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Exploring the Relationship Between Dietary Intake and Clinical Outcomes in Peritoneal Dialysis Patients

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Exploring the Relationship Between Dietary Intake and Clinical Outcomes in Peritoneal Dialysis Patients:
A 12-Year Follow-Up Using Fine-Grained Electronic Medical Records Data

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Abstract—This study investigates the relationship between dietary intake and mortality risk among patients with End-Stage Renal Disease (ESRD), a population for whom nutritional management is crucial yet challenging due to the disease's complexity and the limitations of existing dietary guidelines. Recognizing the gaps in current research, particularly the lack of detailed evidence-based recommendations for micronutrient intake and a focus predominantly on the early stages of chronic kidney disease, our work seeks to provide evidence-based dietary guidance tailored to the needs of ESRD patients. Through the collection of long-term, longitudinal, and fine-grained dietary records linked with extensive electronic health records (EHR) data over a 12-year period, we have constructed a dataset that offers a detailed view of dietary nutrient intake across 26 types of nutrients. Utilizing a two-stage model, we have analyzed the association between dietary intake and mortality risk. Our model provides comprehensive dietary recommendations for 26 nutrients, with 13 out of these nutrients showing a significant correlation with mortality risk while currently lacking specific guidelines for ESRD patients. This research not only challenges and refines the current dietary recommendations but also highlights the importance of considering non-linear relationships between nutrient intake and health outcomes. By offering a comprehensive perspective on dietary management for ESRD patients, this study contributes to the optimization of nutritional care strategies, aiming to improve patient prognosis and quality of life.

Index Terms—end-stage renal disease; peritoneal dialysis; nutrient; two-stage model

1 Introduction

End-Stage Renal Disease (ESRD) represents a significant global health challenge, profoundly impacting patients' lives through both the disease's direct effects and the substantial socioeconomic costs associated with management and treatment [9]. Patients with ESRD experience a diminished quality of life, grappling with the burdens of chronic illness and the rigors of treatment regimens, including Peritoneal Dialysis (PD) [10]. This backdrop sets the stage for our exploration of dietary and nutritional management as a pivotal aspect of ESRD care. Given the chronic nature of ESRD and the reliance on dialysis, dietary interventions emerge as crucial for slowing disease progression and preventing adverse outcomes, underscoring the importance of tailored nutritional guidance in this context [11], [12].

However, current dietary and nutritional management guidelines for ESRD are coarse and lack real-world clinical evidence. They are primarily based on a foundation that, while comprehensive in areas such as protein, energy, potassium, and phosphorus intake, often lacks in-depth recommendations for micronutrients. Besides, most evidence grades of dietary recommendations are low (level C or opinion-level) [13]. Even broader research rarely explores the relationship between diet and prognosis in ESRD patients. Most dietary research focuses on the early stages (Stages 1-3) of chronic kidney disease (CKD) [1], [2], [3], [4], [5] and related comorbidities [7], thus limiting the scope and applicability of findings to the later stages of the disease. As a result, the evidence level regarding prognosis and dietary recommendations remains low, with conclusions that are provisional and lack a unified model for cohesive guidance. This gap underscores the need for research based on high-quality, large-scale data that can provide robust, survival-outcome-oriented nutritional recommendations. However, collecting such high-quality dietary data for ESRD patients presents significant challenges:

- **Data collection method**: Data collection based on patient recall through questionnaires often introduces significant inaccuracies because it relies on the patient's own recall and understanding of the questionnaire, which may lead to memory errors or misinterpretation of terminology and definitions in the questionnaire. In contrast, dietary data collection supervised by experts, using methods such as the three-day or seven-day food record, can substantially enhance data accuracy [14]. Nonetheless, the majority of studies rely on questionnaires for nutritional information collection because it is much easier and requires fewer resources [2], [3], [4], with only a minority utilizing expert-monitored methods like three-day or seven-day dietary records [6], [8]. Therefore, collecting long-term high-quality dietary

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TABLE 1: Comparison of existing dietary and nutritional research for ESRD patients

		Research Question	Data Quality				Method Capability	Result Precision
Author/Year	Patient Cohort	Ending-Dependent	Long-term Follow-up Dietary Data Records Supervised by Professional Experts	Dietary Record Follow-up Duration	Dietary Record Follow-up Frequency	Nutrition Granularity	Non-linear Relationship	Precise Threshold
Marie 2018 [1]	CKD	✗	✗	Once	Once	Fine-grained (Single Nutrition)	✓	✗
Tanushree 2019 [2]	CKD	✗	✗	Once	Once	Coarse-grained	✗	✗
Orlando 2014 [3]	CKD	✗	✗	Once	Once	Coarse-grained	✗	✗
Banerjee 2015 [4]	CKD	✗	✗	Once	Once	Coarse-grained	✗	✗
Wagner 2022 [5]	CKD	✗	✗	Once	Once	Fine-grained (Single Nutrition)	✗	✗
Virojanawat 2021 [6]	PD	✗	✗	Once	Once	Fine-grained (Single Nutrition)	✗	✗
Angela 2019 [7]	PD	✗	✗	Once	Once	Fine-grained (Single Nutrition)	✗	✗
Xu 2019 [8]	PD	✓	✓	3-year	Every 3 months	Fine-grained (Single Nutrition)	✓	✗
Ours	PD	✓	✓	12-year	Every 3 months	Fine-grained (Multiple Nutrients)	✓	✓

Abbreviations: CKD: Chronic Kidney Disease;PD: Peritoneal Dialysis

records is a pivotal step toward a precision dietary recommendation.

- **Data longitudinality and granularity**: The long-term impact of dietary nutrition on mortality risk is a critical area of interest. However, the importance of longitudinal data is frequently overlooked, with many studies focusing only on baseline dietary information without considering the long-term dietary effects [1], [2], [7]. Furthermore, existing research often lacks in-depth analysis of multiple nutrients simultaneously. While some studies focus on specific dietary patterns, such as the DASH diet [2], these approaches tend to be coarse-grained and may not provide detailed conclusions about individual nutrients, thereby limiting their utility for clinical dietary guidance. Conversely, studies that quantify individual nutrients, like protein and sodium, offer fine-grained analyses but often neglect the complex interrelationships between various nutrients. A comprehensive study that quantifies multiple nutritional elements simultaneously not only provides a more holistic view but also enhances interpretability and can offer more precise guidance for clinical practice. Achieving this requires extensive long-term tracking of patients' dietary intake and health status, a task that is often unfeasible for short-term dietary studies.
- **Data linkage**: Merely having long-term dietary records is insufficient to formulate evidence-driven dietary recommendations for ESRD patients. It's essential to integrate these dietary records with comprehensive health records and outcome data. This integration facilitates a more accurate understanding of how diet influences ESRD progression and patient outcomes. Effective data linkage involves meticulous matching of dietary intake data with patient health records over extended periods, requiring sophisticated data management and analysis techniques. This process is vital for identifying correlations between dietary patterns and health outcomes, enabling the development of targeted nutritional guidelines that can significantly improve the quality of life and prognosis for ESRD patients.

To address these challenges and provide comprehensive dietary recommendations for patients with ESRD, we have collected long-term, longitudinal, and fine-grained dietary records of patients and linked them with extensive electronic health record (EHR) data. Our study's dataset encompasses both dietary and EHR data over a span of 12 years, with regular follow-up intervals averaging every three months per patient. This temporal depth and frequency afford a richer, more dynamic view of the patients' conditions compared to many existing datasets. The linked dataset comprises comprehensive clinical features, including dietary nutrition intake, laboratory tests, comorbidities, baseline information, and clinical outcomes. Furthermore, our dataset includes detailed records of dietary nutrient intake across 26 types of nutrients, providing a level of granularity that significantly surpasses the limited scope of previous research. The "3-day dietary record method" is utilized to collect the dietary data, ensuring high data quality. The data collection process is meticulously conducted through detailed interviews by experienced physicians, rather than being self-reported by patients.

Our dietary recommendation analysis is centered on elucidating the relationship between dietary intake and the likelihood of clinical outcomes of peritoneal dialysis patients. This study introduces a two-stage model that incorporates a Cox proportional hazards model to predict mortality risk based on clinical features and a cubic spline regression method to model relationships between dietary intake and risk. Leveraging our comprehensive dataset, the proposed model is capable of identifying shifts in health risks associated with varying nutrient intakes. Our findings refine existing medical knowledge and dietary guidelines, providing a more nuanced perspective that enhances the precision of dietary management for PD patients.

In our thorough examination of 26 distinct nutritional elements, we discovered that 14 of these elements (54%), including Vitamin A, Retinol, and Nicotinic, lack corresponding guidelines for patients diagnosed with ESRD and PD. Additionally, our research indicates that 13 out of these 14 elements show a significant association with mortality risk. For nutrients that are emphasized in guidelines and are a focal point of significant research, our findings for the entire patient cohort closely align with these recommendations. For instance, while guidelines suggest a Dietary Protein Intake (DPI) of 1.0-1.2g/kg/day [13], our research identifies an intake range of 0.88-1.13g/kg/day as being associated with a lower risk of mortality. Similarly, Dietary Energy

Intake (DEI) guidelines recommend 25-35 kcal/kg/day [13], and our study finds that a range of 26-42 kcal/kg/day is correlated with a reduced risk of death. We further stratified patients into different subgroups for a more detailed analysis, which both validates and complements existing research. We observed that the non-linear relationship is more pronounced in patient cohorts with poor health status, with 69% of the relationships between dietary intake and outcome being non-linear. This underscores the importance of the proposed non-linear modeling approach in improving health outcomes and reducing losses. Table 1 highlights the advantages of our research over existing studies.

2 Dataset and Method

We designed a two-stage model to evaluate the mortality risk for patients and to obtain precise dietary nutritional ranges associated with low mortality risk for each dietary nutrient. Figure 1 shows the pipeline of our method. In this section, we will elaborate on the problem formulation, data collection, and methodological details.

2.1 Problem formulation

Let P denote the population of patients with Peritoneal Dialysis (PD). Our objective is to identify a daily dietary arrangement that optimizes health benefits for this group.

A *beneficial dietary arrangement* is characterized as one that extends the *survival time* T for patients with analogous baseline attributes. The survival time T is the period from the *initiation of the follow-up* t_0 to its *conclusion* t_{end}, which may be due to death, loss to follow-up, or the termination of the study period.

To ensure precision and practicality in a clinical setting, the diet is quantified in terms of nutrient granularity. Let $D = \{d_1, d_2, \ldots, d_n\}$ denote the collection of *daily nutrient intakes* for all patients, with each d_i representing the mean dietary intake for an individual during the follow-up, inclusive of N distinct nutrients $\{t_1, t_2, \ldots, t_N\}$.

The initial step involves pinpointing a dietary intake $d^* \in D$ that enhances the follow-up duration t to the greatest extent, denoted by the *dietary evaluation score* s.

The dietary benefit for a patient is assessed with the function f:

$$f : b \times d \times t \times \omega \to s$$

where:

- b represents the *baseline characteristics* of the patient, encompassing demographic markers such as gender and age, as well as clinical metrics like creatinine, albumin, blood pressure, etc.
- d is the average *dietary intake* throughout the follow-up period.
- t signifies the *follow-up duration*, calculated as $t = t_{end} - t_0$.
- ω denotes the *outcome* (e.g., alive, lost to follow-up, or deceased).
- $f(b, d, t, \omega)$ yields the *dietary evaluation score* for a patient, defined as s, where $s \in S$.

This produces the set of diet and *dietary evaluation score* pairs for all patients $P = \{(d, s) \mid d \in D, s \in S\}$. By disaggregating d into its components, we obtain the collection of all patients' records for the i-th nutrient and its corresponding evaluation $P_i = \{(t_i, s)\}$.

Proceeding to the second step, to extrapolate from individual findings to the broader population, we introduce a function g that processes the set of all patient diet-evaluation pairs and identifies the *range of nutrient intakes* linked with prolonged survival times:

$$g : t \times s \to r$$

where:

- t is the intake of a specific dietary nutrient.
- s represents the *dietary evaluation score* corresponding to that nutrient intake.
- r denotes the intake range of the dietary nutrient associated with low mortality risk which can extend survival times as much as possible, given by $r = (r_{min}, r_{max})$, where r_{min} denotes the minimum intake that meets the condition, r_{max} denotes the maximum one.

Hence, we determine the specific intake range for each dietary nutrient t_1 to t_N connected with a lower mortality risk:

$$R = \{r^i = \{r^i_{min}, r^i_{max}\} \mid 1 \leq i \leq N\}$$

2.2 Data source and collection

2.2.1 Data source

A retrospective analysis of real-world data is conducted, involving 656 patients aged between 16 and 97 years, all diagnosed with Stage 5 Chronic Kidney Disease (CKD). These patients are tracked at a Peritoneal Dialysis Clinic from January 2006 to January 2018. All patients underwent long-term regular follow-ups until they are lost to follow-up, deceased, or until data collection ended on October 31, 2018. The duration of treatments varied from 2 months to 10 years. The specific distribution of follow-up times is illustrated in Figure 6, with a total of 656 individuals included.

All data are obtained from routine clinical records at the same Peritoneal Dialysis Clinic, encompassing baseline data at the onset of dialysis, follow-up data, and outcome data. During the data collection and processing phase, patient information is de-identified, and individual identities are linked exclusively to patient IDs.

Demographic and clinical lab tests, such as age, gender, diabetes status, blood pressure, blood glucose, blood phosphorus, albumin, and more, are collected during each patient visit. The results from the initial visit served as baseline values, with their time-averaged values calculated subsequently. The specific methodology is detailed in Section 2.2.2.

During the follow-up, all patients are required to visit the peritoneal dialysis (PD) clinic regularly with their three-day dietary records. These records are initially collected at the sixth month of dialysis to establish baseline data and subsequently at least every three months. After verification using food models by the dietitian, the dietary information from the diaries is entered into a computer software program (PD Information Management System, Peking University Third Hospital), and dietary intake is calculated accordingly [15]. DPI and daily energy intake (DEI) are normalized by ideal body weight (IBW), which is defined as body height (cm) minus 105 (modified Broca method) [16].

