examination Survey, RDA=Recommended Daily Allowances

Table 5. Studies comparing dietary intake between HIV-infected (HIV+) and HIV-uninfected (HIV-) U.S. adults

Study	Sample	Dietary intake	Micronutrient intake/ prevalence of deficiency	Relationship to serum concentration
Sharkey et al. 1992 (66)	Homosexual M [a] HIV+ (n=28) HIV- (n=9)	7-day weighed food record	No differences by HIV status in any nutrient intakes	NA
Beach et al. 1992 (68)	Homosexual M (20-55 y) HIV+ (n=100); HIV- (n=42)	Willett FFQ Current supplement use was reported	*Mean intakes > RDA for all subjects *Intakes for HIV+ > HIV- *Percent < RDA: HIV+: Vit E (10%), Zinc (21%) HIV-: Riboflavin & vitamin B_6 (>15%), zinc (48%), vitamin E (26%) folate (35%)	Vitamin A, riboflavin, copper serum values ↓ for HIV+ vs. HIV- after adjusting for intake.
Baum et al. 1994 (69)	Homosexual M HIV+ (n=88) HIV- (n= 108) (20-55 y)	Willett FFQ Current supplement use was reported	*Mean intakes HIV+ > HIV- *Supplement use greater in HIV+ *Majority of all participants consumed at least the RDA for most essential nutrients	*HIV-: Intake of RDA associated with normal serum concentration *HIV+ required intakes >RDA to achieve normal plasma concentration
Smit et al. 1996 (70)	IV drug abusers: HIV+ (n=45) HIV- (n=59) 36% F (Mean age 40 y)	FFQ & 24-hour recall	*Energy, total fat, saturated fat, vitamins B_6 & B_{12}, panothenic acid, phosphorus, & selenium intake: HIV+ > HIV- *Vitamins A & E, calcium, & zinc > RDA for both groups	NA
Hogg et al. 1996 (71)	Homosexual M HIV+ (n=139) HIV- (n=145) (24-64 y)	24-hour recall	Energy, protein, carbohydrates & cholesterol intake: HIV+ > HIV- (micronutrient intake-NA)	NA
McDermid et al. 2002 (72)	HIV+ (n=58) HIV- (n=31) M & F (Mean age 27 y)	24 hour recall & modified FFQ	M: Vitamin C & E intakes: HIV+ > HIV-	NA

[a] Age not available; Abbrevations: F=Female, FFQ=Food frequency questionnaire, IV=Intravenous, M= Male, NA=Not available, RDA=Recommended Daily Allowances

Despite different dietary intake methods, findings from the above studies (Table 4, 5) were relatively consistent. These dietary intake studies included primarily homosexual adult men. More recently, studies have included more minority groups and women (67,70). However, little is known about the dietary intake patterns of adolescent and young adults with HIV infection.

Metabolic and body composition changes related to HIV and drug therapy

Wasting

Wasting was a common occurrence with HIV-infection. Even in early stages of HIV-infection, depletion of lean body mass has been seen among HIV-infected adults without a significant change in BMI or body weight (77). Timing of death in persons with AIDS has been predicted as body cell mass reaches 55% of normal weight or when body weight nears 66% of ideal body weight (78). While the exact mechanism of wasting among HIV-infected individuals is unknown, some potential mechanisms for weight loss and decreased lean body mass are inadequate intake, gastrointestinal malabsorption, and abnormal energy utilization (79).

Inflammation and ulcers of the mouth, esophagus, and stomach can lead to decreased intake and anorexia. Pancreatic and biliary tract disease can result in vomiting and abdominal pain. HIV encephalopathy can affect the ability to consume adequate energy due to decline in physical coordination and mental function (80). Furthermore, medications commonly used among HIV-infected individuals can result in gastrointestinal side effects resulting in decreased intake (79). Malabsorption is prevalent among advanced HIV infection regardless of other opportunistic pathogens (81). Small intestine dysfunction has been identified by abnormal D-xylose absorption tests, Schilling tests, and by presence of steatorrhea reflecting malabsorption of carbohydrates, vitamin B_{12}, and lipids, respectively (81).

Increased energy metabolism has been reported among HIV-infected individuals (82,83). Increased energy expenditure may be due in part to increased cytokine production. Cytokine production has been associated with "futile cycling" where fatty acids are transported from adipose tissue to the liver and then back to adipose tissue resulting in greater energy requirements to meet energy demands (79).

Antiretroviral therapies

Since some of the metabolic and body composition changes have been associated with antiretroviral therapy (ART), the three main categories of ART currently used for treatment will be briefly discussed: 1) Nucleoside Reverse Transcriptase Inhibitors (NRTI) and Nucleotide reverse transcriptase inhibitors (NtRTI), 2) Protease inhibitors (PI), and 3)

Nonnucleoside reverse transcriptase inhibitors (NNRTI) (84). Often these medications are given in combination of three or more different drugs referred to as highly active antiretroviral therapy (HAART) (39). NRTIs and NtRTIs inhibit the replication of HIV through incorporation into the DNA by viral reverse transcriptase and thus terminate the DNA chain (85). PIs inhibit the HIV protease and prevent the maturation of the virus. NRTIs bind directly to the reverse transcriptase and block RNA and DNA causing a disruption of the enzymes site.

Patients with advanced HIV disease clearly benefit from ART, which slows the progression of disease, improves survival rate, and reverses some opportunistic infections (86). While use of PIs has improved the morbidity and mortality of HIV infection, several metabolic side effects have been identified, such as include insulin resistance, lipodystrophy or fat redistribution, and hyperlipidemia (86-89). These complications are similar to those associated with the metabolic syndrome known as Syndrome X (90). While the majority of the metabolic and body composition changes have been associated with ART, they have also been seen among HIV-infected individuals not receiving ART (91-93).

Insulin resistance

Insulin resistance occurs at the cellular level preventing insulin from exerting its effect on glucose metabolism (35). Insulin resistance has been associated with development of glucose intolerance, type 2 diabetes mellitus, and cardiovascular disease (94,95). Individuals with diabetes have increased risk of developing a number of long-term complications, which include coronary heart disease, peripheral vascular disease, cerebral vascular disease, dyslipidemia, hypertension, retinopathy, nephropathy and neuropathy. Insulin resistance has been seen with ART in HIV-infected individuals (96,97). PI-induced insulin resistance can develop in as soon as four weeks after starting therapy and often precedes lipodystrophy (discussed below) (87). Although insulin resistance has been associated with PI therapy, hyperglycemia is relatively rare since most patients may be able to compensate for insulin resistance by increasing insulin secretion to maintain adequate glucose levels (86).

A possible mechanism for PI-induced insulin resistance is the inhibition of the insulin-responsive glucose transporter (GLUT-4) (98). Insulin signaling to the intracellular

GLUT-4 causes plasma membrane translocation. GLUT-4 translocation accounts for the majority of the glucose uptake stimulated by insulin in fat and muscles cells (99). Murata et al. (98) studied the effect of PI therapy on insulin resistance in frogs. PI therapy inhibited GLUT-4 activity by 45% compared to frogs not receiving PI therapy. They speculated that PI inhibited the transport function of GLUT-4 resulting in increased insulin resistance seen in HIV individuals. Further, the PI effect on GLUT-4 may account for the observed lipodystrophy in addition to the insulin resistance since GLUT-4 activity is involved in *de novo* peripheral adipogenesis primarily from glucose while abdominal adipocytes may obtain lipids primarily from circulating triglycerides.

Lipodystrophy and fat redistribution

Clinical features of lipodystrophy, or fat redistribution syndrome, include peripheral fat loss especially in the face, limbs, and buttocks with a central fat accumulation in the abdominal region, breasts, and over the dorsocervical vertebrate (referred to as the "buffalo hump") (39,86). Peripheral wasting and/or central adiposity have been noted among patients receiving PI. (89,91,96,97).

Carr et al. (100) looked at the occurrence of lipodystrophy in 116 HIV-infected, otherwise healthy patients receiving at least one PI, 32 HIV-infected PI naïve patients, and 47 healthy male control subjects. Lipodystrophy was observed in 64% of the HIV-infected taking PI for an average of 13.9 months but in only one HIV-infected PI naïve individual ($p=0.0001$). The median time for onset of lipodystrophy was 10 months. Patients with PI-induced lipodystrophy had significantly longer duration of PI therapy than those without lipodystrophy (15.2 and 10.9 months, respectively; $p=0.0001$). Higher triglyceride, total cholesterol, insulin, and C-peptide levels were also seen in patients with lipodystrophy compared to those without lipodystrophy.

Hyperlipidemia

While increased triglyerides have been seen in HIV-infected patients not taking ART (91,101,102), an increase in dyslipidemia has been observed in patients on ART (103,104). Hypertriglyceridemia has been seen most frequently although increased very low density lipoprotein (VLDL) and low density lipoproteins (LDL) has been noted (104). Henry et al.

(103) have identified elevated lipid concentrations in 33% of the 124 HIV-infected patients on PI.

While the exact mechanism of development of hyperlipidemia has not been determined, several possible mechanisms have been proposed relating to: 1) apoprotein B (Apo B), 2) cytoplasmic retinoic acid binding protein 1 (CRABP-1), and 3) mitochondrial toxicity. Hyperlipidemia could be related to synthesis and secretion of apoprotein B (Apo B), which is found in low-density lipoprotein (LDL), very-low-density lipoprotein (VLDL) and intermediate-density lipoprotein (IDL) cholesterol. In the presence of lipids, Apo B increases VLDL assembly and secretion, while in the absence of lipids the protein is degraded and no VLDL production results (105). The rate of apo B synthesis was doubled in HIV-infected individuals with ART compared to healthy HIV-uninfected individuals (104). PI may interfere with the degradation of apo B leading to increased lipoprotein secretion and subsequent increased serum lipid concentrations.

Adipocyte differentiation and lipid metabolism are impaired due to the interaction of protease inhibitors with cytoplasmic retinoic acid binding protein 1 (CRABP-1) and the lipoprotein receptor-related protein (LRP), a receptor essential for lipoprotein remnant removal from the plasma (86,100). The resulting hyperlipidemia could result in abdominal redistribution, insulin resistance, and secondary type 2 diabetes (86,104). NRTI-induced mitochondrial toxicity could impair oxidative phosphorylation, thus resulting in increased triglycerides and subsequent insulin resistance (104).

Before ART, prognosis for individuals with AIDS was poor; concerns about other long-term health problems seemed irrelevant (103). In the early years of the HIV epidemic, morbidity (<7%) and mortality (1.1%) related to cardiovascular disease was relatively low (106). Between 1994 and 1996, cardiac disease was the primary cause of death in 9.1% of HIV-infected individuals (107). An increasing number of clients will require medical nutrition therapy for co-morbidities associated with HIV infection such as renal disease, hyperlipidemia, pancreatic dysfunction, diabetes, and liver disease (108).

Review of methodologies

Various methodologies were used in this study. To better understand these methods, a review of literature has been included for the following: food frequency questionnaires,

Dietary Reference Intakes, Healthy Eating Index, BMI, assessment of overweight and obesity, television watching, self-reported health status, and Revised Children's Manifest Anxiety.

Assessment of dietary intake using a food frequency questionnaire

The importance of accurate assessment of dietary intake is essential for appropriate identification of population deficiencies for research and public health purposes as well as individual dietary patterns for planning education strategies. There are several commonly used methods to assess food intake such as food records, 24-hour recalls, and food frequency questionnaires (FFQ). FFQs are commonly used in epidemiological studies with the premise that the average long-term diet is conceptually more important than intake on a few specific days (109). The questionnaire can be given in an interview format or self-administered though the later method requires literate individuals (110). FFQs consist of a food list and a frequency response section. A more comprehensive food list is typically preferred due to the additional information about dietary variety and usual intake.

There are several advantages to using a FFQ. Individuals are able to describe their usual frequency of consuming various foods easier than describe what foods were eaten at a specific meal. FFQs are also less expensive and easier to administer. Nutrient analysis of questionnaires is often computerized allowing for the processing of large numbers of subjects. FFQs have another advantage over 24-hour recalls or food records. One limitation of the 24-hour recall or food record is that dietary intake varies from day to day; any 24-hour recall or food record from a specific day may not give an appropriate representation of the overall diet. For example, 106 days would need to be represented for the Vitamin A intake to fall within 20% of the true mean intake (111). The additional time, subject commitment, and nutritional expertise needed for multiple 24-hour recalls or food records are much higher than that required for administration of a food frequency questionnaire.

Food frequency questionnaires have some limitations. Food items on the questionnaire must be representative of an individual's usual diet otherwise dietary intake will be inaccurately assessed. In addition, many foods are often combined into one response category and nutrient contents may vary considerably. Another concern is that individuals may report recent intake rather than usual intake during the time indicated by the FFQ (i.e.

past month, six months, or past year) (112). FFQs do not provide a direct quantitative assessment of amounts consumed of individual foods with food lists often limited to those considered primary contributors of major nutrients (113,114). FFQs tend to overestimate consumption (109). Previous studies with dietary intake and HIV infection have used various forms of FFQs (52,53,68-70,72,115), including the Health Habits and History Questionnaire (HHHQ), an early version of the Block food frequency questionnaire (BFFQ) (55).

Block food frequency questionnaire

Early versions of the HHHQ were developed in the early 1980's by Dr. Gladys Block at the National Cancer Institute for studying the role of diet in disease and health. The food list was designed to adequately assess a wide range of nutrients consumed in the typical U.S. diet. Foods included in the list represented at least 90% of 18 major nutrients and 93% of energy intake based on U.S. consumption from NHANES II data (114).

The 98.2 Block food frequency questionnaire (BFFQ) is a revision based on the NHANES III food intake data (116) (Appendix C). Foods normally eaten by different ethnic groups and low-fat foods were added to reflect current trends in food consumption. In addition, pictures depicting food portions were included with each questionnaire to help subjects visualize portion sizes. The 98.2 version is a computerized scanned questionnaire and nutrient analysis is completed by Block Dietary Data Systems (Berkeley, CA). The questionnaire has been used in over 700 research groups and universities (117). Other studies have found earlier versions of the BFFQ to be reasonably valid for estimating a group's mean actual intake for a variety of nutrients (118,119); the questionnaire was validated with black/non-Hispanic populations (120).

Under-reporting of food intake

With any method of assessing dietary intake, subjects may either underestimate or overestimate intake. Over- or under-reporting might be more likely with FFQs than diet records due to the greater standard deviations for energy intake seen with FFQs compared to other methods (121). Various studies have found under-reporting of energy intake more common among overweight or obese subjects (122-127) with similar findings seen among adolescents (123,128,129). Under-reporting among obese subjects was seen with different

dietary intake methods including food records (122,123,129,130), 24-hour recalls (125,126,128), and food frequency questionnaires (124,127). Other factors associated with under-reporting included being female (126,127), black/non-Hispanic (126), older (126), and of lower SES (125,126).

Some possible reasons for under-reporting of food intake in obese individuals include: 1) a failure to record food eaten in order to misrepresent a lower energy intake or a 'healthy' diet; 2) a failure to record because it is time consuming and inconvenient or due to difficulty qualifying food portions; 3) a failure to record due to memory laps across all or selective food items; and 4) accurate food recording but an alteration in habitual intake due to alterations in eating habits or dieting (122,131). These reasons may be particularly strong in the obese due to negative social attitudes towards overweight and consequent guilt about either the quantities or types of food that they are consuming (131).

Dietary Reference Intakes

The new Dietary Reference Intakes (DRI), established by the Institute of Medicine's (IOM) Food and Nutrition Board, differ conceptually from the 1989 Recommended Dietary Allowances (RDA) (132). First, where sufficient data were available, reduction of risk for chronic disease was considered in addition to the absence of deficiency signs when setting recommendations. Second, the upper levels of intake were established where sufficient evidence suggest risk of adverse health effects due to excessive intake. Third, components of foods that did not met the traditional concept of a nutrient but may have possible health benefits were reviewed and reference intakes established where sufficient data were available. Finally, the DRIs include four components: the Estimated Average Requirement (EAR), RDA, Adequate Intake (AI) and Tolerable Upper Intake Level (UL). Each nutrient has a set of DRIs if adequate information were available, including either an EAR and an RDA, or an AI. Figure 2 shows the EAR, RDA, and UL in relationship to risk of inadequate intake or risk of excessive intake.

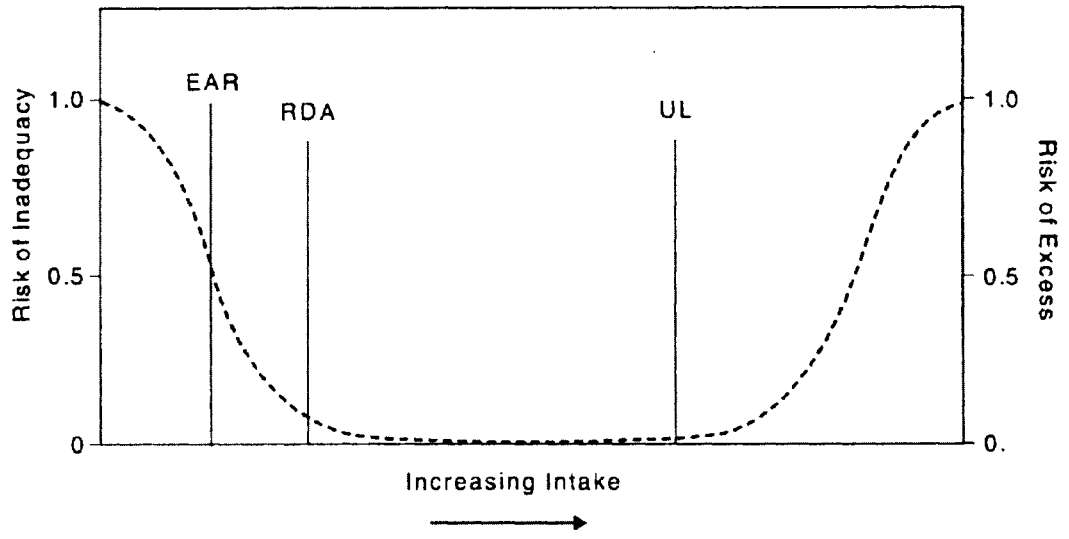

Figure 2. Dietary Reference Intakes including EAR, RDA, and UL (132)

DRI components

Estimated Average Requirement

The Estimated Average Requirement (EAR) is defined as "the median usual intake level estimated to meet the requirement of half the healthy individuals in a particular life stage and gender group" (132). The EAR is based on specific criteria related to reduction of disease risk as well as other health parameters from careful review of literature. The EAR is then used to determine the RDA.

Recommended Dietary Allowances

The RDA refers to "the average daily nutrient intakes levels sufficient to meet the nutrient requirement of nearly all (97 to 98 percent) healthy individuals in a particular life stage and gender group" (132). The RDA is a recommended guideline for daily intake of individuals and is not appropriate for assessment of group intakes. Under assumption of normality, the RDA is calculated from the EAR and the standard deviation of the requirements (SD_{EAR}) using the following equation: $RDA=EAR + 2\ SD_{EAR}$. Since the RDA is derived from the EAR, if no EAR is available, the RDA cannot be set.

Adequate Intake

When an EAR and subsequently an RDA cannot be determined due to insufficient data, an AI is determined for the nutrient. The AI is the "recommended average daily

nutrient intake level based on observed or experimentally determined approximations or estimates of nutrient intake by group (or groups) of apparently healthy people that are assumed to be adequate" (132). The AI would not consistently relate to the EAR and its RDA even if they could be established. Since the AI is expected to exceed the EAR and often the RDA for a nutrient, the AI can be used as the goal for an individual's intake. However, there are limitations to the use of the AI in assessment, which will be discussed later.

Tolerable Upper Intake Level

The UL has been defined as "the highest average daily nutrient intake level likely to pose no risk of adverse health effects to almost all individuals in the general population. As intake increases above the UL, the potential risk of adverse consequences increases" (132). For individuals, this can serve as a recommended upper intake level to avoid adverse effects related to excessive intake. For group assessment, the UL can be used to estimate the proportion of the population at potential risk related to excessive intake.

Assessment of group intakes

For assessing intakes of groups, the EAR is the most appropriate indicator to estimate the prevalence of inadequate intakes within a group. The RDA should not be used to assess group intake because it overestimates the proportion of individuals with inadequate intake (132). There are two methods available to estimate prevalence of inadequacy using the EAR: the probability approach and the EAR cut-point method. The probability approach uses the distributions of usual intakes and the distribution of requirements to estimate the prevalence of inadequate intakes in a group. This approach is based on the concept that at very low intakes the risk of inadequacy is high while at very high intakes the risk of inadequacy is negligible. Two key assumptions are necessary with the probability approach. First, intakes and requirements are independent, and second, the distribution of requirements is known.

The EAR cut-point method is a simplified version of the probability approach (Figure 3) (132). Instead of estimating the risk of inadequacy for each individual's intake level, the number of individuals in the group with intakes below the EAR is counted. This is based on the assumption that the population proportion with intakes above the EAR but below their own individual requirements (Figure 4-triangle A) are the same as the proportion with

intakes less than the EAR yet above their own individual requirement (Figure 4-triangle B). In addition to the assumptions required for the probability approach, the cut-point method also assumes that the distribution of the requirements has a symmetrical distribution. The mean or median intakes are not appropriate for assessing groups using the EAR because the prevalence of inadequacy depends on the shape and the distribution of the usual intake distribution, not the mean or median intake.

When an EAR is not available then the AI can be used but with limitations (132). If the mean usual intake is at or above the AI, a low prevalence of inadequate intakes can be assumed. However, when the usual intake of a nutrient is below the AI, no conclusions can be drawn. Since intake at or about the AI would represent a low prevalence of inadequacy, the prevalence of adequacy could be estimated by counting the number of individuals above the AI (Carriquiry, personal communication, January 2002). In addition, it is not appropriate to estimate an EAR from an AI. The UL can estimate the prevalence of a population at potential risk for adverse effects associated with excessive intakes by determining the percentage with usual intakes exceeding the UL (132).

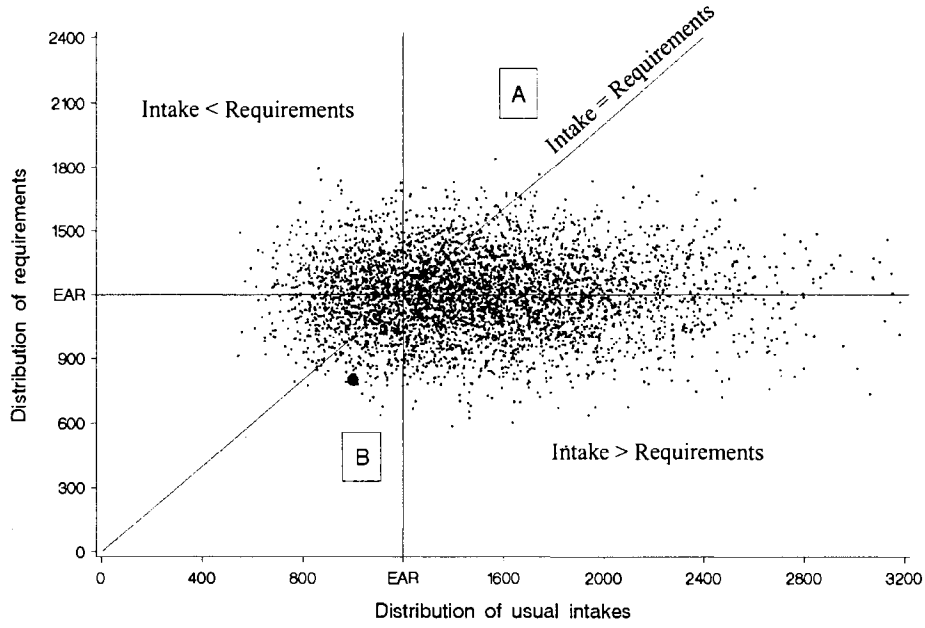

Figure 3. Example of EAR cut-point method (132)

Assessment of selected micronutrients using the new DRIs

The criteria for adequacy have been determined for each nutrient and may be gender and life-stage specific. For some nutrients, new conversion factors have been developed based on current bioavailability research. Special considerations with selected micronutrients are discussed below.

Vitamin A

Dietary sources of vitamin A come from preformed vitamin A and provitamin A carotenoids. Retinol activity equivalents (RAE) were developed based on research related to the absorption and bioconversion of provitamin A carotenoids to retinol (133). For example, dietary β-carotene has a lower bioavailability than a β-carotene supplement since the food matrix affects intestinal absorption. In addition, α-carotene and β-cryptoxanthin have approximately half the vitamin A activity of β-carotene. The RAE conversion factors for β-carotene, α-carotene, and β-cryptoxanthin are set at 12, 24, and 24 μg, respectively. Based on these new conversion factors, vitamin A intake has been overestimated in the past.

Vitamin E

The 2000 DRIs (134) have included Vitamin E activity only for α-tocopherol in the naturally occurring form (RRR-) and the other three 2R stereoisomeric forms (RSR-, RRS-, RSS-). Other forms (β-, δ-, γ-) are poorly recognized by the α-tocopherol transfer protein and do not contribute to meeting the vitamin E requirement. Dietary vitamin E intake as mg of α-tocopherol can be estimated from α-tocopherol equivalents (α-TE) x 0.8. For supplements, a conversion factor is used based on the form of vitamin E in the supplement. For RRR-α-tocopherol and for *all rac*-α-tocopherol, the conversion factor of 0.67 and 0.45 mg/IU are used, respectively.

Folate

Fortification of enriched grain products with folic acid began in 1998 in the U.S. with fortification levels set at 140 μg of folic acid/100 g of flour (135). Folic acid intakes are expected to increase women's average intake by 80-100 μg/day. Dietary folate equivalents (DFE) were developed to account for the increased bioavailability of fortification of folic acid from foods and supplements. 1 DFE = 1 μg food folate = 0.6 μg of folic acid from fortified food or as a supplement consumed with food = 0.5 μg of a supplement taken on an

empty stomach. Due to fortification of folic acid in food products as well as the new conversion factors based on bioavailability, food composition tables often do not provide up-to-date accurate information regarding folate content of foods. This poses a challenge in using the new DRIs to accurately assess prevalence of inadequacy in the population.

Iron

The EAR for iron was set using the factorial method using basal iron losses, menstrual losses, fetal requirements in pregnancy, increased requirement during growth for the expansion of blood volume, and increased tissue and storage iron (133). Information on the distribution of iron requirements is needed to estimate the prevalence of inadequacy in a population. Because the iron requirement distributions are not symmetrical for all age and sex groups, the proportion of individuals with intake below the EAR will not reflect the population prevalence of inadequacy. Thus, the full probability approach is recommended. An example of this method is shown (Table 6). For this approach, a risk of inadequate intake is assigned to each range. The number of individuals with intake in a specific range is multiplied by the assigned risk for that range to estimate the number of individuals with inadequate intake. The numbers of individuals with inadequate intake for each range are totaled. For the example provided, 165 out of 1000 women had inadequate iron intake (16.5% prevalence of inadequacy).

Table 6. Example of EAR probability approach (133)

Range of usual iron intake (mg/d)	Risk of inadequate intake	Number of women with intake in range	Number of women with inadequate intake
<4.42	1.0	1	1
4.42 – 4.88	0.985	1	0.985
4.89 – 5.45	0.925	3	2.775
5.46 – 6.22	0.85	10	8.5
6.23 – 6.87	0.75	15	11.25
6.88 – 7.46	0.65	20	13.0
7.47 – 8.07	0.55	23	12.65
8.08 – 8.76	0.45	27	12.15
8.77 – 9.63	0.35	50	17.5
9.64 – 10.82	0.25	150	37.5
10.83 – 13.05	0.15	200	30.0
13.06 – 15.49	0.075	175	13.125
15.50 – 18.23	0.015	125	4.625
>18.23	0.0	200	0
Total		1000	165

Limitations using the DRIs

One of the most important keys to assessing nutrient intakes is obtaining accurate data on intake. Using intakes collected by FFQs for use with the DRIs has several limitations. FFQs do not have a direct quantitative assessment of amounts consumed of individual foods (113). Foods included in a FFQ are typically limited to those that are considered the primary contributors to the major nutrients (114). In addition, many foods are often combined into one response category and the content of the different nutrients may vary considerably. For example on page 5 of the BFFQ (version 98.2), after listing 13 specific vegetable categories, a category labeled "any other vegetable, like okra, squash, cooked green peppers" is listed (Appendix C). This type of categorization can lead to inaccurate representation of the specific micronutrient content. The vitamin A content in a ½ cup serving of raw winter squash is considerably higher than of a ½ cup serving of raw green peppers, 235 verses 32 μg retinol equivalents, respectively (136). Thus, the IOM (132) suggested that repeated 24-hour recalls or diet records are the most appropriate data collection tools for use with the DRIs; however, FFQs may be appropriate when accurate recall or repeated measurements may be difficult to obtain.

Since the DRIs are recommendations that should be applied to healthy populations, using these recommendations should be used cautiously when assessing populations with certain disease states or altered nutritional needs. With these populations, the DRIs are most appropriate to use for comparison purposes between groups within a population. Finally, new conversion factors have been established for various micronutrients based on current research on bioavailability. Most available food composition tables are not based on these new conversion factors; and the increasing number of products with fortification poses a challenge to achieve an accurate up-to-date representation of food composition.

The USDA's Healthy Eating Index

The United States Department of Agriculture (USDA) developed the Healthy Eating Index (HEI) in 1995 as a way to measure overall dietary quality (137-139). The HEI measures how well American's eating habits meet the recommendations of the Dietary Guidelines (140) and the Food Guide Pyramid (141). The index is a sum of 10 dietary components representing various aspects of a healthy diet with each component having equal

weight (Table 7). Each component of the HEI has a maximum score of 10 and a minimum score of 0 with the maximum overall score being 100. Higher scores represent intakes closer to the recommended goals and lower scores indicate less compliance with dietary recommendations. A HEI score above 80 implies a good diet, a score between 51 and 80 indicates that the diet needs improvement, and a score below 51 implies a poor diet.

Table 7. Components of the Healthy Eating Index and scoring system (138)

	Score Range[1]	Criteria for Maximum Score of 10	Criteria for Minimum Score of 0
Grain	0 to 10	6 – 11 servings[2]	0 servings
Vegetable	0 to 10	3 – 5 servings[2]	0 servings
Fruit	0 to 10	2 – 4 servings[2]	0 servings
Milk	0 to 10	2 – 3 servings[2]	0 servings
Meat	0 to 10	2 – 3 servings[2]	0 servings
Total fat	0 to 10	≤30% energy from fat	≥45% energy from fat
Saturated fat	0 to 10	<10% energy from saturated fat	≥15% energy from saturated fat
Cholesterol	0 to 10	≤300 mg	≥450 mg
Sodium	0 to 10	≤2400 mg	≥4800 mg
Food variety	0 to 10	≥8 different items/day	≤3 different items/day

[1] People with intake between maximum and minimum amounts are assigned scores proportionally

[2] Number of servings depends on recommended energy allowance—see Table 8

HEI components

USDA Food Guide Pyramid components

The first five components are based on the five major groups in the Food Guide Pyramid: grains, vegetables, fruit, milk and meat. The recommended number of servings is dependent on a person's energy requirement. Table 8 shows the recommended number of servings for the five groups based on age and sex. Scores were computed proportionally based on these recommendations. For example, if the recommended number of servings for a food group was four and the individual consumed two, then they would be assigned a score of five points.

