Dietary Patterns and Health Outcomes among African American Maintenance Hemodialysis Patients

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Abstract: The association between dietary patterns and health outcomes, such as quality of life (QOL), in maintenance hemodialysis (MHD) patients with certain racial backgrounds has not been studied in detail. QOL is a powerful outcome measure in which dietary patterns could be a modifying factor. This study is a secondary analysis examining the association between dietary patterns and health outcomes in 101 African American (AA) maintenance hemodialysis (MHD) patients participating in the Palm Tocotrienols in Chronic Hemodialysis (PATCH) study. Quality of life (QOL) was assessed using the Kidney Disease Quality of Life 36-item survey (KDQOL-36™). Blood samples were analyzed for lipids, lipoprotein subfractions, and inflammatory markers. Food intake was measured using six non-consecutive 24-h dietary recalls over 15 months. Implausible energy intake reports were screened out by comparing reported energy intake (rEI) with predicted total energy expenditure (pTEE). Cluster analysis, using the k-means algorithm, identified two distinct dietary patterns in the study population: a high “sugar sweetened beverage” pattern (hiSSB) and a low “sugar sweetened beverage pattern” (loSSB). In the hiSSB group, consumption of SSB accounted for ~28% of energy intake, while SSB represented only 9% of energy intake in the loSSB group. The hiSSB group was characterized by a higher intake of total calories, sugar and percentage of kilocalories from carbohydrates, whereas the percentage of kilocalories from protein and fat was lower. While additional micronutrient intakes differed between groups (vitamin C, zinc, chromium), these were significantly lower than recommended values in the entire cohort. Patients in the hiSSB group presented with lower high-density lipoprotein cholesterol (HDL-C), lower large HDL particles and smaller low density lipoprotein (LDL) particle diameters. Antidepressant usage was significantly higher in the hiSSB group. Patients in the hiSSB group scored lower across all five KDQOL domains and scored significantly lower in the mental composite domain. MHD patients following a hiSSB dietary pattern had smaller dense LDL particles, lower HDL-C, and a lower QOL. Suboptimal intakes of fruits, vegetables, and grains as well as key micronutrients were evident in both patterns.

Keywords: hemodialysis; maintenance hemodialysis; dietary patterns; cluster analysis; quality of life; lipoproteins; inflammation
1. Introduction

The global prevalence of chronic kidney disease (CKD) is estimated to be 11%–13%, [1] affecting approximately 30 million Americans. With an increasing prevalence of CKD, the impact of treatment is projected to confer a significant economic and social burden [2]. In the United States, African Americans (AA) suffer a disproportionate burden of end stage renal disease (ESRD), comprising 35% of all dialysis patients, as they are 3.7 times more likely to progress to ESRD than whites [3,4]. This increase in risk is partially attributed to higher rates of hypertension (HTN), diabetes mellitus (DM), and cardiovascular disease [5]. Primary prevention strategies focused on preventing the development of CKD include dietary modification. “Unhealthy” dietary patterns, such as those high in fat and sugar [6], have been associated with significantly increased CKD incidence and progression risk [7], whereas healthier diet patterns, which include higher intakes of vegetables, fruits, legumes, nuts, whole grains, fish, and low-fat dairy, and lower intakes of red and processed meats, sodium, and sugar-sweetened beverages, have been associated with a lower incidence of CKD [8].

Patients requiring maintenance hemodialysis (MHD) are expected to make major lifestyle changes, including adherence to a conventional renal diet limited in fruits, vegetables, nuts, legumes, dairy, and whole grains due to concerns about both phosphorus and potassium [9]. However, there are few studies examining the association between dietary patterns and health outcomes, especially for the AA ESRD population [10].

Two common analytical approaches used to identify dietary patterns in nutritional epidemiology are à priori and à posteriori methods [11]. À priori methods are based on indices of diet quality or scores defined by nutritional health, whereas à posteriori methods, such as factor and cluster analyses, use multivariate statistical techniques to derive dietary patterns [12]. By reducing diet data into mutually exclusive patterns, a cluster analysis approach separates individuals with similar mean dietary intakes into non-overlapping groups from which health outcomes can be compared [13,14]. This study therefore applied an à posteriori approach to evaluate dietary patterns of AA patients on MHD (AA-MHD) to understand the impact of diet on health outcomes.

2. Materials and Methods

2.1. Subjects

Data were collected by trained research personnel from subjects participating in the Palm Tocotrienols in Chronic Hemodialysis (PATCH) Study (NCT02358967) and were extracted for the secondary analysis of the current study. Briefly, the PATCH Study was a randomized, double-blind, placebo-controlled trial evaluating the effects of daily supplementation with 300 mg of a vitamin E tocotrienol-rich fraction (TRF) on markers of inflammation, oxidative stress, and blood lipids in patients undergoing MHD at multiple dialysis clinics, both within Michigan, USA, and overseas. The criteria for study participation included patients with ESRD receiving thrice-weekly hemodialysis for at least 120 days, aged over 18 years, with a life expectancy of over one year, and the ability to understand and provide informed consent. Exclusion criteria included a history of poor adherence to hemodialysis, active malignancy, AIDS, and patients receiving nutritional support. This current report is based on the data collected from the Michigan cohort (135 subjects enrolled). Subjects were enrolled between June 2017 and February 2018, with all study procedures completed by April 2019. The study was approved by the ethics boards of participating dialysis units and Wayne State University’s Institutional Review Board. All subjects provided written informed consent.

2.2. Collection of Clinical Information

We used baseline clinical laboratory data, heights, and post-dialysis weights, which were obtained from patients’ medical records collected from the original study. Body mass index was calculated based on Quetelet’s Index [15].
2.3. Blood Sampling and Lipid Measurement

In the original study, pre-dialysis non-fasting blood samples were collected in Ethylenediaminetetraacetic acid (EDTA) or lithium heparin tubes and kept on ice. Plasma samples were transported to Wayne State University within two hours and centrifuged at 3500 rpm for 10 min to separate plasma. Aliquots were stored at −80 °C. C-Reactive Protein (CRP), Interleukin 6 (IL-6), Interleukin 18 (IL-18), and Monocyte Chemoattractant Protein-1 (MCP-1) samples were analyzed in duplicates in 384-well AlphaPlates™ (PerkinElmer®). IL-6 values lower than the detection limit (1.3 pg/mL) were assigned a value of 0.01 pg/mL. The plasma total cholesterol (TC) and triglycerides (TAG) were determined by enzymatic assays (Pointe Scientific Inc., Canton, MI, USA). High density lipoprotein cholesterol (HDL-C) was measured in the supernatant after precipitation of apoB-containing lipoproteins by dextran sulfate and magnesium ions (Pointe Scientific Inc., Canton, MI, USA). Low density lipoprotein cholesterol (LDLC) was calculated using the Friedwald equation by difference (LDLC = TC − HDLC − TAG/5). HDL and LDL subfractions from plasma were also measured via polyacrylamide gel electrophoresis using the Lipoprint™ System (Quantimetrix Corporation, Redondo Beach, CA, USA). Using the manufacturer’s proprietary software, HDL and LDL subfractions were quantitated after electrophoresis. Both HDL and LDL were then grouped into large, intermediate, and small subfractions. The Lipoprint™ system is U.S. Food and Drug Administration (FDA) certified for LDL measurements; however, values for HDL are for research purposes only.