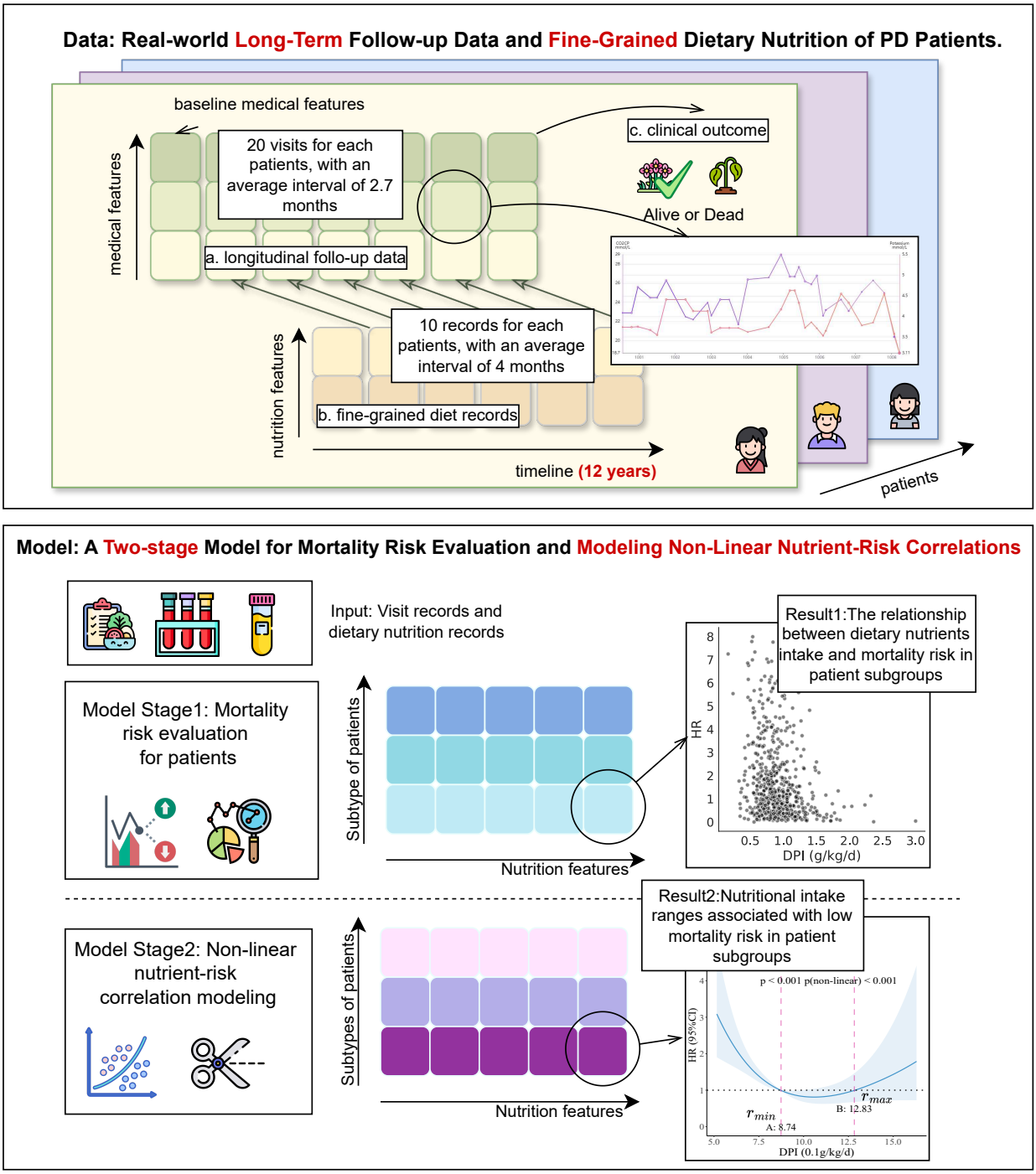


Fig. 1: The pipeline of our method.We collect an over 12-year, long-term, and real-world clinical EHR dataset of PD patients, consisting of static baseline information, longitudinal multivariable records and clinical outcomes. We develop a two-stage to evaluate the mortality risk for patients and to model the non-linear relationship: i) Prior to stage one, we categorize patients into distinct subtypes. This stratification aids in identifying more granular patterns. Consequently, the results generated in stages one and two are grounded within specific subtypes rather than the entire patient population, allowing for more precise analysis. ii) In the first stage, we use the Cox model to predict patients' mortality risk with patients' dietary nutrition and medical examination records. The Cox model is used to assess the dietary evaluation score for each nutritional element. iii) In the second stage, we design a non-linear Restricted Cubic Spline (RCS) model to process the results from the first stage. The RCS model calculates the beneficial intake range for each dietary nutrient associated with low mortality risk. The pink dashed lines in the result 2 figure for the second stage indicate the ranges $r^i = (r^i_{min}, r^i_{max})$.

2.2.2 Data preprocess

The time-averaged calculations of biochemistry and nutrients in the models are conducted based on the measurements taken over a 6-month period [8].

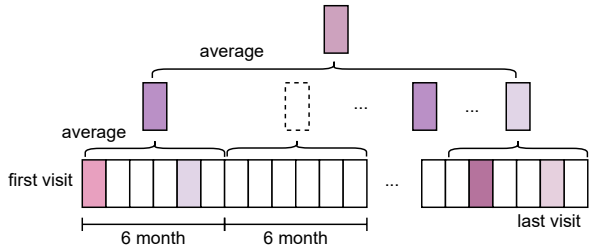


Fig. 2: Time-averaged data processing method. The white rectangles represent months for which there are no records, while the purple rectangles indicate that there are records for the corresponding months, different colored rectangles represent different records. The dashed boxes denote event windows where no records exist, and to ensure data accuracy, we discard such time windows. The final 'time-average' data represents the time-averaged result of the original records.

The detailed methodology can be described as follows, with reference to Figure 2: Patient follow-up records are segmented into intervals of 6 months. Within each 6-month interval, the data for all patient visits are averaged to create a single average record for that 6-month period. Depending on the varying follow-up times, there might be multiple "6-month average" follow-up records available, represented as 'n' records. To obtain the overall average follow-up record, the data from these 'n' records are averaged together. In cases of missing follow-up data, no imputation is performed to ensure data diversity and accuracy. When calculating the average, any corresponding records with missing data are excluded from the calculation. Please note that Figure 2 likely contains a visual representation of this process for a clearer understanding. All records of patient follow-up are included to calculate the time-averaged data.

2.3 A two-stage model for evaluating mortality risk and modeling non-linear nutrient-risk correlations

2.3.1 Patient subtyping

To facilitate a more fine-grained observation and enhance its applicability in clinical practice, we have chosen serum albumin as the key biomarker for patient subtyping. This decision is underpinned by several clinical findings:

- **Serum Albumin for Prognosis Prediction:** Extensive research has identified serum albumin as the most significant biomarker for predicting patient outcomes. For example, hypoalbuminemia has been recognized as a potent predictor for PD-related peritonitis, which is a leading cause of infection-related mortality and peritonitis in patients [17]. Furthermore, some models have assigned high attention weights to albumin levels in patients who have survived, indicating that low-risk scores are correlated with higher albumin levels [18], [19], [20].
- **Serum Albumin for Nutritional Intake Evaluation:** Current studies have revealed a strong correlation between serum albumin levels and the nutritional status of patients with ESRD. Malnutrition in ESRD patients is typically associated with lower serum albumin levels, whereas higher levels usually suggest better nutritional status [12], [21].
- **Availability of Serum Albumin in Clinical Practice:** Serum albumin is a routinely measured biomarker in the regular lab tests conducted during the clinical follow-up of most ESRD patients. This widespread availability ensures that our findings can be easily implemented in clinical settings, including in regions with underdeveloped healthcare infrastructure. By utilizing serum albumin as the biomarker for patient subtyping, we aim to make our research outcomes more accessible and practical.

This study serves as an initial investigative research project to confirm the relationship between dietary intake and clinical outcomes. In our future work, we plan to incorporate more comprehensive methods for patient subtyping, such as unsupervised patient clustering techniques.

2.3.2 Model stage-1: mortality risk evaluation

To explore the range of dietary nutrient intake in different types of patients, we first assess the associations between various dietary nutrients and patients' mortality risk across different patient subtype groups. For the mortality risk assessment model, we choose either statistic-based models, such as the Cox proportional hazards model [22] and the logistic regression model [23]; machine learning-based models, such as the random forest model [24]; or deep learning-based models, such as neural network survival models or neural network prediction models [18], [19], [25], [26]. In this study, we opted for the multivariate Cox proportional hazards regression model to predict and assess mortality risk, which, compared to other models, does not require model training and is widely used in the medical field due to its better stability and credibility.

The Cox proportional hazards regression model is employed to adjust for well-recognized confounding factors and determine the corresponding mortality risk associated with time-averaged dietary nutrient intake. We presented the multivariate-adjusted hazard ratios (HRs) along with their 95% confidence intervals. The full names of these covariates, along with additional information, can be found in Table 5.

2.3.3 Model stage-2: non-linear nutrient-risk correlation modeling

As discussed in the introduction, extreme intake levels of certain dietary nutrients, either excessively high or low, may pose health risks to patients. This understanding has led us to investigate the nonlinear relationship between dietary nutrient intake and mortality risk.

In the initial stage, delineated in Section 2.3.2, we established the correlation between patients' dietary nutrient intake and mortality risk. For any specific dietary nutrient intake, we estimate the patients' mortality risk utilizing the model. By sampling in accordance with the patients' actual

conditions, we generate several "x-y" pairs representing the relationship between dietary nutrient intake and mortality risk, as illustrated in Figure 3.

To more accurately establish the beneficial dietary nutrient intake threshold, we recognize that a nonlinear correlation may exist between dietary nutrients and mortality risk. Consequently, we further utilize the Restricted Cubic Spline (RCS) method [27], [28] to fit the results previously mentioned. In the fitted results, we identify the nutrient range (r_{min}, r_{max}) that corresponds to an HR value less than 1 (i.e., the corresponding intake range is a protective factor) and designate this as the suitable nutrient intake range. Subsequently, we utilize the ANOVA method to test for non-linearity. If the p-value indicates a significant non-linear relationship, it implies that the intake of the dietary nutrient in question has a non-linear association with mortality risk. This suggests that the identified range is meaningful. Conversely, if the non-linearity is not significant, it may indicate that there is no apparent non-linear relationship, and the range may only delineate an upper or lower limit. In such cases, the results of the linear test from stage one can be considered as a reference.

3 RESULTS

In the results, we design experiments to answer the following research questions.

- RQ1. How do clinical characteristics vary in patients with different baseline albumin levels?
- RQ2. How does dietary nutrition affect the mortality rate in patients?
- RQ3. What are the beneficial dietary intake ranges for each nutrition?

Continuous data are represented by mean ± standard deviation (SD) or interquartile range (IQR), depending on the data distribution. Discrete data (categorical variables) are presented as frequencies or percentages. Group comparisons are conducted using one-way analysis of variance (ANOVA) [29], [30], Tukey's Honestly Significant Difference (HSD) test [31], [32], and the chi-square (χ^2) test [33], [34] for discrete data to compare differences in variables among groups. The log-rank test [35], [36] is employed to evaluate differences in survival curves among the three groups.

The computations are performed using Python 3.7.13 and Lifelines 0.27.2, as well as R-base 4.2.2 software. All statistical tests are two-tailed, and the threshold for statistical significance is established at $p < 0.05$. In instances where $p < 0.01$ or $p < 0.001$, special symbols denote these heightened levels of significance.

3.1 How do clinical characteristics vary in patients with different baseline albumin levels

The distribution of baseline clinical indicators and dietary nutrient intake of the patients is evaluated, as shown in Table 2 and Table 3, respectively. To discern differences among patients with varying albumin levels, we classify them into three categories by using tertiles [7]: low, medium, and high, using their baseline albumin levels. The ranges for these categories are below 35.2g/L, 35.2-39.5g/L, and above 39.5g/L, respectively, with the groups consisting of 217, 222, and 217 individuals. The distribution of baseline clinical indicators and dietary nutrient intake for these groups are also presented in Table 2 and Table 3.

The average age of the entire sample is 59.1 years (SD=15.8), and the average ages for the high, medium, and low albumin groups are 53.6 years (SD=15.8), 60.27 years (SD=15.9), and 63.39 years (SD=14.0), respectively. Among the total sample, 50% are female, and 37% had been diagnosed with diabetes.

Significant disparities are observed in most baseline clinical indicators and demographic characteristics across the different albumin groups, except for blood chloride levels, white blood cell count, systolic blood pressure, gender, height, and glomerular filtration rate (GFR). In contrast, the variations in the majority of baseline dietary nutrient intake indicators among the albumin groups did not reach statistical significance ($p \geq 0.05$), with the notable exceptions of carbohydrates, calories, dietary fiber, copper, and manganese. Intergroup comparisons revealed marked differences in the intake of carbohydrates, calories, copper, and manganese between the high and low albumin groups ($p < 0.05$), as well as in copper intake between the high and medium albumin groups ($p < 0.05$).

Table 4 presents the time-averaged dietary nutrient intake results. The high concordance of our time-averaged results with the guidelines' recommended values attests to the reliability and quality of our data.

When synthesizing information from Tables 2, 3, and 4, it becomes evident that under different serum albumin stratifications, the majority of baseline clinical indicators show significant differences (73%), whereas only a minority of baseline dietary nutrient intakes are significantly different (20%). In contrast, a substantial proportion (85%) of time-averaged dietary nutrient intakes exhibit significant differences.

3.2 How does dietary nutrition affect the mortality rate in patients

We performed a mortality risk association analysis for 26 commonly utilized nutrients. Table 5 presents the multivariate Cox regression analysis of mortality risk. Without considering the albumin indicator, 20 nutrients (77%) exhibit an association with mortality risk ($p < 0.05$).

However, this association alters when patients are classified based on their albumin levels. For the group with low albumin levels, 4 nutrients (15%) demonstrate an association with mortality risk. In the group with medium albumin levels, 3 nutrients (12%) show an association, while in the group with high albumin levels, 23 nutrients (88%) indicate an association. Specific data can be found in Table 5. This suggests that when a patient's albumin level is high, the majority of dietary nutrients have a sensitive impact on the patient's health status. Conversely, when a patient's albumin level is intermediate or low, only certain key dietary nutrients are closely related to their health status.

Among the three patient groups, intake levels of carbohydrates, vitamin A, niacin, dietary protein intake (DPI), and dietary energy intake (DEI) are all associated with an increased risk of mortality across multiple groups ($p < 0.05$). Consequently, it is imperative to carefully monitor

TABLE 2: Baseline Demographic Characteristics and Clinical Laboratory Findings by Albumin Level Groups[1].

