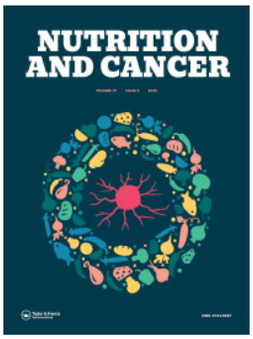

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Ketogenic Diets Are Associated with an Elevated Risk for All Cancers: Insights from a Cross-Sectional Analysis of the NHANES 2001–2018

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ABSTRACT

Ketogenic diet (KD) has increasingly been applied in anti-cancer therapy in recent years; however, its effect on cancer development risk remains controversial. We examined the association between dietary ketogenic ratio (DKR) and cancer incidence using cross-sectional data from the National Health and Nutrition Examination Survey (NHANES) conducted between 2001 and 2018. Dietary intake information was collected *via* a detailed 24-h dietary recall survey, and DKR values were calculated using a specialized formula. Multivariate logistic regression analysis was performed to evaluate the correlation between DKR and tumor occurrence, with restricted cubic splines (RCS) utilized to assess potential nonlinear relationships. Furthermore, a two-stage linear regression analysis was carried out to determine the inflection point. Furthermore, subgroup analyses were conducted stratified by demographic variables, including age, gender, race, body mass index (BMI), smoking status, and diabetes mellitus. A significant association was observed between DKR and cancer risk in multivariate logistic regression models fully adjusted for all potential confounding factors (OR, 1.58; 95%CI: 1.08, 1.54; $p = 0.049$). Moreover, individuals in the highest quartile of DKR exhibited a significantly increased risk for all cancers compared to those in the lowest quartile (Q4: OR, 1.29; 95%CI: 1.08, 1.34; $p = 0.005$). The RCS analysis revealed a non-linear relationship between DKR and cancer risk ($p < 0.001$, P for nonlinear trend = 0.003), with a turning point identified at 0.44 units on the scale used in this study. Piecewise regression analysis based on this threshold indicated that DKR values below 0.44 (DKR < 0.44) were significantly associated with an increased risk for all cancers within the context of this investigation (OR, 1.08; 95%CI: 1.04, 1.12; $p < 0.001$), while no significant correlation was observed for DKR values above this threshold (DKR ≥ 0.44) (OR, 1.01; 95%CI: 0.95, 1.07; $p = 0.77$). Furthermore, the findings from the subgroup analyses were consistent with the overall results. Therefore, we conclude that a KD might elevate the risk for all cancers, and further studies are warranted to validate this hypothesis.

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Introduction

The ketogenic diet (KD) was originally designed for weight management in obese individuals, but it has recently attracted significant attention due to its potential anti-cancer effects (1). The KD is characterized as a high-fat, low-carbohydrate dietary regimen that induces ketosis, thereby shifting the body's metabolic processes to rely on fats rather than carbohydrates as the primary energy source (2). Preclinical studies have shown that KDs possess anti-inflammatory and antioxidant properties (3), and they may also inhibit tumor growth (4). Nevertheless, the influence of the KD on cancer development risk within human populations remains inconclusive (5).

A recent comprehensive Mendelian randomization study on cancer revealed a significant association between elevated plasma levels of β-hydroxybutyrate (β-HB) and a decreased risk of diffuse large B-cell lymphoma, hepatocellular carcinoma, bile duct malignancy, as well as primary lymphoid and hematopoietic malignancies (6). Moreover, compared to a conventional diet, the ketogenic diet (KD) exhibited superior efficacy in reducing blood glucose, insulin, triglyceride levels, body weight, body mass index (BMI), and fat

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mass while improving emotional function in cancer patients (7). However, other studies have demonstrated that in a mouse model of cancer cachexia, although the KD slowed tumor growth, it led to cortisol deficiency and accelerated cachexia progression, thereby shortening survival time (8). Additionally, one study reported that β-HB generated by the KD enhanced the invasiveness and metastatic potential of pancreatic ductal adenocarcinoma (PDA) cells (9).

Given the growing interest in unraveling the potential benefits and risks of KDs for cancer prevention and treatment, there is an urgent need for further research to clarify the relationship between KDs and cancer risk in human populations. The present study aims to address this gap by examining the association between adherence to KDs and cancer incidence using data from the National Health and Nutrition Examination Survey (NHANES) collected between 2001 and 2018.

Materials and Methods

Data Source and Study Population

Based on a retrospective cohort study conducted from 2001 to 2018, we utilized data from nine cycles of the NHANES, each cycle incorporating at least one dietary assessment (https://www.cdc.gov/nchs/nhanes/). NHANES is a nationally representative cross-sectional survey initiated by the National Center for Health Statistics (NCHS) in 1999, designed to assess the health and nutritional status of the non-institutionalized U.S. population. Figure 1 illustrates the participant selection process. Initially, 91,351 participants were included in this study; however, individuals younger than 20 years old ($N = 41,150$), those with missing tumor-related data ($N = 55$), incomplete dietary information ($N = 5,705$), and insufficient covariate data ($N = 603$) were excluded from the analyses. Consequently, the final analytic sample consisted of 43,838 adults with complete data. All participants provided written informed consent, and the study protocol was approved by the ethics review board of the NCHS.