2.4. Assessment of Kidney Disease Quality of Life (KDQOL)

As a measure of health-related quality of life (HRQOL), Kidney Disease Quality of Life 36-item surveys (KDQOL-36™) were administered at baseline [16,17] by the same research team member for a subset of the original study population. The KDQOL-36 is comprised of five subscales calculated separately: 1) SF-12 physical component summary (PCS), 2) SF-12 mental component summary (MCS), 3) burden of kidney disease, 4) symptoms of kidney disease, and 5) effects of kidney disease. Subscale scores range from 0 to 100, with lower scores indicating poor self-reported QOL [18,19].

2.5. Assessment of Dietary Intake

In the original study, six 24-h dietary recalls taken on non-dialysis days were collected in person by the same registered dietitian quarterly over a 15-month time period [20,21] using the U.S. Department of Agriculture (USDA) five-pass method [22]. Reported energy intake (rEI) and nutrient analysis were calculated using the Food Processor SQL software package (version 11.2, 2016, ESHA Research, Salem, OR, USA). To reduce the effect of confounding from physiologically implausible rEI, dietary reports from over and under-reporters were screened out [23]. Using the method introduced by McCrory et al., which accounts for within-subject errors in rEI and predicted total energy expenditure (pTEE) without estimation of physical activity level, a 2 standard deviation cutoff was used to classify 24HR less than 56% or more than 144% of estimated energy needs as implausible [24]. Edema-free adjusted body weight was used for patients whose weight was less than 95% or greater than 115% of standard body weight [25].

2.6. Statistical Analysis

Diet analyses revealed thirty-three food groups, which were converted to percent contribution of total daily energy (%TE) intakes [26]. Cluster analysis was performed using the k-means algorithm, a nonhierarchical clustering method which classifies participants into non-overlapping groups based on Euclidian distance. A set of two clusters was selected as the solution that provided a sample size large enough to allow for analyses of distinct groups. Cluster membership was concordant for 84.2% of all subjects in clusters 1 and 2 (κ = 0.674). The two clusters were named according to the food group providing the greatest total energy intake (hiSSB and loSSB) [12,27].
Differences in nutrient intakes, health characteristics, and KDQOL across the food intake patterns were compared according to data distribution, using the t test or Mann–Whitney U test for continuous variables and Pearson chi-square test for categorical variables. For all tests, the level of significance was set as \( p < 0.05 \). All analyses were performed using SPSS (version 25, SPSS Inc., Chicago, IL, USA).

3. Results

3.1. Clusters Identified for Dietary Patterns

We identified two major dietary patterns: cluster 1, with a high “sugar sweetened beverage” pattern (hiSSB) and a low “sugar sweetened beverage” pattern (loSSB). As illustrated in Table 1, the hiSSB dietary pattern was characterized by higher energy contributions from calorically sweetened soft and juice drinks \( (p < 0.001) \) and poultry \( (p < 0.05) \), whereas the greatest energy contributors to the loSSB group were unprocessed red meat \( (p < 0.05) \), fish and shellfish, \( (p < 0.05) \), and custard style desserts such as puddings, ice cream, and cheesecake \( (p < 0.05) \). A total of 47 patients were classified in the hiSSB pattern and 54 in the loSSB pattern.

Table 1. Percentage of energy contribution of food groups.

| Food Groups                                      | Pattern 1 (hiSSB) | Pattern 2 (loSSB) | \( p \)  |
|-------------------------------------------------|-------------------|-------------------|--------|
| Sugar sweetened beverages                       | 27.97 ± 9.27      | 9.45 ± 5.65       | <0.001 |
| Unprocessed red meat                            | 0.95 ± 1.49       | 2.17 ± 3.49       | 0.022  |
| Poultry                                         | 4.67 ± 5.99       | 2.51 ± 2.47       | 0.024  |
| Fish and shellfish                              | 0.49 ± 0.93       | 1.42 ± 2.87       | 0.028  |
| Puddings, ice cream, cheesecake                  | 0.24 ± 0.78       | 0.76 ± 1.70       | 0.049  |
| Processed and cured meats (bacon, sausage, hot dogs) | 2.67 ± 2.47       | 3.62 ± 3.39       | 0.106  |
| Dairy, low-fat and 2%                            | 1.75 ± 4.61       | 0.59 ± 2.30       | 0.122  |
| Egg and egg dishes                              | 2.43 ± 2.60       | 3.29 ± 3.08       | 0.132  |
| Vegetables, canned, fresh and frozen            | 3.25 ± 4.20       | 4.45 ± 4.11       | 0.151  |
| Fast foods, frozen and convenience entrees      | 4.46 ± 6.67       | 2.92 ± 4.87       | 0.194  |
| Pizza, pasta and lasagna                        | 2.77 ± 4.95       | 4.25 ± 6.58       | 0.200  |
| Butter, margarine, animal fats                  | 0.25 ± 0.41       | 0.41 ± 0.95       | 0.256  |
| Potatoes, mashed and salad                      | 0.87 ± 2.16       | 1.39 ± 2.55       | 0.270  |
| Beans and legumes                               | 0.43 ± 1.25       | 0.75 ± 1.92       | 0.315  |
| Potatoes, fried and hash browns                 | 1.39 ± 2.26       | 0.96 ± 2.18       | 0.330  |
| Fruit, canned, fresh and dried                  | 2.02 ± 4.61       | 1.28 ± 2.53       | 0.335  |
| Oils (vegetable, olive, canola)                 | 0.03 ± 0.09       | 0.05 ± 0.20       | 0.398  |
| Crackers, chips and popcorn                     | 2.12 ± 4.80       | 1.60 ± 2.57       | 0.509  |
| Candy                                           | 0.47 ± 1.11       | 0.34 ± 1.01       | 0.542  |
| Sauce and condiments, savory                    | 0.83 ± 1.50       | 0.69 ± 0.92       | 0.597  |
| Nuts and seeds and nut butters                  | 0.24 ± 0.84       | 0.17 ± 0.86       | 0.665  |
| Dairies, full-fat and creamer                   | 1.00 ± 3.00       | 0.75 ± 3.18       | 0.691  |
| Cakes, cookies, pie, donuts, and rich dough     | 1.71 ± 2.34       | 1.78 ± 2.70       | 0.885  |
| Grains                                          | 6.38 ± 8.63       | 6.62 ± 8.26       | 0.888  |
| Pork                                            | 0.71 ± 2.22       | 0.76 ± 1.65       | 0.889  |
| Sauces and condiments, sweet                    | 0.40 ± 0.62       | 0.39 ± 0.68       | 0.945  |

Values are mean ± SD; \( n = 47 \) for hiSSB and 54 for the loSSB group. %TE food: the percentage total energy contribution from food.