		All		Low Albumin Group		Medium Albumin Group		High Albumin Group		
Variables	Unit	Mean	Std	Mean	Std	Mean	Std	Mean	Std	P[2]
Age	year	59.11	15.78	63.39	14.03	60.27	15.85	53.64	15.84	<0.001
Gender	-	female 327 (50%)		female 100 (46%)		female 117 (53%)		female 110 (51%)		0.34
Height	cm	162.60	9.46	162.86	8.68	161.89	10.47	163.08	9.10	0.81
Weight	kg	61.17	11.95	59.95	12.13	60.44	11.58	63.13	11.95	<0.01
BMI	-	23.19	5.89	22.50	3.66	23.42	8.71	23.66	3.67	0.04
Albumin	g/L	37.15	4.90	31.58	3.00	37.56	1.20	42.29	2.12	<0.001
Diab	-	diabetes 244 (37%)		diabetes 121 (56%)		diabetes 76 (34%)		diabetes 47 (22%)		<0.001
SBP	mmHg	135.65	21.38	137.24	21.97	135.15	21.69	134.58	20.44	0.19
DBP	mmHg	78.97	14.20	75.74	14.55	79.37	14.42	81.79	12.98	<0.001
WBC	$\times 10^9$/L	6.88	2.28	6.88	2.46	6.77	2.40	7.00	1.94	0.58
Hb	g/L	108.11	21.78	104.12	20.54	109.05	21.70	111.15	22.55	<0.001
Urea	mmol/L	21.26	6.54	20.02	6.91	21.13	5.82	22.63	6.62	<0.001
Scr	umol/L	720.38	254.87	616.76	225.81	734.61	243.01	809.44	258.08	<0.001
K	mmol/L	4.33	0.78	4.23	0.87	4.32	0.73	4.45	0.70	<0.01
Na	mmol/L	139.17	4.87	138.51	7.06	139.13	3.66	139.88	2.71	<0.01
Cl	mmol/L	101.48	6.33	101.63	6.31	100.76	7.47	102.06	4.86	0.48
Ca	mmol/L	2.24	0.84	2.09	0.28	2.25	0.29	2.39	1.39	<0.001
P	mmol/L	1.61	0.49	1.52	0.45	1.63	0.44	1.69	0.55	<0.001
Hs-CRP	mg/L	5.66	13.29	7.45	18.04	5.73	11.19	3.78	8.69	<0.01
Glucose	mmol/L	6.19	2.94	6.74	3.88	6.09	2.13	5.74	2.45	<0.001
CO2CP	mmol/L	25.87	4.44	26.56	4.33	26.13	4.42	24.93	4.43	<0.001
GFR	ml/min	0.06	0.60	0.12	1.04	0.04	0.01	0.03	0.02	0.14

Abbreviations: Cl, Chloride; CO2CP, Carbon Dioxide Combining Power; WBC, White Blood Cells; Hb, Hemoglobin; Urea; Ca, Calcium; K, Potassium; Na, Sodium; Scr, Serum Creatinine; P, Phosphorus; Hs-CRP, High Sensitivity C-Reactive Protein; Glucose; Weight; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; Diab, Diabetes; BMI, Body Mass Index; GFR, Glomerular Filtration Rate.

[1] The study subjects were divided into three albumin level groups: Low Albumin Group (albumin levels below 35.2 g/L, n=217), Medium Albumin Group (albumin levels between 35.2 and 39.5 g/L, n=222), and High Albumin Group (albumin levels above 39.5 g/L, n=217).

[2] The term "P" represents the overall test difference. The threshold for statistical significance was established at $P < 0.05$. In instances where $P < 0.01$ or $P < 0.001$, special symbols will denote these heightened levels of significance.

these dietary components throughout the progression of the disease.

Moreover, in patients with high albumin levels, a significant correlation with mortality risk ($p < 0.05$) is observed for the intake of water, protein, fat, carbohydrates, calories, dietary fiber (DF), potassium (K), sodium (Na), magnesium (Mg), phosphorus (P), iron (Fe), zinc (Zn), copper (Cu), manganese (Mn), vitamin A, carotene, vitamin E, thiamine, riboflavin, niacin, ascorbic acid, dietary protein intake (DPI), and dietary energy intake (DEI). In those with medium albumin levels, carbohydrate intake, DPI, and DEI also demonstrated a significant correlation with mortality risk ($p < 0.05$). For patients presenting with low albumin levels, the intake of carbohydrates, retinol, vitamin A, and niacin is significantly correlated with mortality risk ($p < 0.05$).

Figure 3 illustrates the sampling of the relationship between fat intake and mortality risk, based on the Cox model. It is evident that patients with different albumin levels display varying patterns between fat intake and mortality risk. The relationship does not definitely follow a linear pattern and warrants further investigation, despite its statistical significance in the multivariate Cox correlation analysis where it is often interpreted as linear.

3.3 What are the beneficial dietary intake ranges for each nutrition

According to the conclusions drawn in section 3.2, we understand that to a certain extent, dietary nutrition affects the mortality risk of PD patients. The impact of dietary nutrition varies among patients with different albumin levels. Tables 6 presents a comprehensive overview of the specific intake ranges for various dietary nutrients that are beneficial for patients in different albumin groups (corresponds to an HR value less than 1), as determined by the two-stage method.

The tables are designed to provide comprehensive insights into the dietary patterns across these groups, highlighting nutritional components such as water, protein, fat, carbohydrates, and other macronutrients, as well as micronutrients such as copper, phosphorus, and vitamin A, among others. For each nutrient, the table presents: (1) the unit of measurement for dietary intake; (2) the dietary intake ranges correlated with lower mortality risk across various albumin level groups—low, medium, and high—as well as for the overall patient cohort. Here we present the identified beneficial intake ranges associated with lower mortality risk ($HR < 1$) for DPI and protein in Figure 4 and Figure 5. Figures for all nutrients can be found in the supplementary materials 5.

In most cases, points A and B indicate the nutrient intake range associated with low mortality risk, as illustrated in Figures 4(a) and 4(d). In some instances, point B may not exist, as shown in Figure 4(c), where the range associated with low mortality risk could be [A, -). Alternatively, there may be a point C, where the dietary nutrition intake ranges associated with low mortality risk could be [A,B] and [C, -). In our results, no scenarios involve ranges defined by more than three points; specific cases should be interpreted

TABLE 3: Baseline dietary profiles by albumin level groups[1].

		All		Low Albumin Group		Medium Albumin Group		High Albumin Group		
Variables	Unit	Mean	Std	Mean	Std	Mean	Std	Mean	Std	P[2]
Water	g/d	1520.81	572.28	1486.04	561.38	1490.19	585.03	1586.90	566.77	0.07
Protein	g/d	52.01	18.30	51.40	17.36	50.96	18.89	53.69	18.57	0.19
Fat	g/d	58.32	24.01	57.85	23.15	56.65	22.79	60.49	25.93	0.25
Carbohydrate	g/d	214.39	82.84	197.58	70.78	214.50	82.95	231.09	90.52	<0.001
Calories	kcal/d	1544.92	523.92	1471.84	477.13	1528.79	518.59	1634.50	562.02	<0.01
Df	g/d	9.89	5.90	9.18	4.37	10.04	5.86	10.44	7.10	0.03
K	mg/d	1475.64	591.13	1431.76	543.80	1457.93	625.32	1537.64	598.10	0.06
Na	mg/d	2307.36	1182.15	2432.96	1285.46	2267.54	1207.20	2222.48	1034.19	0.06
Mg	mg/d	246.99	98.91	239.63	89.11	244.99	100.59	256.40	105.96	0.08
Ca	mg/d	445.67	230.32	457.24	248.81	423.02	213.19	457.28	227.29	1.00
P	mg/d	792.38	283.65	779.30	264.41	783.71	282.33	814.33	302.94	0.20
Fe	mg/d	15.29	6.58	14.86	5.75	15.36	7.16	15.65	6.72	0.21
Zn	mg/d	7.87	3.33	7.80	3.15	7.89	3.57	7.93	3.26	0.67
Cu	mg/d	1.33	0.89	1.19	0.76	1.28	0.63	1.51	1.16	<0.001
Mn	mg/d	3.93	1.86	3.64	1.58	3.97	1.94	4.18	2.00	<0.01
Se	ug/d	36.77	21.34	35.62	16.06	35.34	15.07	39.39	29.68	0.07
Retinol	ug/d	166.30	207.21	177.98	179.76	165.28	245.23	155.67	190.00	0.26
VitaminA	ugRAE/d	463.23	392.35	461.16	401.21	471.56	416.81	456.78	357.71	0.91
Carotene	ug/d	1743.30	1978.37	1671.51	2068.62	1769.53	2071.96	1788.24	1785.82	0.54
VitaminE	ug/d	20.96	12.88	20.74	14.13	20.10	11.63	22.05	12.75	0.29
Thiamine	mg/d	1.66	10.08	1.98	12.96	1.16	3.71	1.86	11.22	0.90
Riboflavin	mg/d	1.73	9.85	2.11	12.86	1.22	3.38	1.88	10.80	0.81
Nicotinic	mgNE/d	11.98	6.00	11.61	5.17	12.13	6.76	12.20	5.94	0.31
Ascorbic	mg/d	88.58	72.44	83.90	71.42	85.61	68.44	96.31	76.99	0.07
DPI	g/kg/d	0.91	0.30	0.89	0.29	0.89	0.31	0.93	0.29	0.19
DEI	kcal/kg/d	26.93	8.58	25.60	8.12	26.88	8.71	28.32	8.72	<0.001

Abbreviations: Df, Dietary Fiber; K, Potassium; Na, Sodium; Mg, Magnesium; Ca, Calcium; P, Phosphorus; Fe, Iron; Zn, Zinc; Cu, Copper; Mn, Manganese; Se, Selenium; Thiamine, Vitamin B1; Riboflavin, Vitamin B2; Nicotinic, Nicotinic Acid; Ascorbic, Ascorbic Acid; DPI, Dietary Protein Intake; DEI, Dietary Energy Intake.

[1] The study subjects were divided into three albumin level groups: Low Albumin Group (albumin levels below 35.2 g/L, n=217), Medium Albumin Group (albumin levels between 35.2 and 39.5 g/L, n=222), and High Albumin Group (albumin levels above 39.5 g/L, n=217).

[2] The term "P" represents the overall test difference. The threshold for statistical significance was established at $P < 0.05$. In instances where $P < 0.01$ or $P < 0.001$, special symbols will denote these heightened levels of significance.

with reference to points A, B, and C, along with their corresponding p-values.

As demonstrated in Table 6, the dietary nutrients observed to correlate with a lower risk of mortality, based on our method, are consistent with the recommended dietary nutrient intakes suggested by the guidelines.

In the group with low albumin levels, a nonlinear correlation is observed between the risk of mortality and the intake of most dietary nutrients (69%), including protein, fat, carbohydrates, calories, dietary fiber, potassium, sodium, magnesium, phosphorus, iron, zinc, selenium, Vitamin A, Carotene, Vitamin E, Thiamine, Nicotinic, and DPI. For example, the optimal intake is determined to be 194.82-296.37g/d for carbohydrates and 9.44-14.6g/d for dietary fiber. This implies that for patients presenting low albumin levels, it is necessary to strictly regulate the intake of many nutrients, keeping in mind both the upper and lower limits, in order to prevent excessive or insufficient consumption.

For the group with intermediate albumin levels, only certain dietary (35%) nutrients exhibited a correlation with mortality, such as protein, fat, potassium, sodium, calcium, Retinol, Vitamin A, Carotene, and Nicotinic Acid. Dietary fiber, Riboflavin, and DPI are found to have a correlation with the risk of mortality, but the nonlinear test did not demonstrate statistical significance. For patients with intermediate albumin levels, only the intake of key dietary nutrients might significantly impact their health, and thus, should be regulated within the appropriate range. The intake of other dietary nutrients may not necessitate particular attention, or they may refer to the results of the 'Regardless of Albumin Group' as presented in Table 6.

In the group with high albumin levels, most dietary nutrients (81%) exhibited an association with the risk of mortality, but only a subset (15%) demonstrated statistical significance in their nonlinear correlation, such as Df, Mg, Vitamin A, and Ascorbic Acid, among others. This suggests that during periods of relatively high albumin levels in patients, the regulation of dietary nutrient intake is crucial. However, for most dietary nutrients, it may only be necessary to consider either their upper or lower limits. For example, the DPI should preferably not fall below 0.82g/(kg·d), which is in accordance with the guidelines provided by Ikizler et al. [13].

For each dietary nutrient and its association with the risk of mortality, we can generate four figures, such as Figure 5 and Figure 4. The figures provide a visual representation of the dietary nutrient ranges correlated with reduced mortality risk, as detailed in Table 6, and include the 95% confidence intervals.

Specifically, Figure 5 delineates the influence of protein intake on the mortality risk of patients across different protein groups. In all protein groups, total protein intake exhibits a significant association with mortality risk ($p<0.05$). Notably, the correlation between protein

TABLE 4: Time-average dietary profiles by albumin level groups[1].