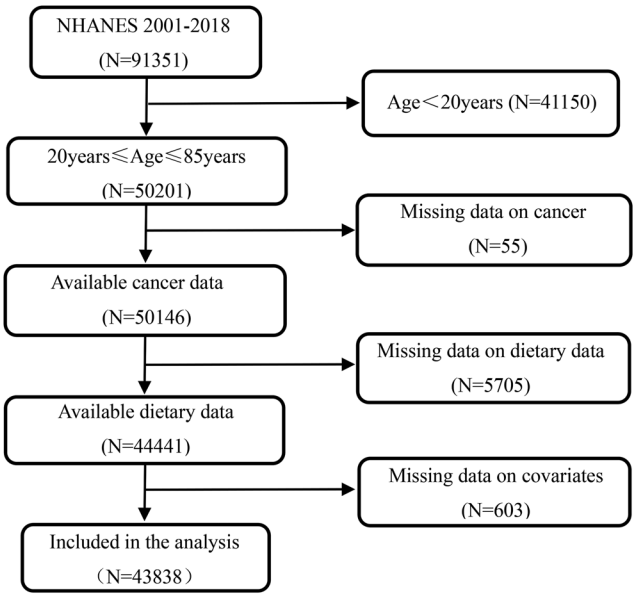

Figure 1. Flow diagram of the study participant selection. NHANES: National Health and Nutrition Examination Survey.

History of Cancer

The data on self-reported cancer history were obtained from the "Medical Conditions" section of the NHANES survey. This section involves comprehensive individual interviews with adults, during which participants self-report or provide proxy reports of their health status and disease history. Cancer survivors were identified based on their affirmative response to the following question: "Have you ever been told by a doctor or other health professional that you had cancer or a malignancy of any kind?"

Dietary Intake Data

The collection of dietary intake data was conducted through two separate 24-h NHANES surveys, administered by trained professionals specializing in dietetics. The process began with an in-person interview, followed by a telephone interview conducted 3 to 10 days later. During the follow-up interview, participants were asked to recall and report detailed information regarding the types and quantities of food items and beverages consumed on the previous day. Their overall dietary intake was then estimated based on the average of the two 24-h recalls (10). The energy content and nutritional composition of all food items were determined using a specialized Food and Nutrient Database developed from extensive studies on dietary patterns (11).

The Dietary Ketogenic Ratio(DKR)

In order to assess the predictive potential of dietary patterns in inducing nutritional ketosis, we calculated the DKR based on macronutrient ratios with ketogenic and anti-ketogenic properties. The DKR is determined using an equation formulated by Withrow (12). Specifically, this index is calculated by dividing ($0.9 \times$ grams of fat + $0.46 \times$ grams of protein) by ($0.1 \times$ grams of fat + $0.58 \times$ grams of protein + grams of net carbohydrates), yielding values that range from

zero to nine. A higher DKR value indicates a greater likelihood of achieving diet-induced nutritional ketosis.

Covariates

Obtain sociodemographic and lifestyle characteristics, as well as medical history including diseases, from demographic and questionnaire data. Specifically, categorize participants into age groups (≤40 years old; 41–60 years old; ≥61 years old), document gender and race/ethnicity (Mexican American, Non-Hispanic White, Non-Hispanic Black, Other Hispanic, Other Race), record educational attainment (Less than High School, High School/GED, More than High School), assess poverty-income ratio (PIR), and capture marital status (Married, Widowed, Divorced, Separated, Never Married, Living with Partner). Evaluate smoking status and BMI (<24 kg/m^2, 24–28 kg/m^2, ≥28 kg/m^2). Participants who affirmatively respond to the question "Have you smoked at least 100 cigarettes in your lifetime?" are classified as smokers; those who respond negatively are classified as nonsmokers. Height (cm) and weight (kg) measurements are obtained at mobile examination centers to calculate BMI using the formula: weight (kg) divided by height squared (m^2). Participants diagnosed with heart failure or a history of myocardial infarction, angina pectoris, or coronary heart disease (CHD) by healthcare professionals are considered to have a history of cardiovascular disease. Stroke and cancer conditions are determined through questionnaire surveys. Diabetes is identified based on the following criteria: HbA1c ≥6.5%, self-reported diagnosis of diabetes, or current use of antidiabetic medication.

Statistical Analysis

Continuous variables were typically summarized as mean (average) ± standard deviation (SD) or median with interquartile range (IQR), while categorical variables were presented as percentages. Weighted one-way analysis of variance (ANOVA) was utilized to analyze continuous variables, and weighted chi-square tests were applied for categorical variables. Multivariate logistic regression was performed to assess the association between DKR and cancer risk, with odds ratios (OR) and 95% confidence intervals (CI) estimated accordingly. To address potential confounding factors, three hierarchical models were constructed: Model A without covariate adjustment; Model B adjusted for gender and age; and Model C further adjusted for race, BMI, smoking status, diabetes history, dietary intake of vitamin A, vitamin C, magnesium, selenium, protein, carbohydrate, total fat, energy, and dietary fiber based on Model B. Additionally, restricted cubic spline (RCS) curve analysis was employed to investigate potential nonlinear relationships in detail. The highest inflection point of the inverted U-shaped curve identified *via* RCS served as the basis for piecewise regression analysis to explore more nuanced patterns within the data. Interaction effects among individual variables were comprehensively evaluated through subgroup analyses stratified by key dimensions such as gender, age, race, smoking status, BMI, and diabetes history.