3.2. Baseline Characteristics

Subjects were 100% African American, 59% male, aged 25–87, and with an average Body Mass Index (BMI) of 27 kg/m². About 2/3 had a diagnosis of DM and 1/3 were tobacco users (Table 2).
### Table 2. Patient characteristics at baseline according to diet cluster.

| Age, year (n = 100) | hiSSB (n = 47) | loSSB (n = 53) | p Value between Groups |
|---------------------|---------------|---------------|------------------------|
| 60 (53–66)          | 60 ± 14       |               | 0.662                  |

| Ethnicity | All (n = 100) | hiSSB (n = 47) | loSSB (n = 53) | p Value between Groups |
|-----------|---------------|---------------|---------------|------------------------|
| African American, n (%) | 100 (100) | 47 (47) | 54 (53) | —                      |
| Males, n (%) | 59 (59) | 29 (29) | 30 (30) | 0.605                  |

| BMI Category, n(%) | All (n = 100) | hiSSB (n = 47) | loSSB (n = 53) | p Value between Groups |
|--------------------|---------------|---------------|---------------|------------------------|
| Underweight (BMI < 18.5) | 3 (3) | 1 (1) | 2 (2) | 0.028                  |
| Normal Weight (18.5–24.9) | 31 (31) | 8 (8) | 23 (23) |                         |
| Overweight (BMI 25–29.9) | 31 (31) | 19 (19) | 12 (12) |                         |
| Obese (BMI > 30) | 35 (35) | 19 (19) | 16 (16) |                         |

| Cause of Kidney Failure | All (n = 100) | hiSSB (n = 47) | loSSB (n = 53) | p Value between Groups |
|-------------------------|---------------|---------------|---------------|------------------------|
| Diabetes Mellitus, n (%) | 47 (47) | 24 (24) | 23 (23) | 0.663                  |
| Hypertension, n (%) | 37 (37) | 18 (18) | 19 (19) |                         |
| Glomerulonephritis, n (%) | 5 (5) | 2 (2) | 3 (3) |                         |
| SLE, n (%) | 2 (2) | 0 (0) | 2 (2) |                         |
| HIV-Nephropathy, n (%) | 2 (2) | 0 (0) | 2 (2) |                         |
| Others, n (%) | 5 (5) | 2 (2) | 3 (3) |                         |
| Unknown, n (%) | 2 (2) | 1 (1) | 1 (1) |                         |
| Insulin Use, n (%) | 30 (30) | 20 (20) | 10 (10) | 0.010                  |

| Oral Hypoglycemic Agent Use, n (%) | All (n = 100) | hiSSB (n = 47) | loSSB (n = 53) | p Value between Groups |
|-----------------------------------|---------------|---------------|---------------|------------------------|
| Tobacco Use, n (%) | 29 (29) | 14 (14) | 15 (15) | 0.870                  |

| Vascular Access | All (n = 100) | hiSSB (n = 47) | loSSB (n = 53) | p Value between Groups |
|-----------------|---------------|---------------|---------------|------------------------|
| Arteriovenous fistula, n (%) | 59 (59) | 28 (28) | 31 (31) |                         |
| Arteriovenous graft, n (%) | 26 (26) | 12 (12) | 15 (14) | 0.993                  |
| Catheter, n (%) | 15 (15) | 7 (7) | 8 (8) |                         |

| Blood Pressure, mmHg (post-sitting) | All (n = 100) | hiSSB (n = 47) | loSSB (n = 53) | p Value between Groups |
|------------------------------------|---------------|---------------|---------------|------------------------|
| Systolic 138 (118–155) | 141 ± 24 | 139 ± 21 | 0.620 |                         |
| Diastolic 77 (70–83) | 78 ± 10 | 80 ± 22 | 0.483 |                         |
| 2 Kt/V 1.5 (1.4–1.6) | 1.5 ± 0.2 | 1.6 ± 0.3 | 0.084 |                         |
| Antidepressant use, % | 17 (17) | 12 (12) | 5 (5) | 0.032                  |
| Renal vitamin * use, % | 56 (56) | 24 (24) | 32 (32) | 0.349                  |
| Serum Potassium (mEq/L) | 4.6 (4.1–5.1) | 4.7 ± 0.6 | 4.6 ± 0.6 | 0.632                  |
| Serum Phosphorus (mg/dL) | 5.0 (4.3–5.8) | 5.2 ± 1.5 | 5.0 ± 1.0 | 0.285                  |
| Albumin (g/dL) | 3.8 (3.6–4.0) | 3.8 ± 0.4 | 3.8 ± 0.3 | 0.948                  |
| CRP (mg/L) | 6.1 (3.1–8.4) | 6.2 ± 4.8 | 6.0 ± 3.2 | 0.745                  |
| IL-6 (pg/mL) | 1.7 (0.01–5.3) | 10.9 ± 36 | 13.2 ± 54.4 | 0.799                  |
| IL-18 (pg/mL) | 238 (172–320) | 276 ± 16 | 259 ± 163 | 0.594                  |
| MCP-1 (pg/mL) | 113 (89–160) | 155 ± 127 | 134 ± 97 | 0.373                  |
| Total Cholesterol (mg/dL) | 148 (113–188) | 147 ± 41 | 154 ± 47 | 0.471                  |
| Triglycerides (mg/dL) | 81 (53–125) | 106 ± 52 | 87 ± 51 | 0.069                  |
| LDL-C (mg/dL) | 76 (46–104) | 80 (8) | 80 ± 45 | 0.987                  |
| HDL-C (mg/dL) | 47 (39–62) | 46 ± 16 | 56 ± 21 | 0.007                  |
| Large HDL (mg/dL) | 17.0 (11.0–31.8) | 17.9 ± 12.7 | 27.2 ± 17.8 | 0.003                  |
| Intermediate HDL (mg/dL) | 23.0 (18.3–26.0) | 21.7 ± 5.4 | 23.8 ± 6.1 | 0.081                  |
| Small HDL (mg/dL) | 6.0 (4.0–8.0) | 6.7 ± 3.1 | 5.5 ± 3.4 | 0.076                  |
| Intermediate LDL (mg/dL) | 11.0 (7.0–16.0) | 13.7 ± 7.8 | 11.8 ± 8.4 | 0.251                  |
| Small LDL (mg/dL) | 2.0 (0.0–5.3) | 4.9 ± 6.1 | 3.1 ± 4.8 | 0.103                  |
| Mean LDL Size (Å) | 270.0 (266.0–273.0) | 268.0 ± 4.6 | 270.3 ± 4.1 | 0.009                  |
| LDL Pattern A, n (%) | 62 (61.4) | 22 (21.8) | 40 (39.6) | 0.050                  |
| LDL Pattern B, n (%) | 16 (15.8) | 10 (9.9) | 6 (5.9) | 0.050                  |