			All		Low Albumin Group		Medium Albumin Group		High Albumin Group		
Variables	Unit	Guidelines[2]	Mean	Std	Mean	Std	Mean	Std	Mean	Std	P[3]
Water	g/d	500 mL + previous day's urine output + previous day's net peritoneal dialysis ultrafiltration amount [37]	1512.00	522.61	1441.07	471.74	1518.35	495.35	1574.88	585.45	<0.01
Protein	g/d	see DPI	52.52	15.78	49.91	13.48	52.50	14.43	55.09	18.50	<0.001
Fat	g/d	25% to 35% of total calories [38]	61.28	17.76	58.63	17.49	61.71	16.84	63.45	18.61	<0.01
Carbohydrate	g/d	the recommended carbohydrate energy contribution is between 55% and 65% [38].	215.34	68.68	192.29	54.77	216.08	64.83	237.06	77.04	<0.001
Calories	kcal/d	see DEI	1582.30	450.26	1451.05	374.35	1593.96	423.44	1698.71	507.52	<0.001
Df	g/d	19.8 g/d [39]	10.18	3.75	9.03	3.42	10.52	3.86	10.96	3.69	<0.001
K	mg/d	normal 2000 mg/d [40]	1493.33	481.10	1388.68	428.49	1525.76	503.01	1563.75	492.45	<0.001
Na	mg/d	<2300 mg/d [13]	2595.51	925.01	2511.73	892.66	2628.97	879.93	2644.62	994.49	0.13
Mg	mg/d	normal 300-330mg/d [40]	253.39	80.37	236.79	75.59	257.28	76.78	265.78	85.72	<0.001
Ca	mg/d	800-1000 mg/d, <2000mg/d [13]	437.14	168.42	447.75	184.32	441.10	163.93	422.98	155.83	0.12
P	mg/d	800-1000 mg/d [13]	799.52	244.67	764.02	220.35	804.01	227.36	829.70	277.75	<0.01
Fe	mg/d	normal 10-18 mg/d, <42 mg/d [40]	15.70	5.17	14.72	4.94	15.75	4.75	16.59	5.60	<0.001
Zn	mg/d	normal 8.5-12mg/d, <40mg/d [40]	8.18	2.87	7.77	2.51	8.26	2.65	8.49	3.34	<0.01
Cu	mg/d	normal 0.7-0.8 mg/d, < 8 mg/d [40]	1.26	0.44	1.15	0.38	1.27	0.41	1.37	0.49	<0.001
Mn	mg/d	normal 4.0-4.5 mg/d, < 11 mg/d [40]	3.92	1.56	3.51	1.27	3.96	1.38	4.28	1.87	<0.001
Se	ug/d	normal 60 ug/d, <400ug/d [40]	35.71	11.36	34.44	10.95	34.96	10.25	37.68	12.50	<0.01
Retinol	ug/d	-	166.44	152.01	193.04	208.15	152.79	99.26	153.78	124.27	<0.01
VitaminA	ugRAE/d	normal 600-770 ugRAE/d, < 30 ugRAE/d [40]	469.28	272.27	473.60	314.27	469.66	248.05	464.70	251.00	0.73
Carotene	ug/d	-	1705.64	1228.01	1503.12	1238.72	1786.06	1211.35	1824.82	1214.32	<0.01
VitaminE	ug/d	normal <700 ug/d [40]	21.99	9.31	19.82	7.89	22.43	8.67	23.68	10.71	<0.001
Thiamine	mg/d	normal 1.2-1.4 mg/d [40]	1.24	3.73	1.90	6.25	0.85	1.16	0.96	1.07	<0.01
Riboflavin	mg/d	normal 1.2-1.4 mg/d [40]	1.32	3.58	2.01	6.00	0.96	1.05	1.00	0.96	<0.01
Nicotinic	mgNE/d	normal 12-15 mg NE/d [40]	12.45	5.15	11.59	4.41	12.43	4.51	13.32	6.19	<0.001
Ascorbic	mg/d	Male90,Famale75 [13]	88.43	48.69	80.11	51.27	91.20	45.00	93.84	48.69	<0.01
DPI	g/kg/d	1.0-1.2 g/kg/d [13]	0.92	0.26	0.88	0.24	0.93	0.24	0.94	0.29	<0.01
DEI	kcal/kg/d	25-35kcal/kg/d [13]	27.60	7.37	25.44	6.70	28.29	7.13	29.04	7.76	<0.001

Abbreviations: Df, Dietary Fiber; K, Potassium; Na, Sodium; Mg, Magnesium; Ca, Calcium; P, Phosphorus; Fe, Iron; Zn, Zinc; Cu, Copper; Mn, Manganese; Se, Selenium; Thiamine, Vitamin B1; Riboflavin, Vitamin B2; Nicotinic, Nicotinic Acid; Ascorbic, Ascorbic Acid; DPI, Dietary Protein Intake; DEI, Dietary Energy Intake.

[1] The study subjects were divided into three albumin level groups: Low Albumin Group (albumin levels below 35.2 g/L, n=217), Medium Albumin Group (albumin levels between 35.2 and 39.5 g/L, n=222), and High Albumin Group (albumin levels above 39.5 g/L, n=217).

[2] In the "Guidelines" column, the table lists the recommended dietary nutrition intake for patients with chronic kidney disease as suggested by existing guidelines. In the absence of specific guidance for this patient group, the table provides the recommendations for the general population, which are denoted by 'normal'.

[3] The term "P" represents the overall test difference. The threshold for statistical significance was established at $P < 0.05$. In instances where $P < 0.01$ or $P < 0.001$, special symbols will denote these heightened levels of significance.

[4] No clear numerical value. For patients with stable fluid status, the daily fluid intake should be 500 ml plus the previous day's urine output and the previous day's net peritoneal dialysis ultrafiltration amount (2D).

intake and mortality risk in the low-protein and medium-protein groups is nonlinear. The protein intake range associated with low mortality risk for the low-protein group is 52.23g/d-60.02g/d, while for the medium-protein group, it is 46.99g/d-48.48g/d. Conversely, the association between protein intake and mortality risk in the high-protein group is linear, with a hazard ratio of 0.94 (95% CI 0.91,0.97), p <0.001. Without distinguishing between protein groups, the suitable intake range is 50.29g/d-74.76g/d.

Figure 4 illustrates the impact of DPI intake on the mortality risk of patients across different protein groups. In all protein groups, DPI intake shows a significant correlation with mortality risk (p<0.05). The relationship between DPI intake and mortality risk in the low-protein group is nonlinear, with the recommended intake range being 0.87g/(kg·d)-1.11g/(kg·d).

4 Discussion

4.1 How the proposed two-stage model helps Cox identify non-linear relationship

The methodology of the proposed two-stage model displays substantial practicality. While the majority of existing studies have merely established correlations between dietary nutrition and mortality risk, they have often neglected nonlinear associations [1], [8]. In instances where nonlinear relationships are present, as reported in previous studies [1], [12], [41], linear methods such as the Cox model may become ineffective and produce outcomes that are inconsistent with the actual dynamics observed. However, our approach is capable of capturing these nonlinear associations fully and providing sound conclusions.

For example, in Table 5, the Cox analysis results show a significant correlation between DPI and mortality risk across the entire population. However, in subtypes, only the high albumin group shows a significant correlation between DPI and mortality risk, while the low and medium albumin groups do not exhibit a significant association between DPI intake and mortality risk. This is inconsistent with clinical practices in that patients with low albumin levels are typically advised to more stringently control their protein intake. The failure of the linear method may likely stem from the presence of nonlinear correlations that it cannot adequately capture. Our two-stage method, on the other hand, is designed to fully capture such nonlinear relationships.

As demonstrated in Figure 4, our method reveals that, for both the low albumin group (p<0.01, p-non-linear <0.01) and the medium albumin group (p=0.03, p-non-

TABLE 5: Evaluation of Mortality Risk Associated with Dietary Nutrient Intake in Different Albumin Level Groups[1].

		All			Low Albumin Group			Medium Albumin Group			High Albumin Group		
Variables[2]	Unit	HR	95%CI	P[3]	HR	95%CI	P	HR	95%CI	P	HR	95%CI	P
Water	100g/d	0.95	0.92-0.99	<0.01	0.98	0.93-1.04	0.56	0.97	0.91-1.03	0.34	0.86	0.77-0.95	<0.01
Protein	g/d	0.97	0.96-0.98	<0.001	0.98	0.96-1.00	0.09	0.98	0.96-1.01	0.15	0.94	0.91-0.97	<0.001
Fat	10g/d	0.82	0.74-0.92	<0.001	0.97	0.81-1.16	0.72	0.84	0.71-1.01	0.06	0.58	0.44-0.76	<0.001
Carbohydrate	g/d	0.99	0.99-1.00	<0.001	0.99	0.99-1.00	0.05	0.99	0.99-1.00	0.04	0.99	0.98-1.00	<0.01
Calories	10kcal/d	0.99	0.98-0.99	<0.001	0.99	0.98-1.00	0.07	0.99	0.99-1.00	0.09	0.98	0.96-0.99	<0.001
Df	g/d	0.90	0.86-0.95	<0.001	0.93	0.86-1.00	0.06	0.92	0.85-1.00	0.06	0.80	0.70-0.90	<0.001
K	10mg/d	0.99	0.99-1.00	<0.001	0.99	0.99-1.00	0.08	1.00	0.99-1.01	0.64	0.98	0.97-0.99	<0.001
Na	100mg/d	0.97	0.95-0.99	<0.01	1.00	0.97-1.03	0.76	0.97	0.94-1.00	0.09	0.90	0.86-0.95	<0.001
Mg	10mg/d	0.95	0.93-0.97	<0.001	0.97	0.93-1.01	0.12	0.98	0.94-1.02	0.36	0.87	0.81-0.93	<0.001
Ca	100mg/d	0.89	0.81-0.98	0.02	0.96	0.84-1.11	0.58	0.90	0.77-1.05	0.18	0.79	0.61-1.01	0.06
P	10mg/d	0.98	0.97-0.99	<0.001	0.99	0.98-1.00	0.12	0.99	0.98-1.00	0.19	0.97	0.95-0.99	<0.01
Fe	mg/d	0.94	0.91-0.98	<0.01	0.97	0.91-1.04	0.41	0.98	0.93-1.03	0.36	0.77	0.69-0.87	<0.001
Zn	mg/d	0.86	0.80-0.93	<0.001	0.91	0.81-1.02	0.10	0.97	0.87-1.09	0.66	0.61	0.49-0.76	<0.001
Cu	mg/d	0.68	0.46-0.99	0.05	0.88	0.47-1.66	0.70	0.78	0.43-1.43	0.43	0.25	0.08-0.73	0.01
Mn	mg/d	0.80	0.70-0.92	<0.01	0.89	0.74-1.07	0.20	0.98	0.78-1.23	0.85	0.41	0.26-0.64	<0.001
Se	mg/d	1.00	0.98-1.01	0.61	1.02	0.99-1.04	0.22	0.99	0.96-1.01	0.24	0.99	0.95-1.03	0.63
Retinol	10ug/d	1.01	0.99-1.02	0.36	1.02	1.01-1.03	<0.001	0.98	0.96-1.01	0.20	0.99	0.96-1.02	0.56
Vitamin A	100ugRAE/d	0.98	0.92-1.05	0.60	1.09	1.00-1.18	0.04	1.01	0.91-1.12	0.85	0.72	0.60-0.88	<0.001
Carotene	100ug/d	0.99	0.98-1.01	0.45	1.00	0.98-1.02	0.77	1.02	0.99-1.04	0.21	0.91	0.87-0.96	<0.001
Vitamin E	ug/d	0.97	0.95-1.00	0.04	1.01	0.98-1.05	0.48	0.97	0.93-1.01	0.14	0.93	0.87-0.99	0.02
Thiamine	mg/d	1.02	0.99-1.04	0.18	1.02	0.99-1.05	0.23	0.99	0.85-1.14	0.85	1.22	1.01-1.47	0.04
Riboflavin	mg/d	1.02	0.99-1.05	0.17	1.02	0.99-1.05	0.22	0.97	0.82-1.15	0.76	1.24	1.02-1.50	0.03
Nicotinic	mgNE/d	0.92	0.88-0.96	<0.001	0.93	0.87-0.99	0.03	0.99	0.93-1.06	0.80	0.75	0.67-0.85	<0.001
Ascorbic	mg/d	0.99	0.99-1.00	<0.01	1.00	0.99-1.00	0.15	1.00	1.00-1.01	0.21	0.97	0.96-0.98	<0.001
DPI	0.1g/kg/d	0.85	0.79-0.92	<0.001	0.92	0.81-1.03	0.15	0.91	0.80-1.04	0.16	0.71	0.59-0.86	<0.001
DEI	kcal/kg/d	0.94	0.92-0.97	<0.001	0.97	0.92-1.01	0.13	0.96	0.92-1.01	0.10	0.87	0.81-0.94	<0.001

Abbreviations: Df, Dietary Fiber; K, Potassium; Na, Sodium; Mg, Magnesium; Ca, Calcium; P, Phosphorus; Fe, Iron; Zn, Zinc; Cu, Copper; Mn, Manganese; Se, Selenium; Thiamine, Vitamin B1; Riboflavin, Vitamin B2; Nicotinic, Nicotinic Acid; Ascorbic, Ascorbic Acid; DPI, Dietary Protein Intake; DEI, Dietary Energy Intake.

[1] The study subjects were divided into three albumin level groups: Low Albumin Group (albumin levels below 35.2 g/L, n=217), Medium Albumin Group (albumin levels between 35.2 and 39.5 g/L, n=222), and High Albumin Group (albumin levels above 39.5 g/L, n=217).

[2] The confounding variables we adjusted for include: Chloride, Carbon Dioxide Combining Power, White Blood Cells, Hemoglobin, Urea, Calcium, Potassium, Sodium, Serum Creatinine, Phosphorus, Albumin, High Sensitivity C-Reactive Protein, Glucose, Weight, Systolic Blood Pressure, Diastolic Blood Pressure, Diabetes, Body Mass Index, and Glomerular Filtration Rate. See more details at Table 2.

[3] The term "P" represents the overall test difference. The threshold for statistical significance was established at $P < 0.05$. In instances where $P < 0.01$ or $P < 0.001$, special symbols will denote these heightened levels of significance.

linear =0.05), there exists a significant and nonlinear relationship between DPI and mortality risk. For the low albumin group, our findings suggest that a protein intake of 0.87-1.12 g/kg/day is beneficial for patient prognosis. For the medium albumin group, an intake of 0.85-0.86 g/kg/day is associated with better outcomes. In the high albumin group, our approach indicates a significant relationship between DPI and mortality risk ($p<0.01$); however, the non-linearity is not significant (p-non-linear=0.68), suggesting that an intake of <0.89 g/kg/day is associated with lower mortality risk, which aligns with the conclusions drawn from linear analyses.

Overall, our method introduces the exploration of non-linear relationships between variables, which, compared to solely using linear methods, yields more robust and precise conclusions.

4.2 The value of long-term, expert-supervised dietary nutrition data

Investigations into the dietary nutrition of PD patients within the ESRD demographic have been notably limited. This scarcity of research is, in part, attributable to the prolonged disease progression characteristic of PD, which necessitates an extended period for data collection [42]. Moreover, the process of acquiring high-quality dietary data, under the supervision of experts, is inherently complex, requiring active participation from patients in record-keeping, as well as precise and targeted documentation by dietitians [43]. The dataset utilized in this study consists of high-quality, fine-grained longitudinal data, meticulously collected under expert supervision, and is both comprehensive and detailed, encompassing 12 years of clinical indicators and dietary logs, with follow-ups conducted at least quarterly. This data has been processed with utmost care, providing a critical foundation for subsequent analysis and experimentation.