To evaluate the associations between DKR quartiles and cancer risk at baseline, we employed logistic regression to estimate ORs, a metric appropriate for binary outcomes such as prevalent cancer diagnosis at enrollment. In contrast to hazard ratios (HR), which measure time-to-event risks in longitudinal survival analyses, OR provides insight into the likelihood of having cancer at a specific time point, aligning with our cross-sectional study design. Due to the absence of longitudinal follow-up data for incident cancer cases, HR was not applicable in this analysis.

The statistical significance was determined using a two-sided p value of less than 0.05. Statistical analysis was performed using SPSS 27 software, while data generation utilized GraphPad Prism 9.1 and R version 3.4.1.

After applying multiple imputation techniques to address missing values in covariates with a low missing rate of less than 10%, we determined the final set of variables based on the flowchart illustrated in Figure 1, where all variables exhibited missingness below 10%.

Results

Participants Characteristics

The demographic characteristics of the study participants are summarized in Table 1. Study participants were categorized into four groups according to DKR quartiles, and significant differences were observed across these groups in various baseline characteristics, including age, gender, race, education, marital status, PIR, BMI, and smoking status. Furthermore, substantial variations in co-morbidities such as hypertension, diabetes, and cancer were noted among the groups. However, no significant differences were detected in cardiovascular conditions, including coronary heart disease, angina pectoris, heart failure, myocardial infarction, or stroke. Additionally, a higher DKR value was associated with an increased likelihood of being male, older, belonging to the non-Hispanic white population, having a higher educational attainment but poorer marital status, possessing a higher PIR and

Table 1a. Patient demographics and baseline characteristics.

Characteristic	DKR Q1 (0.08,0.29), N = 10,960	Q2 [0.29,0.36), N = 10,959	Q3 [0.36,0.46), N = 10,959	Q4 [0.46,2.47], N = 10,960	p-value
Gender, n (%)					<0.001
Male	4,971 (45.4%)	5,214 (47.6%)	5,341 (48.7%)	5,646 (51.5%)	
Female	5,989 (54.6%)	5,745 (52.4%)	5,618 (51.3%)	5,314 (48.5%)	
Age, years, n (%)					<0.001
<40	4,114 (37.5%)	3,936 (35.9%)	3,712 (33.9%)	3,419 (31.2%)	
40 – 60	3,390 (30.9%)	3,345 (30.5%)	3,574 (32.6%)	3,784 (34.5%)	
≥60	3,456 (31.5%)	3,678 (33.6%)	3,673 (33.5%)	3,757 (34.3%)	
Ethnicity, n (%)					<0.001
Mexican American	2,016 (18.4%)	1,907 (17.4%)	1,781 (16.3%)	1,574 (14.4%)	
Other Hispanic	1,075 (9.8%)	1,008 (9.2%)	853 (7.8%)	689 (6.3%)	
Non-Hispanic White	4,424 (40.4%)	4,797 (43.8%)	5,088 (46.4%)	5,364 (48.9%)	
Non-Hispanic Black	2,281 (20.8%)	2,246 (20.5%)	2,319 (21.2%)	2,450 (22.4%)	
Other Race	1,164 (10.6%)	1,001 (9.1%)	918 (8.4%)	883 (8.1%)	
Education Level, n (%)					<0.001
Less than High school	3,403 (31.0%)	2,811 (25.7%)	2,586 (23.6%)	2,353 (21.5%)	
High school/GED	2,470 (22.5%)	2,564 (23.4%)	2,661 (24.3%)	2,523 (23.0%)	
More than high school	5,087 (46.4%)	5,584 (51.0%)	5,712 (52.1%)	6,084 (55.5%)	
Marital Status, n (%)					<0.001
Married	5,524 (50.4%)	5,763 (52.6%)	5,881 (53.7%)	5,861 (53.5%)	
Widowed	963 (8.8%)	931 (8.5%)	882 (8.0%)	834 (7.6%)	
Divorced	1,117 (10.2%)	1,090 (9.9%)	1,132 (10.3%)	1,231 (11.2%)	
Never married	409 (3.7%)	376 (3.4%)	330 (3.0%)	331 (3.0%)	
Separated	2,082 (19.0%)	1,918 (17.5%)	1,898 (17.3%)	1,856 (16.9%)	
Living with partner	865 (7.9%)	881 (8.0%)	836 (7.6%)	847 (7.7%)	
PIR, n (%)					<0.001
<1.5	4,360 (39.8%)	3,753 (34.2%)	3,466 (31.6%)	3,189 (29.1%)	
1.5 – 3	3,226 (29.4%)	3,214 (29.3%)	3,267 (29.8%)	3,029 (27.6%)	
≥3	3,374 (30.8%)	3,992 (36.4%)	4,226 (38.6%)	4,742 (43.3%)	
BMI, kg/cm2, n (%)					<0.001
<24	2,735 (25.0%)	2,568 (23.4%)	2,435 (22.2%)	2,111 (19.3%)	
24 – 28	3,185 (29.1%)	3,113 (28.4%)	2,947 (26.9%)	2,845 (26.0%)	
≥28	5,040 (46.0%)	5,278 (48.2%)	5,577 (50.9%)	6,004 (54.8%)	
Smoke, n (%)					<0.001
Yes	4,773 (43.5%)	4,765 (43.5%)	4,952 (45.2%)	5,464 (49.9%)	
No	6,187 (56.5%)	6,194 (56.5%)	6,007 (54.8%)	5,496 (50.1%)	

Abbreviations: DKR: dietary ketogenic ratio; PIR: Ratio of family income to poverty; BMI: body mass index.