1 Excludes 1 hypertriglyceridemic subject. Values are expressed as median (Q1–Q3), means ± SDs, or percentages. Statistics: 2-sample t test, or Pearson chi-square test. BMI: Body Mass Index; SLE: Systemic lupus erythematosus; Kt/V is a number used to quantity hemodialysis (K, dialyzer clearance of urea; t, dialysis time; V, volume of distribution of urea, approximately equal to patient’s total body water); * Renal vitamins included Nephrocare, Renal Caps, and Nephrocare brands; CRP: C-reactive protein; IL-6: Interleukin 6; IL-18: Interleukin 18; MCP-1: Monocyte Chemotactic Protein-1; HDL: high density lipoprotein cholesterol; LDL: low density lipoprotein cholesterol; Å: angstrom; 1 BP hiSSB (n = 44), loSSB (n = 43); 2 KuV hiSSB (n = 43), loSSB (n = 45); 3 Albumin hiSSB (n = 46), loSSB (n = 50); 4 CRP, 5 IL-6, 6 MCP-1; 7 hiSSB (n = 46), loSSB (n = 52); 8 IL-18 hiSSB (n = 46), loSSB (n = 49); 9 large, 10 intermediate, and 11 small LDL, 12 mean LDL size and 13 LDL pattern hiSSB (n = 46), loSSB (n = 52).
3.3. Characteristics of Patients According to Dietary Cluster

We compared the baseline characteristics of the subjects by diet clusters. To minimize statistical bias as an influential outlier, one subject with hypertriglyceridemia (plasma TG 943 mg/dL) was excluded in subsequent analyses. Kidney specific clinical parameters did not differ significantly between the two diet clusters. Patients in the hiSSB group had a significantly higher BMI and were more likely to be prescribed an antidepressant than those patients in the loSSB group. Both total HDL cholesterol and large HDL subfractions were significantly lower in the hiSSB group than in the loSSB group. Patients in the hiSSB group were 2.4 times more likely to have a pattern B phenotype, characterized by predominantly smaller and denser LDL subfractions. The hiSSB dietary pattern di

3.4. Nutrient Intake According to Dietary Cluster

Table 3 outlines the nutrient intake of the subjects according to their diet cluster. The macronutrient distribution was significantly different between the two dietary clusters, with a larger proportion of energy intake from carbohydrate in the hiSSB group and from fat and protein in the loSSB group. Macronutrient intakes for total energy and sugar were significantly higher in the hiSSB group and intakes for cholesterol were significantly higher in the loSSB group. Micronutrient intakes differed significantly between groups for vitamin C, zinc (Zn), chromium (Cr), and selenium (Se). With the exception of vitamin C, intakes for these nutrients were lower in the hiSSB group compared to the loSSB group.

Table 3. Mean daily nutrient intake according to diet cluster.

| Nutrient               | All (n = 100) | hiSSB (n = 47) | loSSB (n = 53) | P Value between Groups | Nutrient Recommendations |
|------------------------|--------------|---------------|---------------|------------------------|-------------------------|
| Energy, kcals          | 2027 ± 414   | 2123 ± 432    | 1941 ± 381    | 0.029                  | Per renal Rx            |
| Protein, g             | 86 ± 26      | 83 ± 21       | 88 ± 29       | 0.311                  | Per renal Rx            |
| % kcals from protein   | 17 ± 4       | 16 ± 4        | 18 ± 4        | 0.008                  | 10–35 *                 |
| Fat, g                 | 90 ± 24      | 89 ± 25       | 91 ± 24       | 0.713                  |                         |
| % kcals from CHO       | 40 ± 7       | 38 ± 6        | 42 ± 7        | 0.001                  | 20–35 *                 |
| % kcals from CHO       | 43 ± 8       | 47 ± 7        | 40 ± 7        | <0.001                 | 45–65 *                 |
| % kcals from CHO       | 12 ± 4       | 13.2 ± 3.9    | 11.8 ± 4.5    | 0.095                  | 30 *                    |
| Sugars, g              | 87 ± 45      | 115 ± 47      | 63 ± 23       | <0.001                 | <50 **                  |
| Phosphorus, mg         | 833 ± 303    | 797 ± 260     | 864 ± 335     | 0.263                  | Per renal Rx            |
| Iron, g                | 11.5 ± 4.5   | 11.3 ± 4.1    | 11.6 ± 4.9    | 0.800                  | Per renal Rx            |
| Magnesium, mg          | 149 ± 64     | 150 ± 65      | 147 ± 64      | 0.852                  | 420 *                   |
| Sodium, mg             | 2996 ± 961   | 3036 ± 1013   | 2960 ± 921    | 0.695                  | <2300 **                |
| Potassium, mg          | 1500 ± 585   | 1487 ± 636    | 1512 ± 542    | 0.834                  | Per renal Rx            |
| Zinc, mg               | 8.5 ± 4.1    | 7.4 ± 2.9     | 9.4 ± 4.8     | 0.017                  | 11 *                    |
| Vitamin C, mg          | 58 ± 46      | 69 ± 35       | 48 ± 33       | 0.023                  | 90 *                    |
| Vitamin E, mg          | 4.3 ± 3.1    | 4.4 ± 3.4     | 4.1 ± 2.9     | 0.628                  | 15 *                    |
| Chromium (µg)          | 4.4 ± 6.9    | 2.9 ± 4.5     | 5.7 ± 8.2     | 0.031                  | 30 *                    |
| Selenium (µg)          | 88.2 ± 37.8  | 79.6 ± 35.3   | 95.8 ± 38.7   | 0.023                  | 55 *                    |
| Folic acid, mg         | 225 ± 108    | 230 ± 112     | 221 ± 105     | 0.707                  | 400 *                   |
| Cholesterol, mg        | 412 ± 177    | 368 ± 153     | 452 ± 188     | 0.016                  | Per renal Rx or (<200) [28] |