4.3 Key findings regarding the dietary nutritional intake range associated with a reduced mortality risk

Leveraging comprehensive and precise data, combined with a two-stage model methodology, our research investigates a broad spectrum of dietary nutrients pertinent to patients with ESRD. The key findings yielded by our approach are summarized below.

- **Protein or DPI.** Protein intake is a critical clinical metric, and it is generally recommended that patients with peritoneal dialysis (PD) consume 1.0-1.2 g/kg/day of protein [13]. Our experimental results indicate that for PD patients, a protein intake ranging from 0.87 to 1.28 g/kg/day is associated with a reduced risk of mortality, which is consistent with the guidelines. Moreover, extending beyond the guidelines, we observed that excessive protein intake may pose risks for patients with hypoalbuminemia, as illustrated in Figure 4(a).

TABLE 6: Nutritional intake ranges associated with low mortality risk in patients across albumin levels[1]

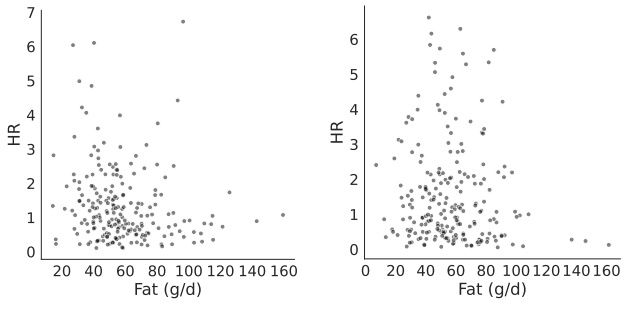
		All					Low Albumin Group					Medium Albumin Group					High Albumin Group				
Variables	Unit	A[2]	B[2]	C[2]	P[3]	P-NL[3]	A	B	C	P	P-NL	A	B	C	P	P-NL	A	B	C	P	P-NL
Water	100g/d	14.11	-	-	<0.01	0.10	12.76	13.72	-	0.15	0.08	13.79	-	-	0.81	0.84	14.45	-	-	0.02	0.29
Protein	g/d	50.29	74.76	-	<0.001	<0.001	52.23	62.02	-	<0.01	<0.01	46.99	48.48	-	<0.01	0.01	51.29	-	-	<0.01	0.21
Fat	10g/d	5.91	7.72	-	<0.001	<0.001	5.88	7.36	-	<0.001	<0.001	5.78	5.84	-	<0.001	<0.01	6.04	-	-	<0.001	0.49
Carbohydrate	g/d	206.26	382.04	-	<0.001	0.02	194.82	296.37	-	<0.01	<0.001	201.57	-	-	0.22	0.81	216.48	-	-	0.02	0.63
Calories	10kcal/d	152.47	238.33	-	<0.001	<0.001	150.10	199.18	-	<0.001	<0.001	148.13	197.21	-	0.06	0.10	156.05	-	-	<0.001	0.42
Df	g/d	9.73	15.45	-	<0.001	<0.001	9.44	14.60	-	<0.001	<0.001	9.20	9.90	13.56	0.04	0.15	10.17	16.00	-	<0.001	<0.01
K	10mg/d	144.09	235.32	-	<0.001	<0.001	142.06	213.51	-	0.03	0.05	133.14	138.99	-	0.04	0.02	148.89	-	-	<0.001	0.07
Na	100mg/d	24.89	24.89	-	<0.001	<0.001	21.63	24.47	-	<0.001	<0.001	20.91	24.44	-	<0.001	<0.001	25.04	-	-	<0.001	0.87
Mg	10mg/d	24.05	40.57	-	<0.001	<0.001	23.67	36.19	-	0.02	0.01	23.95	36.13	-	0.75	0.73	24.23	-	-	<0.001	0.02
Ca	100mg/d	4.10	7.74	-	<0.001	0.01	4.20	-	-	0.81	0.85	3.94	4.07	-	<0.01	0.01	4.01	6.61	-	0.12	0.27
P	10mg/d	76.06	117.12	-	<0.001	<0.001	77.96	98.67	-	<0.01	<0.01	73.77	101.97	-	0.28	0.33	77.26	-	-	<0.01	0.06
Fe	mg/d	14.81	26.70	-	<0.001	<0.001	14.27	20.09	-	<0.01	<0.001	14.37	24.64	-	0.59	0.56	15.34	-	-	<0.001	0.48
Zn	mg/d	7.58	12.27	-	<0.001	<0.001	7.39	11.05	-	<0.01	<0.01	7.57	8.49	-	0.21	0.11	7.61	-	-	<0.001	0.44
Cu	mg/d	1.18	2.28	-	0.04	0.21	1.14	1.14	-	0.13	0.06	1.12	1.14	1.38	0.93	0.97	1.28	-	-	0.03	0.49
Mn	mg/d	3.64	7.24	-	<0.001	<0.01	3.53	6.27	-	0.05	0.04	2.40	3.57	5.57	0.99	0.95	3.70	-	-	<0.001	0.63
Se	ug/d	34.60	37.70	-	<0.001	<0.001	34.44	38.76	-	<0.001	<0.001	29.75	33.09	-	0.09	0.10	36.24	53.34	-	0.21	0.11
Retinol	10ug/d	14.06	14.59	-	<0.001	<0.001	9.19	15.95	-	<0.001	0.23	11.97	12.86	-	0.02	0.02	9.92	13.00	-	0.16	0.09
Vitamin A	100ugRAE/d	4.17	7.18	-	<0.001	<0.001	4.20	6.65	-	<0.001	<0.001	4.05	4.22	-	0.04	0.02	3.45	4.17	6.26	<0.001	0.04
Carotene	100ug/d	14.59	32.94	-	<0.001	<0.001	13.50	36.53	-	<0.001	<0.001	14.28	21.08	-	<0.01	<0.01	15.82	-	-	<0.01	0.60
Vitamin E	ug/d	20.47	33.62	-	<0.001	<0.001	20.19	22.67	-	<0.001	<0.001	20.19	28.31	-	0.19	0.21	21.40	-	-	0.06	0.93
Thiamine	mg/d	0.71	0.71	-	<0.001	<0.001	0.70	0.72	-	<0.01	<0.01	0.56	0.63	-	0.98	0.91	0.69	-	-	0.05	0.07
Riboflavin	mg/d	-	-	-	<0.001	<0.001	0.75	0.85	-	0.11	0.18	0.77	0.78	-	0.08	0.03	0.81	1.89	-	0.06	0.10
Nicotinic	mgNE/d	11.57	20.03	-	<0.001	<0.001	11.19	19.37	-	0.02	0.02	11.28	14.54	-	<0.01	<0.01	12.19	-	-	<0.001	0.18
Ascorbic	mg/d	79.27	-	-	<0.01	0.10	75.89	-	-	0.39	0.93	78.41	79.74	-	0.06	0.11	82.74	-	-	<0.001	0.04
DPI	0.1g/kg/d	8.75	12.83	-	<0.001	<0.001	8.73	11.18	-	<0.01	<0.01	8.49	8.61	-	0.03	0.05	8.94	-	-	<0.01	0.68
DEI	kcal/kg/d	26.22	41.93	-	<0.001	<0.01	25.86	34.28	-	<0.01	<0.001	25.69	41.98	-	0.19	0.37	26.67	27.68	28.14	<0.01	0.13

Abbreviations: Df, Dietary Fiber; K, Potassium; Na, Sodium; Mg, Magnesium; Ca, Calcium; P, Phosphorus; Fe, Iron; Zn, Zinc; Cu, Copper; Mn, Manganese; Se, Selenium; Thiamine, Vitamin B1; Riboflavin, Vitamin B2; Nicotinic, Nicotinic Acid; Ascorbic, Ascorbic Acid; DPI, Dietary Protein Intake; DEI, Dietary Energy Intake.

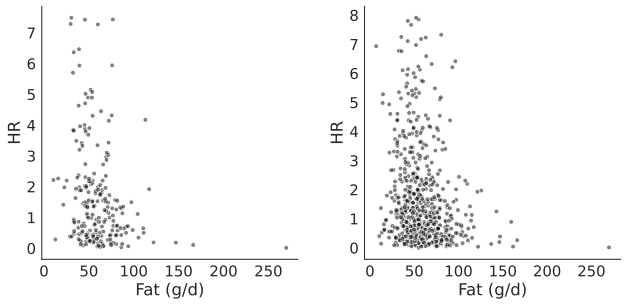
[1] The study subjects were divided into three albumin level groups: Low Albumin Group (albumin levels below 35.2 g/L, n=217), Medium Albumin Group (albumin levels between 35.2 and 39.5 g/L, n=222), and High Albumin Group (albumin levels above 39.5 g/L, n=217).

[2] Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

[3] The term P denotes the significance of the association between nutritional elements and mortality risk, whereas P for non-linear(P-NL) represents the statistical significance of the potential nonlinearity in the correlation between nutritional intake and mortality risk. The nonlinear relationship between nutritional elements and mortality risk is considered statistically significant only when both P and P for non-linear are significant. It should be noted that * represents $p<0.05$, ** signifies $p<0.01$, and *** indicates $p<0.001$.

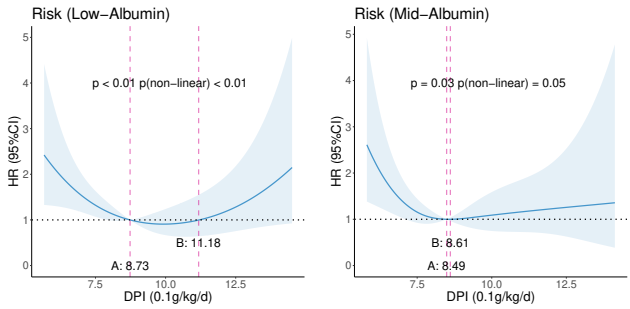


(a) Low (b) Medium

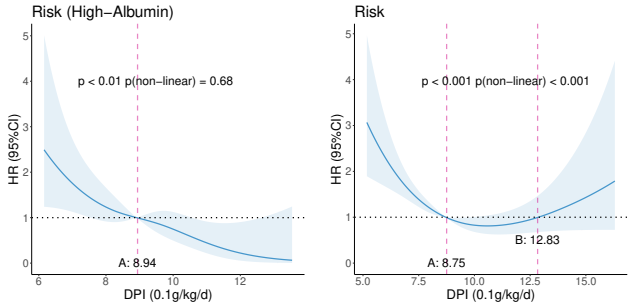


(c) High (d) All

Fig. 3: The correlation between fat intake and mortality risk among patients grouped by different albumin levels, sampled from the results of model stage 1.



(a) Low (b) Medium

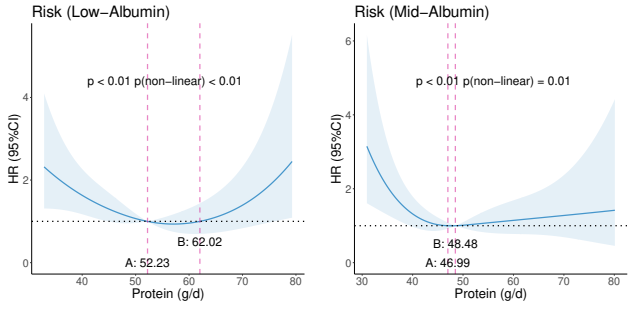


(c) High (d) All

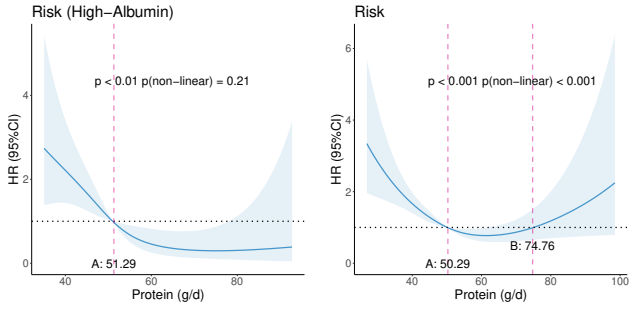
Fig. 4: Appropriate DPI intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

However, for patients with medium or high serum albumin levels, increased protein consumption does not correlate with heightened risk, as shown in Figures 4(b-c).

- **Calories or DEI.** Clinical guidelines generally recommend a caloric intake of 25-35 kcal/kg/day for patients with ESRD [13]. Our research findings suggest that for patients with hypoalbuminemia, an energy intake of 26-42 kcal/kg/day is associated with a lower risk of mortality. In contrast, for those in the medium and high serum albumin groups, an intake of over 27 kcal/kg/day correlates with a reduced mortality risk, with no clearly identified upper limit of energy intake. ESRD patients often face risks of malnutrition due to factors such as appetite loss, toxin accumulation, chronic inflammation, and socioeconomic factors. It is also noted that patients on dialysis may spontaneously decrease their protein and calorie consumption owing to uremic toxins, increases in leptin and other cytokines, as well as delayed gastric emptying [12], underscoring the importance of ensuring a minimum level of energy intake. Furthermore, some ESRD patients may also have comorbidities such as diabetes or cardiovascular diseases, necessitating blood sugar control and reduced cardiovascular risk, which might influence the upper limits of caloric intake.
- **Fat.** Regarding the specific intake of fats and carbohydrates, the guidelines do not stipulate exact amounts but rather recommend proportions of these macronutrients in relation to total caloric intake, as detailed in Table 4. Our experimental findings demonstrate a



(a) Low (b) Medium



(c) High (d) All

Fig. 5: Appropriate protein intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

significant nonlinear relationship between both fat and carbohydrate intake and mortality risk in patients with chronic kidney disease. An intake of 59-77 grams per day of fats and 206-382 grams per day of carbohydrates is associated with a lower mortality risk. More granular results can be found in Table 6. Limiting the minimum intake of fats is necessary because fats are essential components of a healthy diet, providing energy, aiding in the regulation of cholesterol and blood pressure, and facilitating the absorption of vitamins. However, excessive consumption of fat, particularly harmful types, can lead to accumulation in the blood vessels, heart, and kidneys. Saturated and trans fats, in particular, can elevate cholesterol levels and contribute to vascular occlusion. Therefore, it is also necessary to limit the maximum intake of fats [44].