Table 8. Recommended number of USDA Food Guide Pyramid servings/day (138)

Age/sex category	Energy (kcal)	Grains	Vegetables	Fruit	Milk	Meat[1]
Children 2-3 [2]	1300	6	3	2	2	2
Children 4-6	1800	7	3.3	2.3	2	2.1
Females 51+	1900	7.4	3.5	2.5	2	2.2
Children 7-10	2000	7.8	3.7	2.7	2	2.3
Females 11-24	2200	9	4	3	3	2.4
Females 25-50	2200	9	4	3	2	2.4
Males 51+	2300	9.1	4.2	3.2	2	2.5
Males 11-14	2500	9.9	4.5	3.5	3	2.6
Males 19-24	2900	11	5	4	3	2.8
Males 25-50	2900	11	5	4	2	2.8
Males 15-18	3000	11	5	4	3	2.8

[1] One serving equals 2.5 ounces of lean meat

[2] Portion sizes reduced to 2/3 of adult servings except for milk for children age 2-3 y.

Fat and saturated fat components

Minimum and maximum scores for total fat and saturated fat are shown in Table 7. The percentages of total fat and saturated fat needed for the maximum score were based on the Dietary Guidelines for Americans (140). The upper percentages for fat (45%) and saturated fat (15%) were based on consultation with nutrition researchers and exploration of the consumption distribution of these components. Intake between the percentages assigned to the minimum and maximum scores was computed proportionally.

Cholesterol and sodium components

Both cholesterol and sodium scores were based on the amount consumed per day in milligrams. Table 7 shows the intake amounts assigned to maximum and minimum scores with intake between these ranges scored proportionally. The amount assigned to the maximum score was based on the recommendations of the Committee on Diet and Health of the National Research Council (3). Intakes assigned to the minimum score were determined based on consultations with nutrition researchers and exploration of the consumption distribution of each component.

Variety component

The importance of variety in the diet is stressed by the National Research Council's diet and health report, the Dietary Guidelines, and the Food Guide Pyramid (3,140,141); however, there is no consensus on how to quantify variety (138). For the HEI, the number of

different foods that a person consumed in a day determined variety. For a food item to count towards variety, at least one-half of a serving needed to be consumed. Foods that differed only by preparation method were grouped together and counted as one food type. For example, baked, fried, or boiled potatoes were counted only once. Different types of foods were grouped separately. For example, each type of fish such as mackerel, tuna, and trout were considered different foods. Food mixtures were divided into their food ingredients and assigned to the appropriate food category. The upper and lower limit amounts are shown in Table 7 and were based on consultation with nutrition researchers. A modified version of the USDA's HEI using 9 of the original 10 components was developed (142). The scores for each included component ranged from 0 to 10 with intermediate scores computed proportionally; the variety component was excluded. The total score of the modified HEI was adjusted to a 100-point scale.

Studies utilizing HEI as a measure of dietary quality

Several researchers have used the Healthy Eating Index as a measure of dietary quality. Variyam et al. (139) applied the HEI to the 1989-1990 USDA's CSFII. The companion Diet and Health Knowledge Survey (DHKS) provided additional information about nutrient content knowledge and diet-health awareness information. Higher education and incomes were related to higher knowledge of nutrient content of foods, more awareness of diet-health problems, and higher HEI scores. People over age 69 had HEI scores that were 10 points higher than individuals under 30 years. Women had higher HEI scores, nutrient knowledge, and diet-health awareness levels than men. Whites had higher HEI scores on average than blacks, but Hispanic and non-Hispanic scores were not different.

Bowman et al. (138) utilized the USDA's 1994-1996 CSFII to examine dietary quality. The mean HEI score was 63.6 in 1994, 63.5 in 1995, and 63.5 in 1996. Between 1994 and 1996, the diets of most people (70%) needed improvement. About 12% had a good diet and about 18% had a poor diet. The highest mean HEI component was for cholesterol. The cholesterol score was 7.8 out of 10. Variety received the second highest mean score of 7.6 out of 10. The fruit and milk components had the lowest mean scores of 3.9 and 5.4, respectively. The average scores on the other components were between 6 and 7. Young children (2-3 y) had an average HEI of 74, which was the highest score among all children as

well as among all age/sex groups. Adolescent females and adolescent males had HEI scores of 60.8 and 60.7, respectively.

HEI scores varied by demographic and socioeconomic characteristics. Females had slightly higher scores than males (138). Both males and females 51 years and older had higher HEI scores than other adults. Whites had a higher average HEI score than black/non-Hispanics (64 vs. 59) with black/non-Hispanics scoring lower on dairy and fat components. Both income and education level were positively associated with improved dietary quality. These findings were similar to those seen by Variyam et al. (139). In addition, regional differences were seen with people in the Northeast having higher dietary quality and those in the South having lowest dietary quality (138). HEI scores were compared by BMI. For both females and males, better dietary quality was associated with lower BMI.

Hann et al. (143) calculated the HEI on 340 women aged 21-80 y involved in a case control study of diet and breast cancer. The HEI scores were compared to dietary intakes as well as plasma carotenoids, vitamin C and folate. The average total HEI of this population was 77.3. Significantly higher HEI scores were seen among those who were better educated, had higher incomes, and were married. Strong correlations between the HEI and dietary intake were found for energy, macronutrients, cholesterol, sodium, vitamin C, folate, and fiber. When compared to plasma levels, the HEI was positively correlated with vitamin C and all carotenoids except lycopene. Folate had a positive correlation though not significant. Plasma cholesterol had a negative significant correlation with HEI.

For epidemiological studies, FFQs are an effective way to collect dietary intake information especially for micronutrients were repeated 24-hour recalls are not feasible. Caution must be used when interpreting dietary intake information since under-reporting has been documented with various groups including females and those who are obese. Dietary reference intakes are useful in assessing micronutrient inadequacies and the HEI provides an indicator of overall dietary quality. In the next section, current guidelines for assessment of weight status among adolescents and adults using BMI will be reviewed.

Methodology for assessment of overweight and obesity

Adiposity is defined as excessive body fat expressed either as total fat mass or as a percent of the total body mass (144). Overweight indicates excess body weight in children or

adolescents (144) while obesity refers to excessive body fat in adults (24). Defining overweight and obesity requires a cut-off based on morbidity or mortality risks associated with increased adiposity. Since morbidity and mortality associated with overweight in children is less prevalent than in adults, the persistence of obesity into adulthood has been used as an indicator. The criteria for a suitable measurement method include accuracy in measurement of body fat with small measurement error, acceptable to the subject, and practical for the clinic or field setting (145). Several methods are available for determining or calculating total body fat including densitometry, total body water, total body potassium, bioelectric impedance, dual-energy X-ray absorptiometry, and skinfold thicknesses. In this section, the use of body mass index (BMI) for assessment of adiposity will be discussed including current recommendations for both adults and adolescents and the advantages and limitations of using BMI with both populations.

Body mass index

BMI is defined as body weight in kilograms divided by height in meters squared (kg/m^2) (146). Even though accurate methods for assessing body composition are available, these techniques are often expensive and are not readily available in most clinics (24). Although often impractical in many clinic and field settings, densitometry, or hydrostatic weighing, is the generally accepted standard for measuring body composition (or adiposity). (147).

Willett (147) compared numerous epidemiological studies that correlated densitometry to BMI (Table 9). Correlations ranged from 0.58 to 0.85. For adolescent males, the correlation between densitometry and BMI was 0.61, while the correlation was 0.77 for adolescent females. While these correlations seem high, many of the studies compared had not been adjusted for age. Since BMI increases with age, not adjusting for age would lead to an overestimation in this correlation. Thus, Willett estimated that the correlation of true percent body fat composition in general populations is approximately 0.5 or 0.6 for men and slightly higher for women.

Use of BMI for assessing weight status is attractive in many health care and field settings since it is based on commonly measured anthropometric measurements that are reliable, non-intrusive, and require little specialized equipment. BMI can be considered an

indirect indicator of body fatness and has become widely accepted as a screening tool for obesity in the adult population (24). Recently, BMI-for-age growth charts were developed by the CDC (19) for use in assessing weight status in children and adolescents from 2- to 20-years old (Appendix A).

Table 9. Studies comparing correlation coefficients (Pearson *r*) between densitometry estimates of body fat composition and BMI (147)

Study	Subjects	*r*
Keys et al. 1972	180 students	0.85
	249 executives	0.67
Womersley and Durnin 1977	245 men	0.71
	324 women	0.82
Roche et al. 1981	68 boys (6-12 y)	0.68
	49 girls (6-12 y)	0.55
	63 boys (13-17 y)	0.61
	81 girls (13-17 y)	0.77
	141 men (18-49 y)	0.77
	135 women (18-49 y)	0.76
Revicki and Israel 1986	474 men	0.71
	Age-adjusted	0.58

Classification of overweight and obesity in adults

The National Heart, Lung, and Blood Institute (NHLBI) developed recommendations for the classification of overweight and obesity by BMI (Table 10) (24). This classification scheme provides an initial categorization upon which further assessment can be conducted. Among adults, there are some limitations with using BMI that could result in inappropriate classification. BMI can overestimate body fat in individuals who are very muscular and underestimate body fat in individuals with muscle loss such as the elderly. In addition, very short individuals (<5 feet) may have high BMIs but not be overweight or have excessive body fat. Women may have higher percent body fat yet have the same BMI as men. However, these differences do not have a huge influence on the validity of the BMI cutoffs for classifications of individuals into broad categories of overweight and obesity.

Table 10. Classification of overweight and obesity by BMI (24)

	BMI (kg/m^2)	Obesity class
Underweight	<18.5	
Normal	18.5-24.9	
Overweight	25.0-29.9	
Obesity	30.0-34.9	I
	35.0-39.9	II
Extreme Obesity	≥40	III

Classification of overweight status in adolescents

Since growth during adolescence varies by sex, age, and sexual maturation, developing a standard for assessing weight status has been a challenge. Growth charts have been used to help assess individual growth through childhood and adolescence by comparing to a reference population. The 1977 National Center for Health Statistics (NCHS) growth charts were limited to assessing weight-for-height in prepubescent children under 145 cm tall and younger than 11.5 years for boys and 10 years for girls. (20).

BMI-for-age growth charts

The BMI-for-age growth charts were developed with national survey data from 1963-1994 excluding data from the 1988-1994 NHANES III survey for children older than six years (20). This exclusion eliminated the influence of an increased body weight among older children in the NHANES III compared to previous national surveys that would have raised the 85^{th} and 95^{th} percentiles and underestimated the prevalence of risk for overweight and overweight, respectively. There are several advantages for using BMI-for-age growth charts as a screening tool for those who should receive further medical assessment (148). There are several advantages for using BMI-for-age as a screening tool. Since BMI is a commonly used in adults, use with adolescents provides the opportunity to track weight status into adulthood. In addition, adolescent BMI has been a predictor of adulthood BMI (6).

In addition to some of the BMI limitations mentioned with adults, individual and population differences in timing and rate of the adolescent growth spurt and sexual maturation should be considered when interpreting BMI (149). Since there is the potential of misclassifying individuals as being overweight despite their normal body fat percentage, BMI-for-age should be used as a preliminary screening tool. Additional assessment of the

individual's body composition and other health risks should be taken into account for determining the appropriate intervention.

Weight classification in a population including adolescents and young adults

In the United States, there are currently two separate accepted guidelines based on age discussed above. While the development of BMI-for-age growth charts were designed to allow for easier transition from adolescent weight assessment into adult standards above age 20, some problems have been identified. First, the classification terminology is inconsistent. Overweight under 20 years refers to $\geq 95^{th}$ percentile associated with the highest level of weight classification. Adult classification uses the word "overweight" to define BMI between 25 and 30, while "obesity" is used to classify the highest level of weight. With the word "overweight" meaning a different level of weight classification for under age 20 and over age 20, use of the consistent terminology in a population of adolescents and young adults poses a challenge. Second, The cut-offs for the 85^{th} and 95^{th} percentile at 20 y are greater than the adult BMI cut offs of 25 and 30. Under age 20 y, the 85^{th} and 95^{th} percentile cutoffs are 27.0 and 30.6 kg/m^2 for males and 26.5 and 31.8 kg/m^2 for females, respectively (150). This poses a challenge in having a smooth classification standard across a population of both adolescents and young adults. The expert committee on Clinical Guidelines for Overweight in Adolescent Preventative Services recommended that overweight be defined as BMI $\geq 95^{th}$ percentile for age and sex or when BMI ≥ 30 kg/m^2 (which ever is smaller) with a similar recommendation based on the $\geq 85^{th}$ and $< 95^{th}$ percentile or BMI ≥ 26 and < 30 (which ever is smaller) for classifying risk for overweight (151).

Physical activity, television watching and obesity

Numerous researchers have studied the relationship between physical activity, television watching and obesity particularly among children and adolescents. Television viewing has been correlated with between meal snacking, consumption of foods advertised on television and the children's attempts to influence their mother's food purchases (152). Television viewing may affect both energy intake and expenditure. Energy expenditure may be lower for watching television than other recreational activities such as playing tag or riding a bicycle. Television viewing tended to increase energy intake by increasing calorie-dense, between-meal snacking. Others have shown a positive relationship between watching

television and fat intake (153). With television being such a pervasive influence, children may not be able to decrease energy intake at other meals or increase energy expenditure enough to maintain energy balance (152).

Epidemiological studies

In 12-17 year old adolescents from NHANES II and III, the prevalence of obesity increased by 2% for each additional hour of television viewed (152). Results from the 1990 Youth Risk Behavior Survey of 11631 American high school students revealed that 37% reported at least 20 minutes of vigorous physical activity three or more times per week (154). Physical activity was higher among males than females ($p<0.01$) and higher among white students than among other race and ethnic groups ($p<0.01$). More than 70% of students reported spending at least one-hour watching television each school day and more than 35% reported watching television ≥3 hours each school day. Although an inverse relationship between television watching and vigorous physical activity was not found among male students, more active females were less likely to watch one to three hours of television/day compared with less active girls. Black females were the least active (38% sedentary) and spent the greatest amount of time watching television with 60.6% reported three or more hours per school day compared to other groups (154). A similar inverse relationship was found between minutes of physical activity and hours television watching ($r = -0.34$, $p = 0.04$) among Hispanic girls aged 9 to 12 years (155).

Several researchers examined the relationship between television watching, physical activity, and weight status among U.S. males and females aged 8 to 16 years from the NHANES III study (153,156,157). Females who were watched more television were more likely to be overweight (≥95th percentile BMI-for-age) than those who watched less television. Among all children in this study, 26% watched ≥4 hours of television per day with the highest rates among black/non-Hispanic children (42%) (157) with more black/non-Hispanic females being overweight compared with white/non-Hispanic females (156). Children, regardless of sex, who watched ≥4 hours of television each day had higher body fat ($p<0.001$) and BMI ($p<0.001$) than those who watched >2 hr/day (157). Among females, this relationship between television watching and overweight persisted even after controlling for age, race/ethnicity, family income, weekly physical activity, and energy intake (153). In

addition, females with larger families and males from low-income families were less likely to be overweight (156). While 80% of U.S. children reported vigorous activity three or more times each week, the prevalence was much lower among black/non-Hispanic and Hispanic girls (69% and 73%, respectively) (157). More non-Hispanic white boys reported participating in physical activity five or more times per week than any other race/ethnic or sex group (153).

A strong dose response relationship between the prevalence of overweight and hours of television viewed among 746 youths aged 10 to 15 years (158). The odds of being overweight were 4.6 times greater for youth watching more than five hours of television per day compared with those watching for zero to two hours. Similar results were seen when adjusting for previous overweight status, maternal weight, SES, ethnicity, and maternal and child aptitude test scores. Approximately 60% of overweight incidence could be linked to excess television watching.

Some studies on television watching, physical activity, and health outcomes have been conducted. Among adult men, physical activity and television watching were significantly associated with several biochemical markers of obesity and cardiovascular disease including HDL cholesterol, LDL cholesterol, apolipoprotein A1, leptin, and C-peptide (159). Health perceptions were negatively associated with the amount of time spent watching television for women only (160).

Intervention studies

Researchers have studied if overweight could be reduced or prevented through decreasing television watching. Faith et al. (161) studied the effects of contingent television on physical activity and television viewing in 10 obese children over 12 weeks. In this randomized pilot study, television viewing was contingent on pedaling a stationary cycle ergometer for experimental participants but not with the control participants. The experimental group pedaled 64.4 minutes per week on average compared to 8.3 minutes with the controls. Television viewing was significantly less among the experimental group compared to the control (1.6 vs. 21.0 hours per week). Total time pedaling was correlated with greater decreases in percent body fat ($r = -0.68$). Thus, contingent television may be one method to help treat childhood obesity.

Robinson et al. (162) used a randomized controlled school-based intervention incorporating 18 lessons into the standard curriculum to be taught by 3rd and 4th grade teachers. Each lesson included self-monitoring and self-reporting of television, videotape, and video game use to motivate children to want to reduce the time they spent in these activities. The purpose of this intervention was to decrease media use alone without specifically promoting more activity as a replacement. Children who received the intervention had significant decreases in BMI, triceps skinfold thicknesses, waist circumference, and hip-to-waist ratio compared with the controls ($p<0.01$). These changes were accompanied by statistically significant decreases in reported television viewing and meals eaten in front of the television in the intervention group relative to the controls. Television watching has been associated with decreased physical activity and increased prevalence of overweight adolescents, interventions targeted at decreasing television viewing can be an effective way to help prevent or reduce the prevalence of overweight during adolescence as well as establish behaviors to continue into adulthood.

Self-reported health status

The BFFQ (version 98.2) included a question relating to self-reported health status. The question stated "would you say your health is..." and five response categories (excellent, very good, good, fair, poor) were provided (116). Despite the simplicity of this question, answers were usually "robust predictors of later health outcomes" (163). The relationship between self-reported health status and mortality was studied among over 700,000 individuals over 18 years old from different racial/ethnic groups in the National Health Interview Survey (164). Participant responses to self-reported health status were grouped into a dichotomous variable (excellent, very good, or good responses vs. fair or poor responses). Females were more likely to report fair or poor health than males, while blacks and Native Americans were more likely to report fair or poor health compared to other racial/ethnic groups. For all groups, a greater percentage of those who were older and less educated reported poorer health compared to those who were younger or more educated. Poor self-reported health has been related to subsequent mortality among various racial/ethnic and national groups with those reporting poor health having a higher mortality rate than those reporting better health.

Idler et al. (163) compared results from several different studies that analyzed the relationship between self-reported health status and mortality by sex. In six studies, mortality risks were greater for those with poor health compared to excellent health among women, although several of the differences were slight. In 10 studies, risks of poor health compared to excellent health were higher for men than women with six of these studies showing no significant effect of women at all. Perceived health may mean something different to men and to women, which could affect their own pre-existing knowledge of their health and their subsequent health risk behavior.

RCMAS anxiety score

The Revised Children's Manifest Anxiety Score (RCMAS), adapted from an earlier anxiety scale by Reynolds and Richmond (165), is a questionnaire to assess anxiety symptoms in children and adolescents. The questionnaire consists of items relating to subjective and psychological indexes, which are summed to form a general anxiety score. The RCMAS is divided into the following subscales: physiological anxiety, worry/oversensitivity, and social concerns/concentration.

The RCMAS anxiety index was used to assess anxiety among HIV-infected adolescents (n=230) in the REACH study (166). Approximately 20% of this study showed moderate to high levels of anxiety and of depression. Stressful life events such as being prescribed medications, family financial problems, and parental abuse of alcohol were associated with higher levels of anxiety. Contrary to expectations, neither social support nor adaptive coping decreased anxiety. At the time of this study, the majority of the participants were healthy with low viral loads. HIV-infected adolescents with ineffective social support and coping mechanisms were at higher risk for developing depressive symptoms.

Conceptual framework and study objectives

The manuscripts in this thesis are from a supplemental cross-sectional study of dietary intake and nutritional status of HIV-infected and HIV-uninfected adolescents and young adults. Study participants were part of the "Reaching for Excellence in Adolescent Care and Health" (REACH) study, which was a prospective, observational cohort study of the progression of HIV infection in adolescents in 15 U.S. clinical sites in 13 cites (167,168). Based on this review of literature, an *a priori* model was developed to provide the conceptual

framework for the study design and statistical analysis used for following manuscripts (Figure 4). This model was based on individual characteristics, environmental factors, and health behaviors, which in turn affect dietary intake and weight status.

The first manuscript (Chapter 3) focused on the association between micronutrient intakes and HIV infection among participants in the REACH study. Both macronutrient and micronutrient intakes are presented. The prevalence of inadequacy for micronutrient intakes was determined using the new DRIs. The original hypothesis was:

> When controlling for other determinants, advanced stages of HIV infection would be negatively associated with the intake of the following micronutrients: vitamins A, C, and E, iron, and zinc.

These micronutrients were selected because of their relationship to immune function and known inadequacy among adolescents. Additional results not presented in this manuscript are provided in Appendix D.

The second manuscript (Chapter 4) focused on the relationship between dietary quality, weight, and HIV-infection among the REACH study participants. Dietary quality was determined using the modified Healthy Eating Index (HEI) (142). Overweight and obesity were determined using the BMI-for-age percentiles for adolescents (20) and the adult BMI classification guidelines (24). The original hypotheses for this manuscript were:

> When controlling for other determinants, advanced stages of HIV infection would be negatively associated with 1) dietary quality (HEI) and 2) obesity.

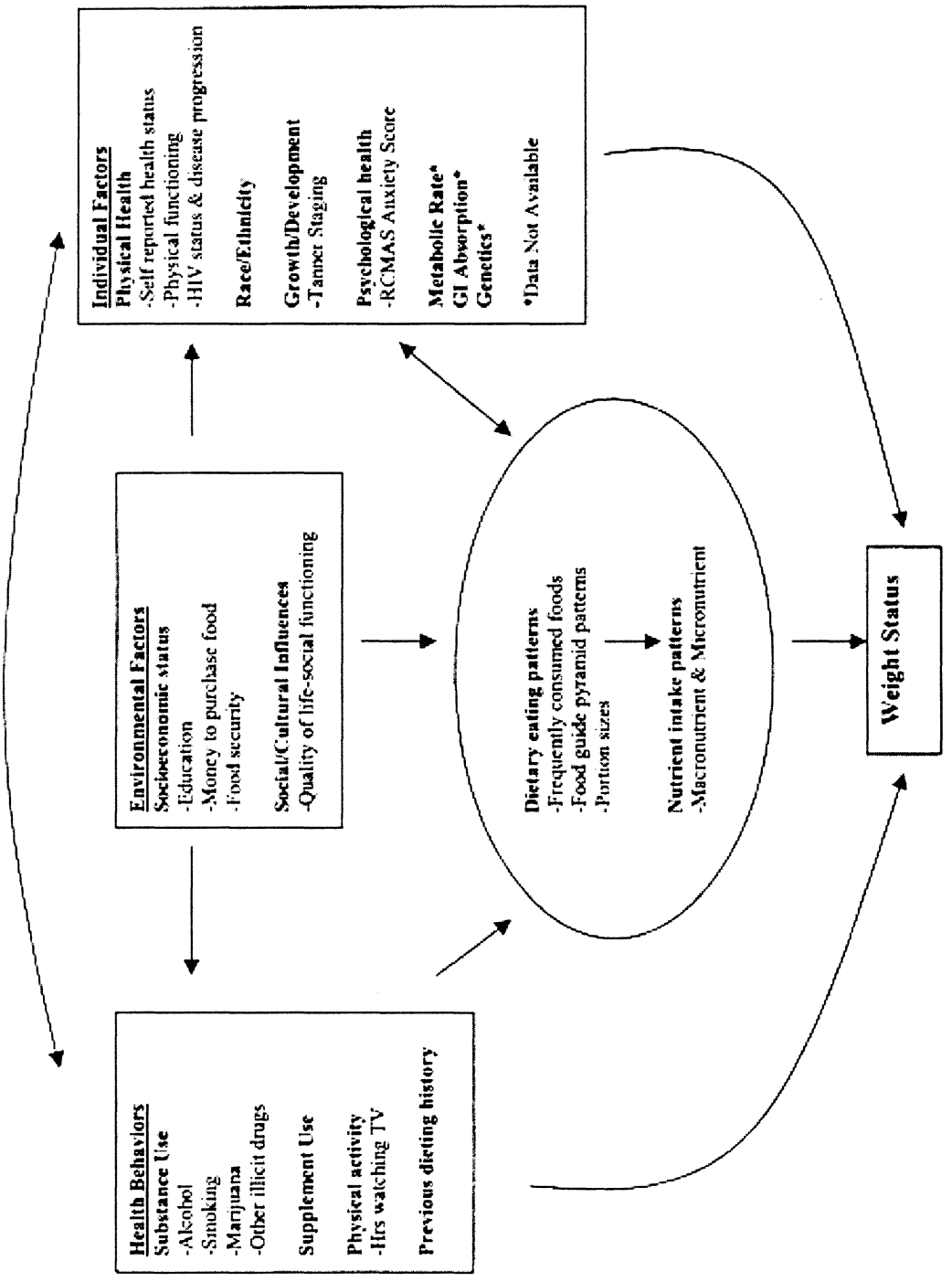

Figure 4. ***A priori*** **model for the REACH study**

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CHAPTER 3. MACRO- AND MICRONUTRIENT INTAKES DIFFER BETWEEN HIV-INFECTED AND HIV-UNINFECTED MALE ADOLESCENTS AND YOUNG ADULTS IN THE REACH COHORT STUDY

A paper to be submitted to the American Journal of Clinical Nutrition

Abstract

Background: Adolescents with HIV are at increased nutritional risk because of the nutrient demands of growth as well as disease.

Objective: We examined the association between micronutrient intakes and HIV infection in adolescents and young adults.

Design: A cross-sectional dietary intake study was conducted with 264 HIV-infected and 127 HIV-uninfected adolescents and young adults from the REACH cohort study (67% black/non-Hispanic; 75% female). Biochemical, clinical and sociodemographic data were collected through the REACH network. Dietary intake data were collected using the Block Food Frequency Questionnaire (98.2). CD4+ T-cells were stratified for HIV-infected participants: ≥500, 200-499, and <200 cells/μL. Generalized liner regression was used to determine predictors of vitamins A, C, and E, iron, and zinc intake.

Results: Almost half (49%) of HIV-infected participants had CD4+ T-cells ≥500 cells/μL and were overweight or obese (53% HIV-infected, 55% HIV-uninfected participants). HIV was associated with dietary intake differences only among males. Intake of energy and protein intake (adjusted by weight), fat, saturated fat, cholesterol, and vitamins C and E were higher among HIV-infected males than HIV-uninfected males ($p<0.05$). However, HIV was not associated with differences in nutritional inadequacies. Energy intake was a positive predictor for vitamins A, C, and E, iron, and zinc intakes ($p<0.001$). After controlling for other factors, HIV-infected participants with CD4+ ≥500 had decreased iron intake ($p<0.05$). HIV amplified the positive effect of anxiety on vitamin A intake.

Conclusions: Energy intake was the strongest predictor of micronutrient intake. After controlling for energy intake, early HIV infection was associated with a slightly lower micronutrient intake.

Introduction

Adolescents with human immunodeficiency virus (HIV) infection may be at increased risk nutritionally due to the energy and nutrient demands necessary to support growth and development as well as compensate for infection. Adolescents and young adults often have lifestyle and eating habits that may lead to poor quality diets. For example, analysis of a nationally representative survey demonstrated inadequate intakes of calcium, zinc, iron, and vitamins A, B_6, C, and E among U.S. adolescents (1).

Some insight has been gained concerning the relationship between diet and HIV in adults. Woods et al. (2) reported that macronutrient intakes increased with decreasing CD4+ T-cells. Micronutrient intakes from food and supplements did not differ significantly by CD4+ T-cells, although vitamin and mineral supplement use was greatest among those with CD4+ T-cells <200 cells/μL. Progression of HIV infection to AIDS and survival rate has been associated with specific micronutrient intakes during early HIV infection (3-6). Intakes of thiamin, riboflavin, niacin, vitamins A, B_6, C, and E, and iron during early HIV infection have been positively associated with CD4+ T-cells and survival after a 6-8 y follow-up period (3-5). In contrast, vitamin A and β-carotene intake during early HIV infection had a U-shaped relationship with risk of developing AIDS (4,5); both deficiencies and excessive intakes had a subsequent negative association with immune function and disease progression. A similar U-shaped relationship has been suggested for zinc intake as well.

Little is known about the relationship between dietary intake and HIV infection among adolescents and young adults. The primary objective of this study was to examine the association between micronutrient intakes and HIV infection in adolescents and young adults. Micronutrients of interest included vitamins A, C, and E, iron, and zinc because of their relationship to immune function and known inadequacy among adolescents.

Subjects and methods

Study population

The "Reaching for Excellence in Adolescent Care and Health" (REACH) study was a prospective, observational, cohort study on the progression of HIV infection in adolescents in 15 U.S. clinical sites (7,8). A standardized protocol was developed through the Adolescent Medicine HIV/AIDS Research Network. Between March 1996 and November 1999, 325

adolescents between 12 and 18 years of age who had acquired HIV infection through sexual activity or intravenous drug use were recruited. In addition, 171 HIV-uninfected adolescents were recruited from the same clinic sites based on risk-behavior profiles and demographic characteristics similar to HIV-infected individuals. This paper describes a supplemental cross-sectional study of individual dietary intake and nutritional status was conducted during one study visit between January and October 2000. One site did not participate due to logistical difficulties. Among the 436 participants active in the REACH network at the time of the dietary intake study, 391 agreed to participate (264 HIV-infected and 127 HIV-uninfected). The study received approval by the human subjects review boards at Iowa State University, University of California at Davis, University of Alabama at Birmingham, and at each clinic site. All participants provided informed written consent.

Data collection

REACH data were collected with face-to-face interviews, interactive computer interviews, medical record abstractions, and physical and laboratory examinations. HIV-infected subjects were seen every three months while HIV-uninfected subjects were seen every six months. A detailed description of the REACH study protocol is described elsewhere (7,8).

Dietary intake

The Block Food Frequency Questionnaire (version 98.2) (BFFQ) (9) was used to estimate usual dietary intake patterns over the past year (10,11,12) (Endnote 1). The BFFQ was administered in an interview format by trained clinic staff and was completed within 1.4 days (95% CI: - 0.6 to 2.1 days) of the study visit (Endnote 2). In addition to the two-dimensional serving size pictures included with each questionnaire, each site was provided with pictures, plates, and cups to help participants visualize portion sizes (Endnote 3). A registered dietitian reviewed the questionnaires and interviewers were contacted regarding missing information, unusual responses, or discrepancies prior to scanning. Results from computerized scanning included average intakes of macronutrients, vitamins, minerals, antioxidant nutrients, food group servings, and nutrients from supplements.