USDA My plate recommendations (%)

|       |       |       |       |       |       |
|-------|-------|-------|-------|-------|-------|
| Grain | 76 ± 33 | 75 ± 37 | 77 ± 30 | 0.749 |
| Vegetable | 33 ± 27 | 30 ± 25 | 35 ± 28 | 0.369 |
| Fruit | 24 ± 33 | 32 ± 41 | 16 ± 21 | 0.023 |
| Dairv | 13 ± 14 | 15 ± 14 | 12 ± 15 | 0.306 |
| Protein | 148 ± 64 | 132 ± 59 | 162 ± 65 | 0.019 |

1 Excludes 1 hypertriglyceridemic subject. Values are means ± SDs, or percentages. Statistics: 2-sample t test, or Pearson chi-square test. * DRI, Dietary Reference Intakes for males ages 51–70 [30]; CHO, carbohydrate; Rx, prescription; ** 2015–2020 Dietary Guidelines for Americans recommends <10 percent of calories per day from added sugars or 50 grams for a 2000 kcal [31].
Based on the Recommended Dietary Allowance (RDA), both clusters exceeded the Acceptable Macronutrient Distribution Range (AMDR) for fat [32] and the Dietary Guidelines for Americans’ (DGA) recommendations for both sodium and sugars [31]. Intakes for fiber, magnesium (Mg), Zn, vitamins C and E, Cr, and folic acid fell below RDA guidelines for both groups [32]. USDA My Plate recommendations fell below the recommended minimum serving amounts for all subjects for grains (76%), vegetables (33%), fruits (24%), and dairy (13%), while intakes exceeded minimum recommended servings of protein for both the hiSSB (132%) and loSSB (162%) clusters. [29].

3.5. KDQOL among MHD Patients According to Diet Clusters among MHD Patients

Those patients following a hiSSB diet pattern scored lower baseline values on all five KDQOL domains and significantly lower on the SF12 mental composite domain (p < 0.026) (Table S1) compared to those following a loSSB pattern (Figure 1). Additionally, in a univariate analysis we compared KDQOL scores across each domain for additional variables (Table S2), and found that symptoms and effects scores were positively associated with age, the burden score was positively associated with vintage, and the PCS score was negatively associated with vintage.

Our findings on this AA patient group on maintenance HD (AA-HD) are consistent with those of previous studies in non-CKD populations, which found that obesogenic dietary behaviors—such as a greater consumption of refined carbohydrates and fast foods, and low intakes of fruits and vegetables—were associated with lower self-reported QOL [33]. Alternatively, adherence to a healthy diet pattern, such as the Mediterranean diet, has been associated with better HRQOL, which may be partially explained by higher antioxidant content [34,35]. MHD patients are often instructed to avoid dairy products, fruits, vegetables, and whole grains in an effort to reduce phosphorus and potassium intake. At the same time, they are encouraged to consume protein rich foods, especially
animal products with high biological value proteins. To meet energy requirements, juices, “clear” sugar sweetened carbonated beverages, and higher fat non-dairy products are often suggested as acceptable choices.

It is worth noting that only 3% of the entire study population consumed at least four servings of fruits and vegetables per day, and thus the majority failed to achieve the minimum four to five daily servings recommended by the USDA for women and men aged 31–50 [31]. Dietary intakes for fiber, Mg, Zn, Cr, folic acid, and vitamins C and E fell below RDA guidelines for both groups, which may be attributed to the low consumption of micronutrient rich foods. The percentage of calories from carbohydrates fell below the AMDR range for the loSSB group and was at the lower end of the range for the hiSSB group. Both groups failed to meet the USDA recommendations for grains, with refined carbohydrates exceeding DGA guideline upper limits.

Micronutrient intakes for Zn, Cr, and Se were significantly lower in the hiSSB. Previous studies have found that low intakes of Zn and Se have been associated with lower QOL indicators. Low Zn status has been associated with impaired QOL due to lower physical ability and fatigue in non-CKD patients [36,37]. Zn deficiency can contribute to disturbances in taste (dysgeusia) and smell, which may lead to poor nutritional intake, a commonly observed issue in CKD patients [38–40]. Non-CKD elderly persons with lower Se intakes and serum levels reported poorer self-perceived health and chewing ability [41]. Poor intakes of nutrients such as Zn and Se may have played a role in the lower QOL scores observed in the hiSSB group.

Inadequate micronutrient intake may have also contributed to the lower total HDL cholesterol, large HDL subfractions, and the atherogenic pattern B phenotype observed in the hiSSB group. Low Zn status has been associated with lipid peroxidation and inflammation in those with CKD [42]. Lower Cr levels have been associated with malnutrition in HD patients [43], and with inflammation, increased cardiovascular risk, and lower levels of HDL in non-CKD populations [44,45]. Lower concentrations of Se have been associated with higher rates of hospitalization and death among HD patients [46]. The hiSSB group tended to have higher levels of CRP, IL-18, and MCP-1, although the difference in these inflammatory markers between the loSSB and hiSSB groups was not significant. Given that low grade inflammation may play a role as both a cause and consequence of low HDL-C levels [47,48], and higher HDL-C levels may act as a buffer against low-grade inflammation [49–51], the lower levels of HDL-C observed in the hiSSB group may have been a consequence of proinflammatory factors modulated by a high sugar dietary pattern.

Although the burden of following a restrictive diet has the potential to impair QOL [52], it has been found that HD patients who control their diet benefit from reduced symptom burden and enhanced general health and wellbeing [53]. In non-dialysis populations, following healthy dietary patterns has also been associated with improved QOL [54,55]. In the African American Study of Kidney Disease and Hypertension [30], both lower mental and physical health scores were associated with increased risk of cardiovascular events among blacks with hypertensive CKD. Researchers postulated that the lower QOL scores may have been attributed to several factors including poor self-care, resulting in poor compliance to treatment regimens such as prescribed medical nutrition therapy [56,57]. Additionally, Feroze et al. found that not only were lower mental health scores the most powerful predictors of mortality—with each 10-unit lower score associated with an approximately 12% higher death risk—but that these low scores were better mortality predictors among AA-HD patients compared to whites. Potential contributors to reduced QOL for MHD patients include surrogates of protein energy wasting, obesity, and higher levels of proinflammatory cytokines [58,59].