Table 1b. Patient demographics and baseline characteristics.

Characteristic	DKR Q1 (0.08,0.29), N = 10,960	Q2 [0.29,0.36), N = 10,959	Q3 [0.36,0.46), N = 10,959	Q4 [0.46,2.47], N = 10,960	p-value
Hypertension, n (%)					<0.001
Yes	3,597 (32.8%)	3,728 (34.0%)	3,883 (35.4%)	4,115 (37.5%)	
No	7,363 (67.2%)	7,231 (66.0%)	7,076 (64.6%)	6,845 (62.5%)	
Diabetes, n (%)					<0.001
Yes	1,070 (9.8%)	1,185 (10.8%)	1,328 (12.1%)	1,734 (15.8%)	
No	9,890 (80.2%)	9,774 (89.2%)	9,631 (87.9%)	9,226 (84.2%)	
CHD, n (%)					0.478
Yes	434 (4.0%)	458 (4.2%)	431 (3.9%)	470 (4.3%)	
No	10,526 (96.0%)	10,501 (95.8%)	10,528 (96.1%)	10,490 (95.7%)	
Angina, n (%)					0.526
Yes	317 (2.9%)	300 (2.7%)	281 (2.6%)	301 (2.7%)	
No	10,643 (97.1%)	10,659 (97.3%)	10,678 (97.4%)	10,659 (97.3%)	
Heart attack, n (%)					0.098
Yes	452 (4.1%)	477 (4.4%)	414 (3.8%)	479 (4.4%)	
No	10,508 (95.9%)	10,482 (95.6%)	10,545 (96.2%)	10,481 (95.6%)	
Heart failure, n (%)					0.307
Yes	330 (3.0%)	349 (3.2%)	328 (3.0%)	371 (3.4%)	
No	10,630 (97.0%)	10,610 (96.8%)	10,631 (97.0%)	10,589 (96.6%)	
Stroke, n (%)					0.425
Yes	418 (3.8%)	386 (3.5%)	430 (3.9%)	402 (3.7%)	
No	10,542 (96.2%)	10,573 (96.5%)	10,529 (96.1%)	10,558 (96.3%)	
Cancer, n (%)					<0.001
Yes	921 (8.4%)	1,068 (9.7%)	1,040 (9.5%)	1,119 (10.2%)	
No	10,039 (91.6%)	9,891 (90.3%)	9,919 (90.5%)	9,841 (89.8%)	

Abbreviations: DKR: dietary ketogenic ratio; CHD: coronary heart disease.

Table 1c. Patient demographics and baseline characteristics.

Characteristic	DKR				*p*-value
	Q1 (0.08,0.29), *N* = 10,960	Q2 [0.29,0.36), *N* = 10,959	Q3 [0.36,0.46), *N* = 10,959	Q4 [0.46,2.47], *N* = 10,960	
Energy (kcal)					<0.001
Median (IQR)	1,750 (1,278, 2,376)	1,968 (1,460, 2,604)	2,021 (1,507, 2,695)	2,009 (1,482, 2,689)	
Protein (gm)					<0.001
Median (IQR)	56 (39, 77)	72 (52, 96)	80 (59, 107)	89 (64, 120)	
Carbohydrate (gm)					<0.001
Median (IQR)	271 (200, 364)	259 (192, 341)	233 (173, 308)	181 (129, 245)	
Total fat (gm)					<0.001
Median (IQR)	46 (30, 66)	68 (49, 93)	81 (58, 110)	96 (69, 131)	
Dietary fiber (gm)					<0.001
Median (IQR)	14 (9, 21)	15 (10, 22)	15 (10, 21)	13 (8, 19)	
Vitamin A (mcg)					<0.001
Median (IQR)	394 (187, 715)	465 (257, 775)	493 (280, 794)	489 (281, 785)	
Vitamin C (mcg)					<0.001
Median (IQR)	69 (24, 149)	61 (25, 128)	53 (22, 112)	42 (18, 91)	
Vitamin E (mg)					<0.001
Median (IQR)	4.8 (3.0, 7.5)	6.3 (4.1, 9.4)	7.0 (4.6, 10.3)	7.6 (4.9, 11.5)	
Magnesium (mg)					<0.001
Median (IQR)	241 (170, 337)	267 (196, 360)	273 (199, 368)	268 (194, 372)	
Zinc (mg)					<0.001
Median (IQR)	8 (5, 11)	10 (7, 14)	10 (7, 15)	11 (8, 16)	
Selenium (mcg)					<0.001
Median (IQR)	77 (52, 110)	97 (69, 134)	108 (77, 148)	115 (82, 159)	
Total Folate (mcg)					<0.001
Median (IQR)	344 (225, 508)	364 (247, 521)	353 (241, 503)	311 (213, 445)	

Abbreviations: DKR: dietary ketogenic ratio; IQR: interquartile range.