Reported micronutrient intakes from food sources alone and food and supplement sources combined were compared to the Estimated Average Requirements (EAR) or

Adequate Intakes (AI) in the Dietary Reference Intakes (DRI) (13-16). The EAR Cut-Point Method was used to determine the prevalence of inadequacy for all nutrients except iron, calcium and vitamin D (17). With this method the number of individuals below the EAR for age, sex, and pregnancy status were counted to determine the prevalence of inadequacy. Since iron requirements are skewed, the probability approach was used to determine the prevalence of inadequacy (16). An EAR has not been established for calcium and vitamin D; therefore, the AI was used instead (13). The prevalence of adequacy was estimated by counting the number of individuals above the AI.

Retinol activity equivalents (RAE) were calculated from retinol and carotenoid intake using current conversion guidelines (16). The Block Dietary Data Systems provided vitamin E intake in α-tocopherol equivalents (α-TE) and mg of α-tocopherol were estimated by α-TE x 0.8 (15). Since the form of the vitamin E supplement reported on the BFFQ was not known, a conservative estimate was made by mg of vitamin E from supplement x 0.45. Although the amount of folic acid fortification in foods was not available through the BFFQ, folic acid from supplements was converted to dietary folate equivalents (DFE) by μg folic acid x 1.7 (14). The folate intake was compared to the EAR; however, this may lead to an overestimation of the prevalence of inadequacy due to the higher bioavailability of fortified folic acid in foods.

Anthropometric measurements

Height and weight were taken during the physical examination at the study visit. Participants were gowned and weighed using digital scales accurate to one-tenth decimal place. Heights were measured using calibrated stadiometers installed at each study site. Body mass index (BMI) was calculated for each participant as weight (kg)/height $(m)^2$. Body mass indices >50kg/m^2 were verified with clinic staff for accuracy. BMI-for-age percentiles for adolescents (18) and adult BMI classification guidelines (19) were used to categorize weight status. Adult classification guidelines were used for participants ≥ 20 years old or when the BMI-for-age percentile cut-off met or exceeded the BMI adult classification guidelines. Participants with BMI ≥25 and <30kg/m^2 or BMI-for-age≥85th and <95th percentile were classified as overweight and those with BMI ≥ 30 kg/m^2 or BMI-for-age ≥95th percentile were obese. The term "obese" is not recommended for use with

adolescents due to growth and body composition changes that occur as part of normal maturation and development and the negative connotation associated with this term (20). However, obesity is used within this study to provide consistency in classification terminology across the study group since 87% of the participants were ≥18 years of age at the time of this cross-sectional study.

HIV related information

Laboratory tests were performed at local clinic sites according to REACH protocol described elsewhere (7,8). HIV serum antibody tests and CD4+ T-cells were obtained on all study participants. Quantitative immunophenotyping of CD4+ T-cells were determined at the individual clinical sites in certified laboratories using AIDS Clinical Trials Group (ACTG) standardized flow cytometry protocols. HIV status was used as a dichotomous variable. Absolute CD4+ T-cell counts for HIV-infected participants were stratified based on Centers for Disease Control and Prevention criteria for HIV/AIDS classification: ≥500, 200-499, and <200 cells/μL (21). Other HIV related information including date of first HIV seropositive test and use of antiretroviral therapies were obtained through face-to-face interview and medical record abstraction.

Demographic, health, and behavior characteristics

Demographic characteristics including race/ethnicity, family financial situation, living arrangements, and education were obtained through face-to-face interviews and interactive computer interviews. Other variables obtained from the BFFQ included vitamin/mineral and herbal supplement use, previous dieting history, hours spent watching television, cigarette smoking, pregnancy status, and self-reported health status. Hours of television watching served as an indirect indicator of physical inactivity (22). Self-reported health status ranged from poor to excellent. A Revised Children's Manifest Anxiety Scale (RCMAS), including two subscales of physiologic anxiety and worry/oversensitivity, (23) has been used previously in analysis of the REACH study (24). The RCMAS scale ranged from 0-21 with higher scores indicating greater anxiety. Data for the RCMAS scale was collected on all REACH participants through an interactive computer interview. Detailed information regarding psychological health among this population has been reported elsewhere (24).

Statistical analysis

A model for statistical analysis was developed *a priori* based on a review of the literature and data available through the REACH study. Macronutrient and micronutrient intakes were reported as mean ± standard error of mean (SE) and percentiles. For continuous variables, significant differences by HIV infection were determined by two-sample Student's *t*-tests. Differences by CD4+ T-cell stratification were determined using Analysis of Variance (ANOVA) with post-hoc differences using Bonferroni multiple comparisons. For categorical variables, differences were determined by Chi-squared test of goodness-of-fit.

Generalized linear regression analysis was used to describe the associations between micronutrient intake of selected nutrients and the explanatory variables. The dependent variables, vitamins A, C, and E, iron and zinc, were skewed and, therefore, transformed using natural logarithms. Initial regression models were based on the *a priori* model and included the following explanatory variables: health behaviors such as supplement use, substance use, and television watching; environmental factors such as family financial situation, living arrangements and food security; participant characteristics such as sex, self-reported health status, race/ethnicity, education, and psychological health; and dietary intake information such as Food Guide Pyramid servings and macronutrient intake. Total energy was centered around the mean and the squared term was included in the equation. Variables with a significance of $p<0.10$ remained in the model. Statistical analysis was repeated without outliers. Since beta-coefficients and p-values did not change, all individuals were included in the models presented. SYSTAT (version 10.0) (25) and SPSS (version 10.0.5) (26) statistical software were used for all data analysis. Significance was set at $p<0.05$ unless otherwise indicated.

Results

Participant characteristics

The BFFQ was completed with 391 participants (78.8% of the total REACH cohort); 68% were HIV-infected (Table 1). The majority (96%) of the subjects were black/non-Hispanic or Hispanic and three-quarters were female. More males were Hispanic compared to females (34.0% vs. 15.9%, $p<0.001$) while more females were black/non-Hispanic compared to males (71.1% vs. 55.7%, $p<0.01$). HIV-infected participants were older

($p<0.001$), more likely to live on their own ($p<0.001$), and were less likely to have completed or be enrolled in high school or GED program than HIV-uninfected participants ($p<0.01$).

HIV-related characteristics

Of those with HIV infection (n=264), approximately half (48.9%) had CD4+ T-cells ≥500, 38% between 200-499, and 12.5% below 200 cells/μL. Almost half (46.2%) of the HIV-infected group were not on antiretroviral therapy (ART). Of those on ART, 34.5% were on a mono- or combination therapy without a protease inhibitor (PI) while 19.3% were on a combination therapy that included a PI. Almost all (96.7%) of the HIV-infected participants reported the date of their first HIV seropositive test. Of these, the mean number of years that individuals had known they were HIV-infected was 3.6 ± 0.1 years (range: 0.5 - 10.3 years). The mean age that individuals first learned they were HIV-infected was 16.4 ± 0.1 years (range: 11.7 - 19.0 years).

Health and behavior characteristics

About one-fifth (21.7%)of the participants were overweight and one-third (32.2%) were obese (Table 2). More females were overweight or obese compared to males (62.9% vs. 26.7% respectively, $p<0.001$). Over half (55%) of the HIV-uninfected participants and 53% of the HIV-infected participants were overweight or obese. Among HIV-infected participants, the prevalence of overweight or obesity progressively decreased with decreasing CD4+ T-cell strata (60.5%, 51.0%, and 30.4% respectively, $p<0.01$).

There was no significant difference in self-reported health between HIV-infected and HIV-uninfected participants; however, the proportion of fair or poor responses increased with decreasing CD4+ T-cell strata among HIV-infected participants (30.2%, 33.3%, 60.6%, respectively; $p<0.01$). Over one-third of participants reported using a vitamin or mineral supplement. Multivitamin/mineral supplements (n=119) and the single nutrient supplements of iron (n=39) and vitamin C (n=32) were most frequently consumed while only three participants reported taking a calcium supplement. Although there was no significant difference in vitamin/mineral supplement or herbal supplement use by HIV status, more males (15.5%) than females (1.4%) used herbal supplements ($p<0.001$). Of those taking herbal supplements, over two-thirds consumed two or more different supplements with ginseng (n=12), ginkgo (n=10) and echinacea (n=9) being the most commonly consumed.

Energy and macronutrient intake

Reported energy and macronutrient intakes were compared by sex. When energy and protein intake were adjusted by body weight, males had higher intakes than females ($p<0.01$). No differences were noted in the percent of energy from macronutrients. For the total population, 48.9 ± 0.4% of the energy was from carbohydrates, 13.4 ± 0.2% from protein, 38.1 ± 0.3% from fat, and 11.9 ± 0.1% from saturated fat.

Fat (170.1 ± 6.4 vs. 146.7 ± 7.4 g), saturated fat (53.1 ± 2.0 vs. 44.9 ± 2.2 g; $p<0.01$), dietary cholesterol (491.1 ± 18.6 vs. 410.0 ± 23.1 mg; $p<0.01$) and percent of energy from saturated fat (12.1 ± 0.1 vs. 11.4 ± 0.2%; $p<0.01$) were significantly higher for HIV-infected participants than HIV-uninfected participants. Saturated fat (55.3 ± 3.2 vs. 44.9 ± 2.2 gm) and cholesterol intake (509.1 ± 31.6 vs. 410.0 ± 23.1 mg) were significantly higher among those with CD4+ T-cells between 200-499 cells/μL compared to HIV-uninfected participants.

To understand the differences in intakes, results were compared by HIV infection separately for males and females (Table 3). Total fat, saturated fat, and dietary cholesterol intake were significantly higher only among HIV-infected compared to HIV-uninfected males. When adjusted by body weight, energy and protein intake was significantly higher among HIV-infected compared to HIV-uninfected males.

Food Guide Pyramid servings were compared by sex and HIV status. For the total population, mean servings are reported for each food group: 8.2 ± 0.3 for grains, 1.5 ± 0.1 for fruits, 3.1 ± 0.1 for vegetables, 1.8 ± 0.1 for dairy, and 4.1 ± 0.1 for meat. Grains, vegetables, and dairy servings were higher among males than females with the smallest difference seen in dairy (0.3 servings) and the greatest difference seen in grain intake (1.5 servings). No significant differences were seen by HIV infection for males or females when compared separately.

When adjusted by body weight, energy and protein intakes were lower among obese than non-obese participants (35.8 ± 1.9 vs. 61.3 ± 2.0 kcal/kg and 1.2 ± 0.1 vs. 2.0 ± 0.1 g/kg, respectively; $p<0.001$). A significant inverse relationship was seen between BMI and energy intake (Pearson's $r^2= -0.15$).

Micronutrient intake

Vitamin, mineral, and carotenoid intakes from food are reported by sex and HIV status (Tables 4, 5, and 6). Males had significantly higher intake of almost all of the micronutrients than females. Only zinc intake was significantly higher among HIV-infected compared to HIV-uninfected participants. Vitamin C and E intakes were significantly higher among HIV-infected males compared to HIV-uninfected males; no differences were seen among females. No significant differences were seen in micronutrient intakes when compared by CD4+ T-cell strata.

Intake from both food and supplements were compared to the EAR or AI (Table 7). Almost 40% of all participants had vitamin E intakes below the EAR. Approximately 14% of all participants had inadequate folate intake; however, this is an overestimation since folic acid fortification of food was not available from the BFFQ. Inadequate intakes of vitamin A, magnesium, and zinc were reported among 18.7%, 28.4%, and 10.5% of participants, respectively. Prevalence of inadequacy of other micronutrients that were compared to the EAR was less than 10%. When prevalence of inadequacy was compared by sex, significantly more females had inadequate intake of iron compared to males. There were no significant differences in prevalence of inadequacy for micronutrients when compared by CD4+ T-cell strata or by HIV status. Almost half of all participants had calcium intakes above the AI and over 60% had vitamin D intakes above the AI. Significantly more HIV-infected individuals had calcium intake above the AI compared to HIV-uninfected individuals.

Individual generalized linear regression models were used to determine predictors of intakes of vitamins A, C, and E, iron, and zinc (Table 8). Increased energy intake was associated with higher micronutrient intake for all models. Positive associations between Food Guide Pyramid serving intake and micronutrient intakes varied: dairy and vegetable intake was associated with vitamin A intake, fruit with vitamin C intake, meat and vegetable with iron intake, and dairy and meat servings with zinc intake. The percent of energy from fat was positively associated with vitamin E intake. Vitamin and mineral supplement use was associated with higher intakes from food for vitamins A and E and zinc. While significant difference in micronutrient intakes were seen by sex, after controlling for energy intake, being female was associated with significantly lower zinc intake. Being black, non-

Hispanic was associated with higher intakes for vitamin C. Living independently from family was associated with higher intakes of vitamins C and E.

Being HIV-infected with CD4+ T-cells above 500 cells/μL was associated with lower intake of iron. The effect of anxiety on vitamin A intake was modified by HIV infection. When controlling for all other factors among HIV-uninfected participants, there was a 32 RAE increase in Vitamin A intake associated with high anxiety (75^{th} percentile RCMAS score) compared to those with low anxiety (25^{th} percentile RCMAS score). This difference was approximately 6-fold higher (197 RAE) among HIV-infected participants.

Discussion

This is the first study to examine the associations of HIV infection and dietary intakes among adolescents and young adults. This study showed two indicators of poor quality diet: 1) excessive intake of fat, saturated fat, and cholesterol and 2) inadequate intakes of vitamins A and E that are important for maintenance of a healthy immune response and have been associated with a slowed progression of HIV disease (3-6). Between 13-54% of the total REACH population had inadequate intakes of vitamins A and E, folic acid, zinc and magnesium when compared to the EAR. Woods et al. (2) reported similar results with high median intakes of many micronutrients but a significant proportion of subjects having intakes less than 75% of the DRI with a higher prevalence of inadequacy seen among women compared to men. This difference was similar to results seen in this study (Endnote 4).

The median intakes of several micronutrients were above intakes reported by same age and sex subjects in the Third National Health and Nutrition Examination Survey (13,14,15,16). This would indicate that the prevalence of inadequacy is lower among this study compared to the general population. This discrepancy may be related to 1) measurement error associated with the food frequency questionnaire, 2) higher intakes related to HIV infection, or 3) a combination of factors. Other HIV dietary intake studies have reported intakes of several micronutrients above the RDA (3-5,27-29). In these studies, zinc was the most common nutrient that fell short of the recommendations. Several studies have reported higher intakes among HIV-infected subjects compared to HIV-uninfected subjects (27,28,30-32). A similar trend was seen by Sharkey et al. (33); however, did not reach significance.

Over one-third of the subjects in this study reported using a vitamin/mineral supplement. Supplement usage reported among HIV-infected individuals is common and has increased in recent years (3,34,35). Abrams et al. (3) found that multivitamin use was associated with a 31% decrease in risk of progression to AIDS after a 6-year follow-up period. As noted in the present study, they also reported that daily multivitamin users consumed significantly more nutrients from food alone (3). These results suggest that individuals who take supplements make more nutrient-dense food choices compared to those who do not take supplements. Studies on the general population of US adolescents also have shown improved micronutrient intakes from foods among vitamin/mineral supplement users compared to non-supplement users (1,36) (Endnote 5).

HIV status was not a strong predictor of micronutrient intake in the regression analysis. Increased anxiety had a positive effect on vitamin A intake and had the greatest effect increase in vitamin A intake among HIV-infected individuals with CD4+ T-cells between 200-499 cells/μL. Among most macro- and micronutrients, there was a trend towards increased intake among HIV-infected individuals until CD4+ T-cells reached 200 cells/μL, then intakes declined with CD4+ T-cells below 200. In this study, this trend did not reach significance possibly due to the small number of participants with CD4+ T-cells <200 cells/μL. Hogg et al. (32) found that macronutrient intake did not differ by CD4+ T-cells (>200 vs. ≤200 cells/μL) while Woods et al. (2) found micronutrients were generally higher with decreasing CD4+ T-cells.

There may be several explanations for these observed relationships. There may be a conscious effort to eat more nutrient-dense foods among HIV-infected individuals. Dietary quality may increase as disease progresses until intake is affected by complications relating to disease progression that affect eating or appetite such as drug therapy side effects or opportunistic infections. In addition, there may be overeating behaviors associated with anxiety (37), or other factors driving intakes that were not identified in this study.

Serum concentrations of micronutrients were not presented here. Other researchers, however, have compared dietary intake with serum concentrations and have found lower serum concentrations in relation to dietary intake among HIV-infected individuals compared to HIV-uninfected participants (27,28). These discrepancies between dietary intake and

serum concentrations may be related to a variety of mechanisms including altered intestinal absorption, increased needs related to HIV infection and altered immune function, altered utilization related to metabolic changes, or a combination of factors (38,39).

The observed high fat, saturated fat, and cholesterol intake and high prevalence of obesity among HIV-infected participants may have an increased effect on metabolic and body composition abnormalities, such as lipodystrophy, hyperlipidemia, and insulin resistance (40,41). For example, polyunsaturated fats, fiber and alcohol have been strongly associated with insulin resistance and hyperlipidemia among HIV-infected men and women with fat redistribution (41). While these abnormalities were thought to be an effect of ART (42,43), studies have shown metabolic abnormalities among HIV-infected individuals in the absence of ART (41,44). The implications for the obese patient need further investigation.

In our study, there was an inverse relationship between energy intake and BMI suggesting under-reporting among those with increased weight. Other studies have found under-reporting of energy intake more common among overweight or obese subjects (45-47) and adolescents (48-50). Other factors relevant to our study population that have been associated with under-reporting included being female (46,47), being non-Hispanic black (46), and of lower socioeconomic status (45,46) (Endnote 6). Since the 95th percentile was high for several micronutrients, over-reporting is quite possible among some participants in this population. Other studies have shown over-reporting among those wanting to increase weight (47).

Using the new DRIs with this study posed some limitations. The IOM (17) suggested that repeated 24-hour recalls or diet records are the most appropriate data collection tools for use with the DRIs. In addition, DRIs are recommendations that should be applied to healthy population. In this analysis, the DRIs have been used for comparison purposes only between groups. Finally, new conversion factors have been established for folate, vitamin E, and vitamin A based on current research on bioavailability. Most of the available food composition tables are not based on these new conversion factors. The increasing availability of fortification in food products poses a challenge to achieve an accurate up-to-date representation of food composition.

Conclusions

This study on dietary intake among minority adolescents and young adults increases our understanding of relationship between dietary intake and HIV infection. Micronutrient intakes were highest among HIV-infected males and intake decreased among those with more advanced HIV infection. Energy intake was the strongest predictor of micronutrient intake; after controlling for energy intake, early stages of HIV infection was associated slightly lower micronutrient intake. Vitamin/mineral supplement intake was common among this population and was associated with more nutrient-dense food choices. A significant portion of this population was obese creating additional potential health complications in addition to the demands of HIV infection. Obese HIV-infected individuals may be at even greater risk of developing the metabolic abnormalities associated with HIV infection. Nutritionists and healthcare professionals should focus on setting individualized behavioral goals emphasizing improved nutrient density, decreased fat and saturated fat intake and increased physical activity to improve all adolescents' quality of life. Additional research is needed to determine micronutrient needs for HIV-infected youth and determine the effect of excessive micronutrient intake on HIV disease progression.

Endnotes

1. A preliminary study (n=35) to determine the validity of the HHHQ was conducted at two REACH sites. Intake results from the HHHQ were compared to data from one 24-hour dietary recalls. The correlation coefficients were significant for all micronutrients ($p<0.05$) except vitamins C and A (known to have large daily variation) which tended to be significant ($p<0.10$) (Marquis, personal communication). The BFFQ was the most feasible choice for estimating intake in this study compared to other dietary intake methods such as food records or repeated 24-hour recalls due to time constraints during study visits. The number of days needed to accurately estimate micronutrient intakes with repeated food records or 24-hour recalls were also unrealistic with this population. In addition, obtaining accurate and thorough data necessary for food records would have been a challenge with this population.

2. All interviewers received training either in person or via telephone on administering the questionnaires by a registered dietitian. Interviewers were given the opportunity to practice administration of the questionnaire at a training session held October 1999 as well as completing practice questionnaires prior to the start of the nutrition study. A question-by-question guide was provided to each interviewer including information on seasonal intake of foods, common errors encountered, and appropriate questioning format to use during the interview. Laminated quick reference cards were created which included information about seasonality adjustments and response categories.

3. A comment sheet was provided with each questionnaire for the interviewer to make note of any food that was consumed but was unsure of how to code on the BFFQ or any observations regarding the interview process. Foods written on the comment sheet were added to the BFFQ if an appropriate food grouping was available. When possible, interviewers contacted participants to clarify missing information. Participant dietary intake information and dietary recommendations were returned to study sites.

4. The appropriate and consistent use of reference standards is essential for researchers studying dietary intake. Since different reference values (i.e. 1989 RDA vs. new DRI values) and methods (i.e. percent below 75% of DRI and EAR cut-point method) were used, direct comparisons between these dietary intake studies are not possible.

5. While several micronutrients were significantly higher for males using bivariant analysis, one would expect that these differences would be accounted for by energy and food serving intakes. In regression analysis, being male remained a significant predictor for zinc intake only after controlling for energy intake, dairy and meat servings. This would suggest that the dietary quality of the specific foods consumed were unique among males. Participants living on their own may have different eating habits related to factors such as their parental independence, age, and their cooking abilities. These individuals may select more convenience items that tend to be higher in fat and, thus, higher in vitamin E.

6. Since a majority of the subjects in our study are non-Hispanic black females with increased BMI, underreporting of dietary intake is quite possible; however, actual energy expenditure was not available in this study. In addition, accurate estimation of energy and protein needs was not possible due to limited information regarding activity and other health conditions that affect energy needs. High energy intakes reported in this study suggest that over-reporting occurred among some individuals. Controlling for energy intake in our regression models for micronutrients partially compensated for reporting errors in this study. Other studies have found earlier versions of the BFFQ to be reasonably valid for estimating a group's mean actual intake for a variety of nutrients (10,11) and has been validated with black non-Hispanic populations (12).

Table 1. Participant sociodemographic characteristics by HIV status for the REACH cohort study

n	Total 391	HIV+ 264	HIV- 127
Age, y	19.8 ± 0.1 [a]	20.0 ± 0.1 ***	19.4 ± 0.1 ***
Range	13.8 - 23.2	13.8 - 23.2	14.8 - 22.9
Male	97 (24.8) [b]	68 (25.8)	29 (22.8)
Female	294 (75.2)	196 (74.2)	98 (77.2)
Race/ethnicity			
Hispanic	80 (20.5)	51(19.3)	29 (22.8)
Black/non-Hispanic	263 (67.3)	186 (70.5)	77 (60.6)
White/non-Hispanic	16 (4.1)	8 (3.0)	8 (6.3)
Other	32 (8.2)	19 (7.2)	13 (10.2)
Living arrangements***			
Living in own apartment/house	110 (28.1)	88 (33.3)	22 (17.3)
Parents/family member	220 (56.3)	133 (50.4)	87 (68.5)
Someone else's house/apt	28 (7.1)	19 (7.2)	9 (7.1)
Other [c]	33 (8.4)	24 (9.1)	9 (7.1)
Education**			
No HS diploma or GED	141 (36.1)	113 (42.8)	28 (22.0)
Has HS diploma/GED or enrolled in school	250 (63.9)	151 (57.2)	99 (78.0)
Highest grade completed by mother [d]			
No HS diploma or GED	109 (28.0)	83 (31.5)	26 (20.6)
HS diploma or GED	108 (27.7)	63 (24.0)	44 (35.4)
Trade or tech school	22 (5.6)	18 (6.8)	4 (3.2)
Some college	77 (19.7)	50 (19.0)	27 (21.3)
Don't know/unsure	74 (19.0)	49 (18.6)	25 (19.7)
Household financial situation**			
Struggling to survive	23 (5.9)	21 (8.0)	2 (1.6)
Barely paying bills	21 (5.4)	13 (4.9)	8 (6.3)
Have necessities sometimes	53 (13.6)	38 (14.4)	15 (11.8)
Have necessities but little more	170 (43.4)	122 (46.2)	48 (37.8)
Always have the necessities	124 (31.7)	70 (26.5)	54 (42.5)
Food security [#]			
Food not available	15 (3.8)	14 (5.2)	1 (0.8)
Food available most days	376 (96.2)	250 (94.7)	126 (99.2)

[a] Mean ± standard error; [b] n (%); [c] Living in halfway house, foster or group home, or on the street;
[d] total (n=390), HIV+ (n=263)
***p <0.001; ** p <0.01; * p <0.05; [#] p <0.10 (Chi^2 for categorical variables, t-tests for continuous variables)

Table 2. Health characteristics by sex and HIV status for the REACH cohort study

	Females		Males	
n	HIV+ 196	HIV- 98	HIV+ 68	HIV- 29
Anthropometrics				
Height (cm) [a #]	163.9 ± 0.6 *	162.3 ± 0.7	173.3 ± 1.0 *	173.6 ± 1.7
Weight (kg) [a] ***	79.9 ± 1.8 *	77.4 ± 2.4	70.8 ± 2.3 *	76.9 ± 3.6
Weight classification [b] ***				
Overweight [c]	54 (27.6)	19 (19.4)	7 (10.3)	5 (17.2)
Obese [d]	71 (36.2)	41 (41.8)	8 (11.8)	6 (20.7)
CD4+ T-cells (cells/μL)				
≥500	99 (50.5)	--	30 (44.1)	--
200-499	76 (38.8)	--	26 (38.2)	--
<200	21 (10.7)	--	12 (17.6)	--
Pregnant (% of women)	16 (8.2)	11 (11.2)	--	--
Self-reported health status				
Excellent, very good, or good	125 (63.8)	52 (53.1)	46 (67.6)	20 (69.0)
Fair or poor	71 (36.2)	46 (46.9)	22 (32.4)	9 (37.9)
Vitamin/mineral supplement use	77 (39.3)	33 (44.8)	20 (29.4)	13 (36.6)
Herbal Supplement Use ***	3 (1.5)	1 (1.0)	12 (17.6)	3 (10.3)
Currently smoke	80 (40.8)	31 (45.6)	31 (31.6)	13 (44.8)
Television watching: >3 hr/d	98 (44.9)	44 (44.9)	36 (48.3)	14 (48.3)
History of weight loss attempts *	71 (36.2)	45 (45.9)	16 (23.5)	10 (34.5)
RCMAS anxiety score	7.2 ± 0.4	7.9 ± 0.6 [d]	6.4 ± 0.8	7.5 ± 1.1
Median	6.0	7.0	4.0	8.0

[a] Mean ± standard error; [b] n (%); [c] BMI ≥25 & <30 or BMI-for-age ≥85th and <95th percentile; [c] BMI ≥30 or BMI-for-age ≥95th percentile; [d] n=97

*** p <0.001; ** p <0.01; * p <0.05; [#] p <0.10 (ANOVA for continuous variables, Chi^2 for categorical)

Similar superscripts indicate significant difference of pairwise comparison

Table 3. Macronutrient intake from food by sex and HIV status for the REACH cohort study

	Mean ± SE	Median (5^{th}, 95^{th} percentile)
Energy, kcal		
Females: HIV+ [a]	3691 ± 137	3454.0 (1152.5, 7248.1)
HIV- [b]	3445 ± 191	3016.6 (1329.8, 7392.9)
Males: HIV+ [c]	4507 ± 304 [#]	4013.8 (1711.5, 9975.4)
HIV- [d]	3732 ± 333 [#]	3256.6 (1344.8, 8364.2)
Carbohydrate, g		
Females: HIV+	446.8 ± 16.4	416.3 (137.5, 864.6)
HIV-	427.0 ± 24.2	368.3 (171.4, 834.0)
Males: HIV+	528.7 ± 35.8	451.5 (196.9, 1108.8)
HIV-	466.0 ± 46.3	384.9 (182.8, 1094.9)
Protein, g		
Females: HIV+	121.9 ± 5.0	108.0 (39.7, 285.8)
HIV-	112.3 ± 6.4	94.1 (41.1, 256.6)
Males: HIV+	148.9 ± 10.4 [#]	123.2 (57.3, 336.2)
HIV-	122.1 ± 11.7 [#]	106.8 (52.7, 297.9)
Fat, g		
Females: HIV+	159.8 ± 6.7	138.5 (44.5, 341.3)
HIV-	145.1 ± 8.7	126.4 (54.7, 309.3)
Males: HIV+	200.1 ± 14.8 *	177.2 (64.5, 451.2)
HIV-	152.0 ± 13.2 *	131.6 (45.0, 335.7)
Saturated fat, g		
Females: HIV+	50.2 ± 2.1 [#]	44.6 (13.6, 101.6)
HIV-	44.2 ± 2.6 [#]	38.0 (14.8, 106.0)
Males: HIV+	61.6 ± 4.4 *	56.8 (18.8, 131.4)
HIV-	47.0 ± 4.0 *	40.7 (11.5, 99.2)
Dietary fiber, g		
Females: HIV+	22.1 ± 0.9	19.2 (7.9, 48.8)
HIV-	22.1 ± 1.6	18.3 (6.6, 44.8)
Males: HIV+	26.8 ± 2.2	21.1 (8.3, 62.8)
HIV-	23.9 ± 2.3	22.4 (8.3, 51.8)
Dietary cholesterol, mg		
Females: HIV+	464.6 ± 21.2 [#]	384.6 (136.5, 1141.1)
HIV-	404.3 ± 26.8 [#]	321.8 (118.6, 980.1)
Males: HIV+	567.6 ± 37.1 *	503.4 (169.7, 1220.5)
HIV-	429.4 ± 45.4 *	365.6 (107.6, 1091.1)
Energy, kcal/kg		
Females: HIV+	50.8 ± 2.2	47.4 (12.7, 107.3)
HIV-	49.0 ± 3.2	41.8 (15.4, 108.1)
Males: HIV+	66.4 ± 4.5 *	61.7 (22.9, 162.9)
HIV-	51.1 ± 5.2 *	41.2 (16.5, 127.7)
Protein, gm/kg		
Females: HIV+	1.7 ± 0.1	1.4 (0.4, 4.0)
HIV-	1.6 ± 0.1	1.3 (0.4, 3.5)
Males: HIV+	2.2 ± 0.2 *	1.9 (0.7, 4.9)
HIV-	1.7 ± 0.2 *	1.4 (0.6, 4.6)

[a] n=196; [b] n=98; [c] n=68; [d] n=29; Differences by HIV status for males and females tested separately using two-sample t-tests; * $p < 0.05$; [#] $p < 0.10$

Table 4. Vitamin intake from food by sex and HIV status for the REACH cohort study