- **Sodium and Water.** Sodium is a mineral present in salt that plays a crucial role in regulating body fluid balance. Excessive sodium intake can lead to fluid retention, which may result in high blood pressure and swelling. Such retention places additional strain on the kidneys and heart. In clinical practice, it is generally recommended that a patient's fluid intake is guided by the urine output and dialysis fluid losses from the previous day, as detailed in Table 4, while sodium (Na) intake should typically be less than 2300 mg/day [13]. The Cox linear results from Table 5 indicate that fluid intake is a protective factor for patient prognosis. However, the non-linear results from our proposed method suggest no significant association between fluid intake and patient prognosis, see Table 6. Given the limited capacity of dialysis to remove fluid, excessive water and salt intake increases the risk of volume overload and cardiovascular disease in patients. Therefore, it is of greater importance in clinical practice to guide patients to limit their water and salt intake to maintain volume balance.
- **Vitamin A.** Our research has uncovered a nonlinear relationship between vitamin A and mortality risk, identifying an intake range of 417-718 µg RAE/day associated with lower mortality risk, a topic not extensively discussed in prior studies [45]. However, there is literature that indeed highlights the importance of vitamin A for dialysis patients. For instance, a case study reported a Japanese male hemodialysis patient who was diagnosed with vitamin A deficiency (VAD), emphasizing the potential risk of VAD in dialysis patients, particularly when dietary intake is insufficient [46]. Furthermore, during the process of peritoneal dialysis, patients' vitamin A levels may gradually increase. Despite significant peritoneal transfer and losses into the dialysis fluid, levels of vitamin A and its protein carriers remained elevated throughout the study period [47], [48]. It is crucial to note that excessive levels of vitamin A in the body can lead to toxicity [49].
- **Phosphorus.** Elevated blood phosphorus concentrations are associated with increased mortality in patients with ESRD and contribute to a heightened risk of cardiovascular events, primarily due to their role in vascular calcification [50]. Excessive phosphorus can also lead to secondary hyperparathyroidism, which stimulates the release of calcium from the bone matrix and results in osteodystrophy [50], [51]. Clinically, it is generally recommended that ESRD patients limit their phosphorus intake to 800-1000 mg per day. Our experimental findings are largely in agreement with these guidelines.
- **Dietary Fiber.** Moreover, it is important to highlight that the dietary fiber intake range associated with low mortality risk. Irrespective of albumin levels, we have identified a beneficial range of dietary fiber intake between 9.73 and 15.45 g/d. When albumin levels are factored into the analysis, the data suggest tailored intake ranges: 9.44-14.6 g/d for individuals with lower albumin and 10.17-16.6 g/d for those with higher albumin levels. These intake ranges correspond with, and in some cases extend, the fiber intake levels previously suggested for hemodialysis patients, which are between 10.2 and 12.4 g/d [52], [53]. Considering the proposed fiber intake for chronic kidney disease patients not on hemodialysis is as high as 19.8 g/d [39], our findings support a more generous upper limit. Furthermore, dietary fiber is acknowledged for its capacity to attenuate the glycemic impact of carbohydrate absorption [54], [55], selectively foster beneficial gut microbiota, facilitate the elimination of toxins, and improve lipid profiles, conferring renal advantages [56], [57]. Therefore, advocating for an intake range marginally above that recommended for hemodialysis patients appears substantiated. Interestingly, previous literature has documented that actual cellulose consumption by patients often does not meet these suggested levels [8], a finding that is in line with the patterns we have observed, as depicted in Table 3. This discrepancy suggests that prevalent dietary habits may fall short of supporting optimal health. Nonetheless, our two-stage method successfully delineates appropriate dietary fiber intake ranges, underscoring the robustness and utility of our investigative framework.
- **Calcium.** In the Table 6, we observed that the nutritional intake range of calcium associated with low mortality risk is lower than the recommendations provided by the guidelines. This can be attributed to the fact that for PD patients, non-dietary sources of calcium, such as calcium-phosphate binders, are often used in clinical practice. Therefore, it is normal for the dietary intake of calcium to be below the guideline recommendations for this patient group.
- **Trace Elements.** Trace elements are essential for patients with ESRD. Those undergoing dialysis frequently experience deficiencies in vitamin C, folic acid, vitamin D, iron, and zinc, and may also exhibit insufficient selenium levels, which can lead to a state of antioxidant deficiency [58]. While guidelines do not stipulate specific intake ranges for these micronutrients, our study has identified a correlation between these nutritional elements and mortality risk, as detailed in Table 6.

4.4 Limitations and future works

While our methodology, leveraging reliable long-term data and a new model design, offers numerous advantages, it also presents certain limitations.

Regarding the data, the condition of patients undergoing Peritoneal Dialysis (PD) during each dialysis session is vital information, yet our data does not account for factors such as medication use or nutritional supplements. Additionally, the daily activity levels of patients, significantly associated with dietary nutrition intake and planning [59], [60], are not included in our data. Our cohort also has its limitations. It only includes data from Chinese individuals, despite the extended follow-up period and high-quality records. However, our framework is universal and can be applied to other cohorts. We eagerly anticipate expanding and validating our conclusions in other studies.

In terms of experimental design, this study categorizes patients based on albumin levels, given their significant role in dietary nutrition planning. However, there is a lack of related research in this field. The complex interplay between a patient's dietary nutrition planning and their basal metabolism, underlying diseases, disease progression stages, and various examination indicators necessitates further research and a more personalized approach. A Bayesian or causal inference-based personalized dietary nutrition recommendation could be highly beneficial. Thus, we aim to explore more advanced, user-friendly, and personalized dietary nutrition recommendation methods in our future work.

5 Conclusion

This study advances our understanding of dietary nutrient intake and its association with mortality risk among patients undergoing peritoneal dialysis (PD). Leveraging a comprehensive, long-term dataset and a two-stage analytical model, it clarifies the relationship between 26 dietary nutrients and mortality risk in PD patients, highlighting non-linear associations for certain nutrients. The specific intake thresholds identified in this research are associated with low mortality risk and can inform effective dietary planning and management for this patient population. Furthermore, by associating personalized dietary intake ranges with albumin subtypes, this study not only strengthens existing knowledge but also establishes a groundwork for more tailored and individualized dietary approaches for PD patients. Future research will seek to include additional variables, such as patient medications and daily activity levels, to refine these associations. Additionally, it may investigate individualized dietary nutrition strategies using methods like causal inference or Bayesian models. These endeavors aim to provide more precise, actionable, and personalized nutritional guidance for patients.

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Competing Interests

The authors declare that they have no competing interests.

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APPENDIX: INTRODUCTION TO RESTRICTED CUBIC SPLINES

Restricted Cubic Splines (RCS), also known as natural cubic splines, are a flexible tool for modeling nonlinear relationships between a predictor variable and an outcome variable. RCS are particularly useful in regression models, including Cox proportional hazards models for survival analysis, where the relationship between the predictor and the outcome may not be adequately captured by a simple linear term.

A cubic spline is defined as a piecewise cubic polynomial that is continuous and has continuous first and second derivatives. The points at which the pieces of the polynomial meet are called knots. The 'restricted' part in RCS means that the function is constrained to be linear before the first knot and after the last knot, which prevents extreme extrapolation at the tails of the distribution.

For a given predictor variable x, and k internal knots at positions $\xi_1, \xi_2, \ldots, \xi_k$, the RCS function $f(x)$ can be expressed as:

$$f(x) = \beta_0 + \beta_1 x + \sum_{j=1}^{k} \beta_{j+1} h(x, \xi_j)$$

where:

- $\beta_0, \beta_1, \ldots, \beta_{k+1}$ are the model coefficients to be estimated.
- $h(x, \xi_j)$ are the basis functions defined for each knot ξ_j, given by:

$$h(x, \xi_j) = (x-\xi_j)_+^3 - (\xi_k-\xi_j) \left[\frac{(x-\xi_k)_+^3}{\xi_k - \xi_{k-1}} - \frac{(x-\xi_{k-1})_+^3}{\xi_k - \xi_{k-1}} \right]$$

- $(x-\xi_j)_+^3$ represents the cubic power of the positive part of $x - \xi_j$, i.e., $(x - \xi_j)_+^3 = (x - \xi_j)^3$ if $x > \xi_j$, and 0 otherwise.

The linear component of the model is represented by $\beta_0 + \beta_1 x$, and the nonlinear components are captured by the sum of the basis functions $\sum_{j=1}^{k} \beta_{j+1} h(x, \xi_j)$. The inclusion of these nonlinear terms allows the RCS to flexibly model the shape of the relationship between x and the outcome across the range of x, while maintaining linearity outside the range of the knots.

When using RCS in regression models, it is common to perform hypothesis testing to assess whether the nonlinear components significantly improve the fit of the model compared to a model with only the linear term. This is done by computing the change in the model's deviance, which follows a chi-squared distribution with degrees of freedom equal to the number of nonlinear terms tested.

APPENDIX:FREQUENCY DISTRIBUTION OF PATIENT FOLLOW-UP DURATION.

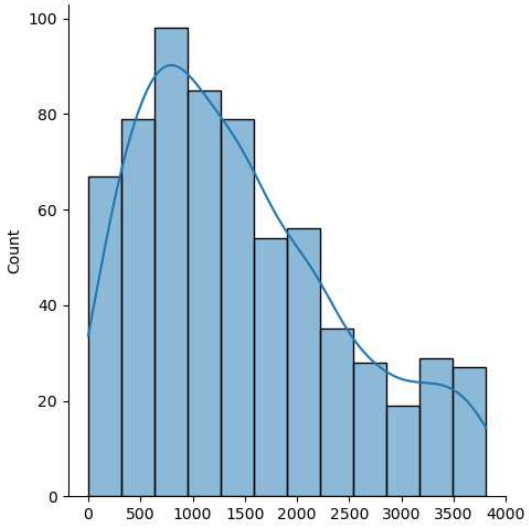
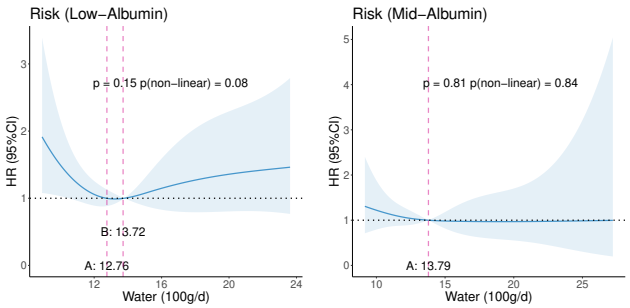


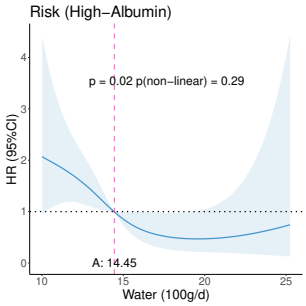
Fig. 6: Frequency distribution of patient follow-up duration. The x-axis represents the follow-up duration (in days), while the y-axis represents the corresponding number of patients.

APPENDIX: RANGES OF NUTRIENT INTAKE ASSOCIATED WITH LOW MORTALITY RISK ACROSS VARYING ALBUMIN LEVELS

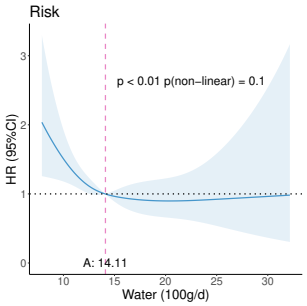


(a) Low

(b) Medium

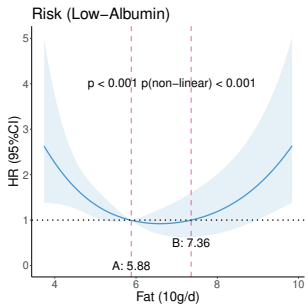


(c) High

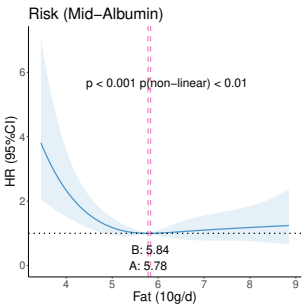


(d) All

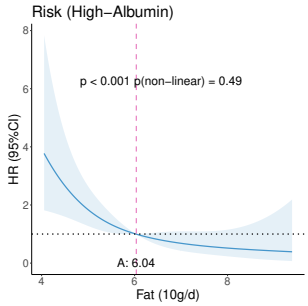
Fig. 7: Appropriate water intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).



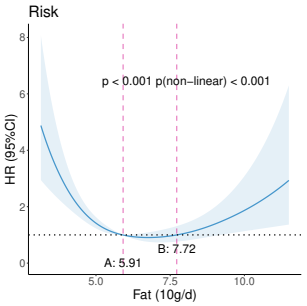
(a) Low



(b) Medium

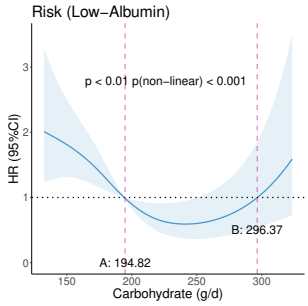


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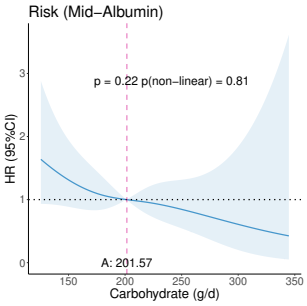


(d) All

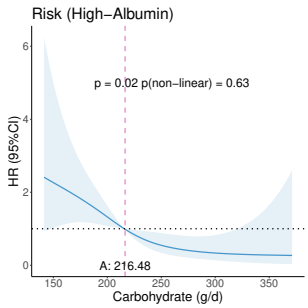
Fig. 8: Appropriate fat intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).