BMI, and engaging in more smoking habits. Notably, the prevalence of diabetes was relatively low, whereas that of stroke was relatively high among the participants. Most importantly, the incidence of tumors exhibited a gradual increase with rising DKR values.

The basic nutrient intake characteristics of the participants are summarized in Table 1. Significant differences were observed among the four groups regarding total energy, carbohydrate, fat, protein, dietary fiber, folate, vitamins, and micronutrient intake. Specifically, with the increase in DKR, there was a notable rise in energy, carbohydrate, and fat intake, whereas a decline was observed in protein, dietary fiber, and folate intake.

Table 2 presents the intake of three key nutrients—carbohydrates, proteins, and fats—detailing their mass, calorie contribution, and proportion in total caloric intake. The analysis revealed that as DKR increased, the mass proportions of proteins and fats progressively rose across the four sample groups, whereas the mass proportion of carbohydrates consistently decreased. Furthermore, the trends in caloric proportions for these three nutrients were consistent with their respective mass proportions.

The Correlation between DKR and Cancer

In this study, we developed three multivariate logistics regression models to investigate the association between DKR and cancer incidence (refer to Table 3). Model A, which was unadjusted for confounding factors, demonstrate a statistically significant association between DKR and cancer risk (OR 1.49, 95%CI 1.21,1.84, $p < 0.001$). However, it is noteworthy that both partially adjusted confounding-factor model B (OR 1.46, 95%CI 1.17,1.83, $p < 0.001$) and fully adjusted confounding-factor model C (OR 1.58, 95%CI 1.08,1.54, $p = 0.049$) confirmed the significant positive correlations between DKR and risk for all cancers. To be precise, each one standard deviation increase in DKR within model C was associated with a significant 12% reduction in mortality risk. Furthermore, multivariate analysis results from models B and C revealed that the hazard ratio for patients in the high quartile was consistently higher than those in the low quartile. Specifically, in model B, the ORs were 1.15 (95% CI: 1.04–1.27, $p = 0.005$) for Q2 and 1.22 (95% CI: 1.11–1.35, $p < 0.001$) for Q4. Similarly, in model C, the ORs were 1.16 (95% CI: 1.04–1.30, $p = 0.008$) for Q2 and 1.29 (95% CI: 1.08–1.34, $p = 0.005$) for Q4. These trends remained robust even after adjusting for all covariates in model C. Notably, higher levels of DKR were significantly associated with an increased cancer risk.

The analysis, utilizing RCS adjusted for model C, demonstrated a statistically significant non-linear relationship between DKR and cancer risk (P for non-linear trend = 0.003). This association followed an overall inverted U-shaped pattern (see Figure 2). An inflection point of 2.09 units was identified in

Table 2. Characteristics of carbohydrates, proteins, and fats as key dietary components.

Characteristic	DKR				*p*-value
	Q1, *N* = 10,960	Q2, *N* = 10,959	Q3, *N* = 10,959	Q4, *N* = 10,960	
Energy (kcal)	1,750 (1,278, 2,376)	1,968 (1,460, 2,604)	2,021 (1,507, 2,695)	2,009 (1,482, 2,689)	<0.001
Protein (gm)	56 (39, 77)	72 (52, 96)	80 (59, 107)	89 (64, 120)	<0.001
Protein-energy (kcal)	223 (154, 307)	286 (209, 384)	319 (235, 429)	354 (258, 479)	<0.001
Protein-energy (%)	13 (10, 15)	14 (12, 17)	16 (13, 19)	18 (15, 21)	<0.001
Carbohydrate (gm)	271 (200, 364)	259 (192, 341)	233 (173, 308)	181 (129, 245)	<0.001
Carbohydrate-energy (kcal)	1,085 (799, 1,456)	1,036 (770, 1,365)	932 (691, 1,234)	724 (515, 981)	<0.001
Carbohydrate-energy (%)	62 (59, 67)	53 (51, 55)	47 (44, 49)	37 (32, 41)	<0.001
Fat (gm)	46 (30, 66)	68 (49, 93)	81 (58, 110)	96 (69, 131)	<0.001
Fat-energy (kcal)	410 (269, 594)	610 (437, 837)	731 (526, 987)	867 (618, 1,180)	<0.001
Fat-energy (%)	24 (20, 28)	32 (29, 34)	37 (33, 39)	43 (39, 47)	<0.001

Table 3. OR and 95% CI for cancer according to according to quartiles of DKR.