	Mean ± SE	Median (5^{th}, 95^{th} percentile)
Vitamin A, RAE [a]		
Females: HIV+ [b]	1025.0 ± 55.3	821.4 (256.5, 2477.8)
HIV- [c]	944.0 ± 72.2	762.6 (231.8, 2244.7)
Males: HIV+ [d]	1241.0 ± 101.2	978.6 (359.4, 3136.4)
HIV- [e]	994.4 ± 139.3	784.8 (349.7, 3262.2)
Thiamin (B_1), mg		
Females: HIV+	2.5 ± 0.1	2.3 (0.7, 5.9)
HIV-	2.4 ± 0.2	2.0 (0.9, 5.1)
Males: HIV+	3.2 ± 0.2	2.7 (1.1, 6.7)
HIV-	2.7 ± 0.3	2.3 (1.1, 6.8)
Riboflavin (B_2), mg		
Females: HIV+	2.7 ± 0.1	2.5 (0.7, 6.2)
HIV-	2.6 ± 0.2	2.1 (0.9, 5.4)
Males: HIV+	3.4 ± 0.2 #	2.8 (1.2, 7.6)
HIV-	2.8 ± 0.3 #	2.4 (1.3, 6.9)
Niacin, mg		
Females: HIV+	36.1 ± 1.5	30.7 (11.1, 85.7)
HIV-	34.4 ± 2.2	28.2 (11.0, 73.3)
Males: HIV+	45.5 ± 3.3	37.5 (15.9, 99.3)
HIV-	38.0 ± 4.1	34.8 (14.1, 106.2)
Vitamin B_6, mg		
Females: HIV+	2.9 ± 0.1	2.6 (0.9, 6.2)
HIV-	2.7 ± 0.2	2.3 (0.9, 5.6)
Males: HIV+	3.5 ± 0.2	3.0 (1.1, 7.6)
HIV-	3.2 ± 0.4	2.9 (1.1, 8.8)
Vitamin C, mg		
Females: HIV+	246.5 ± 11.4	204.4 (54.8, 562.9)
HIV-	255.0 ± 16.8	255.0 (52.8, 585.2)
Males: HIV+	285.1 ± 22.1 *	244.2 (52.3, 646.6)
HIV-	275.2 ± 31.5 *	282.6 (67.2, 656.6)
Folic Acid, µg		
Females: HIV+	588.0 ± 24.5	503.5 (184.5, 1300.3)
HIV-	586.0 ± 40.1	586.0 (185.5, 1166.5)
Males: HIV+	719.5 ± 48.6	628.6 (251.3, 1525.4)
HIV-	620.7 ± 55.4	530.9 (256.3, 1421.1)
Vitamin D, IU		
Females: HIV+	257.6 ± 18.5	174.0 (33.3, 818.1)
HIV-	236.7 ± 22.7	162.8 (35.1, 667.8)
Males: HIV+	323.4 ± 32.2	228.4 (76.3, 973.7)
HIV-	228.7 ± 34.2	165.0 (55.0, 691.2)
Vitamin E, mg [f]		
Females: HIV+	12.8 ± 0.5	11.2 (3.6, 27.6)
HIV-	12.4 ± 0.8	10.0 (4.0, 27.1)
Males: HIV+	16.0 ± 1.1*	14.0 (5.2, 37.1)
HIV-	15.1 ± 1.0 *	10.4 (5.2, 24.1)

[a] Retinol activity equivalents; [b] n=196; [c] n=98; [d] n=68; [e] n=29; [f] As α-tocopherol

Differences by HIV status for males and females tested separately using two-sample t-tests with separate variance; * $p<0.05$; # $p<0.10$

Table 5. Mineral intake from food by sex and HIV status for the REACH cohort study

	Mean ± SE	Median (5^{th}, 95^{th} percentile)
Calcium, mg		
Females: HIV+ [a]	1093.2 ± 43.6	1026.1 (309.1, 2362.6)
HIV- [b]	1056.4 ± 67.6	825.1 (329.6, 2727.5)
Males: HIV+ [c]	1353.1 ± 90.5 [#]	1125.6 (390.1, 2775.2)
HIV- [d]	1107.3 ± 91.0 [#]	994.5 (400.0, 2312.0)
Phosphorus, mg		
Females: HIV+	1887.9 ± 69.6	1745.2 (551.3, 3844.9)
HIV-	1763.6 ± 100.9	1468.1 (653.6, 3892.1)
Males: HIV+	2308.9 ± 155.5 [#]	1883.1 (911.9, 4883.3)
HIV-	1901.7 ± 166.8 [#]	1650.6 (970.5, 4209.2)
Iron, mg		
Females: HIV+	23.9 ± 1.1	20.0 (6.9, 58.3)
HIV-	23.4 ± 1.8	18.8 (8.0, 52.9)
Males: HIV+	31.1 ± 2.5	25.2 (10.3, 86.1)
HIV-	26.0 ± 3.1	23.0 (9.8, 75.1)
Zinc, mg		
Females: HIV+	16.8 ± 0.8	13.7 (4.5, 40.5)
HIV-	14.9 ± 0.9	12.5 (5.3, 34.2)
Males: HIV+	21.9 ± 1.9 [#]	17.0 (7.7, 61.0)
HIV-	17.6 ± 1.3 [#]	16.1 (8.1, 30.1)
Magnesium, mg		
Females: HIV+	397.4 ± 15.1	351.5 (121.3, 866.3)
HIV-	385.5 ± 22.7	323.7 (135.1, 743.2)
Males: HIV+	488.0 ± 34.7	430.3 (165.6, 1106.7)
HIV-	427.7 ± 34.0	417.1 (188.8, 857.9)

[a] n=196; [b] n=98; [c] n=68; [d] n=29; Differences by HIV status for males and females tested separately using two-sample t-tests with separate variance; [#] $p<0.10$

Table 6. Retinol and carotenoid intake from food by sex and HIV status for the REACH cohort study

	Mean ± SE	Median (5^{th}, 95^{th} percentiles)
Retinol, ug		
Females: HIV+ [a]	731.5 ± 44.2	565.6 (137.0, 1830.6)
HIV- [b]	629.2 ± 48.7	521.7 (172.1, 1825.0)
Males: HIV+ [c]	849.7 ± 65.9	653.7 (244.3, 1995.3)
HIV- [d]	683.7 ± 86.6	565.6 (230.1, 2053.5)
Alpha-carotene, µg		
Females: HIV+	369.9 ± 30.9	209.0 (36.6, 1279.7)
HIV-	468.1 ± 99.6	205.2 (45.6, 2015.6)
Males: HIV+	638.9 ± 96.2	384.6 (50.8, 2800.1)
HIV-	423.4 ± 108.1	275.5 (20.3, 2104.6)
Beta-carotene, µg		
Females: HIV+	3214.8 ± 229.3	2263.3 (569.2, 10137.7)
HIV-	3398.1 ± 354.1	2142.1 (520.7, 10837.4)
Males: HIV+	4222.6 ± 626.6	2589.0 (641.5, 14800.4)
HIV-	3352.4 ± 697.9	2256.6 (372.1, 14298.2)
Cryptoxanthin, µg		
Females: HIV+	242.5 ± 16.5	161.0 (16.6, 743.7)
HIV-	289.8 ± 28.0	212.0 (16.2, 830.1)
Males: HIV+	307.1 ± 36.4	208.4 (22.9, 992.5)
HIV-	343.2 ± 52.8	256.0 (62.9, 1080.5)
Lutein, µg		
Females: HIV+	2063.0 ± 187.9	1232.9 (239.9, 6147.7)
HIV-	1818.9 ± 197.0	1205.9 (192.2, 6423.5)
Males: HIV+	1919.4 ± 264.7	1248.0 (179.8, 5669.5)
HIV-	1939.6 ± 553.4	1133.6 (352.1,10255.3)
Lycopene, µg		
Females: HIV+	8877.0 ± 698.9	5311.4 (557.6, 30162.2)
HIV-	8284.5 ± 896.3	5852.9 (1127.0, 25746.3)
Males: HIV+	11569.7 ± 1346.3	7323.3 (1450.9, 40876.6)
HIV-	10856.0 ± 1689.8	7333.8 (597.1, 31531.6)

[a] n=196; [b] n=98; [c] n=68; [d] n=29; No significant differences were found by HIV status for males and females tested separately using two-sample t-tests with separate variance

Table 7. Prevalence of inadequate micronutrient intake from food and supplements for the REACH cohort study

	Total	Female	Male	HIV+	HIV-	CD4+ T-cells (cells/μL) ≥500	200-499	<200
n	**391**	**294**	**97**	**264**	**127**	**129**	**102**	**33**
Prevalence of inadequacy--Percent below Estimated Average Requirement (EAR)								
Vitamins								
Vitamin A	18.7	18.7	18.6	17.8	20.5	16.3	16.7	27.3
Thiamin	4.0	5.1 #	1.0 #	4.1	3.9	3.9	2.9	9.1
Riboflavin	3.8	4.8 #	1.0 #	4.2	3.2	4.7	2.9	6.1
Niacin	3.3	4.1	1.0	3.4	3.2	3.9	2.0	6.1
Vitamin B_6	4.9	5.8	2.1	4.5	5.5	4.7 #	2.0 #	12.1 #
Vitamin C	4.3	4.1	5.2	4.9	3.2	6.2	2.9	6.1
Folic Acid	14.1	15.6	9.2	14.4	13.4	13.2	14.7	18.2
Vitamin E	38.1	39.5	34.0	36.0	42.5	33.3	37.3	42.4
Minerals								
Phosphorus	N/A							
Iron	4.8	6.1 *	1.0 *	4.8	4.9	4.4	4.2	7.8
Zinc	10.5	10.2	11.3	11.0	11.0	9.3	10.8	12.1
Magnesium	28.4	26.5	34.0	26.9	31.5	26.5	25.5	33.3
Prevalence of adequacy--Percent above Adequate Intake (AI)								
Calcium	48.1	45.9	54.6	52.3 *	39.4 *	50.4	52.9	57.6
Vitamin D	61.4	60.5	63.9	63.3	57.5	62.8	63.8	63.6

* $p < 0.05$; # $p < 0.10$; Fisher's exact test for differences by sex and by HIV infection for cells with frequency <5; Chi^2 Goodness-of-fit test for differences by CD4 cell count.

Table 8. Multiple linear regression models predicting the log of micronutrient intakes for the REACH cohort study

Dependant variable (Natural log)	Vitamin A µg RAE	Vitamin C mg	Vitamin E mg	Iron mg	Zinc mg
Explanatory variables			**Coefficients (Std Error)**		
Constant	6.118 (0.069)	4.871 (0.063)	1.876 (0.091)	2.950 (0.047)	2.329 (0.050)
Energy/1000 kcal (centered)	0.158*** (0.016)	0.190*** (0.016)	0.284*** (0.009)	0.263*** (0.014)	0.170*** (0.013)
Female					-0.089** (0.031)
Black/non-Hispanic	0.073 # (0.043)	0.226*** (0.055)			
Living on own		0.155** (0.057)	0.061* (0.030)		
Vitamin/mineral supplement use	0.135** (0.043)		0.090** (0.029)	0.096** (0.032)	
RCMAS anxiety index [a]	0.003 (0.004)				
HIV-infected:CD4+ T-cells ≥500 cells/µL		-0.094# (0.055)	-0.048# (0.029)	-0.079* (0.032)	
HIV-infected: CD4+ T-cells 200-499 cells/µL	-0.032 (0.069)				
Food Guide Pyramid servings					
Dairy	0.161*** (0.017)				0.075*** (0.012)
Vegetables	0.077*** (0.012)			0.026** (0.009)	
Fruit		0.229*** (0.022)			
Meat/other protein				0.020* (0.009)	0.085*** (0.008)
Percent of energy from fat			0.015*** (0.002)		
Interactions					
Energy (centered)^2	-0.011*** (0.003)	-0.014*** (0.003)	-0.017*** (0.002)	-0.019*** (0.002)	-0.018*** (0.002)
RCMAS anxiety* CD4+ T-cells: 200-499 cells/µL	0.014# (0.008)				
n	390	391	391	391	391
Adjusted R^2	0.652	0.508	0.787	0.760	0.804
Std error of estimate	0.393	0.500	0.268	0.295	0.263

[a] Revised Children's Manifest Anxiety Scale

Significance of explanatory variables: *** p <0.001; ** p <0.01; * p <0.05; # p <0.10

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CHAPTER 4. DIETARY QUALITY AND PREVALENCE OF OBESITY AMONG HIV-INFECTED AND HIV-UNINFECTED ADOLESCENTS AND YOUNG ADULTS IN THE REACH COHORT STUDY

A paper to be submitted to the Journal of the American Dietetic Association

Abstract

Objective: Examine the relationship between dietary quality, weight status and HIV infection among HIV-infected and HIV-uninfected adolescents/young adults.

Design: A cross-sectional study of dietary intake with the REACH cohort, a multi-centered observational study of HIV-infected and HIV-uninfected but at-risk adolescents/young adults.

Subjects/setting: 248 HIV-infected and 116 HIV-uninfected participants (73% female; 67% black/non-Hispanic) at 14 U.S. clinic sites.

Methods: Biochemical, clinical, and sociodemographic data were available through the REACH network. Dietary intake was collected using the Block Food Frequency Questionnaire (98.2) and modified Healthy Eating Index (HEI) was calculated to measure dietary quality.

Statistical analysis: χ^2 test, *t*-tests, ANOVA, Pearson's correlation, logistic and generalized linear regression.

Results: Half (50.4%) of HIV-infected and 54.3% of HIV-uninfected participants were overweight or obese. Being female, living independently from parents/family, watching television ≥3 hours/d, previous dieting, and being from the Northeast or South were positively associated with obesity. Obesity was lower among participants with CD4+ T-cells ≥500 cells/μL. For all participants, the modified HEI was 56.2±0.6. Being from the Chicago area, HIV-infected, and watching television ≥3 hr/d were associated with a lower HEI (poorer diet) while being female and having good self-perceived health were associated with a higher HEI (better diet).

Conclusions/applications: Obese, HIV-infected individuals may be at risk of developing metabolic abnormalities associated with HIV. Dietary quality was poor among HIV-infected participants; although, the majority of this young population did not meet dietary

recommendations. Nutrition education should focus on improving dietary quality and physical activity for all adolescents and young adults.

Introduction

The alarming increase in obesity among the U.S. population has become a growing public health concern not only among adults but also among adolescents. Recent findings from the 1999 National Health and Nutrition Examination Survey (NHANES IV) revealed approximately 61% of adults over age 20 were overweight or obese, a 5% increase from NHANES III (1988-1994), and 14% of adolescents aged 12-19 years had body mass index (BMI)-for-age above the 95th percentile, a 3% increase from NHANES III (1,2). Results from NHANES III demonstrated that obesity was highest among ethnic minorities, 38% of black non-Hispanic and 35% of Hispanic women were obese compared to 24% of white women (3). The effect of ethnicity appears to be less pronounced among adolescents; 13-14% of the Hispanics and black non-Hispanics were overweight or obese compared to 11% of white adolescents.

Adolescence and young adulthood are critical periods when eating and lifestyle behaviors are established. Behaviors during these periods could have both immediate and long-term health consequences. Overweight teenagers have increased risk of hypertension, hyperlipidemia, development of type 2 diabetes and other chronic diseases during adolescence (4-6). Overweight adolescents are more likely to be overweight as adults (7-10) and have increased morbidity associated with hypertension, cardiovascular disease, type 2 diabetes, stroke, gallbladder disease, osteoarthritis, respiratory problems, sleep apnea and some cancers (11). For obese adults, the risk of mortality from all causes especially cardiovascular disease increased by 50-100% over those with a normal BMI. Besides the physiologic consequences of obesity, numerous psychosocial consequences such as social stigmatization, depression, binge eating disorder, and altered body image have been identified in both adolescents and adults (4,11)

Many chronic conditions associated with obesity have been linked to modifiable lifestyle and dietary behaviors. Researchers have examined dietary quality and intake among adolescents and young adults. In a nationally representative study, 25% of adolescents had intakes of calcium, zinc, iron, and vitamins A, B6, C and E <75% of the US Recommended

Dietary Allowance (12) while over 30% of adolescents had total fat intake above 30% of energy and saturated fat intake above 10% of energy (3). The USDA developed the Healthy Eating Index (HEI) (13) as a measure of diet quality based on the Food Guide Pyramid (14) and Dietary Guidelines for Americans recommendations (15.). The average score for both males and females aged 15-19 years (HEI score: 61 out of 100) indicated dietary intake fell short of these recommendations (13).

In addition to the nutritional concerns of dietary intake and increased weight, human immunodeficiency virus (HIV)-infected adolescents and young adults face additional challenges related to the demands of HIV infection. Those who are obese are at even greater risk of developing metabolic and body composition abnormalities such as lipodystrophy, hyperlipidemia, and insulin resistance associated with HIV-infection and antiviral therapies (16,17). Clinical features of lipodystrophy, or fat redistribution syndrome, include peripheral fat loss especially in the face, limbs, and buttocks with a central fat accumulation in the abdominal region, breasts, and over the dorsocervical vertebrate (referred to as the "buffalo hump") (16,18). Little is known about the relationship between nutritional status and health among U.S. adolescents and young adults infected with HIV. The primary purpose of this study was to examine the relationship between dietary quality, weight status, and HIV infection among HIV-infected and HIV-uninfected adolescents and young adults.

Subjects and methods

Study population

The "Reaching for Excellence in Adolescent Care and Health" (REACH) study was a prospective, cohort study of the progression of HIV infection in adolescents in 15 U.S. clinical sites (19,20). A standardized treatment protocol was developed through the Adolescent Medicine HIV/AIDS Research Network. Between March 1996 and November 1999, 325 adolescents between 12 and 18 years of age who had acquired HIV infection through sexual activity or intravenous drug use were recruited. In addition, 171 HIV-uninfected adolescents were recruited from the same clinic sites based on risk-behavior profiles and demographic characteristics similar to HIV-infected individuals. This paper describes a supplemental cross-sectional study of dietary intake and nutritional status was conducted during one study visit between January and October 2000. One site did not

participate in this study due to logistical difficulties with handling laboratory samples. Among the 436 participants active in the REACH network, 391 agreed to participate in the dietary intake study. Pregnant subjects (n=27) were excluded from the present analysis resulting in 364 subjects (248 HIV-infected and 116 HIV-uninfected). The study received approval by the human subjects review boards through Iowa State University, University of California at Davis, University of Alabama at Birmingham, and at each clinic site. All participants provided informed written consent.

Data collection

Data collection for the longitudinal study occurred through face-to-face interviews, interactive computer interviews, medical record abstractions, and physical and laboratory examinations. A detailed description of the REACH study protocol is described elsewhere (19,20).

Dietary intake

The Block Food Frequency Questionnaire (version 98.2) (BFFQ) (21) was used to estimate usual dietary patterns over the past year. The BFFQ was administered in an interview format by trained clinic staff and was completed within 1.4 days (95% CI: - 0.6 to 2.1 days) of the study visit. A registered dietitian reviewed the questionnaires and interviewers were contacted regarding missing information, unusual responses, or discrepancies prior to scanning. Results from computerized scanning included average intakes of macronutrients, vitamins, minerals, antioxidant nutrients, food group servings, and nutrients from supplements.

Anthropometric measurements

Height and weight were taken during the physical examination at the study visit. Participants were gowned and weighed using digital scales accurate to one-tenth decimal place. Weight at the previous 6-mo visit was compared to current weight for assessment of weight change. Body mass index (BMI) was calculated for each participant as weight (kg)/height $(m)^2$. Body mass indices >50 kg/m^2 were verified with clinic staff for accuracy. BMI-for-age percentiles for adolescents (22) and adult BMI classification guidelines (11) were used to categorize weight status. Adult classification guidelines were used for participants ≥20 years old or when the BMI-for-age percentile cut-off met or exceeded the

BMI adult classification guidelines. Participants with BMI ≥25 kg/m^2 and <30 kg/m^2 or BMI-for-age ≥85th and <95th percentile were classified as overweight and those with BMI ≥30 kg/m^2 or BMI-for-age ≥95th percentile were obese. The term "obese" is not recommended for use with adolescents due to growth and body composition changes that occur as part of normal maturation and development and the negative connotation associated with this term (23). However, obesity is used within this study to provide consistency in classification terminology across the study group since 87% of the participants were ≥18 years of age at the time of this cross-sectional study.

Modified Healthy Eating Index

The USDA's HEI, a measure of dietary quality, assesses how well American's eating habits meet the recommendations of the Dietary Guidelines and the Food Guide Pyramid (13). This study used a modified version (24) that included 9 of the original 10 dietary components representing various aspects of a healthy diet with each component having equal weight. Components 1-5 related to meeting the recommended servings based on the Food Guide Pyramid food groups: grains, vegetables, fruit, milk products, and meat and meat alternatives. Component 6 and 7 measured total fat and saturated fat intake as a percent of energy. Component 8 and 9 measured total cholesterol and sodium intake. Since accurate information regarding dietary variety (component 10 of original HEI) was not available through the BFFQ, this component was excluded. Each component of the HEI had a maximum score of 10 and a minimum score of 0. Higher scores represented intakes closer to the recommended goals. Scores between the minimum and maximum values were computed proportionally. All HEI scores on the modified version were adjusted to a 100-point scale. A modified HEI score >80 implied a "good" diet, a score between 51 and 80 implied the diet "needs improvement", and a score of <51 indicated a "poor" diet (25).

HIV related information

Laboratory tests were performed at local clinic sites according to REACH protocol described elsewhere (19,20). HIV serum antibody tests and CD4+ T-cells were obtained on all study participants. Quantitative immunophenotyping of CD4+ T-cells were determined at the individual clinical sites in certified laboratories using AIDS Clinical Trials Group (ACTG) standardized flow cytometry protocols. HIV status was used as a dichotomous

variable. In addition, total CD4+ T-cells for HIV-infected participants were stratified based on Centers for Disease Control and Prevention criteria for HIV/AIDS classification: ≥500, 200-499, and <200 cells/μL (26). Other HIV related information such as use of antiretroviral therapies was obtained through face-to-face interview and medical record abstraction.

Demographic, health and behavior characteristics

Demographic characteristics including race/ethnicity, family financial situation, living arrangements, education, and substance use were obtained through face-to-face interviews and interactive computer interviews. Other variables obtained from the BFFQ included vitamin/mineral and herbal supplement use, hours spent watching television, pregnancy status, previous dieting history, cigarette smoking, alcohol use, and self-reported health status. Hours of television watching served as an indirect indicator of physical inactivity. To determine regional differences in dietary quality, study sites were group by geographic locations. The majority of study sites were located in the Northeast area (New York City area, Philadelphia PA, Baltimore MD, and Washington DC) and South (Florida, Alabama, Tennessee, and Louisiana) with only one study site in the Chicago area and one in the Los Angeles area.

Statistical analysis

Continuous variables were reported as mean ± standard error of mean (SE); significant differences by HIV infection were determined by two-sample Student's *t*-tests. Differences by CD4+ T-cell stratification were determined using Analysis of Variance (ANOVA) with post hoc differences using Bonferroni multiple comparisons. For categorical variables, differences were determined by Chi-squared test of goodness-of-fit. Correlations between continuous variables were determined using Pearson's correlations test.

A model was developed *a priori* based on a review of the literature and data available through the REACH project to explain the relationships between HEI, BMI, and HIV-infection. Generalized linear regression analysis was used to describe the associations between HEI and the explanatory variables; logistic regression was used for risk of obesity. Initial regression models included the following explanatory variables: health behaviors such as supplement use, antiretroviral therapy use, history of previous weight loss attempts, and television watching; environmental factors such as family financial situation, living

arrangements and food security; participant characteristics such as sex, self-reported health status, race/ethnicity, HIV infection and CD4+ T-cell strata; and dietary intake information such as energy intake. Dummy variables were created for categorical variables. Variables that reached a significance of $p<0.10$ or were central to the primary hypothesis remained in the model. SYSTAT (version 10.0) statistical software (27) was used for all data analysis. Significance was set at $p<0.05$ unless otherwise indicated.

Results

Participant characteristics

A total of 364 participants were included in the analysis. HIV-infected participants were older, more likely to on their own, and likely to have completed or be enrolled in high school or GED program than HIV-uninfected participants (Table 1). Almost half (46.8%) of the HIV-infected group were not on antiretroviral therapy (ART). Of those on ART, 34.3% were on a mono- or combination therapy without a protease inhibitor (PI) while 19.0% were on a combination therapy that included a PI. The mean BMI for this population was 28.1 ± 0.5 kg/m^2 (Median: 25.6, range: 15.3-64.6 kg/m^2). The average 6-mo weight gain for all participants was 1.5 ± 0.3 kg. Obese participants gained significantly more weight over the past six months than non-obese participants (2.7 ± 0.6 vs. 0.9 ± 0.3 kg, $p<0.01$). Due to growth differences and BMI differences by age and sex during adolescents, interpretation of absolute BMI values in this population was difficult. Weight classification described earlier was used to provide a basis for equivalent comparisons in this population. Among all participants in this study, over half were overweight or obese (Table 1). More females were overweight or obese compared to males (60.6% vs. 26.7% respectively, $p<0.001$). Over half (54.3%) of the HIV-uninfected participants and 49.4% of HIV-infected participants were overweight or obese. Among HIV-infected participants, the prevalence of overweight or obesity decreased with decreasing CD4+ T-cell strata (59.7%, 45.7%, and 28.1% respectively, $p<0.01$). The prevalence of overweight and obesity was compared by CD4+ T-cell strata and by sex (Figure 1). There was no significant difference in prevalence of overweight when compared by CD4+ T-cell strata for either males or females. When the prevalence of obesity was compared by CD4+ T-cell strata, no significant difference was seem among males; however, females had a significant decrease in obesity as CD4+ T-cells

decreased. A higher prevalence of obesity was seen in the South (37.3%) and the Northeast (32.0%) than the Chicago and Los Angeles area (14.3%, $p<0.05$). A significant inverse relationship was found between energy and BMI (Pearson's $r = -0.15$). Additional information about participant characteristics, including macro- and micronutrient intakes for this population was reported elsewhere (Chapter 3).

Using logistic regression, HIV-infected participants with CD4+ T-cells ≥500 cells/μL were 15% more likely to be obese compared to the HIV-uninfected participants; however, the risk of obesity decreased progressively as the CD4+ T-cells dropped below 500 cells/μL (Table 2). Being female was associated with an almost 3-fold increase in obesity compared to males. Individuals living on their own were almost twice as likely to be obese compared to those who had other living arrangements such as with family members or friends. Watching three or more hours of television per day was associated with a two-fold increase in obesity while having a history of previous weight loss attempts had an over 7-fold increase in the risk of obesity. Participants from the Northeast and the South were about four times more likely to be obese compared to participants from the Chicago or Los Angeles area. Race/ethnicity, financial status, antiretroviral therapy use, modified Healthy Eating Index and energy intake were not significant predictors of obesity and were dropped from the final model.

Modified Healthy Eating Index

The modified HEI score for the REACH population was 56.2 ± 0.6 out of 100. Of the 9 component scores, the highest was the meat score at 9.0 ± 0.1 and sodium, fruit and fat scores were the lowest (3.8 ± 0.2, 4.3 ± 0.2, and 4.6 ± 0.2; respectively). Approximately 68% of the participants had a diet that needed improvement while 31% had a poor diet.

Modified HEI scores varied by HIV status, sex, and region. HIV-infected participants had a lower HEI score (poorer diet) than HIV-uninfected participants (Table 3, $p<0.01$). When comparing HEI component scores, total fat, saturated fat ($p<0.001$), and cholesterol ($p<0.01$) scores were significantly lower (indicating higher intake) for HIV-infected participants than HIV-uninfected participants. HEI component scores were compared by sex; males had a higher average dairy score than females (5.9 ± 0.3 vs. 4.8 ± 0.2, $p<0.01$). Females, however, had significantly higher scores for fruits, cholesterol, and

sodium compared to males (4.6 ± 0.2 vs. 3.6 ± 0.3, 5.1 ± 0.3 vs. 3.5 ± 0.4, and 4.2 ± 0.3 vs. 2.9 ± 0.4, respectively; $p<0.01$) contributing to the overall higher total HEI score seen among females than males (57.1 ± 0.7 vs. 53.5 ± 1.1, $p<0.01$). Total HEI scores did not differ by race/ethnicity. While overall HEI scores did not differ between obese and non-obese participants, fruit and dairy component scores were significantly lower among obese compared to non-obese while cholesterol and sodium component scores were significantly higher (indicating lower intake) among obese than the non-obese. Participants from the Chicago area had lower HEI scores compared to those from the Northeast, South, or Los Angeles area (47.2 ± 2.3 vs. 56.7 ± 0.6, $p<0.001$). HIV-infected females had significantly lower HEI scores than HIV-uninfected females (Table 3); only saturated fat was significantly lower (indicating higher intake) among HIV-infected females compared to HIV-uninfected females ($p<0.01$). HEI scores were lower among HIV-infected males compared to HIV-uninfected males with cholesterol and fat component scores contributing to the overall difference. Vitamin C and folate intakes (data not shown) were positively correlated with HEI ($r=0.38$, $p<0.001$; $r=0.23$, $p<0.01$, respectively) while dietary fiber tended to be positively correlated ($r=0.19$, $p=0.051$).

Predictors of modified HEI using generalized linear regression were identified (Table 4). Being from the Chicago area, HIV-infected, and watching television three or more hours/day were significant negative predictors of overall HEI score while being female and having a self-perceived health status of good, very good, or excellent were positive predictors. In this model, other factors such as living arrangements, weight status, energy intake, family financial status, race/ethnicity, age, and education were not significant. To better illustrate this model, best and worst examples are presented. A HIV-uninfected female from the South with excellent self-perceived health who watched fewer than three hours of television per day had an estimated HEI of 61.8 (diet needs improvement). A HIV-infected male from the Midwest with a poor self-perceived health who watched more than three hours of television per day would have an estimated HEI of 42.2 (poor diet). When the dichotomous HIV infection variable was replaced by the three CD4+ T-cell strata variables (data not shown), the regression coefficients were negative and the largest effect was among those with a CD4+ T-cells 200-499 cells/μL (-3.78; 95% CI: -6.87, -0.68).

Discussion

The high prevalence of overweight and obesity seen in this population is consistent with the increasing prevalence of obesity seen among U.S. adolescents and young adults in recent national surveys (1,2). This trend is particularly evident in black/non-Hispanic and Hispanic adolescents (28,29). In this study population, the majority were black/non-Hispanic females, while 20% of the subjects reported Hispanic ethnicity. Approximately 31% were obese, twice as high as the prevalence among U.S. adolescents from the 1999 NHANES IV (2).

Many factors no doubt contribute to this high prevalence of obesity. One possibility is that female subjects in this present study see a larger body size as desirable. Anecdotal reports from clinicians in our study tend to support this supposition, as they suggest that weight gain occurred in some subjects after being diagnosed with HIV infection in reaction to the fear of weight loss associated with advanced HIV infection (Wilson, personal communications, 2002). While information relating to body image and attitudes are not available in this study, others have reported ethnic differences that could influence BMI among women with HIV infection (30). Black, HIV-infected women were more likely to want a larger body size while Caucasian, HIV-infected women wanted to lose weight. These findings did not depend on stage of illness or history of opportunistic infections. These ethnic differences in body image and obesity reported among HIV-infected women were similar to those seen among other black non-Hispanics adults and adolescents (31-33). While the strong association of obesity with weight loss attempts seen in our subjects argues against intentional weight gain, the possibility remains that BMIs in the overweight and obese range are not seen as negatively by subjects in this study as they are by other population groups.

Regional differences were seen in the prevalence of obesity in the REACH subjects. In this study, participants from the South were more likely to be black/non-Hispanic, a group with high rates of obesity (3). Subjects from the South also reported lower food security and family financial situation compared to participants from other regions (data not shown). Food insecurity has been associated with higher BMIs in U.S. women (34). When BMI was compared by region, the South had significantly higher mean BMI than the Northeast area

and the Los Angeles site. The Northeast area was as significant predictor of obesity. This region had the second highest prevalence of obesity following the South. Participants from the Northeast had more educated mothers and tended to be more educated themselves. There were no other sociodemographic differences seen in participants from this area compared to the other regions. It is unclear what factors in this region are influencing obesity.

