International Journal of General Practice Nursing, 2025, Volume 3, No Special
https://ojs.bbwpublisher.com/index.php/IJGPN
ISSN Online: 2981-9423
ISSN Print: 2981-9415

# A Nomogram for Predicting the Risk of Chronic Kidney Disease among Chinese Adults

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**Abstract:** *Objective:* By evaluating the associations between some factors and chronic kidney disease (CKD), a predictive nomogram is established to identify the high-risk population of CKD. *Methods:* A retrospective survey was conducted on the physical examination population in two hospitals in Shanghai and one hospital in Jiangsu Province from October 2022 to April 2023. Variables with *P*-values < 0.05 in the univariate analyses were selected as independent predictors for a multivariate logistic regression. The nomogram prediction model for the risk of CKD was established and validated using Stata15.0. *Results:* The prediction model in our study had the best explanatory power. In our prediction model, the influencing factors and hazard ratios were: age 1.85 (1.108–3.088), family history of hypertension 2.057 (1.248–3.392), family history of diabetes 2.675 (1.623–4.407), family history of CKD 4.142 (2.526–6.793), high-salt diet 3.814 (2.343–6.208), creatinine 1.996 (1.225–3.254), glucose 6.874 (4.129–11.443), triglyceride 4.104 (2.464–6.853), C-reactive protein 4.861 (2.817–8.387), and sodium 4.281 (2.617–7.003). The area under the curve values for the performance of the modeling and validation groups for the test nomogram were 0.9225 and 0.9466 and 0.9616, respectively. The Hosmer–Lemeshow test was: modeling set: ($\chi^2$ = 2.237, $P$ = 0.973); verification set (1): ($\chi^2$ = 5.380, $P$ = 0.716); verification set (2): ($\chi^2$ = 6.752, $P$ = 0.564). The nomogram calibration curves predicting the risk showed good consistency between the modeling and verification groups. *Conclusion:* A nomogram is established and verified internally and externally, which can help identify individuals with increased risk of CKD and provide a reference for the public and decision-makers to formulate primary prevention of CKD.

**Keywords:** Chronic kidney disease; Behavioral; Biochemical detection; Nomogram

**Online publication:** December 31, 2025

## 1. Introduction

Chronic kidney disease (CKD) refers to chronic kidney structure or dysfunction caused by many factors [1], which has the characteristics of high prevalence rate, low awareness rate, and high medical expenses [2]. In recent years, with the increasing aging of the world's population, the incidence and mortality of CKD have increased year by

year, which has become a global public health problem [3]. In 2020, according to the statistics of the World Health Organization, the global prevalence of CKD is 10.1–13.3%, surpassing diabetes, chronic obstructive pulmonary disease, depression, and other diseases [4]. The mortality rate rose from 8.13% in 2000 to 13% in 2019. It is estimated that it will become the fifth largest cause of death in the world in 2040 [5].

Studies in Australia and New Zealand show that the first-year survival rate of patients with CKD is 77%, and the five-year survival rate is only 23% [6]. Studies in Scotland show that the risk of acute kidney injury (AKI) in patients with CKD is twice that in patients without CKD [7]. In China, the incidence of CKD is increasing rapidly year by year, and the medical expenses are also increasing year by year [8]. Therefore, controlling risk factors can effectively reduce the morbidity and mortality of CKD.

In order to formulate and optimize the preventive strategy of CKD, it is necessary to understand and properly quantify the contribution of its key risk factors [9]. However, it is complicated to evaluate and manage the risk factors of CKD. Effective management measures for high-risk groups can reduce the exposure to risk factors and prevent high-risk groups from turning into CKD patients [10]. Such a strategy can optimize the allocation of health resources and make the limited resources play the greatest role. In addition, studies have shown that the levels of creatinine, glucose, triglyceride, cholesterol, C-reactive protein, and sodium in biochemical detection are related to the risk of CKD [11]. In the past, there were few discussions about biochemical detection indexes in prediction models, so it is necessary to include biochemical detection indexes in screening risk factors of CKD [12].