Unfortunately, there has been little improvement in HRQOL among HD patients over the past decade [60,61]. HD patients who are consuming an energy dense diet which relies heavily on hiSSB and fast foods may be lacking in several micronutrients [62]. Most renal specific vitamins, including the brands prescribed to the patients in this study, provide the water-soluble B vitamins and vitamin C lost through dialysis. Only a few renal specific vitamins provide additional micronutrients such as vitamin E, Se, and Zn. These patients may benefit from targeted nutritional intervention or supplementation.
Our study has important strengths, such as the inclusion of patients from multiple HD centers and the use of rigorous analytical and laboratory methods. However, this study has some limitations. First, diet data was not collected for dialysis days, and may not represent usual intake. Second, the small sample size and homogenous population can impact the ability to robustly detect differences between groups. Third, the KDQOL is not validated to specifically measure the impact of diet on health; however, to our knowledge, there is no specific instrument that measures dietary contribution to HRQOL for CKD patients. Fourth, causality cannot be determined due to the cross-sectional nature of this study. Therefore, it is unclear whether dietary patterns may have influenced QOL or if self-perceived QOL affected dietary choices. Finally, micronutrient values may have been underestimated, since not all commercial products in ESHA food processor software have complete information.

5. Conclusions

Lower KDQOL scores among MHD patients were associated with a dietary pattern characterized by high intakes of sugar sweetened beverages and reduced intakes of protein foods and vegetables. The clinical impact of excessive refined sugar intake was also associated with significantly lower intakes of several micronutrients, including Zn, Cr, and Se, potentially contributing to the lower KDQOL scores observed as well as lower levels of HDL cholesterol and large HDL subfractions. Further studies examining the role of dietary micronutrients as antioxidants with lipoprotein modification is warranted.

6. Practical Implication

Renal dietary guidelines generally focus on specific nutrients and foods to avoid in an effort to control serum phosphorus, potassium, and fluid, while at the same time encouraging adequate protein intake to avoid negative nitrogen balance. Less attention is given to diet-derived micronutrient intake. We found that a dietary pattern high in refined sugars can have a negative impact on several health outcomes, including QOL and lipid profiles in an African American HD cohort. Future studies examining diet patterns associated with improved outcomes in the HD population are needed.

Supplementary Materials: The following are available online at http://www.mdpi.com/2072-6643/12/3/797/s1, Table S1: KDQOL scores according to diet cluster. Table S2: Comparison of KDQOL score by selected variables.

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References
1. Hill, N.R.; Fatoba, S.T.; Oke, J.L.; Hirst, J.A.; O’Callaghan, C.A.; Lasserson, D.S.; Hobbs, F.D. Global Prevalence of Chronic Kidney Disease—A Systematic Review and Meta-Analysis. PLoS ONE 2016, 11, e0158765. [CrossRef] [PubMed]
2. Wang, V.; Vilme, H.; Maciejewski, M.L.; Boulware, L.E. The Economic Burden of Chronic Kidney Disease and End-Stage Renal Disease. Semin. Nephrol. 2016, 36, 319–330. [CrossRef] [PubMed]
3. The United States Renal Data System. Chapter 1: Incidence, Prevalence, Patient Characteristics, and Treatment Modalities. Am. J. Kidney Dis. 2017, 69, S261–S300. [CrossRef]
4. NKF. African Americans and Kidney Disease. Available online: https://www.kidney.org/news/newsroom/factsheets/African-Americans-and-CKD (accessed on 9 January 2020).

5. Harding, K.; Mersha, T.B.; Webb, F.A.; Vassalotti, J.A.; Nicholas, S.B. Current State and Future Trends to Optimize the Care of African Americans with End-Stage Renal Disease. *Am. J. Nephrol.* 2017, 46, 156–164. [CrossRef] [PubMed]

6. Asghari, G.; Momenan, M.; Yuzbashian, E.; Mirmiran, P.; Azizi, F. Dietary pattern and incidence of chronic kidney disease among adults: A population-based study. *Nutr. Metab.* 2018, 15, 88. [CrossRef]

7. Ajjarapu, A.S.; Hinkle, S.N.; Li, M.; Francis, E.C.; Zhang, C. Dietary Patterns and Renal Health Outcomes in the General Population: A Review Focusing on Prospective Studies. *Nutrients* 2019, 11, 1877. [CrossRef]

8. Bach, K.E.; Kelly, J.T.; Palmer, S.C.; Khalesi, S.; Strippoli, G.F.M.; Campbell, K.L. Healthy Dietary Patterns and Incidence of CKD: A Meta-Analysis of Cohort Studies. *Clin. J. Am. Soc. Nephrol.* 2019. [CrossRef]

9. Biruete, A.; Jeong, J.H.; Barnes, J.L.; Wilund, K.R. Modified Nutritional Recommendations to Improve Dietary Patterns and Outcomes in Hemodialysis Patients. *J. Ren. Nutr.* 2016. [CrossRef]

10. Kelly, J.T.; Palmer, S.C.; Wai, S.N.; Ruospo, M.; Carrero, J.J.; Campbell, K.L.; Strippoli, G.F. Healthy Dietary Patterns and Risk of Mortality and ESRD in CKD: A Meta-Analysis of Cohort Studies. *Clin. J. Am. Soc. Nephrol.* 2017, 12, 272–279. [CrossRef]

11. Ocke, M.C. Evaluation of methodologies for assessing the overall diet: Dietary quality scores and dietary pattern analysis. *Proc. Nutr. Soc.* 2013, 72, 191–199. [CrossRef]

12. Devlin, U.M.; McNulty, B.A.; Nugent, A.P.; Gibney, M.J. The use of cluster analysis to derive dietary patterns: Methodological considerations, reproducibility, validity and the effect of energy mis-reporting. *Proc. Nutr. Soc.* 2012, 71, 599–609. [CrossRef] [PubMed]

13. Sauvageot, N.; Schritz, A.; Leite, S.; Alkerwi, A.; Stranges, S.; Zannad, F.; Stree1, S.; Hoge, A.; Donneau, A.F.; Albert, A.; et al. Stability-based validation of dietary patterns obtained by cluster analysis. *Nutr. J.* 2017, 16, 4. [CrossRef] [PubMed]

14. Newby, P.K.; Tucker, K.L. Empirically derived eating patterns using factor or cluster analysis: A review. *Nutr. Rev.* 2004, 62, 177–203. [CrossRef] [PubMed]

15. Garrow, J.S.; Webber, J. Quetelet’s index (W/H2) as a measure of fatness. *Int. J. Obes.* 1985, 9, 147–153. [PubMed]

16. Peipert, J.D.; Hays, R.D. Using Patient-Reported Measures in Dialysis Clinics. *Clin. J. Am. Soc. Nephrol.* 2017, 12, 1889–1891. [CrossRef] [PubMed]

17. Hays, R.D.; Kallich, J.D.; Mapes, D.L.; Coons, S.J.; Carter, W.B. Development of the kidney disease quality of life (KDQOL) instrument. *Qual. Life Res.* 1994, 3, 329–338. [CrossRef]

18. Hays, R.D.; Kallich, J.D.; Mapes, D.L. Kidney Disease Quality of Life Short Form (KDQOL-SF™), Version 1.3: A Manual for Use and Scoring; RAND: Santa Monica, CA, USA, 1997.