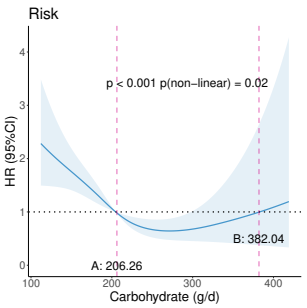
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(b) Medium

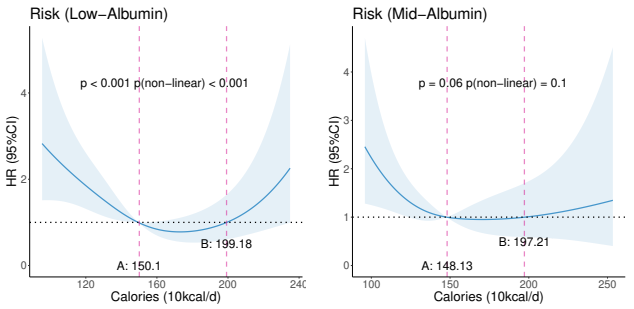


(c) High

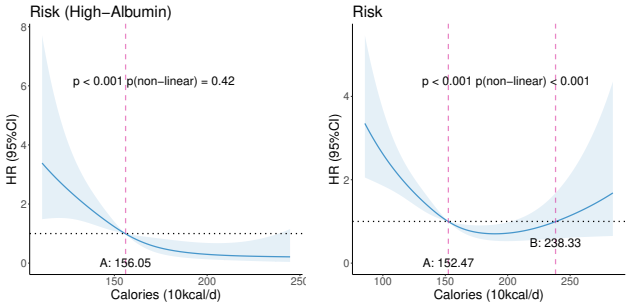


(d) All

Fig. 9: Appropriate carbohydrate intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

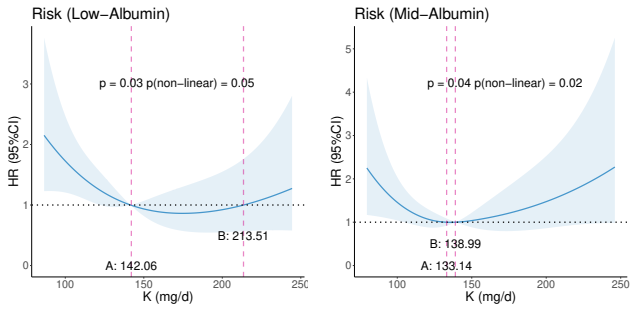


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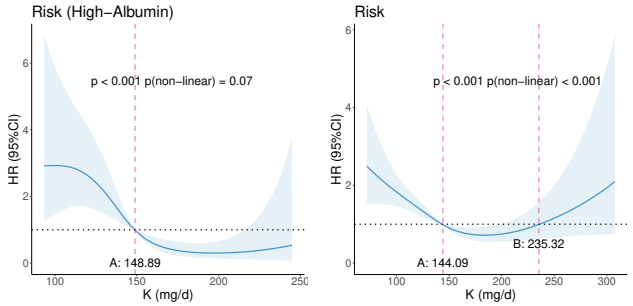


(c) High (d) All

Fig. 10: Appropriate calories intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

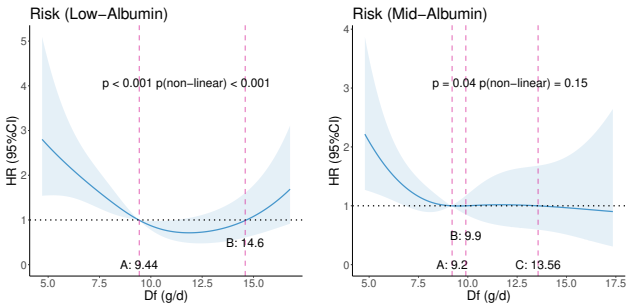


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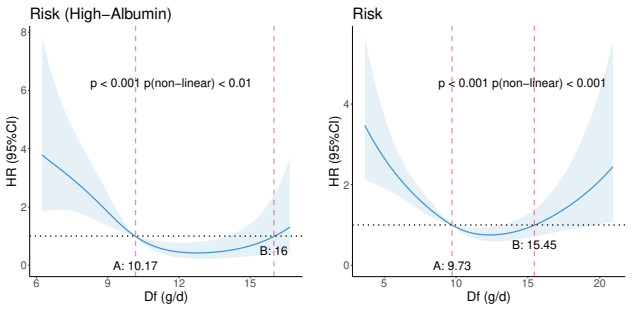


(c) High (d) All

Fig. 12: Appropriate K intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

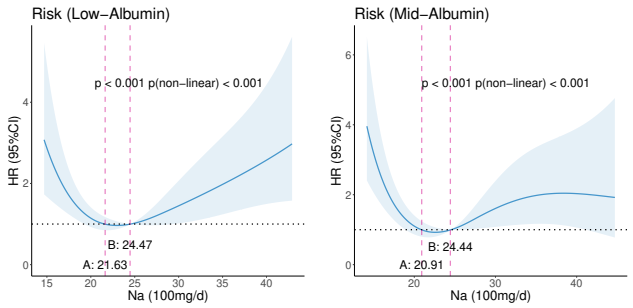


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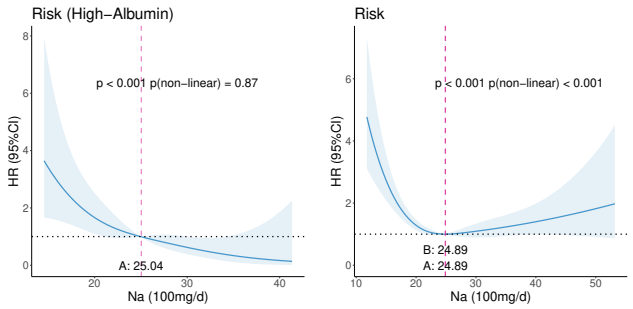


(c) High (d) All

Fig. 11: Appropriate df intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

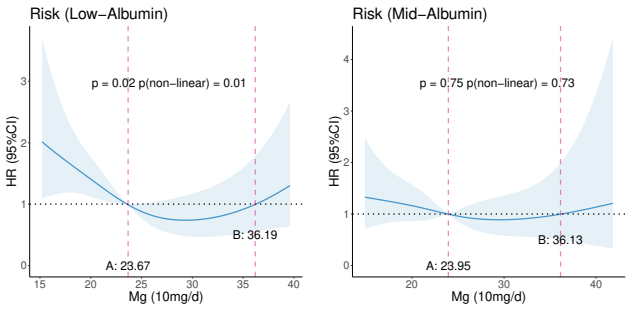


(a) Low (b) Medium

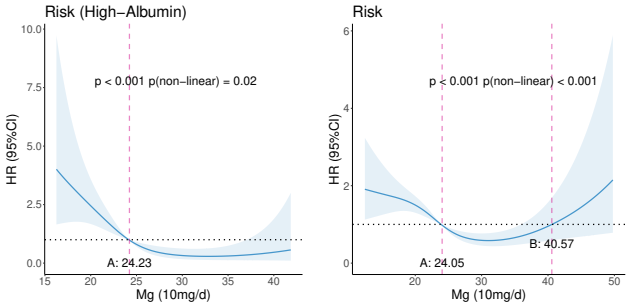


(c) High (d) All

Fig. 13: Appropriate Na intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

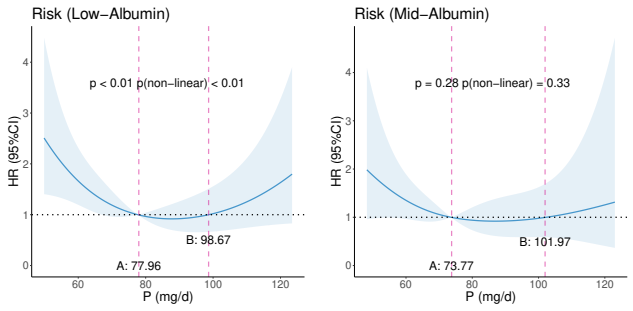


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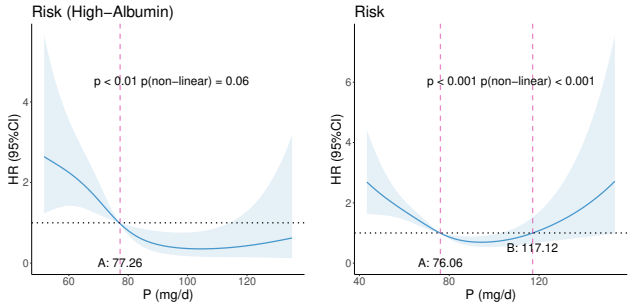


(c) High (d) All

Fig. 14: Appropriate Mg intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

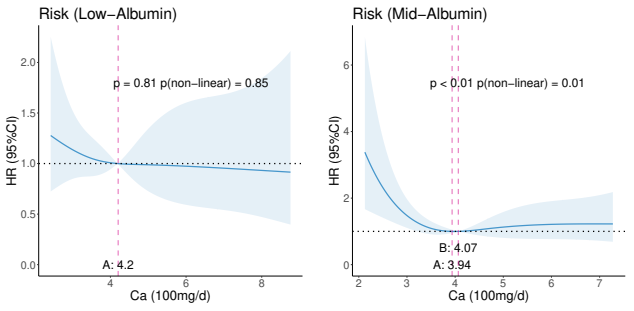


(a) Low (b) Medium

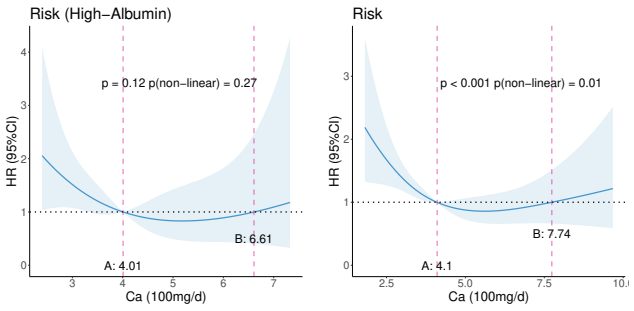


(c) High (d) All

Fig. 16: Appropriate P intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

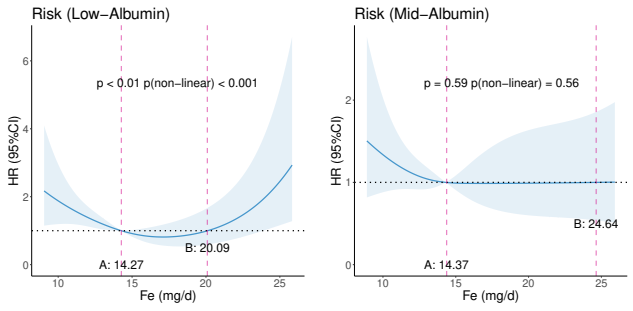


(a) Low (b) Medium

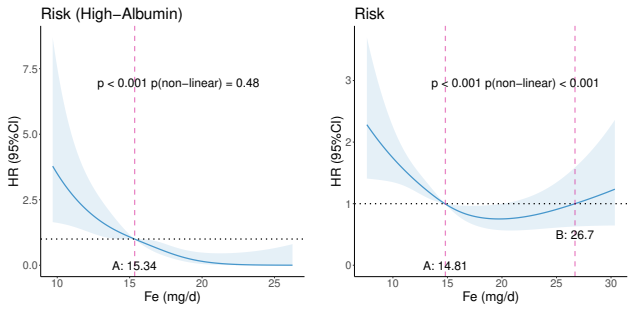


(c) High (d) All

Fig. 15: Appropriate Ca intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

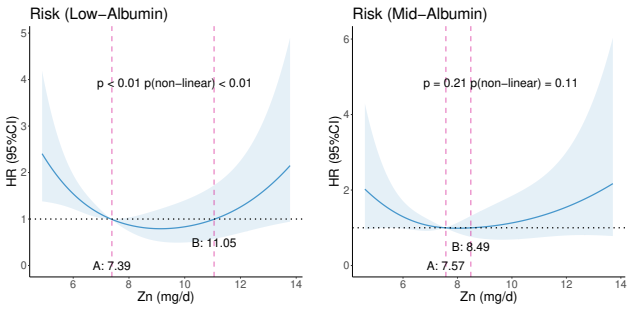


(a) Low (b) Medium



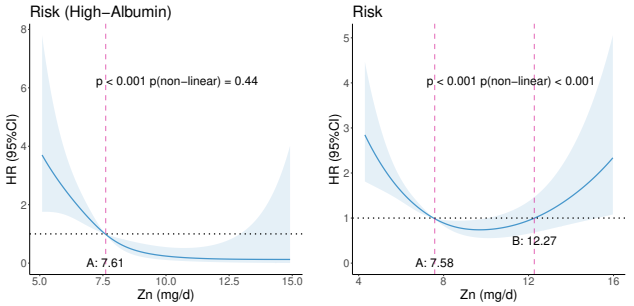
(c) High (d) All

Fig. 17: Appropriate Fe intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).



(a) Low

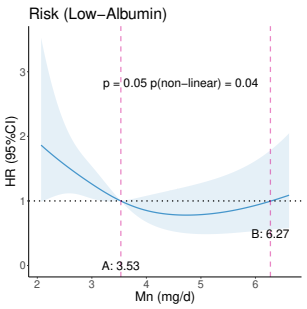
(b) Medium



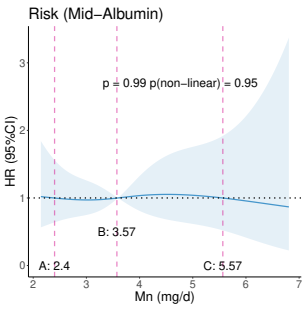
(c) High

(d) All

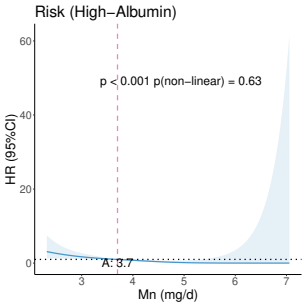
Fig. 18: Appropriate Zn intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).



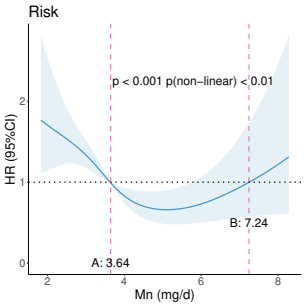
(a) Low



(b) Medium

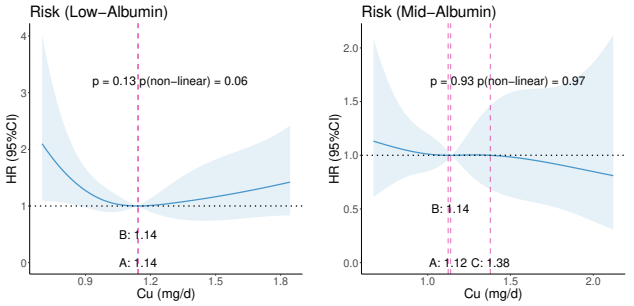


(c) High



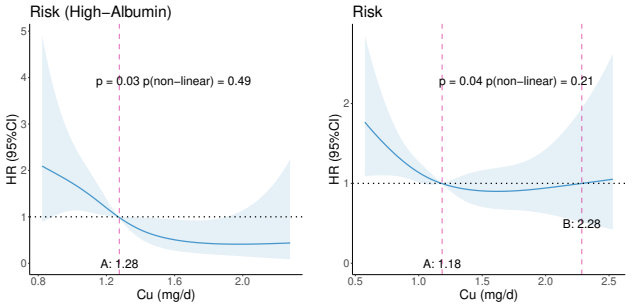
(d) All

Fig. 20: Appropriate Mn intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).