Characteristic	Model A			Model B			Model C		
	OR	95% CI	*p*-value	OR	95% CI	*p*-value	OR	95% CI	*p*-value
Q1	Ref.		–	Ref.		–	Ref.		–
Q2	1.18	1.07,1.29	<0.001	1.15	1.04,1.27	0.005	1.16	1.04,1.30	0.008
Q3	1.14	1.04,1.25	0.005	1.11	1.00,1.22	0.042	1.13	0.99,1.28	0.080
Q4	1.24	1.13,1.36	<0.001	1.22	1.11,1.35	<0.001	1.29	1.08,1.34	0.005
DKR	1.49	1.21,1.84	<0.001	1.46	1.17,1.83	<0.001	1.58	1.08,1.54	0.049

Abbreviations: OR: odds ratio; CI: confidence interval; DKR: dietary ketogenic ratio.
Model A: unadjusted covariates.
Model B: adjusted by age and gender.
Model C: adjusted by age, gender, race, BMI, smoke, diabetes, energy, protein, carbohydrate, total fat, dietary fiber, vitamin A, vitamin C, magnesium, and selenium.

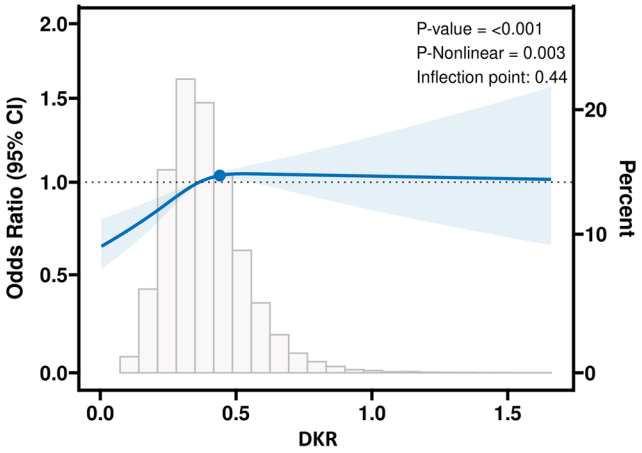

Figure 2. Association between DKR levels and the risk of all cancers was assessed using restricted cubic splines (RCS) and identifying the inflection point. DKR: dietary ketogenic ratio; CI: confidence interval; OR: odds ratio.

Table 4. Effect of DKR on cancer: Adjusted ORs from segmented logistic regression analysis.

Characteristic	OR	95% CI	*p*-value
KR (<0.44)	1.08	1.04, 1.12	<0.001
KR (≥0.44)	1.01	0.95, 1.07	0.77

Abbreviations: DKR: dietary ketogenic ratio; OR: odds ratio; CI: confidence Interval.
Model C: adjusted by age, gender, race, BMI, smoke, diabetes, energy, protein, carbohydrate, total fat, dietary fiber, vitamin A, vitamin C, magnesium, and selenium.

both Figure 2 and Table 4, which justified the application of a two-stage regression analysis to explore the impact of DKR on cancer risk. The piecewise regression analysis revealed that below the threshold value of 0.44 for DKR, there was a positive correlation with cancer risk (OR, 1.08; 95% CI: 1.04, 1.12; $p < 0.001$). In contrast, no significant correlation was observed when DKR ≥ 0.44 (OR, 1.01; 95% CI: 0.95, 1.07; $p = 0.77$). Notably, within the range of DKR < 0.44, the risk of all cancers progressively increased as DKR levels rose.

Subgroup Analysis

The subgroup analysis of the forest plot reveals several significant findings: Among female participants (OR 2.01, 95% CI 1.02–3.94, $p = 0.043$), individuals with BMI ≥ 28 (OR 2.06, 95% CI 1.14–3.72, $p = 0.016$), and non-coronary heart disease (CHD) individuals (OR 1.74, 95% CI 1.09–2.78, $p = 0.021$), a positive correlation was observed between DKR levels and cancer risk. In contrast, no such correlation was detected in male participants (OR 1.55, 95% CI 0.81–2.99, $p = 0.187$), individuals with BMI < 28 (OR 1.14, 95% CI 0.56–2.32, $p = 0.725$), or CHD patients (OR 0.47, 95% CI 0.08–2.77, $p = 0.408$). Similarly, this correlation was not observed across all racial groups, regardless of smoking status or diabetes status.

The fully adjusted Model C was utilized for subgroup analysis, accounting for potential confounding factors including age, gender, race, BMI, smoking status, diabetes, and CHD (Figure 3). The construction of the subgroup forest plot yielded several significant

findings: a positive correlation between DKR levels and cancer risk was observed in female participants (OR 2.01, 95%CI 1.02–3.94, $p = 0.043$), individuals with BMI ≥ 28 (OR 2.06, 95%CI 1.14–3.72, $p = 0.016$), and non-CHD individuals (OR 1.74, 95%CI 1.09–2.78, $p = 0.021$). In contrast, no such association was detected in male participants (OR 1.55, 95%CI 0.81–2.99, $p = 0.187$), individuals with BMI < 28 (OR 1.14, 95%CI 0.56–2.32, $p = 0.725$), or CHD patients (OR 0.47, 95%CI 0.08–2.77, $p = 0.408$). Additionally, significant interactions were exclusively identified between DKR and CHD variables (P for interaction = 0.009), whereas no significant interactions were observed between DKR and age (P for interaction = 0.184), gender (P for interaction = 0.097), race (P for interaction = 0.634), BMI (P for interaction = 0.734), smoking status (P for interaction = 0.327), or diabetes (P for interaction = 0.768). Collectively, this study provides robust evidence elucidating the relationship among DKR, diverse patient characteristics, and clinical outcomes through comprehensive subgroup analyses and interaction evaluations.