19. Kidney Disease Quality of Life Instrument (KDQOL): Scoring the KDQOL-36. Available online: https://www.rand.org/health/surveys_tools/kdqol.html (accessed on 31 August 2019).

20. De Keyzer, W.; Huybrechts, I.; De Vriendt, V.; Vandevijvere, S.; Slimani, N.; Van Oyen, H.; De Henauw, S. Repeated 24-hour recalls versus dietary records for estimating nutrient intakes in a national food consumption survey. *Food Nutr. Res.* 2011, 55. [CrossRef]

21. Bailey, R.L.; Gutschall, M.D.; Mitchell, D.C.; Miller, C.K.; Lawrence, F.R.; Smiciklas-Wright, H. Comparative strategies for using cluster analysis to assess dietary patterns. *J. Am. Diet. Assoc.* 2006, 106, 1194–1200. [CrossRef]

22. Raper, N.; Perloff, B.; Ingwersen, L.; Steinfeldt, L.; Anand, J. An overview of USDA’s Dietary Intake Data System. *J. Food Compos. Anal.* 2004, 17, 545–555. [CrossRef]

23. Bailey, R.L.; Mitchell, D.C.; Miller, C.; Smiciklas-Wright, H. Assessing the effect of underreporting energy intake on dietary patterns and weight status. *J. Am. Diet. Assoc.* 2007, 107, 64–71. [CrossRef]

24. McCrory, M.A.; McCrory, M.A.; Hajduk, C.L.; Roberts, S.B. Procedures for screening out inaccurate reports of dietary energy intake. *Public Health Nutr.* 2002, 5, 873–882. [CrossRef] [PubMed]

25. Kopple, J.D. National kidney foundation K/DOQI clinical practice guidelines for nutrition in chronic renal failure. *Am. J. Kidney Dis.* 2001, 37, 566–570. [CrossRef] [PubMed]

26. Hearty, A.P.; Gibney, M.J. Comparison of cluster and principal component analysis techniques to derive dietary patterns in Irish adults. *Br. J. Nutr.* 2009, 101, 598–608. [CrossRef] [PubMed]
27. Anderson, A.L.; Harris, T.B.; Houston, D.K.; Tylavsky, F.A.; Lee, J.S.; Sellmeyer, D.E.; Sahyoun, N.R. Relationships of dietary patterns with body composition in older adults differ by gender and PPAR-gamma Pro12Ala genotype. Eur. J. Nutr. 2010, 49, 385–394. [CrossRef]

28. Foundation, N.K. KDOQI Clinical Practice Guidelines for Managing Dyslipidemias in CKD. Am. J. Kidney Dis. 2003, 41, S1–S91.

29. Choose My Plate. Available online: https://www.choosemyplate.gov/ (accessed on 11 October 2019).

30. Kopple, J.; Wolfson, M. KDOQI Nutrition in Chronic Renal Failure. AJKD 2000, 25, S1–S139. [CrossRef]

31. United States Department of Agriculture. 2015–2020 Dietary Guidelines; USDA: Washington, DC, USA, 2015.

32. Nutrient Recommendations: Dietary Reference Intakes (DRI). Available online: https://ods.od.nih.gov/Health_Information/Dietary_Reference_Intakes.aspx (accessed on 11 October 2019).

33. Duncan, M.J.; Kline, C.E.; Vandelanotte, C.; Sargent, C.; Rogers, N.L.; Di Milia, L. Cross-sectional associations between multiple lifestyle behaviors and health-related quality of life in the 10,000 Steps cohort. PLoS ONE 2014, 9, e94184. [CrossRef]

34. Godos, J.; Castellano, S.; Marranzano, M. Adherence to a Mediterranean Dietary Pattern Is Associated with Higher Quality of Life in a Cohort of Italian Adults. Nutrients 2019, 11, 981. [CrossRef]

35. Bonaccio, M.; Di Castelnuovo, A.; Bonanni, A.; Costanzo, S.; De Lucia, F.; Pounis, G.; Zito, F.; Donati, M.B.; de Gaetano, G.; Iacoviello, L.; et al. Adherence to a Mediterranean diet is associated with a better health-related quality of life: A possible role of high dietary antioxidant content. BMJ Open 2013, 3. [CrossRef]

36. Ribeiro, S.M.F.; Braga, C.B.M.; Peria, F.M.; Martinez, E.Z.; Rocha, J.; Cunha, S.F.C. Effects of zinc supplementation on fatigue and quality of life in patients with colorectal cancer. Einstein (Sao Paulo) 2017, 15, 24–28. [CrossRef]

37. Markiewicz-Zukowska, R.; Gutowska, A.; Borawska, M.H. Serum zinc concentrations correlate with mental and physical status of nursing home residents. PLoS ONE 2015, 10, e0117257. [CrossRef] [PubMed]

38. Manley, K.J. Saliva composition and upper gastrointestinal symptoms in chronic kidney disease. J. Ren. Care 2014, 40, 172–179. [CrossRef] [PubMed]

39. Liu, P.J.; Ma, F.; Wang, Q.Y.; He, S.L. The effects of oral nutritional supplements in patients with maintenance dialysis therapy: A systematic review and meta-analysis of randomized clinical trials. PLoS ONE 2018, 13, e0203706. [CrossRef] [PubMed]

40. Fitzgerald, C.; Wiese, G.; Moorthi, R.N.; Moe, S.M.; Hill Gallant, K.; Running, C.A. Characterizing Dysgeusia in Hemodialysis Patients. Chem. Senses 2019, 44, 165–171. [CrossRef] [PubMed]

41. González, S.; Huerta, J.M.; Fernández, S.; Patterson, A.M.; Lasheras, C. Life-quality indicators in elderly people are influenced by selenium status. Aging Clin. Exp. Res. 2007, 19, 10–15. [CrossRef]