Weight loss and wasting are often associated with advanced HIV infection (35,36). However, most previous studies have not been done in populations with a high prevalence of overweight and obesity. In the REACH population, wasting was rare but the prevalence of obesity was lower in those with the most advanced disease (i.e., lowest CD+ T-cells). This effect could be the result of decreased intake, or altered digestion, absorption, or metabolism of nutrients resulting from HIV infection, opportunistic infections or drug therapy side effects (37,38). Thus the lower prevalence of obesity in REACH subjects with more advanced infection may be due to the same factors that cause wasting in populations where the overall BMI is lower than in the present study. Overweight and obesity in this study population could lead to increased complications relating to obesity, which may be further compounded by metabolic complications such as hyperlipidemia and insulin resistance associated with HIV infection and antiretroviral therapies (16,17).

The HEI scores of this population were lower than those reported from a nationally representative survey. U.S. adolescent females aged 15-18 years had HEI scores of 60.8 while males of the same age were slightly lower at 60.7 (13). Nationally, whites had higher average HEI scores than black/non-Hispanics among all ages; however, no significant difference in HEI scores by race/ethnicity was seen in this study. Regional differences in HEI scores reported in this study differ from those reported by Bowman et al. (13). They found that people from the Northeast region had the highest average HEI scores at 65 while those from the South had the lowest average scores of 62. In our study, participants from the Chicago area had the lowest average HEI scores. They had significantly lower component scores for grains, fruits, fat, and saturated fat, which contributed to their overall lower HEI score. More males were from the Chicago area (43%) compared to other areas (26%); however, after controlling for sex, being from the Chicago area remained a significant negative predictor of dietary quality. No other significant differences in sociodemographic

characteristics were seen between participants from the Chicago area compared to other regions. Fewer participants were obese compared to the Northeast or South. It is difficult to draw any conclusions between these regional variances noted since our sample was not designed to be regionally representative.

Although no relationship was seen between overall HEI scores and BMI in this study, other researchers have reported that a higher HEI score was associated with a lower BMI (13). In our study, there was an inverse relationship between energy intake and BMI suggesting under-reporting among those with increased weight. Factors that are commonly associated with under-reporting that are relevant to this analysis include being obese, female, black non-Hispanic, and of lower socioeconomic status (39-41).

Unlike the results from this study, other researchers have found a positive relationship among income, education and HEI scores (13,42,43). In this study, variables representing these socio-demographic characteristics were not significantly associated with HEI scores. Our study population may have had less diversity in income and education variables than did other studies, thus making it difficult to find an association with HEI. Income was not significant in this study, which could relate to the variable used in the analysis. Participants may have been being unaware of detailed family finances especially for those still living with parents. Thus, variables in this study reflecting household income and education may not be accurately represented. This could explain why only 8% of the total variance was explained using linear regression in this study.

Correlations between vitamin C, folate, fiber and the HEI found in this study are consistent with those reported by Hann et al (43). Although serum micronutrients are not reported here, HEI has been positively correlated with vitamin C and various carotenoids and negatively correlated with serum cholesterol (43). Since the HEI was developed based on current dietary recommendations and has been strongly correlated to certain micronutrient intake and serum concentrations, the HEI served as a useful tool to describe overall dietary quality in this population.

Applications/conclusions

To our knowledge, this is the first study to examine dietary quality using the HEI and the prevalence of overweight and obesity in minority adolescents and young adults with HIV

infection. The growing prevalence of overweight and obesity seen among the U.S. population was also seen among this study population composed primarily of black, non-Hispanic females. Overweight and obese individuals with HIV infection may be at even greater risk of developing metabolic abnormalities associated with HIV infection. Dietary quality was lower among HIV-infected than HIV-uninfected participants, and the majority of this population fell short of the dietary recommendations measured by the modified HEI. Since underreporting has been identified among obese individuals, nutrition educators should utilize various assessment techniques that provide an accurate reflection of dietary intake. In addition, improving self-awareness of eating behaviors through use of food records, food models, and other educational strategies may help promote healthy eating behaviors. Nutrition education should focus on developing individualized behavioral goals emphasizing improved dietary quality and physical activity to improve the quality of life for all adolescents and young adults.

Table 1. Participant sociodemographic characteristics by HIV status for the REACH cohort study [a]

	Total	HIV+	HIV-
n	**364**	**248**	**116**
Age, y	19.8 ± 0.1 [b]	20.0 ± 0.1***	19.4 ± 0.1***
Range	13.8 - 23.2	13.8 - 23.2	14.8 - 22.9
Male	97 (26.6) [c]	68 (27.4)	29 (25.0)
Female	267 (73.4)	180 (72.6)	87 (75.0)
Race/ethnicity [#]			
Hispanic	75 (20.6)	47(19.0)	28 (24.1)
Black/non-Hispanic	242 (66.5)	175 (70.6)	67 (57.8)
White/non-Hispanic	16 (4.4)	8 (3.2)	8 (6.9)
Other	31 (8.5)	18 (7.3)	13 (11.2)
Living arrangements **			
Living in own apartment/house	101 (27.7)	81 (32.7)	20 (17.3)
Parents/family member	207 (56.9)	127 (51.2)	80 (69.0)
Someone else's house/apt	25 (6.9)	17 (6.9)	8 (6.9)
Other [d]	31 (8.5)	23 (9.3)	8 (6.9)
Education ***			
No diploma or GED	127 (34.9)	103 (41.5)	24 (20.7)
Diploma/GED or enrolled in school	237 (65.1)	145 (58.5)	92 (79.3)
CD4+ T-cells (cells/μL)			
≥500		124 (50.0)	--
200-499		92 (37.1)	--
<200		32 (12.9)	--
Weight classification			
Overweight [e]	73 (20.1)	53 (21.4)	20 (17.2)
Obese [f]	115 (31.6)	72 (29.0)	43 (37.1)
Self-reported health status			
Excellent, very good, good	229 (62.9)	160 (64.5)	69 (59.5)
Fair, poor	135 (37.1)	88 (35.5)	47 (40.5)
Vitamin/mineral supplement use	129 (35.4)	89 (35.9)	40 (34.4)
Television watching ≥3 hr/d	177 (48.6)	127 (51.2)	50 (43.1)
History of weight loss attempts *	130 (35.7)	81 (32.7)	52 (44.8)
Currently smoke	151 (41.5)	109 (44.0)	42 (36.2)

[a] Pregnant women excluded; [b] Mean ± standard error; [c] n (%)

[d] Living in halfway house, foster or group home, or on the street

[e] BMI ≥25 and <30 kg/m^2 or BMI-for-age ≥85th and <95th percentile

[f] BMI ≥30 kg/m^2 or BMI-for-age ≥95th percentile

*** p <0.001, ** p <0.01, * p <0.05, [#] p <0.10 (Chi^2 for categorical variables; Two sample *t*-tests for continuous variables)

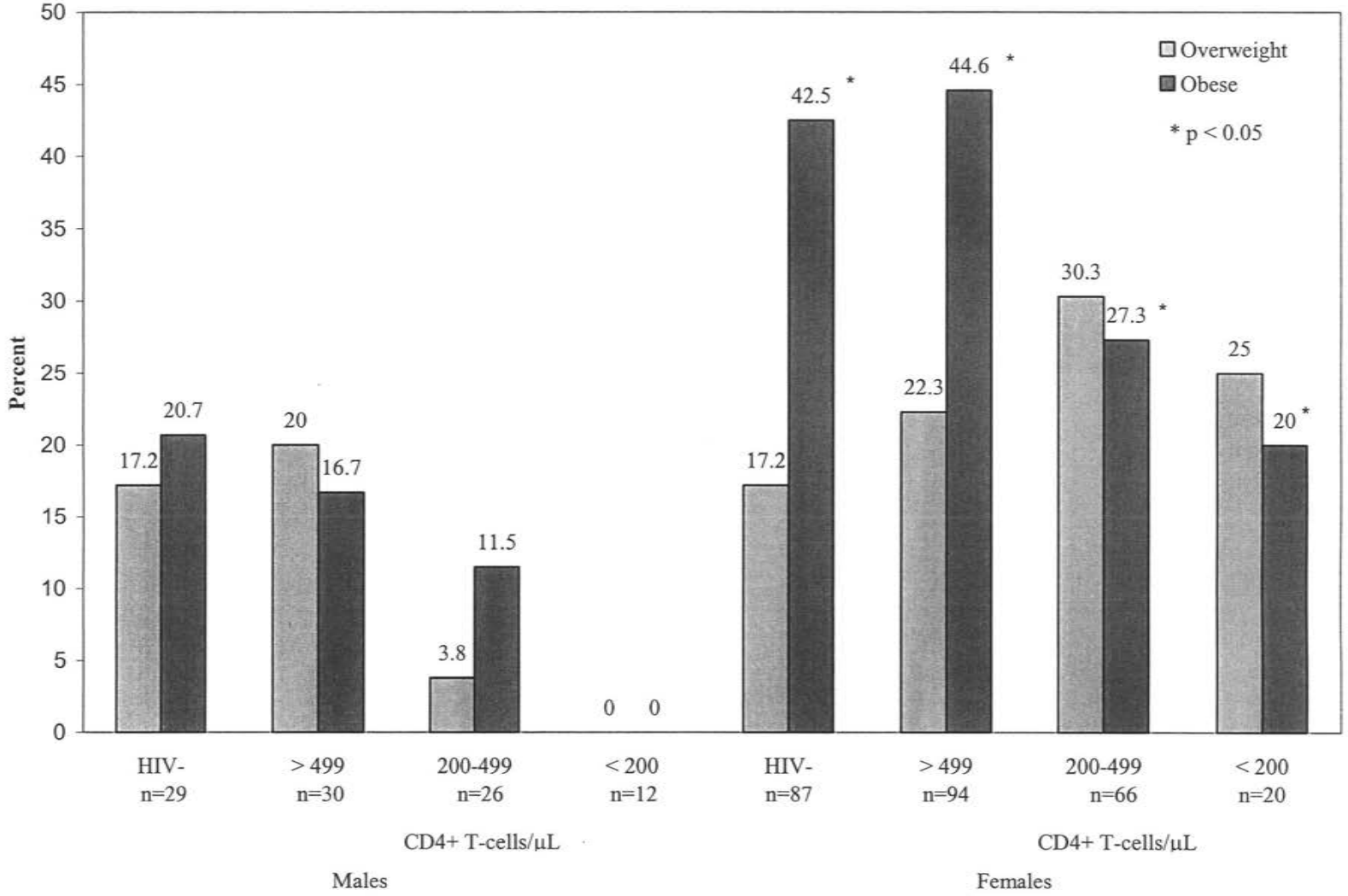

Figure 1. Prevalence of overweight and obesity by CD4+ T-cells and sex in the REACH cohort study

Table 2. Logistic regression model predicting obesity in the REACH cohort study [a] (n=364)

Dependant variable	Obesity			
Independent variables	**Coefficient**	**SE**	**Odds ratio**	**p-value**
Constant	-4.14	0.66		0.000
Female	1.09	0.36	2.97	0.003
Living in own apartment/house	0.64	0.30	1.90	0.030
HIV+/CD4+ T-cells (cells/μL): [b]				
≥500	0.14	0.32	1.15	0.658
200-499	-0.83	0.37	0.44	0.025
<200	-1.16	0.63	0.31	0.066
Northeast region [c]	1.32	0.50	3.74	0.009
South region [d]	1.42	0.52	4.11	0.006
Television watching ≥3 hr/d	0.82	0.28	2.26	0.003
History of weight loss attempts	2.06	0.29	7.83	0.000

[a] Pregnant women excluded

[b] HIV-uninfected participants are 0 for all three CD4+ T-cell variables

[c] Includes New York City area, Philadelphia PA, Baltimore MD, and Washington DC

[d] Includes Florida, Alabama, Tennessee, and Louisiana

Table 3. Modified Healthy Eating Index (HEI) scores by sex and HIV status in the REACH cohort study [a]

	Total		Females		Males	
	HIV+	HIV-	HIV+	HIV-	HIV+	HIV-
n	248	116	180	87	68	29
Component Scores [b]						
Grains	7.0 ± 0.2 [d]	6.8 ± 0.2	6.9 ± 0.2	6.9 ± 0.3	7.1 ± 0.3	6.5 ± 0.5
Fruits	4.2 ± 0.2	4.6 ± 0.3	4.4 ± 0.2	4.8 ± 0.3	3.4 ± 0.3	4.2 ± 0.5
Vegetables	6.2 ± 0.2	5.8 ± 0.3	6.3 ± 0.2	5.8 ± 0.3	6.0 ± 0.3	5.7 ± 0.5
Dairy	5.3 ± 0.2 #	4.6 ± 0.3 #	5.1 ± 0.3 #	4.3 ± 0.3 #	6.1 ± 0.4	5.4 ± 0.6
Meats	9.1 ± 0.1	9.0 ± 0.2	9.0 ± 0.2	8.9 ± 0.2	9.3 ± 0.2	9.3 ± 0.3
Fat	4.4 ± 0.2 *	5.1 ± 0.3 *	4.6 ± 0.3	5.1 ± 0.3	3.9 ± 0.4 *	5.1 ± 0.4 *
Saturated fat	5.6 ± 0.2 ***	6.8 ± 0.3 ***	5.6 ± 0.3 **	6.8 ± 0.3 **	5.4 ± 0.4 #	6.6 ± 0.6 #
Cholesterol	4.2 ± 0.3 **	5.7 ± 0.4 **	4.7 ± 0.3 #	5.8 ± 0.5 #	2.9 ± 0.5 *	5.1 ± 0.8 *
Sodium	3.6 ± 0.3 #	4.4 ± 0.4 #	3.9 ± 0.3	4.6 ± 0.4	2.6 ± 0.5	3.6 ± 0.7
HEI [c]	55.2 ± 0.7 **	58.8 ± 1.1 **	56.1 ± 0.9 *	59.2 ± 1.3 *	51.9 ± 1.3 *	57.3 ± 1.9 *
Good (HEI>80)	1 (0.4) [e]	0 (0.0)	1 (0.6)	0 (0.0)	0 (0.0)	0 (0.0)
Needs improvement (HEI 51-80)	164 (66.1)	85 (73.3)	123 (68.3)	65 (74.7)	41 (60.3)	20 (69.0)
Poor (HEI<51)	83 (33.5)	31 (26.7)	56 (31.1)	22 (25.3)	27 (39.7)	9 (31.0)

[a] Pregnant women excluded
[b] Minimum score=0, Maximum score=10
[c] Excluding variety component based on 100 points
[d] Mean ± standard error
[e] n (%)
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, # $p < 0.10$
Difference by HIV status tested with two sample *t*-tests (males and females tested separately); Chi^2 for categorical variables

Table 4. Multiple linear regression model predicting the modified Healthy Eating Index in the REACH cohort study[a] (n=364)

Dependant variable	Modified Healthy Eating Index			
Independent variables	**Coefficient**	**SE**	***t***	**p-value**
Constant	56.4	1.8	32.1	0.000
Female	3.2	1.3	2.4	0.016
Health status: excellent, very good or good	2.2	1.2	1.8	0.069
HIV-infected	-3.4	1.3	-2.7	0.007
Chicago area	-8.4	2.5	-3.3	0.001
Television watching ≥3 hr/d	-2.4	1.2	-2.0	0.046
N	364			
Adjusted R^2	0.079			
Std error of estimate	11.2			

[a] Pregnant women excluded

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CHAPTER 5. GENERAL CONCLUSIONS

To our knowledge, this study was the first to examine dietary intake among HIV-infected and HIV-uninfected adolescents and young adults. The first study focusing on micronutrient intakes showed two indicators of poor diet quality: 1) excessive fat, saturated fat, and cholesterol, and 2) inadequate intakes of micronutrients that are important for maintenance of a healthy immune response and have been associated with a slowed progression of HIV disease. Energy intake was a significant positive predictor for vitamins A, C, E, iron and zinc. After controlling for energy intake, early HIV infection was associated with a slightly lower micronutrient intake. Vitamin/mineral supplement use was common in this population and was related to more nutrient-dense food choices.

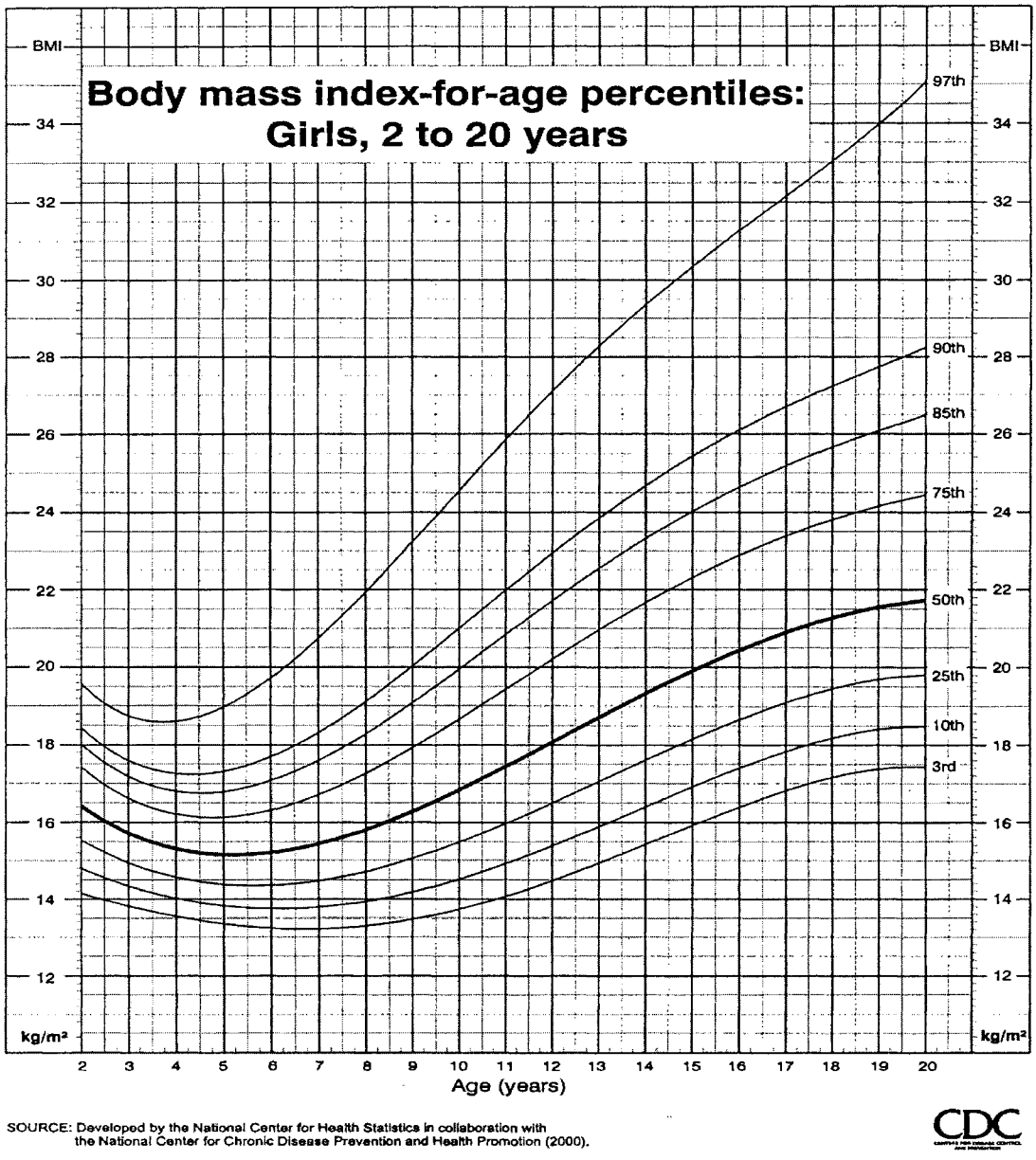
In the second study, overweight and obesity was common in this population composed primarily of black/non-Hispanic females. Prevalence of obesity decreased with decreased CD4+ T-cells. In addition, overweight or obese HIV-infected individuals may have increased weight-related complications, which may further compound metabolic complications such as hyperlipidemia and insulin resistance associated with HIV infection and ART. Overall dietary quality as measured by the HEI was lower among HIV-infected participants. However, the majority of all participants fell short of the dietary recommendations measured by the HEI. Factors positively associated HEI scores included being female and having a higher self-perceived health status, while being HIV-infected and increased television watching were negatively associated with HEI. Another finding in this population was an inverse relationship between energy intake and BMI suggesting under-reporting occurred among those with increased weight.

While this project provides insight on the dietary intake among HIV-infected and HIV-uninfected adolescents and young adults, further research is needed in this area. Additional studies are needed to determine the optimal amounts of micronutrient intake among HIV-infected youth. Since this was a cross-sectional study on dietary intake, we were unable to examine the relationship between dietary intake and disease progression. Since both deficient and excessive micronutrient intakes have been related to advanced disease progression among adults, studies are needed to examine this relationship among HIV-infected adolescents. In addition, biochemical and body composition information was not

available on these subjects to examine the relationship between dietary intake, obesity and complications associated with HIV infection and ART such as insulin resistance, hyperlipidemia, or lipodystrophy. Further investigation on body image and attitudes among HIV-infected individuals of different ethnic backgrounds would also be beneficial. By understanding the psychological reasons for weight preference, health professionals would be better informed for assisting clients in improved their health status. Nutrition education should focus on developing individualized behavioral goals emphasizing improved dietary quality and physical activity to improve quality of life for all adolescents and young adults.

APPENDIX A. BODY MASS INDEX-FOR-AGE GROWTH CHARTS

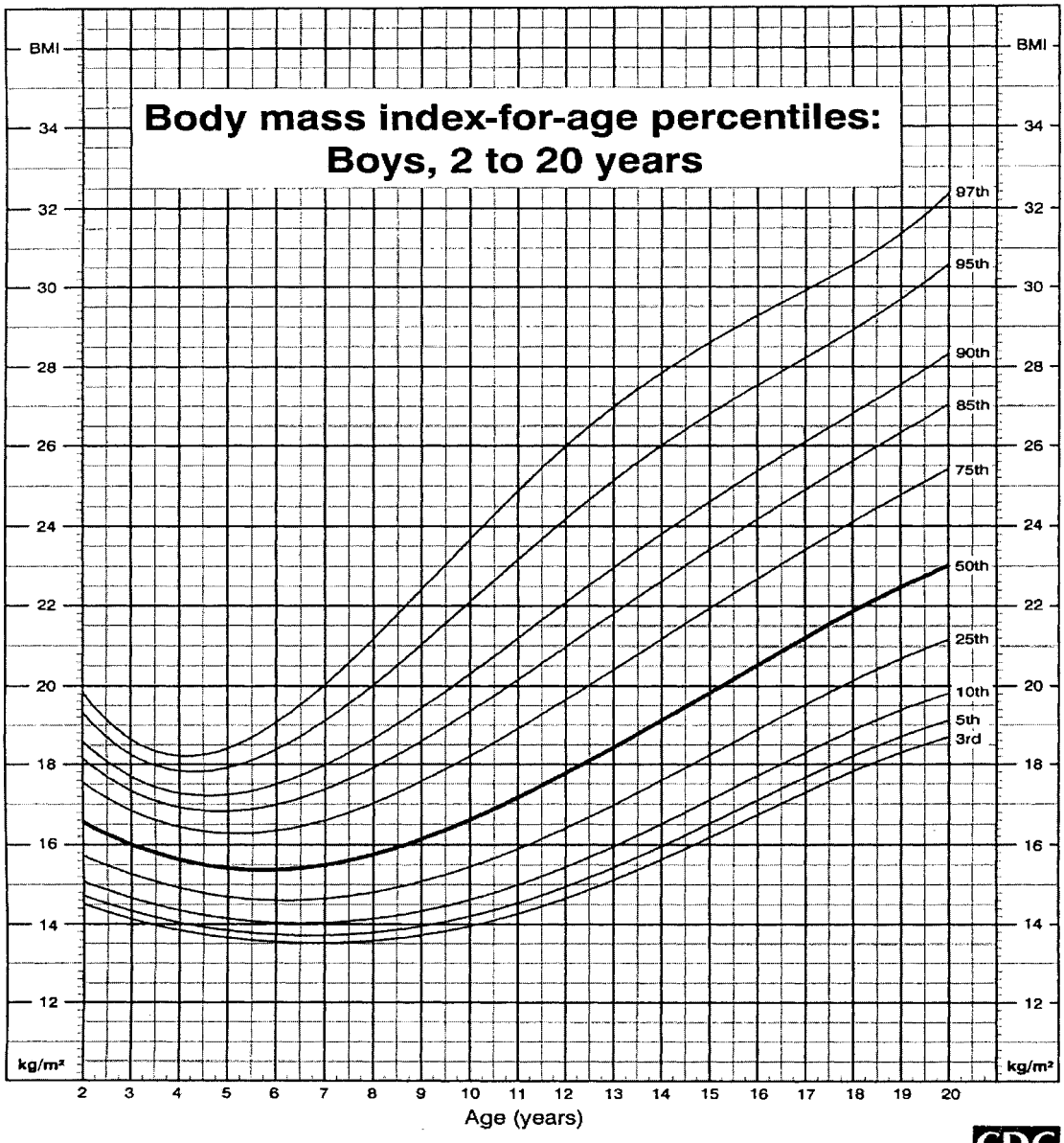
CDC Growth Charts: United States



Reference

Centers for Disease Control and Prevention, National Center for Health Statistics. CDC growth charts: United States. 2000. Available at: http://www.cdc.gov/growthcharts/. Accessed: October 15, 2001.

CDC Growth Charts: United States



Published May 30, 2000.
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).

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APPENDIX B. 1993 REVISED CLASSIFICATION SYSTEM FOR HIV INFECTION

Table 1. 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults ≥13 y. (CDC, 1992).

CD4 T-cell categories	Clinical Categories		
	(A) Asymptomatic HIV infection[a]	(B) Symptomatic HIV infection[b]	(C) AIDS-indicator conditions[c]
(1) ≥500	A1 Early	B1 Early	C1 Intermediate
(2) 200-499	A2 Early	B2 Intermediate	C2 Late
(3) <200	A3 Intermediate	B3 Late	C3 Late

[a] Category A:
One or more of the conditions listed below in an adolescent or adult (>13 y) with documented HIV infection. Conditions listed in categories B and C must not have occurred.

- Asymptomatic HIV infection
- Persistent generalized lymphadenopathy
- Acute (primary) HIV infection with accompanying illness or history of acute HIV infection

[b] Category B:
Symptomatic conditions occurring in an HIV-infected adolescent or adult that are not included among conditions listed in clinical category C, and that meet at least on of the following criteria:

- Bacillary angiomastosis
- Candidiasis, oropharyngeal (thrush)
- Candidiasis, vulvovaginal; persistent, frequent, or poorly responsive to therapy
- Cervical dysplasia (moderate to severe) or cervical carcinoma
- Consititutional symptoms, such as fever (38.5 C) or diarrhea lasting >1 month
- Hairy leukoplakia, oral
- Herpes zoster (shingles), involving at least two distinct episodes or more than one dermatome
- Idiopathic thrombocytopenic purpura
- Listeriosis
- Pelvic inflammatory disease, particularly if complicated by tubo-ovarian abscess
- Peripheral neuropathy

[c] Category C:
Any condition listed in the 1987 surveillance case definition for AIDS and affecting an adolescent or adult. These conditions are strongly associated with severe immunodeficiency, occur frequently in HIV-infected individuals, and cause serious morbidity or mortality. Once a category C condition has occurred, the person will remain in category C.

1. Candidiasis of bronchi, trachea, or lungs

2. Candidiasis, esophageal
3. Cervical cancer, invasive
4. Coccidioidomycosis, disseminated or extrapulmonary
5. Cyrptocovvosis, extrapulmonary
6. Cryptosporidiosis, chronic intestinal (>1 month's duration)
7. Cytomegalovirus disease (other than liver, spleen, or nodes)
8. Cytomegalovirus retinitis (with loss of vision)
9. Encephalopathy, HIV-related
10. Herpes simplex: chronic ulcer(s) (>1 month's duration); or bronchitis, pneumonitis, or esophagitis
11. Histoplasmosis, disseminated or extrapulmonary
12. Isosporiasis, chronic intestinal (>1 month's duration)
13. Kaposi's sarcoma
14. Lymphoma, Burkitt's (or equivalent term)
15. Lymphoma, immunoblastic (or equivalent term)
16. Lymphoma, primary, of brain
17. *Mycobacterium avium* complex of *M. kansasii*, disseminated or extrapulmonary
18. *Mycobacterium tuberculosis*, any site (pulmonary or extrapulmonary)
19. *Mycobacterium*, other species or unidentified species, disseminated or extrapulmonary
20. *Pneumocystis carinii* pneumonia
21. Pneumonia, recurrent
22. Progressive multifocal leukeoencephalopathy
23. *Salmonella* septicemia, recurrent
24. Toxoplasmosis of brain
25. Wasting syndrome due to HIV

Reference

Centers for Disease Control and Prevention. 1993 Revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. *Mor Mortal Wkly Rep CDC Surveill Summ.* 1992;41(No. RR-17):1-17.

APPENDIX C. BLOCK FOOD FREQUENCY QUESTIONNAIRE (98.2)

RESPONDENT ID NUMBER

TODAY'S DATE

Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec | DAY | YEAR: 1998 1999 2000 2001 2002 2003 2004 2005 2006 2007

FOOD QUESTIONNAIRE

This form is about the foods you usually eat.
It will take about 30 - 40 minutes to complete.

- Please answer each question as best you can. Estimate if you aren't sure.
- Use only a No. 2 pencil.
- Fill in the circles completely, and erase completely if you make any changes.

Please print your name in this box.

SEX: Male, Female

If female, are you pregnant or breast feeding? No, Yes, Not female

AGE

WEIGHT pounds

HEIGHT ft. in.

First, a few general questions about what you eat.	AVERAGE USE IN THE PAST YEAR: LESS THAN ONCE per WEEK	1-2 per WEEK	3-4 per WEEK	5-6 per WEEK	1 per DAY	1 1/2 per DAY	2 per DAY	3 per DAY	4+ per DAY
About how many servings of vegetables do you eat, per day or per week, not counting salad or potatoes?	○	○	○	○	○	○	○	○	○
About how many servings of fruit do you eat, not counting juices?	○	○	○	○	○	○	○	○	○
How often do you eat cold cereal?	○	○	○	○	○	○	○	○	○
How often do you use fat or oil in cooking?	○	○	○	○	○	○	○	○	○

What kinds of fat or oil do you usually use in cooking? MARK ONLY ONE OR TWO

- ○ Don't know, or Pam
- ○ Stick margarine
- ○ Soft tub margarine
- ○ Butter
- ○ Butter/margarine blend
- ○ Low-fat margarine
- ○ Corn oil, vegetable oil
- ○ Olive oil or canola oil
- ○ Lard, fatback, bacon fat
- ○ Crisco

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Reference

Block Dietary Data Systems. Food Questionnaire 98.2. Berkeley California: Block Dietary Data Systems, 1998.

During the past year, have you taken any vitamins or minerals regularly, at least once a month?