Discussion

In this study, we analyzed cross-sectional data from the NHANES 2001–2018 to examine the correlation between DKR and cancer incidence. After comprehensively adjusting for all potential confounding factors, a statistically significant association was observed between higher DKR levels and increased cancer risk. Specifically, as DKR increases, there is a gradual rise in the risk of all cancers; however, this association becomes non-significant when DKR exceeds 0.44. The results of the subgroup analysis were consistent with the overall findings. In subsequent research, we aim to further investigate the potential mechanisms by which KD may contribute to an elevated risk of all cancers.

The KD is a dietary pattern characterized by high fat, low carbohydrate, and moderate protein intake, aimed at inducing ketosis through carbohydrate restriction to utilize fat as the primary energy source. This metabolic state results in a significant increase in metabolites such as β-HB, acetoacetate, and

Subgroup	N	Adjusted OR (95% CI)	P value	P for interaction
Overall	43838	1.58 (1.08, 1.54)	0.049	
Gender				0.097
Male	21172	1.55 (0.81, 2.99)	0.187	
Female	22666	2.01 (1.02, 3.94)	0.043	
Age				0.184
< 60	29274	1.77 (0.84, 3.70)	0.132	
≥ 60	14564	1.25 (0.68, 2.27)	0.472	
Race				0.634
Mexican American	7278	3.74 (0.88, 15.95)	0.075	
Other Hispanic	3625	0.23 (0.02, 2.43)	0.224	
Non-Hispanic White	19673	1.58 (0.90, 2.77)	0.111	
Non-Hispanic Black	9296	1.55 (0.52, 4.59)	0.43	
Other Race	3966	3.92 (0.35, 43.87)	0.268	
BMI				0.734
< 28kg/cm²	21939	1.14 (0.56, 2.32)	0.725	
≥ 28kg/cm²	21899	2.06 (1.14, 3.72)	0.016	
Smoke				0.327
Yes	19954	1.34 (0.75, 2.40)	0.324	
No	23884	1.99 (0.95, 4.17)	0.067	
Diabetes				0.768
Yes	5317	1.49 (0.55, 4.05)	0.439	
No	38521	1.62 (0.97, 2.71)	0.065	
CHD				0.009
Yes	1793	0.47 (0.08, 2.77)	0.408	
No	42045	1.74 (1.09, 2.78)	0.021	

Figure 3. Subgroup analysis for the correlation between the DKR and the risk for all cancers in adults. DKR, Dietary ketogenic ratio; CI: confidence interval; OR: odds ratio; BMI: body mass index.

acetone within the body (13). While short-term adherence to the KD may facilitate weight management and improve certain metabolic parameters (7), long-term adherence to this eating pattern could potentially lead to adverse effects, particularly regarding cancer risk (9). Ketones, characteristic metabolic byproducts of the KD, have been implicated in augmenting oxidative stress, an imbalance between free radical production and clearance within the body. Chronic oxidative stress can cause cellular damage to DNA, proteins, and lipids, thereby increasing the risk of cancer development (14). Emerging evidence suggests that ketones may exacerbate oxidative stress either by stimulating free radical generation or inhibiting antioxidant enzyme activity (15). Consequently, it is plausible that elevated ketone levels associated with the KD indirectly contribute to carcinogenesis. Ketone bodies can serve as energy substrates for cancer cells. While the KD aims to reduce the glucose supply to cancer cells by restricting carbohydrate intake, the ketone bodies it induces may be exploited by certain tumor cells as alternative energy sources. Research has demonstrated that cancer cells can metabolize ketone bodies into acetyl-CoA and integrate them into the tricarboxylic acid cycle through the overexpression of ketone body transporter proteins (e.g., MCT1/2) and metabolic enzymes (e.g., OXCT1, ACAT1), thereby sustaining their energy metabolism and proliferation. For instance, pancreatic cancer cells depend on the ketone body-metabolizing enzyme HMGCL to facilitate invasion and metastasis, and knocking out this enzyme significantly suppresses tumor growth (9). Moreover, the KD may enhance the metabolic flexibility of cancer cells, thereby improving their survival. Specifically, the high-fat conditions induced by the KD can stimulate cancer cells to upregulate fatty acid oxidation pathways, enhancing their resistance to apoptosis. In breast cancer cells, for example, a ketone body-rich environment activates the AKT/mTOR signaling pathway, reduces programmed cell death, and promotes tumor progression. Animal experiments have demonstrated that the KD can increase the number of lung metastases in breast cancer mice, potentially due to ketone body-induced epithelial-mesenchymal transition (EMT) and extracellular matrix remodeling (16). Moreover, ketone bodies can enhance the invasive capacity of tumor cells by upregulating metastasis-associated genes (e.g., CXCL12) (17). Notably, the KD may elevate the risk of metabolic heterogeneity in tumor cells. While some tumors depend on glucose for energy, approximately 50% of cancer cells can meet their energy demands through ketone body metabolism, particularly in microenvironments where glycolysis is restricted. In such cases, the KD may serve as an alternative "fuel," thereby facilitating tumor progression and development (18).