The ability to identify individuals at risk of CKD can prevent adverse health outcomes related to CKD. In addition, among the patients diagnosed with CKD, due to the lack of understanding of CKD and its management by clinical medical staff and the uncertainty of the potential risks of CKD progression, appropriate management may also be hindered. When patients enter the end-stage renal disease, they can only undergo renal replacement therapy to survive, which will have a huge economic burden on individuals, families, and society, and affect the quality of life of patients. The risk prediction model has unique clinical value in predicting the risk of disease. A simple risk assessment tool can help clinical medical staff quickly identify the risk of CKD and provide an estimate of the risk, so that better and more targeted monitoring strategies can be formulated, which is very meaningful for the prevention and control of CKD [13]. In this study, the high-risk factors of CKD will be screened out by multivariate regression analysis incorporating biochemical detection indicators, demographic, and behavioral factors. We build a risk prediction model and verify it internally and externally.

## 2. Materials and methods

### 2.1. Study design and participants

The data of two hospitals in Shanghai and one hospital in Jiangsu from October 2022 to April 2023 were retrospectively selected from the hospital information system (HIS). The participants of a hospital in Shanghai were used as the modeling set (internal verification group), and the participants of the other two hospitals were used as the verification set (external verification group). 1,297 participants were included in the model construction and verification (**Figure 1**), including 723 in the modeling set, 274 in the verification set (1), and 300 in the verification set (2).

The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Review Committee of the Shanghai University of Medicine and Health Sciences (No.2023-hxxm-01-612401197903300537). Informed consent was obtained from all participants in the study.

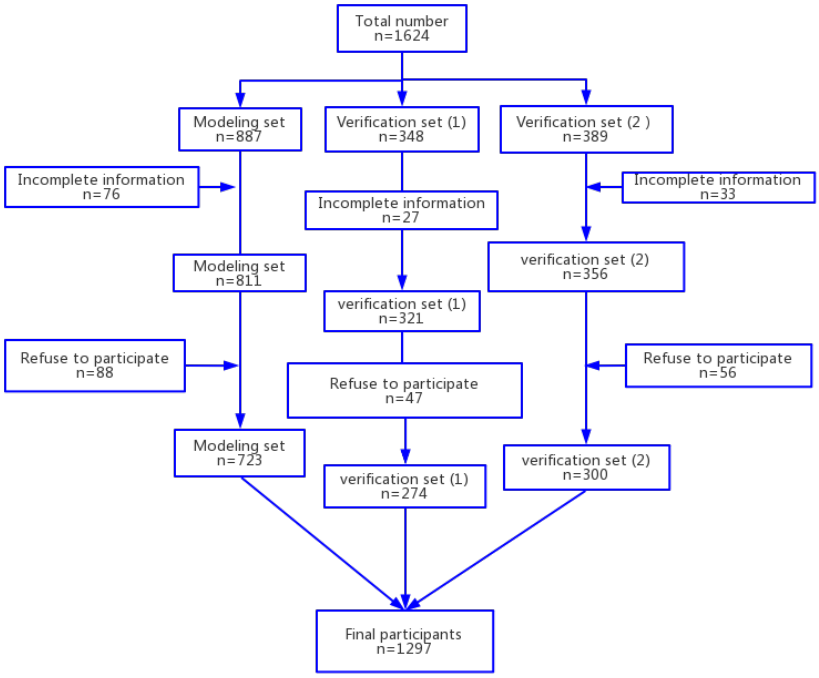


**Figure 1.** Flowchart of sampling method

## 2.2. Diagnostic criteria of CKD

The diagnosis of CKD was a persistent abnormality in kidney structure or function (e.g., glomerular filtration rate <60 ml/min/1.73 m2 or albuminuria ≥30 mg per 24 hours) for more than 3 months [14].