○ No, not regularly ○ Yes, fairly regularly →

(IF YES) WHAT DID YOU TAKE FAIRLY REGULARLY?

VITAMIN TYPE	HOW OFTEN					FOR HOW MANY YEARS?					
	DIDN'T TAKE	A FEW DAYS per MONTH	1-3 DAYS per WEEK	4-6 DAYS per WEEK	EVERY DAY	LESS THAN 1 YR.	1 YEAR	2 YEARS	3-4 YEARS	5-9 YEARS	10+ YEARS
Multiple Vitamins. Did you take...											
Regular Once-A-Day, Centrum, or Thera type	○	○	○	○	○	○	○	○	○	○	○
Stress-tabs or B-Complex type	○	○	○	○	○	○	○	○	○	○	○
Antioxidant combination type	○	○	○	○	○	○	○	○	○	○	○
Single Vitamins (not part of multiple vitamins)											
Vitamin A (not beta-carotene)	○	○	○	○	○	○	○	○	○	○	○
Beta-carotene	○	○	○	○	○	○	○	○	○	○	○
Vitamin C	○	○	○	○	○	○	○	○	○	○	○
Vitamin E	○	○	○	○	○	○	○	○	○	○	○
Folic acid, folate	○	○	○	○	○	○	○	○	○	○	○
Calcium, alone or combined with something else	○	○	○	○	○	○	○	○	○	○	○
Zinc, alone or combined with something else	○	○	○	○	○	○	○	○	○	○	○
Iron	○	○	○	○	○	○	○	○	○	○	○
Selenium	○	○	○	○	○	○	○	○	○	○	○

If you took Once-a-day, Centrum or Thera-type multiple vitamins, did you usually take types that ○ contain minerals, iron, zinc, etc. ○ do not contain minerals ○ don't know

If you took vitamin C or vitamin E:

How many milligrams of **vitamin C** did you usually take, on the days you took it?
○ 100 ○ 250 ○ 500 ○ 750 ○ 1000 ○ 1500 ○ 2000 ○ 3000+ ○ Don't know

How many IUs of **vitamin E** did you usually take, on the days you took it?
○ 100 ○ 200 ○ 300 ○ 400 ○ 600 ○ 800 ○ 1000 ○ 2000+ ○ Don't know

Did you take any of these supplements at least once a month?

○ Ginkgo ○ Ginseng ○ St. John's Wort ○ Kava Kava ○ Echinacea ○ Melatonin ○ DHEA
○ Glucosamine/Chondroitin ○ Something else ○ Didn't take these

---

The next section is about your usual eating habits in the past year or so. This includes all meals or snacks, at home or in a restaurant or carry-out. There are two kinds of questions to answer for each food:

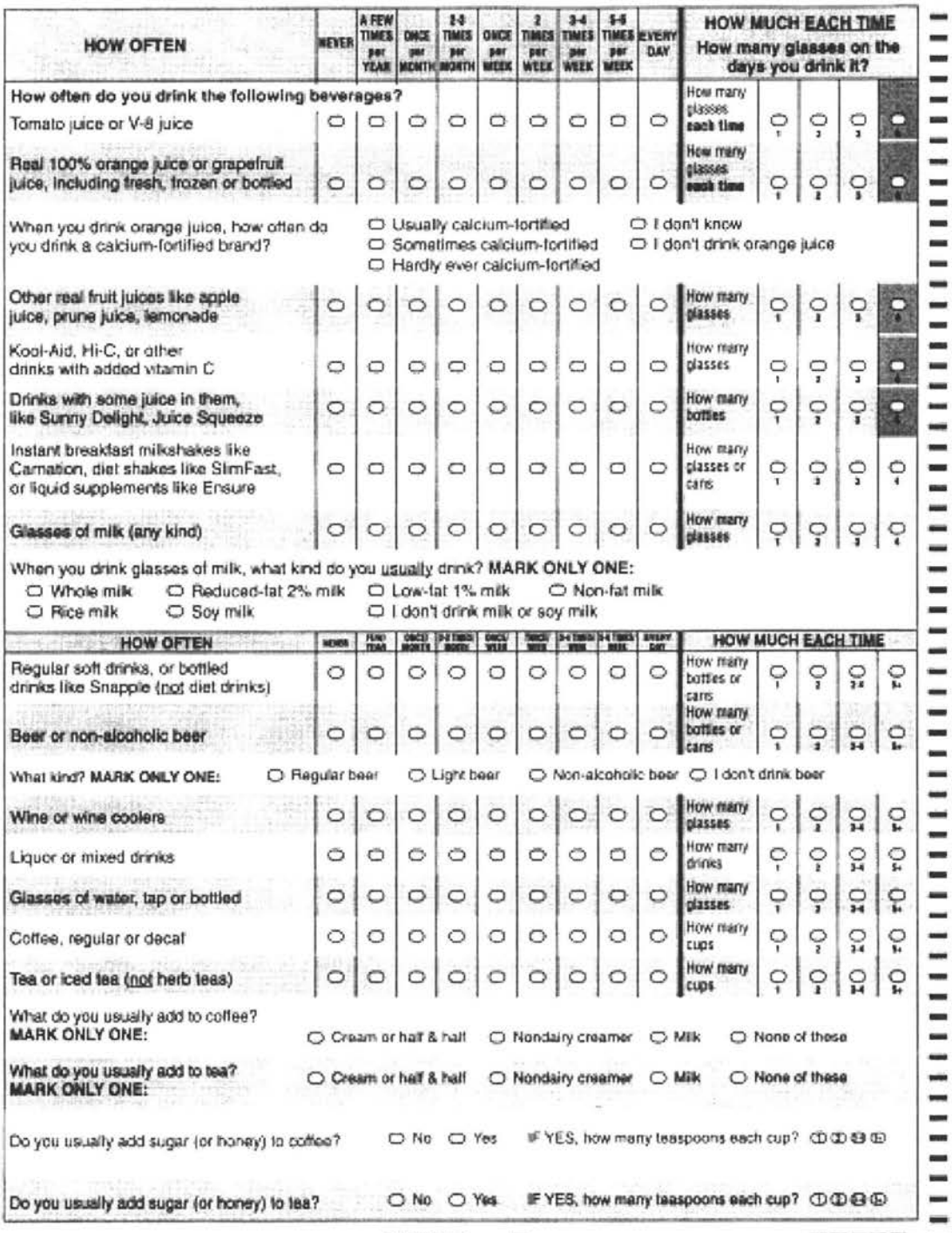
HOW OFTEN, on average, did you eat the food during the past year?
*Please DO NOT SKIP any foods. Mark "Never" if you didn't eat it.

HOW MUCH did you usually eat of the food?

*Sometimes we ask how many you eat, such as 1 egg, 2 eggs, etc., ON THE DAYS YOU EAT IT.
*Sometimes we ask "how much" as A, B, C or D. LOOK AT THE ENCLOSED PICTURES. For each food, pick the picture (bowls or plates) that looks the most like the serving size you usually eat.
(If you don't have pictures: A=1/4 cup, B=1/2 cup, C=1 cup, D=2 cups.)
*Sometimes we made the "D" column a darker color. This is just to remind you to make sure you really eat that large a serving.

EXAMPLE: This person drank apple juice twice a week, and had one glass each time. Once a week he ate a "C" sized serving of rice (about 1 cup).

HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MON.	2-3 TIMES per MON.	ONCE per WEEK	TWICE per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME SEE PORTION SIZE PICTURES FOR A-B-C-D				
Apple juice	○	○	○	○	○	●	○	○	○	How many glasses each time	● 1	○ 2	○ 3	○ 4
Rice	○	○	○	○	●	○	○	○	○	How much each time	○ A	○ B	● C	○ D



HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME — How many glasses on the days you drink it?				
How often do you drink the following beverages?														
Tomato juice or V-8 juice	○	○	○	○	○	○	○	○	○	How many glasses each time	○ 1	○ 2	○ 3	○ 4+
Real 100% orange juice or grapefruit juice, including fresh, frozen or bottled	○	○	○	○	○	○	○	○	○	How many glasses each time	○ 1	○ 2	○ 3	○ 4+
When you drink orange juice, how often do you drink a calcium-fortified brand?	○ Usually calcium-fortified ○ Sometimes calcium-fortified ○ Hardly ever calcium-fortified									○ I don't know ○ I don't drink orange juice				
Other real fruit juices like apple juice, prune juice, lemonade	○	○	○	○	○	○	○	○	○	How many glasses	○ 1	○ 2	○ 3	○ 4+
Kool-Aid, Hi-C, or other drinks with added vitamin C	○	○	○	○	○	○	○	○	○	How many glasses	○ 1	○ 2	○ 3	○ 4+
Drinks with some juice in them, like Sunny Delight, Juice Squeeze	○	○	○	○	○	○	○	○	○	How many bottles	○ 1	○ 2	○ 3	○ 4+
Instant breakfast milkshakes like Carnation, diet shakes like SlimFast, or liquid supplements like Ensure	○	○	○	○	○	○	○	○	○	How many glasses or cans	○ 1	○ 2	○ 3	○ 4
Glasses of milk (any kind)	○	○	○	○	○	○	○	○	○	How many glasses	○ 1	○ 2	○ 3	○ 4

When you drink glasses of milk, what kind do you usually drink? **MARK ONLY ONE:**

○ Whole milk ○ Reduced-fat 2% milk ○ Low-fat 1% milk ○ Non-fat milk
○ Rice milk ○ Soy milk ○ I don't drink milk or soy milk

HOW OFTEN	NEVER	FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME				
Regular soft drinks, or bottled drinks like Snapple (not diet drinks)	○	○	○	○	○	○	○	○	○	How many bottles or cans	○ 1	○ 2	○ 3-4	○ 5+
Beer or non-alcoholic beer	○	○	○	○	○	○	○	○	○	How many bottles or cans	○ 1	○ 2	○ 3-4	○ 5+

What kind? **MARK ONLY ONE:** ○ Regular beer ○ Light beer ○ Non-alcoholic beer ○ I don't drink beer

Wine or wine coolers	○	○	○	○	○	○	○	○	○	How many glasses	○ 1	○ 2	○ 3-4	○ 5+
Liquor or mixed drinks	○	○	○	○	○	○	○	○	○	How many drinks	○ 1	○ 2	○ 3-4	○ 5+
Glasses of water, tap or bottled	○	○	○	○	○	○	○	○	○	How many glasses	○ 1	○ 2	○ 3-4	○ 5+
Coffee, regular or decaf	○	○	○	○	○	○	○	○	○	How many cups	○ 1	○ 2	○ 3-4	○ 5+
Tea or iced tea (not herb teas)	○	○	○	○	○	○	○	○	○	How many cups	○ 1	○ 2	○ 3-4	○ 5+

What do you usually add to coffee? **MARK ONLY ONE:** ○ Cream or half & half ○ Nondairy creamer ○ Milk ○ None of these

What do you usually add to tea? MARK ONLY ONE: ○ Cream or half & half ○ Nondairy creamer ○ Milk ○ None of these

Do you usually add sugar (or honey) to coffee? ○ No ○ Yes IF YES, how many teaspoons each cup? ① ② ③ ④

Do you usually add sugar (or honey) to tea? ○ No ○ Yes IF YES, how many teaspoons each cup? ① ② ③ ④

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HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME SEE PORTION SIZE PICTURES FOR A-B-C-D				
How often do you eat each of the following fruits, just during the 2-3 months when they are in season?														
Raw peaches, apricots, nectarines, while they are in season	○	○	○	○	○	○	○	○	○	How many each time	○ 1/2	○ 1	○ 2	○ 3
Cantaloupe, in season	○	○	○	○	○	○	○	○	○	How much	○ 1/2	○ 1/1	○ 1/2	○ 2
Strawberries, in season	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Watermelon, in season	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Any other fruit in season, like grapes, honeydew, pineapple, kiwi	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
How often do you eat the following foods all year round? Estimate your average for the whole year.														
Bananas	○	○	○	○	○	○	○	○	○	How many each time	○ 1/2	○ 1	○ 2	○ 3
Apples or pears	○	○	○	○	○	○	○	○	○	How many each time	○ 1/2	○ 1	○ 2	○ 3
Oranges or tangerines	○	○	○	○	○	○	○	○	○	How many each time	○ 1/2	○ 1	○ 2	○ 3
Grapefruit	○	○	○	○	○	○	○	○	○	How much	○ 1/2	○ 1	○ 2	○ 3
Canned fruit like applesauce, fruit cocktail, or dried fruit like raisins	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D

HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME				
Eggs, including egg biscuits or Egg McMuffins (Not egg substitutes)	○	○	○	○	○	○	○	○	○	How many eggs each time	○ 1	○ 2	○ 3	○ 4
Bacon	○	○	○	○	○	○	○	○	○	How many pieces	○ 1	○ 2	○ 3	○ 4
Breakfast sausage, including sausage biscuits	○	○	○	○	○	○	○	○	○	How many pieces	○ 1	○ 2	○ 3	○ 4
Pancakes, waffles, French toast, Pop Tarts	○	○	○	○	○	○	○	○	○	How many pieces	○ 1	○ 2	○ 3	○ 4
Breakfast bars, granola bars, Power bars	○	○	○	○	○	○	○	○	○	How many	○ 1	○ 2	○ 3	○ 4
Cooked cereals like oatmeal, cream of wheat or grits	○	○	○	○	○	○	○	○	○	Which bowl		○ B	○ C	○ D
High-fiber cereals like All Bran, Raisin Bran, Fruit-n-Fiber	○	○	○	○	○	○	○	○	○	Which bowl		○ B	○ C	○ D

Which high-fiber cereal do you eat most often? **MARK ONLY ONE:** ○ All Bran or Bran Buds ○ Raisin Bran ○ Fiber One, Fruit-n-Fiber, etc. ○ Something else ○ I don't know ○ I don't eat it

Product 19, Just Right or Total cereal	○	○	○	○	○	○	○	○	○	Which bowl		○ B	○ C	○ D
Any other cold cereal, like Corn Flakes, Cheerios, Special K	○	○	○	○	○	○	○	○	○	Which bowl		○ B	○ C	○ D
Milk or milk substitutes on cereal	○	○	○	○	○	○	○	○	○	How many oz. on cereal	○ 2 oz.	○ 4-5 oz.	○ 6-7 oz.	○ 8+ oz.
Yogurt or frozen yogurt	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Cheese, sliced cheese or cheese spread, including on sandwiches	○	○	○	○	○	○	○	○	○	How many slices	○ ½	○ 2	○ 3	○ 4

When you eat cheese, is it ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ Don't know/don't eat

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HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME SEE PORTION SIZE PICTURES FOR A-B-C-D
How often do you eat the following vegetables, including fresh, frozen, canned or in stir-fry, at home or in a restaurant?										
Broccoli	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Carrots, or mixed vegetables or stews containing carrots	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Corn	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Green beans or green peas	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Spinach	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Mustard greens, turnip greens, collards	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
French fries, fried potatoes or hash browns	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
White potatoes not fried, incl. boiled, baked, mashed & potato salad	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Sweet potatoes, yams (Not in pie)	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Cole slaw, cabbage	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Green salad	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Raw tomatoes, including in salad	○	○	○	○	○	○	○	○	○	How much ○1/4 ○1/2 ○1 ○2
Salad dressing	○	○	○	○	○	○	○	○	○	How many Tbsp. ○1 ○2 ○3 ○4

Is your salad dressing ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ Don't know/don't use

HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME
Any other vegetable, like okra, squash, cooked green peppers	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Refried beans or bean burritos	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Chili with beans (with or without meat)	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Baked beans, black-eye peas, pintos, any other dried beans	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Vegetable stew	○	○	○	○	○	○	○	○	○	Which Bowl ○B ○C ○D
Vegetable soup, vegetable beef, chicken vegetable, or tomato soup	○	○	○	○	○	○	○	○	○	Which Bowl ○B ○C ○D
Split pea, bean or lentil soup	○	○	○	○	○	○	○	○	○	Which Bowl ○B ○C ○D
Any other soup, like chicken noodle, chowder, mushroom, instant soups	○	○	○	○	○	○	○	○	○	Which Bowl ○B ○C ○D
Spaghetti, lasagna or other pasta with tomato sauce	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Cheese dishes without tomato sauce, like macaroni and cheese	○	○	○	○	○	○	○	○	○	How much ○A ○B ○C ○D
Pizza, including carry-out	○	○	○	○	○	○	○	○	○	How many slices ○1 ○2 ○3 ○4

HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME SEE PORTION SIZE PICTURES FOR A-B-C-D				
Do you ever eat chicken, meat or fish? ○ Yes ○ No **IF NO, SKIP TO NEXT PAGE**														
Hamburgers, cheeseburgers, meat loaf, at home or in a restaurant	○	○	○	○	○	○	○	○	○	How much meat	○ 1/8 lb	○ 1/4 lb	○ 1/2 lb.	○ 3/4 lb.
Tacos, burritos, enchiladas, tamales, etc. with meat or chicken	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Beef steaks, roasts, pot roast, or in frozen dinners or sandwiches	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
How do you like beef cooked? ○ Rare ○ Medium ○ Well done ○ I don't eat beef														
Pork chops, pork roasts, or dinner ham	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
When you eat meat, do you ○ Avoid eating the fat ○ Sometimes eat the fat ○ Often eat the fat ○ I don't eat meat														
Veal, lamb or deer meat	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Ribs, spareribs	○	○	○	○	○	○	○	○	○	How many ribs	○ 3-4	○ 5-6	○ 7-8	○ 9+
Liver, including chicken livers or liverwurst	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Gizzard, pork neckbones, chitlins, pigs feet, etc.	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Mixed dishes with beef or pork, like stew, corned beef hash, stuffed cabbage, meat dish with noodles	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Mixed dishes with chicken, like chicken casserole, chicken & noodles, pot pie or in stir-fry	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Fried chicken, at home or in a restaurant	○	○	○	○	○	○	○	○	○	# medium pieces	○ 1	○ 2	○ 3	○ 4
Chicken or turkey not fried, such as baked, grilled, or on sandwiches	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
When you eat chicken, do you ○ Avoid eating the skin ○ Sometimes eat the skin ○ Often eat the skin														

HOW OFTEN	NEVER	FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME				
Oysters	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Other shellfish like shrimp, scallops, crabs	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Tuna, tuna salad, tuna casserole	○	○	○	○	○	○	○	○	○	How much of the tuna	○ A	○ B	○ C	○ D
Fried fish or fish sandwich, at home or in a restaurant	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Other fish, not fried	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Hot dogs, or sausage like Polish, Italian or chorizos	○	○	○	○	○	○	○	○	○	How many	○ 1	○ 2	○ 3	○ 4
Are your hot dogs ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ Don't know/don't eat them														
Bologney, sliced ham, turkey lunch meat, other lunch meat	○	○	○	○	○	○	○	○	○	How many slices	○ 1	○ 2	○ 3	○ 4
Are your lunch meats ○ Usually low-fat or turkey ○ Sometimes low-fat ○ Hardly ever low-fat														

HOW OFTEN	NEVER	A FEW TIMES per YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	HOW MUCH EACH TIME — SEE PORTION SIZE PICTURES FOR A-B-C-D				
Noodles, macaroni, pasta salad	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Tofu, bean curd	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Meat substitutes, such as veggie burgers, Gardenburgers	○	○	○	○	○	○	○	○	○	How many patties	○ 1	○ 2	○ 3	○ 4
Chinese food, Thai or other Asian food, not counted above	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Snacks like potato chips, corn chips, popcorn (not pretzels)	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D

Are these snacks ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ Don't know/don't eat

HOW OFTEN	NEVER	FEW TIMES per YEAR	ONCE/MONTH	2-3 TIMES/MONTH	ONCE/WEEK	2 TIMES/WEEK	3-4 TIMES/WEEK	5-6 TIMES/WEEK	EVERY DAY	HOW MUCH EACH TIME				
Peanuts, other nuts or seeds	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Crackers	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Doughnuts, Danish pastry	○	○	○	○	○	○	○	○	○	How many	○ 1	○ 2	○ 3	○ 4
Cake, sweet rolls, coffee cake	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D

Are they ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ Don't know/don't eat

Cookies	○	○	○	○	○	○	○	○	○	How many	○ 1-2	○ 3-4	○ 5-7	○ 8+

Are your cookies ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ I don't know/don't eat

Ice cream, ice milk, ice cream bars	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D

Is your ice cream ○ Usually low-fat ○ Sometimes low-fat ○ Hardly ever low-fat ○ I don't know/don't eat

Pumpkin pie, sweet potato pie	○	○	○	○	○	○	○	○	○	How many slices	○ 1/2	○ 1	○ 2	○ 3
Any other pie or cobbler	○	○	○	○	○	○	○	○	○	How many slices	○ 1/2	○ 1	○ 2	○ 3
Chocolate candy, candy bars	○	○	○	○	○	○	○	○	○	How many bars	○ small	○ medium	○ large	○ large
Other candy, not chocolate, like hard candy, caramel, jelly beans	○	○	○	○	○	○	○	○	○	How many pieces	○ 1-2	○ 3-5	○ 6-7	○ 8+

PLEASE DO NOT WRITE IN THIS AREA

HOW OFTEN	NEVER OR A FEW TIMES PER YEAR	ONCE per MONTH	2-3 TIMES per MONTH	ONCE per WEEK	2 TIMES per WEEK	3-4 TIMES per WEEK	5-6 TIMES per WEEK	EVERY DAY	2+ TIMES per DAY	HOW MUCH EACH TIME SEE PORTION SIZE PICTURES FOR A-B-C-D				
Biscuits or muffins	○	○	○	○	○	○	○	○	○	How many each time	○ 1	○ 2	○ 3	○ 4
Rolls, hamburger buns, English muffins, bagels	○	○	○	○	○	○	○	○	○	How many each time	○ 1/2	○ 1	○ 2	○ 3
Dark bread like rye or whole wheat, including in sandwiches	○	○	○	○	○	○	○	○	○	How many slices each time	○ 1	○ 2	○ 3	○ 4
White bread or toast, including French, Italian, or in sandwiches	○	○	○	○	○	○	○	○	○	How many slices each time	○ 1	○ 2	○ 3	○ 4
Corn bread, corn muffins	○	○	○	○	○	○	○	○	○	How many pieces	○ 1	○ 2	○ 3	○ 4
Tortillas	○	○	○	○	○	○	○	○	○	How many each time	○ 1	○ 2	○ 3	○ 4
Rice, or dishes made with rice	○	○	○	○	○	○	○	○	○	How much	○ A	○ B	○ C	○ D
Margarine (not butter) on bread or on potatoes or vegetables, etc.	○	○	○	○	○	○	○	○	○	How many pats (tsp.)	○ 1	○ 2	○ 3	○ 4
Butter (not margarine) on bread or on potatoes or vegetables, etc.	○	○	○	○	○	○	○	○	○	How many pats (tsp.)	○ 1	○ 2	○ 3	○ 4
Gravy	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4
Peanut butter	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4
Jelly, jam, or syrup	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4
Mayonnaise, sandwich spreads	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4
Catsup, salsa or chile peppers	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4
Mustard, soy sauce, steak sauce, barbecue sauce, other sauces	○	○	○	○	○	○	○	○	○	How many Tbsp.	○ 1	○ 2	○ 3	○ 4

Did you use the pictures to choose your serving size on this form? ○ Yes ○ No ○ I didn't have any pictures.

Would you say your health is ○ Excellent ○ Very good ○ Good ○ Fair ○ Poor

How many times have you gone on a diet? ○ Never ○ 1-2 ○ 3-5 ○ 6-8 ○ 9 or more

Did you ever drink more beer, wine or liquor than you do now? ○ Yes ○ No

How many hours do you watch television or video, per day or per week on average?
○ None ○ 1-6 hours/week ○ 1 hour/day ○ 2 hours/day ○ 3 hours/day ○ 4+ hours/day

Do you smoke cigarettes now? ○ No ○ Yes
IF YES, On the average about how many cigarettes a day do you smoke now?
○ 1-5 ○ 6-14 ○ 15-24 ○ 25-34 ○ 35 or more

What language do you usually speak at home or with friends?
○ English ○ Spanish ○ Something else ○ English & something else equally

What is your ethnic group? (MARK ONE OR MORE)
○ Hispanic or Latino ○ Black or African American ○ American Indian or Alaska Native
○ White, not Hispanic ○ Asian ○ Native Hawaiian or Other Pacific Islander

Thank you very much for filling out this questionnaire. Please take a minute to go back and fill in anything you may have skipped.

PLEASE DO NOT WRITE IN THIS AREA

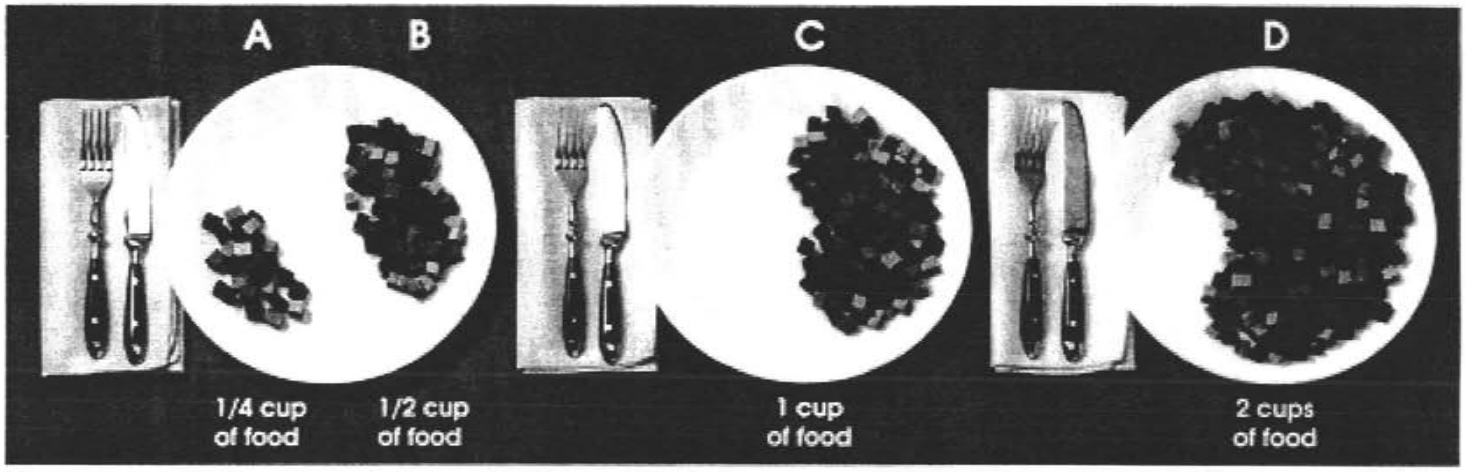
53034

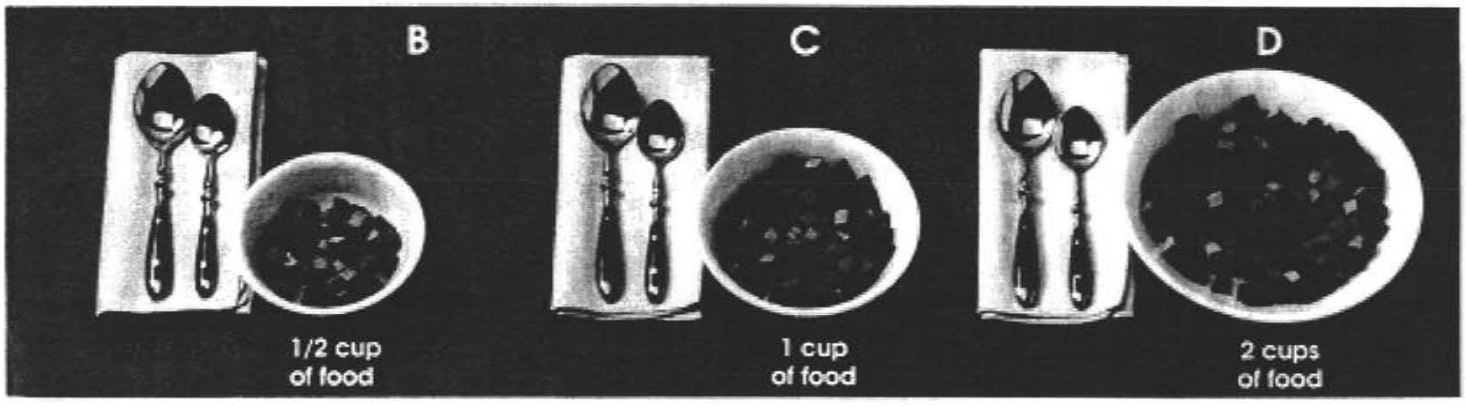
FOOD QUESTIONNAIRE

Serving Size Choices

Keep this in front of you while you are filling out The Food Questionnaire. You may use either the plates or the bowls to help you choose your serving size.