The efficacy of a KD in reducing the production of inflammatory cells and inhibiting the release of inflammatory mediators has been demonstrated by recent studies, thereby mitigating both systemic and tissue-specific inflammation (19–20). Furthermore, the KD exhibits significant anti-inflammatory effects through its modulation of immune cell differentiation and function, suppression of pro-inflammatory factor expression, and promotion of anti-inflammatory factor synthesis (21–22). Recent studies have shown that β-HB at concentrations exceeding 1.2 mM triggers an inflammatory response by upregulating the activity of IKKβ, phosphorylating IκB-α, and increasing the level and expression of p65 protein. This subsequently leads to enhanced expression of NF-κB-regulated cytokines such as TNF-α, IL-6, and IL-1β (23). Similarly, KDs have been associated with prolonged consumption of a high-fat diet, which may induce heightened inflammation, neuropathy, and increased permeability of the blood-brain barrier. These physiological alterations can lead to changes in brain structure and ultimately contribute to cognitive decline (24). The potential influence of ketone bodies on inflammatory response pathways suggests a possible link to oncogenic processes, indicating an indirect association between ketone bodies and cancer development.

The study by Churuangsuk et al. demonstrated a substantial decrease in blood thiamin levels among individuals following a low-carbohydrate diet compared to those consuming a high-carbohydrate diet. Furthermore, the prevalence of hypomagnesemia was markedly higher in the low-carbohydrate group, affecting approximately 20% of participants, compared to 12% in the normal-diet group (25). Similarly, El-Rashidy et al. reported that the implementation of a KD significantly enhanced the overall antioxidant capacity in patients with epilepsy; however, it is important to note that this dietary approach may fail to meet the recommended daily intake of selenium (26). After conducting a comprehensive analysis of baseline data, we have drawn a consistent and robust conclusion: as the degree of ketosis increases, there is a notable decline in the antioxidant properties of vitamins A, C, and E, as well as trace elements such as manganese, zinc, and selenium. Notably, numerous studies have consistently shown that deficiencies in antioxidant vitamins and trace elements can substantially increase the risk of all cancers (27–31).

In conclusion, the KD may potentially elevate the risk of all cancers through multiple mechanisms, such as alterations in metabolic pathways leading to nutrient imbalances and modulating inflammatory states. Nevertheless, it is crucial to emphasize that our observational study is not designed to determine a definitive causal relationship between the KD and cancer development. Consequently, additional high-quality randomized clinical trials and in-depth mechanistic investigations are warranted to confirm the impact of the KD on cancer risk and to clarify the underlying biological processes.

Moreover, compelling subgroup analyses have uncovered potential interactions among smoking, diabetes, stroke history, and the influence of a KD on cancer risk. These findings pave the way for future research to investigate interpopulation variations in response to KDs and elucidate their underlying mechanisms.

Limitations

In order to ensure a comprehensive understanding of the research findings, it is crucial to recognize the limitations inherent in this study. First, although rigorous data processing and statistical analysis indicate that the KD may increase the risk of all cancers, the lack of direct measurement of ketosis levels somewhat constrains our ability to investigate the relationship between ketosis and cancer risk in greater depth. Therefore, future studies should incorporate more precise technical methods for directly measuring ketosis levels, thereby enabling a more thorough assessment of its potential association with all-cause mortality. Additionally, the dietary data obtained from the NHANES database are based on cross-sectional sampling surveys. While this design provides descriptions of population characteristics at different time points through multi-period cross-sectional surveys, each sample only reflects dietary intake and disease status at a single time point. As such, it is not possible to directly evaluate the temporal sequence of "exposure-outcome" (e.g., whether long-term exposure to the KD precedes cancer onset) or to easily account for confounding factors or cumulative effects that vary over time. This limitation is intrinsic to the cross-sectional study design. Although baseline confounding factors were adjusted for in the multivariate model analysis, the issue of time dependence remains unresolved. To address this limitation, we plan to further validate the time-dependent association between the KD and cancer risk in subsequent studies using prospective cohort studies or randomized controlled trials.

Conclusion

In conclusion, the KD may potentially increase susceptibility to cancer, with a proportional rise in risk associated with an elevated DKR. However, further rigorous clinical trials and mechanistic studies are essential to confirm the impact of the KD on cancer risk and clarify its underlying mechanisms. Additionally, it is critical to highlight that individual suitability for the KD varies considerably. When designing dietary plans, factors such as an individual's health status, nutritional needs, and dietary preferences must be carefully evaluated to ensure safety and efficacy.

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Authors' Contributions

All authors contributed to the conception and design of the study. Yuping Liu was responsible for participant recruitment, data collection, data analysis, interpretation of findings, and manuscript writing. Xiaolong Qu participated in data analysis and manuscript writing. Xiaolong Qu and Dongming Yang conducted data analysis and interpretation while also contributing to the writing process. Dongming Yang also played a role in participant recruitment, data collection, and the writing process. Dingsheng Liu played a significant role in analyzing the data and revising the article, ensuring the accuracy and coherence of the final manuscript. Both Yuping Liu and Xiaolong Qu have made equal contributions to this paper. Finally, all authors reviewed and approved the final manuscript submitted for publication.

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Data availability statement

The data can be publicly accessed through the website of the National Center for Health Statistics at https://www.cdc.gov/nchs/nhanes.

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