(a) Low

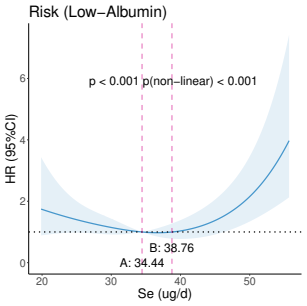
(b) Medium



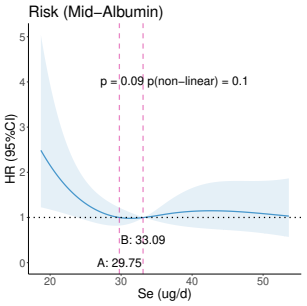
(c) High

(d) All

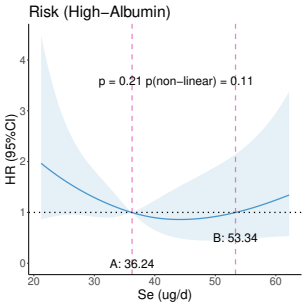
Fig. 19: Appropriate Cu intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).



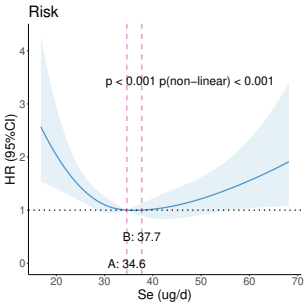
(a) Low



(b) Medium

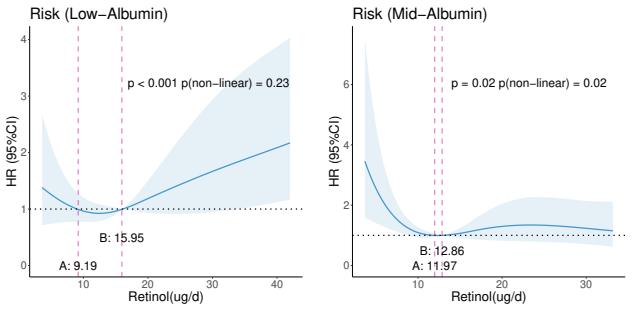


(c) High

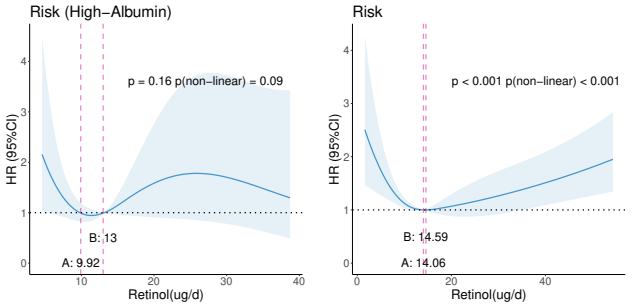


(d) All

Fig. 21: Appropriate Se intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

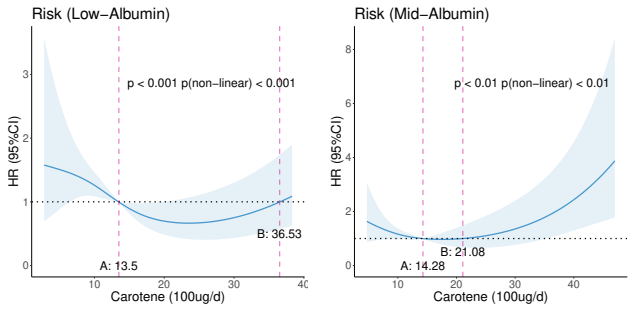


(a) Low (b) Medium

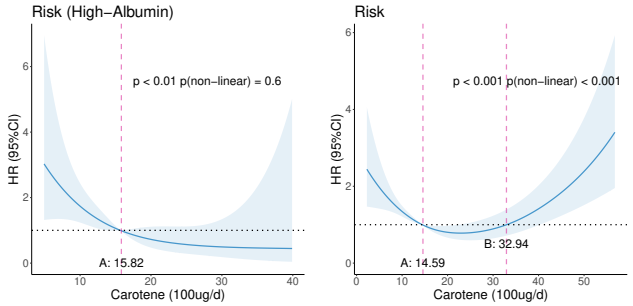


(c) High (d) All

Fig. 22: Appropriate retinol intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

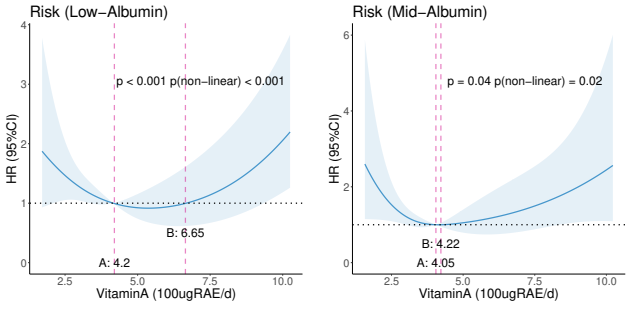


(a) Low (b) Medium

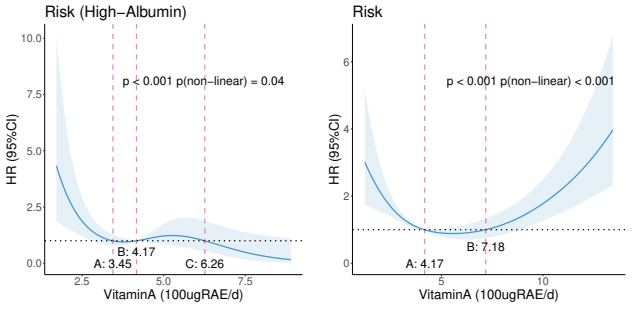


(c) High (d) All

Fig. 24: Appropriate carotene intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

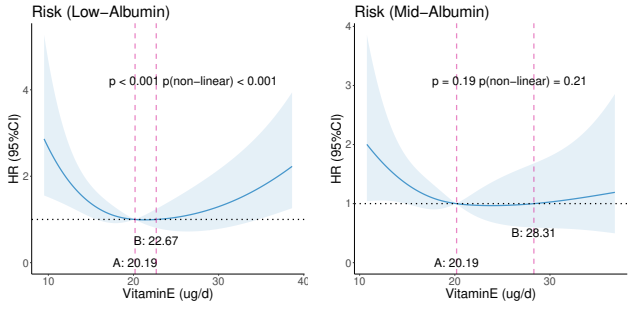


(a) Low (b) Medium

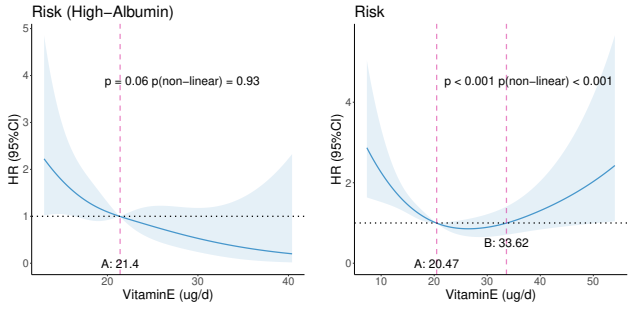


(c) High (d) All

Fig. 23: Appropriate vitaminA intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

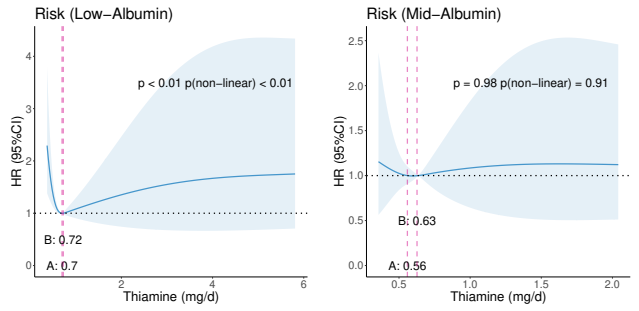


(a) Low (b) Medium

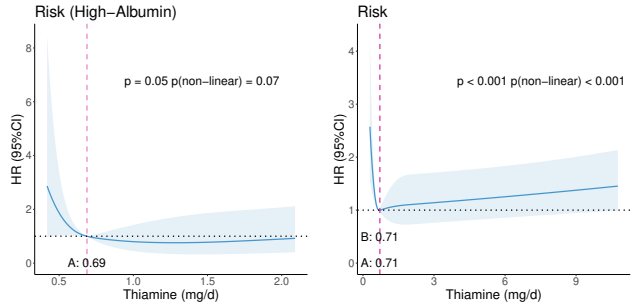


(c) High (d) All

Fig. 25: Appropriate vitaminE intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

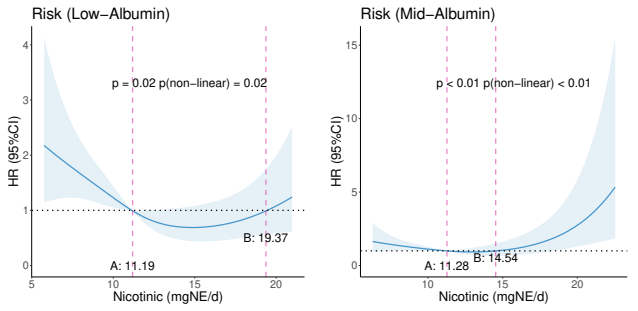


(a) Low (b) Medium

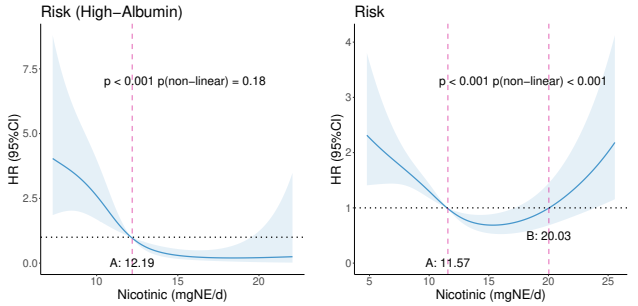


(c) High (d) All

Fig. 26: Appropriate thiamine intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

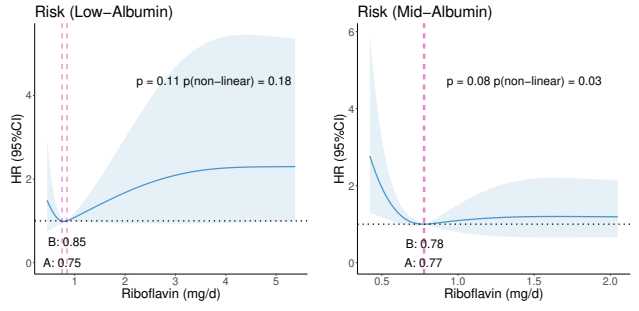


(a) Low (b) Medium

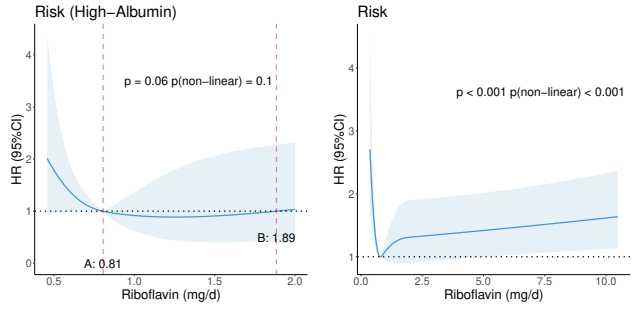


(c) High (d) All

Fig. 28: Appropriate nicotinic intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

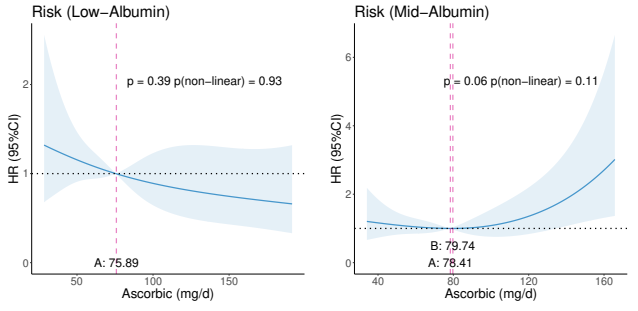


(a) Low (b) Medium

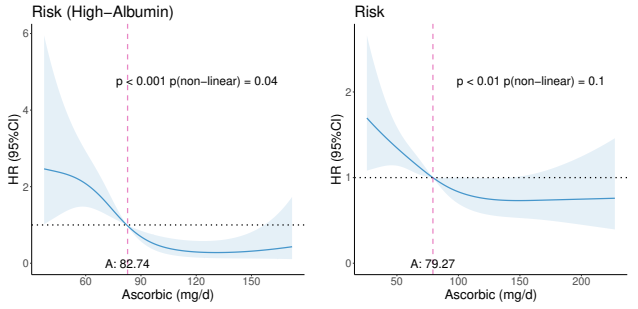


(c) High (d) All

Fig. 27: Appropriate riboflavin intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

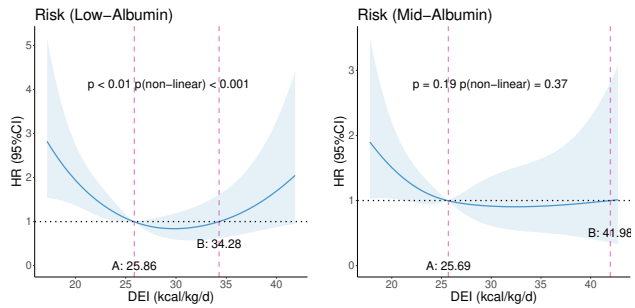


(a) Low (b) Medium



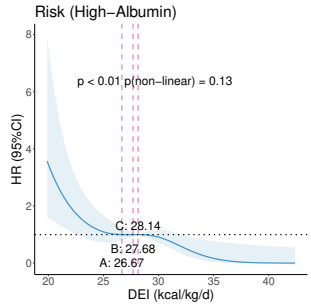
(c) High (d) All

Fig. 29: Appropriate ascorbic intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).

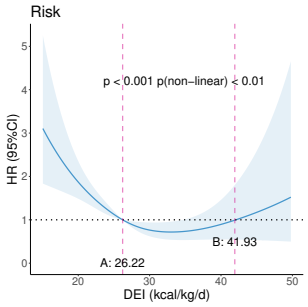


(a) Low

(b) Medium



(c) High



(d) All

Fig. 30: Appropriate DEI intake ranges associated with low mortality risk for patients with different albumin levels. Points A, B, and C define the ranges of dietary nutrition intake associated with low mortality risk, delineated as [A, B] and [C, -).