Choose A, B, C or D: **A** = 1/4 Cup of Food **B** = 1/2 Cup of Food **C** = 1 Cup of Food **D** = 2 Cups of Food





APPENDIX D. ADDITIONAL PARTICIPANT RESULTS

Table 1. Macronutrient and micronutrient intake from food for the REACH cohort study

		Percentiles				
n=391	**Mean ± SE**	**5th**	**25th**	**50th**	**75th**	**95th**
Energy, kcal	3774.5 ± 103.4	1351.7	2254.7	3350.1	4758.9	7439.2
Carbohydrate, gm	457.5 ± 12.5	157.3	282.6	403.9	586.3	896.4
Protein, gm	124.2 ± 3.6	45.0	73.7	107.7	149.1	280.8
Fat, gm	162.5 ± 4.9	53.2	92.9	138.5	207.6	342.2
Saturated fat, gm	50.4 ± 1.5	14.8	29.2	50.4	66.0	106.3
Dietary fiber, gm	23.1 ± 0.7	7.4	9.4	19.4	29.8	52.0
Dietary cholesterol, mg	464.8 ± 14.7	136.1	260.2	464.8	586.4	1134.9
Energy distribution (% of total)						
Carbohydrate	48.9 ± 0.4	36.9	44.6	48.8	53.1	61.1
Protein	13.4 ± 0.2	9.0	11.1	12.9	15.1	19.8
Fat	38.1 ± 0.3	27.8	34.4	38.3	42.3	48.2
Saturated fat	11.9 ± 0.1	8.3	10.4	11.9	13.1	15.5
Adjusted by body weight						
Energy, kcal/kg	53.1 ± 1.6	14.8	28.9	47.3	69.5	112.1
Protein, gm/kg	1.7 ± 0.1	0.5	0.9	1.5	2.2	4.0
Vitamins						
Vitamin A, RAE [1]	1040.0 ± 39.1	268.8	542.9	824.5	1261.2	2455.6
Thiamin (B_1), mg	2.6 ± 0.1	0.9	1.6	2.3	3.4	5.7
Riboflavin (B_2), mg	2.8 ± 0.1	0.9	1.6	2.4	3.6	6.2
Niacin, mg	37.5 ± 1.2	12.6	22.0	31.5	46.0	85.2
Vitamin B_6, mg	3.0 ± 0.1	1.0	1.7	2.6	3.8	6.4
Vitamin C, mg	257.5 ± 8.4	55.5	129.2	211.9	358.3	590.5
Folic Acid, µg	612.8 ± 18.6	207.1	359.5	523.2	761.6	1349.2
Vitamin D, IU	261.6 ± 12.5	40.6	98.5	178.2	330.5	786.4
Vitamin E, mg α-tocopherol	13.2 ± 0.4	4.0	7.8	11.3	16.8	27.7
Carotenoids						
Alpha-carotene, µg	445.2 ± 34.9	38.9	114.1	236.6	519.1	1600.0
Beta-carotene, µg	3446.2 ± 188.8	546.9	1230.2	2313.6	4213.6	10962.7
Cryptoxanthin, µg	273.0 ± 13.2	18.4	84.9	183.7	401.5	798.9
Lutein, µg	1967.7 ± 122.5	237.2	705.5	1233.5	2395.8	6073.7
Lycopene, µg	9343.6 ± 495.5	886.6	2909.0	5813.0	12067.8	31033.6
Minerals						
Calcium, mg	1130.2 ± 32.8	359.2	638.6	994.5	1463.4	2404.2
Phosphorus, mg	1931.1 ± 53.0	703.2	1168.1	1694.3	2380.8	4076.6
Iron, mg	25.2 ± 0.9	8.2	14.3	20.8	30.6	59.8
Zinc, mg	17.3 ± 0.6	5.6	9.6	14.1	21.1	40.4
Magnesium, mg	412.4 ± 11.6	146.6	244.4	357.4	507.8	869.1
Food Pyramid Servings						
Grains	8.2 ± 0.3	1.9	4.4	6.7	10.7	19.1
Vegetables	3.1 ± 0.1	0.8	1.5	2.4	3.7	7.7
Fruits	1.5 ± 0.1	0.2	0.7	1.2	2.0	4.0
Dairy	1.8 ± 0.1	0.2	0.7	1.4	2.6	4.4
Meat	4.1 ± 0.1	1.2	2.3	3.3	5.0	10.1

[1] Retinol activity equivalents

Table 2. Micronutrient intake from food and supplements for the REACH cohort study

		Percentiles				
n=391	**Mean ± SE**	**5th**	**25th**	**50th**	**75th**	**95th**
Vitamins						
Vitamin A, RAE [1]	1452.7 ± 53.4	281.1	662.0	1121.6	2129.6	3499.7
Thiamin (B_1), mg	3.2 ± 0.1	1.0	1.8	2.7	4.0	6.9
Riboflavin (B_2), mg	3.4 ± 0.1	1.0	1.9	3.0	4.4	6.9
Niacin, mg	44.3 ± 1.4	13.4	25.8	38.8	55.9	94.4
Vitamin B_6, mg	3.6 ± 0.1	1.1	2.1	3.2	4.6	7.3
Vitamin C, mg	308.3 ± 11.2	68.2	152.5	241.5	414.3	678.4
Folic Acid, DFE [2]	828.2 ± 26.3	224.4	439.2	691.6	1139.6	1844.8
Vitamin D, IU	369.7 ± 15.4	48.6	122.1	290.8	546.4	934.5
Vitamin E, mg α-tocopherol	17.0 ± 0.7	4.2	8.9	15.1	21.0	34.0
Minerals						
Calcium, mg	1171.0 ± 33.3	364.9	663.6	1052.3	1556.3	2521.0
Phosphorus, mg	NA[3]					
Iron, mg	34.2 ± 1.3	8.5	16.8	27.7	41.2	91.5
Zinc, mg	21.2 ± 0.7	6.0	11.2	19.2	27.7	46.3
Magnesium, mg	439.4 ± 11.9	156.9	277.5	390.0	559.0	885.4

[1] Retinol activity equivalents
[2] Dietary folate equivalents
[3] Not available

Table 3. Macronutrient intake and energy distribution by sex for the REACH cohort study

		Percentiles				
Males (n=97)	**Mean ± SE**	**5th**	**25th**	**50th**	**75th**	**95th**
Energy, kcal	4275.1 ± 237.3 *	1700.0	2659.5	3617.5	5330.4	8981.3
Carbohydrate, gm	509.9 ± 28.7 *	198.0	304.6	437.0	664.5	1098.3
Protein, gm	140.9 ± 8.1 *	56.5	86.3	118.6	179.0	306.1
Fat, gm	185.7 ± 11.3 *	61.4	110.3	160.7	228.8	407.0
Saturated fat, gm	57.2 ± 3.4 *	18.1	33.3	53.4	70.6	121.3
Dietary fiber, gm	25.9 ± 1.7 #	8.3	14.0	22.0	32.9	56.5
Dietary cholesterol, mg	526.3 ± 29.9 *	148.8	317.5	447.4	668.6	1145.5
Energy distribution (% of total)						
Carbohydrate	47.8 ± 0.7 #	38.1	43.9	47.3	51.9	59.0
Protein	13.4 ± 0.3	9.0	11.7	12.9	15.2	17.7
Fat	38.7 ± 0.6	28.3	35.7	38.8	41.9	48.7
Saturated fat	12.0 ± 0.2	8.0	11.0	12.0	13.1	15.4
Adjusted by body weight						
Energy, kcal/kg	61.8 ± 3.6 **	21.1	34.3	52.2	80.3	135.4
Protein, gm/kg	2.0 ± 0.1 **	0.7	1.2	1.7	2.7	4.7
Females (n=294)						
Energy, kcal	3609.3 ± 111.6 *	1230.4	2144.1	3272.9	4732.2	7251.6
Carbohydrate, gm	440.2 ± 13.6 *	153.0	275.2	393.5	570.9	845.8
Protein, gm	118.7 ± 4.0 *	41.0	69.7	106.0	144.2	273.9
Fat, gm	154.9 ± 5.4 *	50.2	87.6	133.9	205.1	334.4
Saturated fat, gm	48.2 ± 1.6 *	14.6	27.4	43.0	64.5	101.8
Dietary fiber, gm	22.1 ± 0.8 #	6.9	13.1	19.0	28.3	47.9
Dietary cholesterol, mg	444.5 ± 16.8 *	134.9	237.1	378.3	548.7	1133.6
Energy distribution (% of total)						
Carbohydrate	49.2 ± 0.4 #	36.3	44.9	49.3	53.6	61.5
Protein	13.4 ± 0.2	8.8	11.0	12.9	15.0	20.0
Fat	38.0 ± 0.4	27.6	34.0	38.2	42.4	47.9
Saturated fat	11.8 ± 0.1	8.3	10.2	11.8	13.1	15.6
Adjusted by body weight						
Energy, kcal/kg	50.2 ± 1.8 **	13.5	26.8	44.6	66.1	107.1
Protein, gm/kg	1.6 ± 0.1 **	0.4	0.8	1.4	2.1	3.7

Differences by sex tested using two-sample t-tests, ** $p < 0.01$; * $p < 0.05$; # $p < 0.10$

Table 4. Macronutrient intake and energy distribution by HIV status for the REACH cohort study

		Percentiles				
HIV+ (n=264)	**Mean ± SE**	**5th**	**25th**	**50th**	**75th**	**95th**
Energy, kcal	3901.3 ± 130.3 #	1296.7	2297.6	3544.2	4932.9	7488.8
Carbohydrate, gm	467.9 ± 15.4	152.8	285.3	423.2	614.6	906.9
Protein, gm	128.9 ± 4.6 #	46.3	73.8	112.4	159.6	288.8
Fat, gm	170.1 ± 6.4 *	49.9	95.5	146.8	219.4	358.3
Saturated fat, gm	53.1 ± 2.0 **	14.9	30.1	47.0	69.8	109.2
Dietary fiber, gm	23.3 ± 0.9	8.2	13.4	19.5	30.0	52.3
Dietary cholesterol, mg	491.1 ± 18.6 **	137.4	282.6	416.4	629.7	1146.5
Energy distribution (% of total)						
Carbohydrate	48.5 ± 0.5	36.2	44.6	48.1	52.8	61.3
Protein	13.4 ± 0.2	8.7	11.1	12.9	15.1	20.0
Fat	38.5 ± 0.4 #	27.6	34.7	38.7	42.7	48.6
Saturated fat	12.1 ± 0.1 *	8.3	10.8	12.2	13.4	15.9
Adjusted by body weight						
Energy, kcal/kg	54.8 ± 2.0	14.3	29.6	49.8	72.7	112.5
Protein, gm/kg	1.8 ± 0.1 #	0.5	0.9	1.6	2.3	4.2
HIV- (n=127)						
Energy, kcal	3510.9 ± 165.7 #	1345.8	2155.2	3069.5	4260.8	7665.1
Carbohydrate, gm	435.9 ± 21.4	173.2	280.8	369.2	550.5	895.2
Protein, gm	114.5 ± 5.6 #	43.6	70.8	94.2	143.9	256.9
Fat, gm	146.7 ± 7.4 *	54.9	88.3	128.6	190.7	321.3
Saturated fat, gm	44.9 ± 2.2 **	14.7	27.4	39.4	56.8	99.8
Dietary fiber, gm	22.5 ± 1.3	6.8	13.4	19.0	29.3	47.7
Dietary cholesterol, mg	410.0 ± 23.1 **	123.9	233.8	335.1	493.6	1019.6
Energy distribution (% of total)						
Carbohydrate	49.6 ± 0.6	40.0	44.5	49.7	53.6	60.8
Protein	13.3 ± 0.3	9.1	11.3	12.8	15.2	18.6
Fat	37.3 ± 0.5 #	28.0	34.2	37.8	40.7	46.9
Saturated fat	11.4 ± 0.2 **	7.9	10.0	11.5	12.7	14.9
Adjusted by body weight						
Energy, kcal/kg	49.5 ± 2.7	15.7	26.7	41.7	62.8	110.8
Protein, gm/kg	1.6 ± 0.1 #	0.5	0.9	1.3	2.0	3.6

Differences by HIV status tested using two-sample t-tests, ** $p < 0.01$; * $p < 0.05$; # $p < 0.10$

Table 5. Macronutrient intake from food by CD4+ T-cells (cells/μL) for the REACH cohort study

		Percentiles				
	Mean ± SE	5th	25th	50th	75th	95th
Energy, kcal						
HIV+ CD4+ ≥500 [a]	3872.1 ± 187.5	1328.0	2333.8	3581.0	4866.2	7610.7
HIV+ CD4+ 200-499 [b]	4025.6 ± 216.6	1377.5	2375.3	3476.2	5506.6	8729.0
HIV+ CD4+ <200 [c]	3631.0 ± 322.5	905.4	2113.5	3582.3	4604.0	7217.9
HIV- [d]	3510.9 ± 165.7	1345.8	2155.2	3069.5	4260.8	7665.1
Carbohydrate, g						
HIV+ CD4+ ≥500	468.7 ± 22.7	147.4	283.6	417.9	627.3	874.0
HIV+ CD4+ 200-499	477.7 ± 24.9	160.4	287.3	434.9	625.8	1069.6
HIV+ CD4+ <200	435.4 ± 38.3	120.0	284.7	398.7	557.4	901.8
HIV-	435.9 ± 21.4	173.2	280.8	369.2	550.5	895.2
Protein, g						
HIV+ CD4+ ≥500	126.1 ± 6.7	42.8	72.9	112.5	155.4	288.9
HIV+ CD4+ 200-499	135.1 ± 7.8	47.2	80.0	112.2	177.9	322.1
HIV+ CD4+ <200	120.5 ± 10.6	23.7	69.1	112.7	164.6	252.4
HIV-	114.5 ± 5.6	43.6	70.8	94.2	143.9	256.9
Fat, g						
HIV+ CD4+ ≥500	168.5 ± 9.0	52.1	93.8	114.8	215.6	357.7
HIV+ CD4+ 200-499	177.9 ± 11.0 #	52.5	98.9	145.9	239.1	404.5
HIV+ CD4+ <200	152.9 ± 14.6	31.5	84.3	147.0	206.2	332.6
HIV-	146.7 ± 7.4 #	54.9	88.3	128.6	190.7	321.3
Saturated fat, g						
HIV+ CD4+ ≥500	52.7 ± 2.8	16.0	30.1	46.6	69.7	114.1
HIV+ CD4+ 200-499	55.3 ± 3.2 *	13.8	31.5	49.4	74.8	118.8
HIV+ CD4+ <200	48.0 ± 4.5	8.7	27.6	45.9	63.6	110.4
HIV-	44.9 ± 2.2 *	14.7	27.4	39.4	56.8	99.8
Dietary fiber, g						
HIV+ CD4+ ≥500	22.6 ± 1.2	7.1	13.2	19.2	29.6	51.9
HIV+ CD4+ 200-499	24.9 ± 1.5	8.7	14.1	20.0	30.9	56.2
HIV+ CD4+ <200	21.0 ± 1.9	5.7	12.0	17.3	31.1	40.7
HIV-	22.5 ± 1.3	6.8	13.4	19.0	29.3	47.7
Dietary cholesterol, mg						
HIV+ CD4+ ≥500	484.4 ± 26.3	133.5	292.0	409.4	629.5	1112.0
HIV+ CD4+ 200-499	509.1 ± 31.6 *	139.1	288.0	428.7	663.5	1169.6
HIV+ CD4+ <200	461.4 ± 44.0	108.9	287.0	387.1	634.2	1065.6
HIV-	410.0 ± 23.1 *	123.9	233.8	335.1	493.6	1019.6
Energy, kcal/kg						
HIV+ CD4+ ≥500	51.4 ± 2.7	14.1	27.6	47.9	69.2	103.5
HIV+ CD4+ 200-499	58.9 ± 3.7	15.2	31.1	50.1	79.5	125.6
HIV+ CD4+ <200	55.5 ± 5.0	11.4	34.1	52.3	65.4	117.6
HIV-	49.5 ± 2.7	15.7	26.7	41.7	62.8	110.8
Protein, gm/kg						
HIV+ CD4+ ≥500	1.7 ± 0.1	0.4	0.8	1.3	2.2	3.7
HIV+ CD4+ 200-499	2.0 ± 0.1 #	0.5	1.0	1.7	2.7	4.6
HIV+ CD4+ <200	1.8 ± 0.2	0.3	1.0	1.8	2.3	3.8
HIV-	1.6 ± 0.1 #	0.5	0.9	1.3	2.0	3.6

[a] n=129; [b] n=102; [c] n=33; [d] n=127; * $p < 0.05$; # $p < 0.10$ (ANOVA) Similar superscripts indicate significant differences by pairwise comparison.

Table 6. Macronutrient intake from food by sex and HIV status for the REACH cohort study

		Percentiles				
	Mean ± SE	5th	25th	50th	75th	95th
Energy, kcal						
Females: HIV+ [a]	3691 ± 137	1152.5	2157.1	3454.0	4706.2	7248.1
HIV- [b]	3445 ± 191	1329.8	2125.8	3016.6	4109.2	7392.9
Males: HIV+ [c]	4507 ± 304 #	1711.5	2795.9	4013.8	5653.3	9975.4
HIV- [d]	3732 ± 333 #	1344.8	2529.0	3256.6	4739.2	8364.2
Carbohydrate, g						
Females: HIV+	446.8 ± 16.4	137.5	273.9	416.3	588.5	864.6
HIV-	427.0 ± 24.2	171.4	276.3	368.3	522.4	834.0
Males: HIV+	528.7 ± 35.8	196.9	306.5	451.5	694.2	1108.8
HIV-	466.0 ± 46.3	182.8	294.4	384.9	561.4	1094.9
Protein, g						
Females: HIV+	121.9 ± 5.0	39.7	70.3	108.0	146.5	285.8
HIV-	112.3 ± 6.4	41.1	69.3	94.1	139.8	256.6
Males: HIV+	148.9 ± 10.4 #	57.3	86.2	123.2	183.5	336.2
HIV-	122.1 ± 11.7 #	52.7	86.6	106.8	146.4	297.9
Fat, g						
Females: HIV+	159.8 ± 6.7	44.5	89.3	138.5	210.5	341.3
HIV-	145.1 ± 8.7	54.7	86.4	126.4	191.1	309.3
Males: HIV+	200.1 ± 14.8 *	64.5	113.3	177.2	239.7	451.2
HIV-	152.0 ± 13.2 *	45.0	103.6	131.6	190.3	335.7
Saturated fat, g						
Females: HIV+	50.2 ± 2.1 #	13.6	28.9	44.6	68.8	101.6
HIV-	44.2 ± 2.6 #	14.8	24.8	38.0	57.0	106.0
Males: HIV+	61.6 ± 4.4 *	18.8	35.1	56.8	74.8	131.4
HIV-	47.0 ± 4.0 *	11.5	33.0	40.7	58.4	99.2
Dietary fiber, g						
Females: HIV+	22.1 ± 0.9	7.9	12.9	19.2	28.7	48.8
HIV-	22.1 ± 1.6	6.6	13.3	18.3	26.4	44.8
Males: HIV+	26.8 ± 2.2	8.3	14.0	21.1	33.8	62.8
HIV-	23.9 ± 2.3	8.3	14.0	22.4	31.3	51.8
Dietary cholesterol, mg						
Females: HIV+	464.6 ± 21.2 #	136.5	263.2	384.6	587.3	1141.1
HIV-	404.3 ± 26.8 #	118.6	230.1	321.8	503.1	980.1
Males: HIV+	567.6 ± 37.1 *	169.7	343.7	503.4	746.6	1220.5
HIV-	429.4 ± 45.4 *	107.6	291.5	365.6	469.3	1091.1
Energy, kcal/kg						
Females: HIV+	50.8 ± 2.2	12.7	27.5	47.4	67.6	107.3
HIV-	49.0 ± 3.2	15.4	25.3	41.8	63.0	108.1
Males: HIV+	66.4 ± 4.5 *	22.9	34.4	61.7	90.0	162.9
HIV-	51.1 ± 5.2 *	16.5	34.3	41.2	62.3	127.7
Protein, gm/kg						
Females: HIV+	1.7 ± 0.1	0.4	0.9	1.4	2.1	4.0
HIV-	1.6 ± 0.1	0.4	0.8	1.3	2.1	3.5
Males: HIV+	2.2 ± 0.2 *	0.7	1.2	1.9	2.9	4.9
HIV-	1.7 ± 0.2 *	0.6	1.1	1.4	1.9	4.6

[a] n=196; [b] n=98; [c] n=68; [d] n=29; Differences by HIV status for males and females tested separately using two-sample t-tests

* $p < 0.05$; # $p < 0.10$

Table 7. Food Guide Pyramid servings by sex for the REACH cohort study

		Percentiles				
	Mean ± SE	5th	25th	50th	75th	95th
Grains						
Males [a]	9.3 ± 0.6 #	2.7	5.0	7.7	13.8	21.5
Females [b]	7.8 ± 0.3 #	1.8	4.2	6.4	10.3	18.0
Vegetables						
Males	3.5 ± 0.2 *	1.0	1.9	2.6	4.4	10.2
Females	2.9 ± 0.1 *	0.6	1.4	2.3	3.6	7.1
Fruits						
Males	1.5 ± 0.1	0.3	0.8	1.2	1.9	4.3
Females	1.5 ± 0.1	0.2	0.7	1.2	2.1	4.0
Dairy						
Males	2.0 ± 0.1 *	0.2	0.8	1.7	2.9	4.6
Females	1.7 ± 0.1 *	0.2	0.6	1.3	2.3	4.4
Meats						
Males	4.6 ± 0.3 #	1.5	2.7	3.9	5.2	10.3
Females	4.0 ± 0.2 #	1.1	2.2	3.2	4.7	9.9

[a] n=97; [b] n=294; * $p < 0.05$; # $p < 0.10$ (two sample t-tests with separate variance)

Table 8. Food Guide Pyramid servings by sex and HIV status for the REACH cohort study

		Percentiles				
	Mean ± SE	5th	25th	50th	75th	95th
Grains						
Females: HIV+ [a]	7.7 ± 0.3	1.7	4.0	6.6	10.8	17.9
HIV- [b]	8.0 ± 6.5	2.3	4.5	6.3	9.0	20.2
Males: HIV+ [c]	9.7 ± 0.7	2.6	5.1	8.1	15.1	21.8
HIV- [d]	8.2 ± 0.9	2.3	4.9	7.1	10.3	21.4
Vegetables						
Females: HIV+	3.0 ± 0.1	0.6	1.4	2.6	3.8	7.3
HIV-	2.7 ± 0.2	0.7	1.4	2.2	3.4	6.4
Males: HIV+	3.0 ± 0.3	1.0	2.0	2.7	4.4	9.1
HIV-	3.7 ± 0.6	1.0	1.7	2.2	5.0	12.6
Fruits						
Females: HIV+	1.5 ± 0.1	0.2	0.6	1.2	2.0	4.1
HIV-	1.6 ± 0.1	0.2	0.7	1.2	2.2	4.1
Males: HIV+	1.5 ± 0.1	0.2	0.6	1.2	1.9	3.9
HIV-	1.7 ± 0.2	0.4	1.1	1.2	2.3	4.6
Meats						
Females: HIV+	4.1 ± 0.2	1.1	2.2	3.4	5.0	10.2
HIV-	3.7 ± 0.3	1.3	2.1	2.9	4.6	9.3
Males: HIV+	4.1 ± 0.4	1.5	2.5	4.1	5.6	12.0
HIV-	4.0 ± 0.5	1.2	2.7	3.2	4.7	10.7
Dairy						
Females: HIV+	1.7 ± 0.1	0.2	0.6	1.4	2.7	4.4
HIV-	1.6 ± 0.1	0.3	0.6	1.2	2.0	4.5
Males: HIV+	1.7 ± 0.2	0.2	0.9	2.0	3.2	5.0
HIV-	1.7 ± 0.2	0.1	0.8	1.5	2.7	3.7

[a] n=196; [b] n=98; [c] n=68; [d] n=29; Differences by HIV status for males and females tested separately using two-sample t-tests-no differences found

Table 9. Vitamin and mineral intake from food by sex for the REACH cohort study

		Percentiles				
	Mean ± SE	5th	25th	50th	75th	95th
Vitamin A, RAE						
Males [a]	1167.2 ± 82.7 #	365.1	627.5	849.3	1376.0	3041.2
Females [b]	998.0 ± 44.0 #	254.7	506.3	814.5	1222.7	2413.0
Thiamin (B_1), mg						
Males	3.0 ± 0.2 **	1.1	1.7	2.6	3.9	6.5
Females	2.5 ± 0.1 **	0.7	1.5	2.2	3.0	5.6
Riboflavin (B_2), mg						
Males	3.3 ± 0.2 **	1.3	1.9	2.8	4.2	7.2
Females	2.7 ± 0.2 **	0.8	1.5	2.3	3.4	5.7
Niacin, mg						
Males	43.3 ± 2.9 *	15.7	25.7	35.6	53.6	97.9
Females	35.6 ± 2.6 *	11.2	20.6	30.1	45.0	81.9
Vitamin B_6, mg						
Males	3.4 ± 0.2 **	1.1	2.1	2.9	4.4	7.5
Females	2.8 ± 0.1 **	0.9	1.7	2.4	3.6	5.9
Vitamin C, mg						
Males	282.1 ± 18.0 #	55.7	148.5	249.4	387.7	634.9
Females	249.3 ± 9.4 #	54.8	126.1	206.9	351.9	564.8
Folic Acid, µg						
Males	690.0 ± 38.0 *	256.4	400.1	593.2	894.1	1459.0
Females	587.3 ± 21.1 *	187.8	349.6	505.4	724.1	1276.4
Vitamin D, IU						
Males	295.1 ± 25.1	62.6	112.7	215.4	385.9	880.0
Females	250.6 ± 14.5	35.0	95.4	172.1	320.5	713.9
Vitamin E, mg						
α-tocopherol						
Males	14.8 ± 0.9 *	5.3	8.6	12.9	18.9	33.5
Females	12.6 ± 0.4 *	3.9	7.3	10.3	16.2	27.2
Calcium, mg						
Males	1279.6 ± 69.7 **	395.3	800.4	1065.6	1714.7	2554.5
Females	1080.1 ± 36.7 **	321.3	613.2	937.6	1435.3	2398.4
Phosphorus, mg						
Males	2187.2 ± 120.9 *	921.8	1280.3	1801.0	2755.8	4501.6
Females	1846.6 ± 57.3 *	582.6	1122.5	1668.3	2257.8	3849.4
Iron, mg						
Males	29.6 ± 2.0 **	10.3	16.4	23.8	36.4	85.0
Females	23.8 ± 0.9 **	7.2	13.5	19.6	29.6	56.5
Zinc, mg						
Males	20.6 ± 1.4 **	7.9	11.0	16.9	25.8	50.4
Females	16.2 ± 0.6 **	4.9	9.3	13.4	19.9	37.9
Magnesium, mg						
Males	470.0 ± 26.4 **	171.0	295.9	422.5	544.1	1020.4
Females	393.4 ± 12.6 **	128.7	236.0	347.7	498.1	854.3

[a] n=97; [b] n=294; ** p <0.01; * p <0.05; # p <0.10 (two sample t-tests with separate variance)

Table 10. Retinol and carotenoid intake from food by sex for the REACH cohort study

		Percentiles				
	Mean ± SE	**5th**	**25th**	**50th**	**75th**	**95th**
Retinol, µg						
Males [a]	799.8 ± 53.3	243.8	417.6	618.2	1015.2	1949.1
Females [b]	697.4 ± 33.7	148.1	331.7	554.9	845.8	1820.5
Alpha-carotene, ug						
Males	574.5 ± 75.1 *	40.7	147.1	306.3	609.4	2483.7
Females	402.6 ± 39.0 *	37.3	104.8	208.8	476.8	1342.4
Beta-carotene, ug						
Males	3962.4 ± 486.0	474.6	1629.8	2492.1	4687.2	13704.6
Females	3275.9 ± 192.8	565.1	1182.0	2225.2	3961.7	10207.8
Cryptoxanthin, ug						
Males	317.9 ± 29.9 #	25.4	89.2	225.1	477.2	994.1
Females	258.2 ± 14.4 #	16.9	78.5	167.1	386.1	761.2
Lutein, ug						
Males	1925.4 ± 246.9	193.4	787.6	1242.6	2406.7	5273.9
Females	1981.6 ± 141.4	238.8	699.2	1232.9	2395.8	6156.8
Lycopene, ug						
Males	11356.3 ± 1066.2 *	1170.2	4179.2	7333.8	15716.9	35230.8
Females	8679.5 ± 552.8 *	737.1	2702.8	5554.4	11248.6	27090.1

[a] n=97; [b] n=294; * $p < 0.05$; # $p < 0.10$ (two sample t-tests with separate variance)

Table 11. Vitamin and carotenoid intake from food by HIV status for the REACH cohort study

		Percentiles				
HIV+ (n=264)	**Mean ± SE**	**5th**	**25th**	**50th**	**75th**	**95th**
Vitamin A, RAE	1080.6 ± 48.9	266.3	563.3	836.2	1351.9	2739.9
Thiamin (B_1), mg	2.7 ± 0.1	0.8	1.6	2.4	3.5	6.1
Riboflavin (B_2), mg	2.9 ± 0.1 #	0.9	1.7	2.6	3.9	6.3
Niacin, mg	38.6 ± 1.4	12.9	22.1	33.4	46.8	86.5
Vitamin B_6, mg	3.0 ± 0.1	1.0	1.7	2.6	3.9	6.6
Vitamin C, mg	256.4 ± 10.2	55.2	129.7	210.4	353.4	601.6
Folic Acid, µg	621.9 ± 22.3	211.8	356.6	523.1	780.9	1404.1
Vitamin D, IU	274.5 ± 16.1	40.0	106.6	187.4	333.0	910.7
Vitamin E, mg α-tocopherol	13.6 ± 0.5	4.2	7.7	10.1	15.4	26.8
Carotenoids						
α-carotene, µg	439.2 ± 34.4	40.1	114.5	250.0	547.4	1593.1
β-carotene, µg	3474.4 ± 235.5	577.3	1231.3	2415.6	4266.4	10558.3
Cryptoxanthin, µg	259.1 ± 15.5	17.9	84.5	163.8	386.7	777.0
Lutein, µg	2026.0 ± 155.1	223.2	724.3	1233.5	2440.3	6081.1
Lycopene, µg	9570.6 ± 626.9	718.0	2905.6	5738.5	12345.7	33864.6
Minerals						
Calcium, mg	1160.2 ± 40.4	337.7	648.1	1093.0	1548.9	2425.7
Phosphorus, mg	1996.3 ± 66.2 #	662.6	1165.8	1784.1	2549.9	4187.4
Iron, mg	25.8 ± 1.0	8.1	10.4	21.0	31.1	63.7
Zinc, mg	18.1 ± 0.8 *	5.1	9.5	14.5	22.6	45.1
Magnesium, mg	420.8 ± 14.5	133.7	241.0	361.2	525.0	883.3
HIV- (n=127)						
Vitamin A, RAE	955.5 ± 63.9	268.6	472.5	775.2	1199.6	2249.4
Thiamin (B_1), mg	2.5 ± 0.1	0.9	1.5	2.1	3.0	5.3
Riboflavin (B_2), mg	2.6 ± 0.1 #	0.9	1.6	2.1	3.2	5.6
Niacin, mg	35.2 ± 1.9	11.6	21.6	29.1	41.7	78.2
Vitamin B_6, mg	2.8 ± 0.1	1.0	1.7	2.4	3.3	5.6
Vitamin C, mg	259.6 ± 14.8	58.0	128.5	212.7	369.4	577.4
Folic Acid, µg	594.0 ± 33.4	205.3	364.2	524.9	709.2	1207.9
Vitamin D, IU	234.8 ± 19.1	39.0	89.4	165.0	320.8	677.1
Vitamin E, mg α-tocopherol	13.2 ± 0.7	3.9	7.8	11.8	17.4	30.0
Carotenoids						
α-carotene, µg	457.9 ± 80.5	36.4	104.3	216.5	456.2	1879.3
β-carotene, µg	3387.7 ± 315.0	511.0	1195.7	2214.5	3812.2	11884.1
Cryptoxanthin, µg	302.0 ± 24.7	18.0	95.0	225.0	495.0	834.9
Lutein, µg	1846.4 ± 196.5	268.3	669.5	1176.4	2247.3	5990.0
Lycopene, µg	8871.7 ± 794.6	1121.8	2960.2	6109.3	11224.6	26413.6
Minerals						
Calcium, mg	1068.0 ± 56.0	366.2	635.5	889.4	1352.5	2400.1
Phosphorus, mg	1795.3 ± 86.5 #	717.2	1168.1	1488.4	2201.6	3974.1
Iron, mg	24.0 ± 1.6	8.8	13.8	20.0	28.8	55.5
Zinc, mg	15.5 ± 0.7 *	5.8	9.6	13.5	19.5	30.2
Magnesium, mg	395.2 ± 19.2	154.5	253.7	339.1	490.6	770.0

* $p < 0.05$; # $p < 0.10$ (t-test with separate variance)

Table 12. Vitamin intake from food by CD4+ T-cells (cells/μL) for the REACH cohort study

		Percentiles				
	Mean ± SE	5th	25th	50th	75th	95th
Vitamin A, RAE						
HIV+ CD4+ ≥500 [a]	1050.6 ± 68.8	261.4	565.2	837.7	1284.8	2810.3
HIV+ CD4+ 200-499 [b]	1161.2 ± 85.8	308.0	608.3	817.8	1539.2	2928.2
HIV+ CD4+ <200 [c]	948.8 ± 100.5	179.2	497.3	865.2	1400.1	2141.2
HIV- [d]	955.5 ± 63.9	268.6	472.5	775.2	1200.6	2249.4
Thiamin (B_1), mg						
HIV+ CD4+ ≥500	2.6 ± 0.1	0.8	1.6	2.4	3.2	5.7
HIV+ CD4+ 200-499	2.9 ± 0.2	0.9	1.6	2.4	3.9	6.8
HIV+ CD4+ <200	2.4 ± 0.2	0.6	1.4	2.3	3.5	4.7
HIV-	2.5 ± 0.1	0.9	1.5	2.1	3.0	5.3
Riboflavin (B_2), mg						
HIV+ CD4+ ≥500	2.8 ± 0.1	0.8	1.6	2.4	3.6	6.1
HIV+ CD4+ 200-499	3.1 ± 0.2 [#]	1.0	1.8	2.7	4.1	6.7
HIV+ CD4+ <200	2.7 ± 0.2	0.6	1.6	2.6	3.6	5.3
HIV-	2.6 ± 0.1 [#]	0.9	1.6	2.1	3.2	5.6
Niacin, mg						
HIV+ CD4+ ≥500	36.9 ± 2.0	11.5	22.0	30.9	45.5	85.8
HIV+ CD4+ 200-499	41.4 ± 2.5	14.1	22.5	34.7	57.9	89.0
HIV+ CD4+ <200	36.3 ± 3.4	8.5	21.4	35.4	45.7	75.4
HIV-	35.2 ± 1.9	11.6	21.6	29.1	41.7	78.2
Vitamin B_6, mg						
HIV+ CD4+ ≥500	2.9 ± 0.1	1.0	1.7	2.6	3.8	6.5
HIV+ CD4+ 200-499	3.2 ± 0.2	1.3	1.9	2.6	4.2	7.3
HIV+ CD4+ <200	2.9 ± 0.3	0.8	1.4	2.7	4.0	5.8
HIV-	2.8 ± 0.1	1.0	1.7	2.4	3.3	5.6
Vitamin C, mg						
HIV+ CD4+ ≥500	253.8 ± 15.1	49.7	113.1	222.1	365.7	603.2
HIV+ CD4+ 200-499	269.8 ± 16.5	72.7	149.3	219.6	349.8	620.4
HIV+ CD4+ <200	225.3 ± 24.9	40.9	114.2	182.9	344.7	559.0
HIV-	259.6 ± 14.8	58.0	128.5	212.7	369.4	577.4
Folic Acid, μg						
HIV+ CD4+ ≥500	602.1 ± 31.0	204.0	366.6	507.7	739.2	1302.1
HIV+ CD4+ 200-499	664.6 ± 39.3	219.0	352.0	551.5	905.1	1482.6
HIV+ CD4+ <200	567.2 ± 49.3	145.7	317.4	529.7	737.7	1084.2
HIV-	594.0 ± 33.4	205.3	364.2	524.9	709.2	1207.9
Vitamin D, IU						
HIV+ CD4+ ≥500	253.8 ± 21.1	37.5	106.2	176.5	308.6	772.4
HIV+ CD4+ 200-499	295.4 ± 28.1	47.3	115.9	221.3	363.3	915.4
HIV+ CD4+ <200	291.1 ± 48.0	29.6	92.8	224.4	371.4	1046.0
HIV-	234.8 ± 19.1	39.0	89.4	165.0	320.8	667.1
Vitamin E, mg						
α-tocopherol						
HIV+ CD4+ ≥500	13.2 ± 0.7	3.9	7.8	11.7	17.5	27.0
HIV+ CD4+ 200-499	14.5 ± 0.9	4.8	8.0	12.2	18.4	33.3
HIV+ CD4+ <200	12.1 ± 1.1	2.6	6.6	12.2	16.8	25.5
HIV-	12.3 ± 0.7	4.2	7.7	10.1	15.4	26.8

[a] n=129; [b] n=102; [c] n=33; [d] n=127; [#] $p < 0.10$ (ANOVA) Similar superscripts indicate significant differences by pairwise comparison.