## 2.3. Predictors

We specified the most likely temporal associations between variables based on prior biological and epidemiologic knowledge and derived predictors, including demographic, behavioral, and biochemical detection factors [15]. Among potential predictors, the demographic variables [16] were age, marital status, years of education, etc. The behavioral variables [17] were living alone, exercise, smoking, alcohol, diet, sleep, etc. The biochemical detection variables [18] were creatinine, urea, uric acid, glucose, triglyceride, cholesterol, C-reactive protein, sodium, potassium, chloride, total protein, hemoglobin, and D-Dimer, etc.

## 2.4. Data analysis

The characteristic differences between the CKD and non-CKD groups were evaluated using the univariate logistic regression. Variables with *P*-values < 0.05 in the univariate analyses were introduced as independent predictors into a multivariate logistic regression. We estimated the strength of the association between CKD risk and predictors by OR and 95% CI. Significant variables were selected and used to construct a nomogram. The total score of the nomogram was classified by quartile to assess the association of the total score with CKD risk. The discriminative ability, predictive accuracy, and clinical application value of the model were assessed using a receiver operating characteristic (ROC) curve, calibration plot, and decision curve analysis. A total of 1,000 bootstrap resamples were employed to reduce overfitting bias. Statistical analyses were performed using SPSS version 25.0 and Stata 15.0 for Windows. Two-sided *P*-values < 0.05 were considered significant.

# 3. Results

## 3.1. Demographic analyses (Table 1)

A total of 1,297 participants were included in this study, including 723 people in modeling set, 274 people in verification set (1), and 300 participants in verification set (2). The age range was 18 to 94 (56.04 ± 14.88) years old. 358/1,297 (27.6%) of participants suffered from CKD, including 190 (26.3%) in the modeling set, 88 (28.5%) in the verification set (1), and 90 (30.0%) in the verification set (2). There was no significant difference in the detection rate of CKD between the modeling group and the two verification groups ($\chi 2 = 0.599$, $P = 0.450$).

**Table 1.** Demographic information [*n* (%)]

| Variables | Modeling set (*n* = 723) | | Verification set (1) (*n* = 274) | | Verification set (2) (*n* = 300) | |
|---|---|---|---|---|---|---|
| | Non-CKD | CKD | Non-CKD | CKD | Non-CKD | CKD |
| Gender | | | | | | |
| Male | 281 (75.70%) | 90 (24.30%) | 60 (77.90%) | 17 (22.10%) | 118 (79.20%) | 31 (20.80%) |
| Female | 252 (71.60%) | 100 (28.40%) | 136 (69.00%) | 61 (31.00%) | 92 (60.90%) | 59 (39.10%) |
| Age in years | | | | | | |
| ≤60 | 249 (83.30%) | 50 (16.70%) | 109 (82.60%) | 23 (17.40%) | 148 (90.20%) | 16 (9.80%) |
| >60 | 284 (67.00%) | 140 (33.00%) | 87 (61.30%) | 55 (38.70%) | 62 (45.60%) | 74 (54.40%) |
| Marital status | | | | | | |
| Married | 294 (72.80%) | 110 (27.20%) | 115 (76.70%) | 35 (23.30%) | 104 (68.90%) | 47 (31.10%) |
| Unmarried | 239 (74.90%) | 80 (25.10%) | 81 (65.30%) | 43 (34.70%) | 106 (71.10%) | 43 (28.90%) |
| Years of education | | | | | | |
| ≤6 | 231 (74.80%) | 78 (25.20%) | 82 (71.30%) | 33 (28.70%) | 93 (69.40%) | 41 (30.60%) |
| 7–12 | 165 (73.00%) | 61 (27.00%) | 61 (71.80%) | 24 (28.20%) | 60 (69.00%) | 27 (31.00%) |
| ≥12 | 137 (72.90%) | 51 (27.10%) | 53 (71.60%) | 21 (28.40%) | 57 (72.20%) | 22 (27.80%) |

## 3.2. Results of logistic regression analysis

In the modeling set, 35 risk factors were subjected to univariate analysis. The variables with statistically significant differences are shown in **Table 2**. Predictive factors with statistically significant differences ($P < 0.05$) in multivariate analysis were included in the model and expressed in a nomogram (**Figure 2**). Independent variable assignments are shown in **Table 3**.