42. Lobo, J.C.; Stockler-Pinto, M.B.; Farage, N.E.; Faulin Tdo, E.; Abdalla, D.S.; Torres, J.P.; Velarde, L.G.; Mafra, D. Reduced plasma zinc levels, lipid peroxidation, and inflammation biomarkers levels in hemodialysis patients: Implications to cardiovascular mortality. Ren. Fail. 2013, 35, 680–685. [CrossRef]

43. Hsu, C.W.; Weng, C.H.; Lee, C.C.; Yen, T.H.; Huang, W.H. Association of serum chromium levels with cardiovascular risk in type 2 diabetes mellitus. A case—Control study. PLoS ONE 2018, 13, e0197977. [CrossRef]

44. Ngala, R.A.; Awe, M.A.; Nsiah, P. The effects of plasma chromium on lipid profile, glucose metabolism and cardiovascular risk in type 2 diabetes mellitus. Health_Information 2017, 11, 10. [CrossRef]

45. Bai, J.; Xun, P.; Morris, S.; Jacobs, D.R., Jr.; Liu, K.; He, K. Chromium exposure and incidence of metabolic syndrome among American young adults over a 23-year follow-up: The CARDIA Trace Element Study. Sci. Rep. 2015, 5, 15606. [CrossRef]

46. Tonelli, M.; Wiebe, N.; Bello, A.; Field, C.J.; Gill, J.S.; Hemmelgarn, B.R.; Holmes, D.T.; Jindal, K.; Klarenbach, S.W.; Manns, B.J.; et al. Concentrations of Trace Elements and Clinical Outcomes in Hemodialysis Patients: A Prospective Cohort Study. Clin. J. Am. Soc. Nephrol. 2018, 13, 907–915. [CrossRef]

47. Maes, M.; Smith, R.; Christophe, A.; Vandoolaeghe, E.; Van Gastel, A.; Neels, H.; Demedts, P.; Wauters, A.; Meltzer, H.Y. Lower serum high-density lipoprotein cholesterol (HDL-C) in major depression and in depressed men with serious suicidal attempts: Relationship with immune-inflammatory markers. Acta Psychiatr. Scand. 1997, 95, 212–221. [CrossRef]

48. Parekh, A.; Smeeth, D.; Milner, Y.; Thure, S. The Role of Lipid Biomarkers in Major Depression. Healthcare (Basel) 2017, 5, 5. [CrossRef] [PubMed]
49. Brites, F.; Martin, M.; Guillas, I.; Kontush, A. Antioxidative activity of high-density lipoprotein (HDL): Mechanistic insights into potential clinical benefit. *BBA Clin.* 2017, 8, 66–77. [CrossRef] [PubMed]

50. Soran, H.; Schofield, J.D.; Durrington, P.N. Antioxidant properties of HDL. *Front. Pharmacol.* 2015, 6, 222. [CrossRef] [PubMed]

51. Santos-Gallego, C.G.; Badimon, J.J.; Rosenson, R.S. Beginning to understand high-density lipoproteins. *Endocrinol. Metab. Clin. N. Am.* 2014, 43, 913–947. [CrossRef]

52. Palmer, S.C.; Hanson, C.S.; Craig, J.C.; Strippoli, G.F.; Ruospo, M.; Campbell, K.; Johnson, D.W.; Tong, A. Dietary and fluid restrictions in CKD: A thematic synthesis of patient views from qualitative studies. *Am. J. Kidney Dis.* 2015, 65, 559–573. [CrossRef]

53. Stevenson, J.; Tong, A.; Gutman, T.; Campbell, K.L.; Craig, J.C.; Brown, M.A.; Lee, V.W. Experiences and Perspectives of Dietary Management Among Patients on Hemodialysis: An Interview Study. *J. Ren. Nutr.* 2018, 28, 411–421. [CrossRef]

54. Govindaraju, T.; Sahle, B.W.; McCaffrey, T.A.; McNeil, J.J.; Owen, A.J. Dietary Patterns and Quality of Life in Older Adults: A Systematic Review. *Nutrients* 2018, 10, 971. [CrossRef]

55. Kim, N.H.; Song, S.; Jung, S.Y.; Lee, E.; Kim, Z.; Moon, H.G.; Noh, D.Y.; Lee, J.E. Dietary pattern and health-related quality of life among breast cancer survivors. *BMJ Women’s Health* 2018, 18, 65. [CrossRef]

56. Porter, A.; Fischer, M.J.; Wang, X.; Brooks, D.; Bruce, M.; Charleston, J.; Cleveland, W.H.; Dowie, D.; Faulkner, M.; Gassman, J.; et al. Quality of life and outcomes in African Americans with CKD. *J. Am. Soc. Nephrol.* 2014, 25, 1849–1855. [CrossRef]

57. Fischer, M.J.; Kimmel, P.L.; Greene, T.; Gassman, J.J.; Wang, X.; Brooks, D.H.; Charleston, J.; Dowie, D.; Thornley-Brown, D.; Cooper, L.A.; et al. Elevated depressive affect is associated with adverse cardiovascular outcomes among African Americans with chronic kidney disease. *Kidney Int.* 2011, 80, 670–678. [CrossRef]

58. Kalantar-Zadeh, K.; Block, G.; McAllister, C. Appetite and inflammation, nutrition, anemia, and clinical outcome in hemodialysis patients. *Am. J. Clin. Nutr.* 2004, 80, 299–307. [CrossRef]

59. Feroze, U.; Noori, N.; Kovesdy, C.P.; Molnar, M.Z.; Martin, D.J.; Reina-Patton, A.; Benner, D.; Bross, R.; Norris, K.C.; Kopple, J.D.; et al. Quality-of-life and mortality in hemodialysis patients: Roles of race and nutritional status. *Clin. J. Am. Soc. Nephrol.* 2011, 6, 1100–1111. [CrossRef]

60. Gabbay, E.; Meyer, K.B.; Griffith, J.L.; Richardson, M.M.; Miskulin, D.C. Temporal trends in health-related quality of life among hemodialysis patients in the United States. *Clin. J. Am. Soc. Nephrol.* 2010, 5, 261–267. [CrossRef]

61. Nissenson, A.R. Improving outcomes for ESRD patients: Shifting the quality paradigm. *Clin. J. Am. Soc. Nephrol.* 2014, 9, 430–434. [CrossRef]

62. Sualeheen, A.; Khor, B.H.; Balasubramanian, G.V.; Sahathevan, S.; Ali, M.S.M.; Narayanan, S.S.; Chinna, K.; Daud, Z.A.M.; Khosla, P.; Gafar, A.H.A.; et al. Habitual Dietary Patterns of Patients on Hemodialysis Indicate Nutritional Risk. *J. Ren. Nutr.* 2019. [CrossRef]

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