Table 13. Mineral intake from food by CD4+ T-cells (cells/μL) for the REACH study

		Percentiles				
	Mean ± SE	**5th**	**25th**	**50th**	**75th**	**95th**
Calcium, mg						
HIV+ CD4+ ≥500 [a]	1154.8 ± 58.9	306.6	657.6	1062.1	1532.5	2356.7
HIV+ CD4+ 200-499 [b]	1207.0 ± 65.7	364.3	648.4	1120.6	1595.5	2586.2
HIV+ CD4+ <200 [c]	1036.3 ± 101.3	165.5	501.8	1055.0	1363.0	2317.3
HIV- [d]	1068.0 ± 56.0	366.2	635.5	889.4	1352.5	2400.1
Phosphorus, mg						
HIV+ CD4+ ≥500	1958.5 ± 94.7	618.0	1138.7	1824.7	2513.8	4220.7
HIV+ CD4+ 200-499	2092.9 ± 111.7	715.6	1198.4	1763.6	2702.1	4548.0
HIV+ CD4+ <200	1845.8 ± 156.7	419.9	1148.7	1806.7	2340.0	3608.0
HIV-	1795.3 ± 86.5	717.2	1168.1	1488.4	2201.6	3974.1
Iron, mg						
HIV+ CD4+ ≥500	24.5 ± 1.4	6.7	13.7	20.6	28.2	60.7
HIV+ CD4+ 200-499	27.9 ± 1.8	8.3	15.3	21.8	36.6	67.7
HIV+ CD4+ <200	24.1 ± 2.29	6.4	12.2	23.0	31.4	51.0
HIV-	24.0 ± 1.6	8.8	13.8	20.0	28.8	55.5
Zinc, mg						
HIV+ CD4+ ≥500	17.4 ± 1.1	4.6	9.4	13.8	20.8	45.5
HIV+ CD4+ 200-499	19.3 ± 1.3	5.9	10.0	15.3	24.2	45.9
HIV+ CD4+ <200	17.4 ± 2.4	2.7	8.8	13.3	23.0	46.8
HIV-	15.5 ± 0.7	5.8	9.7	13.5	19.5	30.2
Magnesium, mg						
HIV+ CD4+ ≥500	405.9 ± 19.8	122.1	238.8	351.4	511.6	854.0
HIV+ CD4+ 200-499	446.9 ± 25.5	162.7	258.7	368.3	596.4	1001.3
HIV+ CD4+ <200	398.3 ± 35.9	94.8	238.8	371.8	527.0	833.9
HIV-	395.2 ± 19.2	154.5	253.7	339.1	490.6	770.0

[a] n=129; [b] n=102; [c] n=33; [d] n=127; No significant differences found using ANOVA

Table 14. Carotenoid intake from food by CD4+ T-cells (cells/μL) for the REACH cohort study

		Percentiles				
	Mean ± SE	5th	25th	50th	75th	95th
Alpha-carotene, μg						
HIV+ CD4+ ≥500 [a]	445.8 ± 54.1	42.2	113.8	236.6	492.9	1934.1
HIV+ CD4+ 200-499 [b]	480.2 ± 55.1	36.6	118.7	252.8	669.8	1581.8
HIV+ CD4+ <200 [c]	286.2 ± 39.6	27.0	99.4	257.0	396.5	739.1
HIV- [d]	457.9 ± 80.5	36.4	104.3	216.5	456.2	1879.3
Beta-carotene, μg						
HIV+ CD4+ ≥500	3419.8 ± 365.9	560.2	1198.1	2150.4	4221.8	10624.2
HIV+ CD4+ 200-499	3641.9 ± 368.4	596.0	1296.5	2514.5	4496.0	12477.6
HIV+ CD4+ <200	3170.1 ± 471.2	285.8	1085.6	2527.2	4176.9	9990.5
HIV-	3387.7 ± 315.0	511.0	1195.7	2214.5	3812.2	11884.1
Cryptoxanthin, μg						
HIV+ CD4+ ≥500	252.3 ± 22.7	16.1	71.9	151.0	397.9	836.1
HIV+ CD4+ 200-499	283.3 ± 24.9	20.7	113.1	218.3	403.5	799.3
HIV+ CD4+ <200	211.0 ± 38.4	14.7	79.3	144.8	252.5	775.8
HIV-	302.0 ± 24.7	18.0	95.0	255.0	495.0	834.9
Lutein, μg						
HIV+ CD4+ ≥500	2156.2 ± 250.4	236.1	706.7	1233.6	2566.4	6631.7
HIV+ CD4+ 200-499	1718.6 ± 171.3	193.4	770.2	1243.4	2171.6	4687.0
HIV+ CD4+ <200	2466.9 ± 544.7	183.4	647.9	1218.1	2871.4	10755.3
HIV-	1846.4 ± 196.5	268.3	669.5	1176.5	2247.3	5990.0
Lycopene, μg						
HIV+ CD4+ ≥500	8880.0 ± 813.8	499.1	2315.1	5096.9	12256.2	28918.5
HIV+ CD4+ 200-499	10571.1 ± 1135.6	976.9	3431.3	6829.0	12690.8	41422.2
HIV+ CD4+ <200	9959.1 ± 1646.4	661.3	4076.7	6148.8	12320.1	37465.0
HIV-	8871.7 ± 794.6	1121.8	2960.2	6109.3	11224.6	26413.6

[a] n=129; [b] n=102; [c] n=33; [d] n=127; No significant differences found using ANOVA

Table 15. Vitamin intake from food by sex and HIV status for the REACH study

		Percentiles				
	Mean ± SE	5th	25th	50th	75th	95th
Vitamin A, RAE						
Females: HIV+ [a]	1025.0 ± 55.3	256.5	512.8	821.4	1254.7	2477.8
HIV- [b]	944.0 ± 72.2	231.8	465.1	762.6	1193.6	2244.7
Males: HIV+ [c]	1241.0 ± 101.2	359.4	648.3	978.6	1490.7	3136.4
HIV- [d]	994.4 ± 139.3	349.7	546.3	784.8	1210.8	3262.2
Thiamin (B_1), mg						
Females: HIV+	2.5 ± 0.1	0.7	1.5	2.3	3.2	5.9
HIV-	2.4 ± 0.2	0.9	1.5	2.0	2.9	5.1
Males: HIV+	3.2 ± 0.2	1.1	1.7	2.7	4.1	6.7
HIV-	2.7 ± 0.3	1.1	1.7	2.3	3.4	6.8
Riboflavin (B_2), mg						
Females: HIV+	2.7 ± 0.1	0.7	1.6	2.5	3.4	6.2
HIV-	2.6 ± 0.2	0.9	1.5	2.1	3.2	5.4
Males: HIV+	3.4 ± 0.2 #	1.2	1.9	2.8	4.4	7.6
HIV-	2.8 ± 0.3 #	1.3	1.9	2.4	3.2	6.9
Niacin, mg						
Females: HIV+	36.1 ± 1.5	11.1	20.9	30.7	45.2	85.7
HIV-	34.4 ± 2.2	11.0	20.2	28.2	42.5	73.3
Males: HIV+	45.5 ± 3.3	15.9	25.0	37.5	61.5	99.3
HIV-	38.0 ± 4.1	14.1	26.4	34.8	42.0	106.2
Vitamin B_6, mg						
Females: HIV+	2.9 ± 0.1	0.9	1.6	2.6	3.7	6.2
HIV-	2.7 ± 0.2	0.9	1.7	2.3	3.3	5.6
Males: HIV+	3.5 ± 0.2	1.1	2.1	3.0	4.5	7.6
HIV-	3.2 ± 0.4	1.1	2.1	2.9	3.7	8.8
Vitamin C, mg						
Females: HIV+	246.5 ± 11.4	54.8	124.6	204.4	344.9	562.9
HIV-	255.0 ± 16.8	52.8	128.0	255.0	369.6	585.2
Males: HIV+	285.1 ± 22.1 *	52.3	153.4	244.2	387.8	646.6
HIV-	275.2 ± 31.5 *	67.2	132.8	282.6	386.7	656.6
Folic Acid, µg						
Females: HIV+	588.0 ± 24.5	184.5	349.1	503.5	729.2	1300.3
HIV-	586.0 ± 40.1	185.5	351.7	586.0	705.3	1166.5
Males: HIV+	719.5 ± 48.6	251.3	406.0	628.6	922.8	1525.4
HIV-	620.7 ± 55.4	256.3	391.0	530.9	745.4	1421.1
Vitamin D, IU						
Females: HIV+	257.6 ± 18.5	33.3	100.0	174.0	321.4	818.1
HIV-	236.7 ± 22.7	35.1	90.8	162.8	320.5	667.8
Males: HIV+	323.4 ± 32.2	76.3	136.8	228.4	460.5	973.7
HIV-	228.7 ± 34.2	55.0	75.6	165.0	363.6	691.2
Vitamin E, mg α-tocopherol						
Females: HIV+	12.8 ± 0.5	3.6	7.1	11.2	16.7	27.6
HIV-	12.4 ± 0.8	4.0	7.5	10.0	15.3	27.1
Males: HIV+	16.0 ± 1.1*	5.2	8.7	14.0	19.9	37.1
HIV-	15.1 ± 1.0 *	5.2	8.1	10.4	15.5	24.1

[a] n=196; [b] n=98; [c] n=68; [d] n=29; Differences by HIV status for males and females tested separately using two-sample t-tests with separate variance; * $p < 0.05$; # $p < 0.10$

Table 16. Retinol and carotenoid Intake from food by sex and HIV status for the REACH cohort study

		Percentiles				
	Mean ± SE	5th	25th	50th	75th	95th
Retinol, ug						
Females: HIV+ [a]	731.5 ± 44.2	137.0	343.2	565.6	886.3	1830.6
HIV- [b]	629.2 ± 48.7	172.1	294.3	521.7	766.9	1825.0
Males: HIV+ [c]	849.7 ± 65.9	244.3	448.2	653.7	1126.2	1995.3
HIV- [d]	683.7 ± 86.6	230.1	382.7	565.6	885.8	2053.5
Alpha-carotene, µg						
Females: HIV+	369.9 ± 30.9	36.6	105.7	209.0	487.8	1279.7
HIV-	468.1 ± 99.6	45.6	104.0	205.2	440.7	2015.6
Males: HIV+	638.9 ± 96.2	50.8	157.4	384.6	681.8	2800.1
HIV-	423.4 ± 108.1	20.3	112.5	275.5	570.2	2104.6
Beta-carotene, µg						
Females: HIV+	3214.8 ± 229.3	569.2	1172.2	2263.3	3919.5	10137.7
HIV-	3398.1 ± 354.1	520.7	1184.1	2142.1	4135.6	10837.4
Males: HIV+	4222.6 ± 626.6	641.5	1653.0	2589.0	5069.1	14800.4
HIV-	3352.4 ± 697.9	372.1	1305.9	2256.6	3461.8	14298.2
Cryptoxanthin, µg						
Females: HIV+	242.5 ± 16.5	16.6	79.0	161.0	365.6	743.7
HIV-	289.8 ± 28.0	16.2	78.2	212.0	455.7	830.1
Males: HIV+	307.1 ± 36.4	22.9	85.7	208.4	454.4	992.5
HIV-	343.2 ± 52.8	62.9	118.1	256.0	512.0	1080.5
Lutein, µg						
Females: HIV+	2063.0 ± 187.9	239.9	706.4	1232.9	2430.3	6147.7
HIV-	1818.9 ± 197.0	192.2	683.6	1205.9	2260.7	6423.5
Males: HIV+	1919.4 ± 264.7	179.8	881.4	1248.0	2453.7	5669.5
HIV-	1939.6 ± 553.4	352.1	628.6	1133.6	2203.6	10255.3
Lycopene, µg						
Females: HIV+	8877.0 ± 698.9	557.6	2593.1	5311.4	11608.5	30162.2
HIV-	8284.5 ± 896.3	1127.0	2794.6	5852.9	9645.0	25746.3
Males: HIV+	11569.7 ± 1346.3	1450.9	4177.4	7323.3	15477.6	40876.6
HIV-	10856.0 ± 1689.8	597.1	3982.9	7333.8	16504.2	31531.6

[a] n=196; [b] n=98; [c] n=68; [d] n=29; Differences by HIV status for males and females tested separately using two-sample t-tests with separate variance-no significant difference found.

Table 17. Mineral intake from food by sex and HIV status for the REACH cohort study

		Percentiles				
	Mean ± SE	**5th**	**25th**	**50th**	**75th**	**95th**
Calcium, mg						
Females: HIV+ [a]	1093.2 ± 43.6	309.1	620.2	1026.1	1444.0	2362.6
HIV- [b]	1056.4 ± 67.6	329.6	580.5	825.1	1356.5	2727.5
Males: HIV+ [c]	1353.1 ± 90.5 [#]	390.1	743.6	1125.6	1786.7	2775.2
HIV- [d]	1107.3 ± 91.0 [#]	400.0	841.3	994.5	1364.5	2312.0
Phosphorus, mg						
Females: HIV+	1887.9 ± 69.6	551.3	1135.0	1745.2	2371.5	3844.9
HIV-	1763.6 ± 100.9	653.6	1115.0	1468.1	2205.6	3892.1
Males: HIV+	2308.9 ± 155.5 [#]	911.9	1361.0	1883.1	2958.3	4883.3
HIV-	1901.7 ± 166.8 [#]	970.5	1236.9	1650.6	2104.0	4209.2
Iron, mg						
Females: HIV+	23.9 ± 1.1	6.9	13.5	20.0	30.0	58.3
HIV-	23.4 ± 1.8	8.0	12.8	18.8	28.5	52.9
Males: HIV+	31.1 ± 2.5	10.3	16.0	25.2	38.7	86.1
HIV-	26.0 ± 3.1	9.8	16.9	23.0	29.9	75.1
Zinc, mg						
Females: HIV+	16.8 ± 0.8	4.5	9.2	13.7	21.0	40.5
HIV-	14.9 ± 0.9	5.3	9.6	12.5	17.2	34.2
Males: HIV+	21.9 ± 1.9 [#]	7.7	10.9	17.0	26.5	61.0
HIV-	17.6 ± 1.3 [#]	8.1	11.4	16.1	24.3	30.1
Magnesium, mg						
Females: HIV+	397.4 ± 15.1	121.3	230.9	351.5	503.4	866.3
HIV-	385.5 ± 22.7	135.1	239.2	323.7	488.4	743.2
Males: HIV+	488.0 ± 34.7	165.6	297.2	430.3	611.7	1106.7
HIV-	427.7 ± 34.0	188.8	293.0	417.1	495.6	857.9

[a] n=196; [b] n=98; [c] n=68; [d] n=29; Differences by HIV status for males and females tested separately using two-sample t-tests with separate variance; [#] $p < 0.10$

Table 18. Vitamin intake from food and supplements by sex and HIV status for the REACH study

		Percentiles				
	Mean ± SE	5th	25th	50th	75th	95th
Vitamin A, RAE						
Females: HIV+ [a]	1469.9 ± 73.9	260.4	678.0	1164.9	2187.8	3328.8
HIV- [b]	1322.3 ± 108.0	231.8	573.6	1014.2	2055.6	3096.0
Males: HIV+ [c]	1587.6 ± 131.2	359.4	683.9	1237.0	2378.7	3812.5
HIV- [d]	1459.9 ± 205.2	354.0	718.3	1199.6	1959.2	4762.2
Thiamin (B_1), mg						
Females: HIV+	3.1 ± 0.1	0.7	1.7	2.7	3.9	6.6
HIV-	2.9 ± 0.2	0.9	1.6	2.4	4.0	5.8
Males: HIV+	3.7 ± 0.3	1.1	2.0	3.1	5.2	7.8
HIV-	3.1 ± 0.3	1.2	2.0	2.8	3.6	8.3
Riboflavin (B_2), mg						
Females: HIV+	3.4 ± 0.2	0.8	2.0	3.1	4.4	6.7
HIV-	3.1 ± 0.2	0.9	1.6	2.7	4.1	6.6
Males: HIV+	4.0 ± 0.3	1.3	2.2	3.6	5.2	8.5
HIV-	3.3 ± 0.3	1.4	2.3	3.0	3.7	8.6
Niacin, mg						
Females: HIV+	43.6 ± 1.9	12.4	25.1	39.1	54.1	92.6
HIV-	40.6 ± 2.6	11.3	23.5	36.0	51.5	85.0
Males: HIV+	51.6 ± 3.6	16.7	28.3	41.7	70.7	115.0
HIV-	44.2 ± 4.8	15.9	30.8	37.8	46.7	126.2
Vitamin B_6, mg						
Females: HIV+	3.5 ± 0.1	0.9	2.1	3.3	4.6	7.3
HIV-	3.2 ± 0.2	1.0	1.8	2.8	4.3	6.2
Males: HIV+	4.0 ± 0.3	1.2	2.3	3.4	5.8	8.0
HIV-	3.8 ± 0.4	1.4	2.8	3.3	4.6	10.8
Vitamin C, mg						
Females: HIV+	289.8 ± 14.0	69.2	150.3	233.0	380.2	644.4
HIV-	297.3 ± 20.9	65.3	140.3	241.5	430.0	663.3
Males: HIV+	356.6 ± 33.6	56.7	167.5	260.8	494.4	968.0
HIV-	357.3 ± 50.9	80.1	163.2	299.5	464.6	1107.2
Folic Acid, µg						
Females: HIV+	836.3 ± 36.4	201.7	441.0	716.3	1159.0	1842.3
HIV-	768.4 ± 57.9	185.5	419.2	590.1	1083.8	1925.3
Males: HIV+	889.5 ± 59.6	262.9	478.8	851.9	1267.2	1846.3
HIV-	889.5 ± 87.9	279.0	481.1	709.2	1023.6	2101.1
Vitamin D, IU						
Females: HIV+	376.2 ± 22.8	40.6	129.6	299.1	542.4	992.8
HIV-	329.4 ± 30.0	35.7	94.1	240.3	517.5	810.9
Males: HIV+	415.9 ± 34.7	76.3	160.2	385.1	603.3	981.6
HIV-	352.8 ± 49.6	55.0	110.7	351.0	512.9	990.6
Vitamin E, mg α-tocopherol						
Females: HIV+	16.4 ± 0.9	3.6	8.8	15.2	20.9	33.0
HIV-	15.0 ± 0.9	4.0	8.8	14.0	19.0	29.1
Males: HIV+	21.3 ± 2.8	6.4	9.9	16.7	25.0	47.2
HIV-	18.0 ± 2.6	6.0	9.4	15.1	22.3	56.0

[a] n=196; [b] n=98; [c] n=68; [d] n=29; Differences by HIV status for males and females tested separately using two-sample t-tests with separate variance—no significant differences found.

Table 19. Mineral intake from food and supplement by sex and HIV status for the REACH cohort study

		Percentiles				
	Mean ± SE	**5th**	**25th**	**50th**	**75th**	**95th**
Calcium, mg						
Females: HIV+ [a]	1131.1 ± 43.8	317.4	653.4	1088.1	1507.3	2382.1
HIV- [b]	1093.8 ± 69.5	329.9	630.2	856.6	1422.7	2733.9
Males: HIV+ [c]	1394.6 ± 91.1	390.1	787.3	1210.1	1920.4	2775.2
HIV- [d]	1177.6 ± 96.8	465.0	856.0	1024.8	1532.5	2377.0
Phosphorus, mg	Supplement information not available					
Iron, mg						
Females: HIV+	35.2 ± 1.8	8.0	18.3	29.4	42.5	95.8
HIV-	32.1 ± 2.9	8.0	15.4	23.7	38.2	100.1
Males: HIV+	35.8 ± 2.7	10.4	11.8	17.5	29.3	86.1
HIV-	30.9 ± 3.7	10.3	17.7	28.3	34.3	93.1
Zinc, mg						
Females: HIV+	21.2 ± 0.9 *	4.6	12.0	20.3	28.1	46.7
HIV-	18.4 ± 1.1 *	5.4	9.9	16.2	25.6	35.9
Males: HIV+	22.2 ± 2.1	7.7	11.6	20.4	31.7	64.5
HIV-	25.0 ± 2.0	8.3	13.6	21.2	27.3	46.1
Magnesium, mg						
Females: HIV+	427.1 ± 15.4	123.6	276.9	379.5	557.2	868.6
HIV-	408.7 ± 23.5	153.2	254.3	358.3	512.6	838.2
Males: HIV+	511.2 ± 35.1	176.1	301.0	451.3	649.2	1157.8
HIV-	458.8 ± 36.1	198.8	298.8	430.3	522.2	957.9

[a] n=196; [b] n=98; [c] n=68; [d] n=29; Differences by HIV status for males and females tested separately using two-sample t-tests with separate variance; * $p < 0.05$

Table 20. Dietary Reference Intakes by age and sex

Nutrient	Age (y)	Males	Females	Pregnant
		Estimated Average Intake (EAR)		
Vitamin A, μg RAE [1]	**9-13**	445	420	--
	14-18	630	485	530
	19-30	625	500	550
Thiamin, mg	**9-13**	0.7	0.7	--
	14-18	1.0	0.9	1.2
	19-30	1.0	0.9	1.2
Riboflavin, mg	**9-13**	0.8	0.8	--
	14-18	1.1	0.9	1.2
	19-30	1.1	0.9	1.2
Niacin, mg NE	**9-13**	9	9	
	14-18	12	11	14
	19-30	12	11	14
Vitamin B_6, mg	**9-13**	0.8	0.8	
	14-18	1.1	1.0	1.6
	19-30	1.1	1.1	1.6
Vitamin C, mg	**9-13**	39	39	
	14-18	63	56	66
	19-30	75	60	70
Folate, μg DFE [2]	**9-13**	250	250	
	14-18	330	330	520
	19-30	320	320	520
Vitamin E, mg	**9-13**	9	9	
α-tocopherol [2]	**14-18**	12	12	12
	19-30	12	12	12
Phosphorus, mg	**9-13**	1055	1055	
	14-18	1055	1055	1055
	19-30	580	580	580
Iron, mg	**9-13**	5.9	5.7	
	14-18	7.7	7.9	23
	19-30	6.0	8.1	22
Zinc, mg	**9-13**	8.0	8.0	
	14-18	8.5	7.5	10.5
	19-30	9.4	6.8	9.5
Magnesium, mg	**9-13**	200	200	
	14-18	340	300	335
	19-30	330	255	390
		Adequate Intakes (AI)		
Calcium, mg	**9-13**	1300	1300	
	14-18	1300	1300	1300
	19-30	1000	1000	1000
Vitamin D, μg	**9-13**	5.0 (200 IU)	5.0	
	14-18	5.0	5.0	5.0
	19-30	5.0	5.0	5.0

Adapted from: Institute of Medicine, Dietary Reference Intakes, 1997, 1998, 2000, 2001.

[1] Retinol Activity Equivalents; 1 RAE=1 μg retinol + (α-carotene + cryptoxanthin)/24 + β-carotene/12

[2]As dietary folate equivalents; 1 DFE =1 μg folate from food; Over-estimation of prevalence of inadequacy since amount of folic acid fortified in foods not available.

[3]As α-tocopherol; 1 mg α-tocopherol= 1 α-tocopherol equivalents from food x 0.8

Table 21. Prevalence of inadequate micronutrient intake from food for the REACH cohort study

						CD4+ T-cell counts		
	Total	Female	Male	HIV+	HIV-	≥500	200-499	<200
n	391	294	97	264	127	129	102	33
Prevalence of inadequacy--Percent below Estimated Average Requirement (EAR)								
Vitamins								
Vitamin A	24.6	24.5	24.7	23.1	27.6	23.3	19.6	33.3
Thiamin	5.9	7.1 #	2.1 #	6.4	4.7	6.2	5.9	9.1
Riboflavin	4.3	5.4 #	1.0 #	4.9	3.2	5.4	3.9	6.1
Niacin	4.3	4.8	1.0	3.8	3.9	4.7	2.0	6.1
Vitamin B6	6.7	8.2 *	2.1 *	6.4	7.1	6.2	4.9	12.1
Vitamin C	6.4	6.1	7.2	7.2	4.7	10.1	2.9	9.1
Folic Acid	20.7	23.1 *	13.4 *	22.0	18.1	20.9	22.5	24.2
Vitamin E	53.5	55.4	47.4	50.4 #	59.8 #	52.7	48.0	48.5
Minerals								
Phosphorus	5.4	6.5	2.1	5.7	4.7	6.2	3.9	9.1
Iron	6.4	8.1 *	1.0 *	6.6	5.9	6.9	5.7	8.5
Zinc	13.3	12.9	14.4	13.3	13.4	12.4	12.7	18.2
Magnesium	34.3	33.0	38.1	33.0	37.0	34.9	30.4	33.3
Prevalence of adequacy--Percent above Adequate Intake (AI)								
Calcium	45.8	44.2	50.5	50.0 *	37.0 *	47.3	52.0	54.5
Vitamin D	46.3	44.6	51.5	48.1	42.5	43.4	52.0	54.5

* $p < 0.05$; # $p < 0.10$; Fisher's exact test for differences by sex and by HIV infection for cells with frequency <5; Chi^2 Goodness-of-fit test for differences by CD4 cell count.

Table 22. Regression models predicting the log of micronutrient intakes with exclusions[1] for the REACH cohort study

Dependant variable (Natural log)	Vitamin A µg RAE	Vitamin C mg	Vitamin E mg	Iron mg	Zinc mg
Explanatory variables			**Coefficients (Std Error)**		
Constant	6.143	4.895	1.893	2.945	2.337
	(0.068)	(0.063)	(0.091)	(0.046)	(0.048)
Energy/1000 kcal	0.161***	0.198***	0.289***	0.262***	0.175***
(centered)	(0.016)	(0.017)	(0.009)	(0.014)	(0.013)
Male					-0.088**
					(0.030)
Black/non-Hispanic	0.064	0.223***			
	(0.042)	(0.055)			
Living on own		0.153***	0.060*		
		(0.056)	(0.030)		
Vitamin/mineral	0.127**		0.091**	0.087**	
supplement use	(0.042)		(0.028)	(0.031)	
RCMAS anxiety index	0.000				
	(0.004)				
HIV+ CD4+ T-cells ≥		-0.104#	-0.054#	-0.080*	
cells/µL		(0.055)	(0.029)	(0.031)	
HIV+ CD4+ T-cells	-0.045				
200-499 cells/µL	(0.068)				
Food pyramid servings					
Dairy	0.165***				0.077***
	(0.017)				(0.011)
Vegetables	0.073***			0.027	
	(0.011)			(0.008)**	
Fruit		0.232**			
		(0.022)			
Meat/other protein				0.027**	0.085***
				(0.008)	(0.008)
Percent of energy from			0.015***		
fat			(0.002)		
Interactions					
Energy (centered)[2]	-0.010**	-0.021***	-0.022***	-0.023***	-0.022***
	(0.003)	(0.004)	(0.002)	(0.003)	(0.002)
RCMAS anxiety *	0.016*				
CD4+ T-cells 200-499	(0.007)				
cells/µL					
N	388	390	390	389	389
Adjusted R^2	0.663	0.512	0.787	0.766	0.820
Std error of estimate	0.383	0.497	0.266	0.288	0.249

Significance of explanatory variables: ***p <0.001; ** p <0.01; * p <0.05; # p <0.10

[1] Participants with high leverage values and outliers were excluded from models

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