**Table 2.** Results of logistic regression analysis

| Variables | Modeling set OR (95% CI) | | Verification set (1) OR (95% CI) | | Verification set (2) OR (95% CI) | |
|---|---|---|---|---|---|---|
| | Univariate analysis | Multivariate analysis | Univariate analysis | Multivariate analysis | Univariate analysis | Multivariate analysis |
| Age | 2.455 (1.704–3.536) | 1.85 (1.108–3.088) | 2.996 (1.707–5.257) | 4.079 (1.484–11.212) | 11.04 (5.961–20.449) | 8.19 (3.091–21.698) |
| Family history of hypertension | 2.446 (1.741–3.436) | 2.057 (1.248–3.392) | 2.945 (1.712–5.065) | 3.088 (1.263–7.549) | 3.548 (2.114–5.955) | 2.492 (1.011–6.14) |

**Table 2 (Continued)**

| Variables | Modeling set OR (95% CI) | | Verification set (1) OR (95% CI) | | Verification set (2) OR (95% CI) | |
|---|---|---|---|---|---|---|
| | Univariate analysis | Multivariate analysis | Univariate analysis | Multivariate analysis | Univariate analysis | Multivariate analysis |
| Family history of diabetes | 2.986 (2.104–4.236) | 2.675 (1.623–4.407) | 2.343 (1.37–4.007) | ------- | 2.807 (1.683–4.68) | 2.173 (0.858–5.506) |
| Family history of CKD | 3.590 (2.542–5.069) | 4.142 (2.526–6.793) | 3.150 (1.828–5.428) | 4.694 (1.839–11.98) | 3.421 (2.045–5.722) | 2.753 (1.095–6.923) |
| High-salt diet | 4.443 (3.124–6.319) | 3.814 (2.343–6.208) | 7.388 (4.05–13.478) | 8.55 (3.205–22.807) | 4.831 (2.835–8.232) | 4.223 (1.654–10.782) |
| Creatinine | 2.235 (1.595–3.130) | 1.996 (1.225–3.254) | 2.593 (1.484–4.529) | 2.818 (1.052–7.549) | 3.510 (2.095–5.881) | 3.204 (1.224–8.384) |
| Uric acid | 1.598 (1.145–2.231) | ------- | 4.300 (2.186–8.46) | ------- | 3.924 (2.228–6.911) | ------- |
| Glucose | 5.849 (4.069–8.407) | 6.874 (4.129–11.443) | 5.715 (3.155–10.355) | 9.260 (3.474–24.684) | 8.026 (4.529–14.222) | 8.161 (3.005–22.16) |
| Triglyceride | 5.898 (4.088–8.511) | 4.104 (2.464–6.835) | 4.210 (2.388–7.424) | ------- | 8.320 (4.752–14.566) | 5.723 (2.137–15.326) |
| Cholesterol | 1.722 (1.229–2.413) | ------- | 2.446 (1.429–4.186) | 7.576 (2.501–22.947) | ------- | 2.457 (0.884–6.825) |
| C-reactive protein | 4.810 (3.282–7.050) | 4.861 (2.817–8.387) | 2.516 (1.415–4.473) | 7.913 (2.565–24.409) | 2.798 (1.6–4.894) | 6.473 (2.125–19.717) |
| Sodium | 4.840 (3.403–6.886) | 4.281 (2.617–7.003) | 16.700 (8.777–31.774) | 18.232 (6.656–49.944) | 14.075 (7.619–26) | 11.137 (4.08–30.399) |

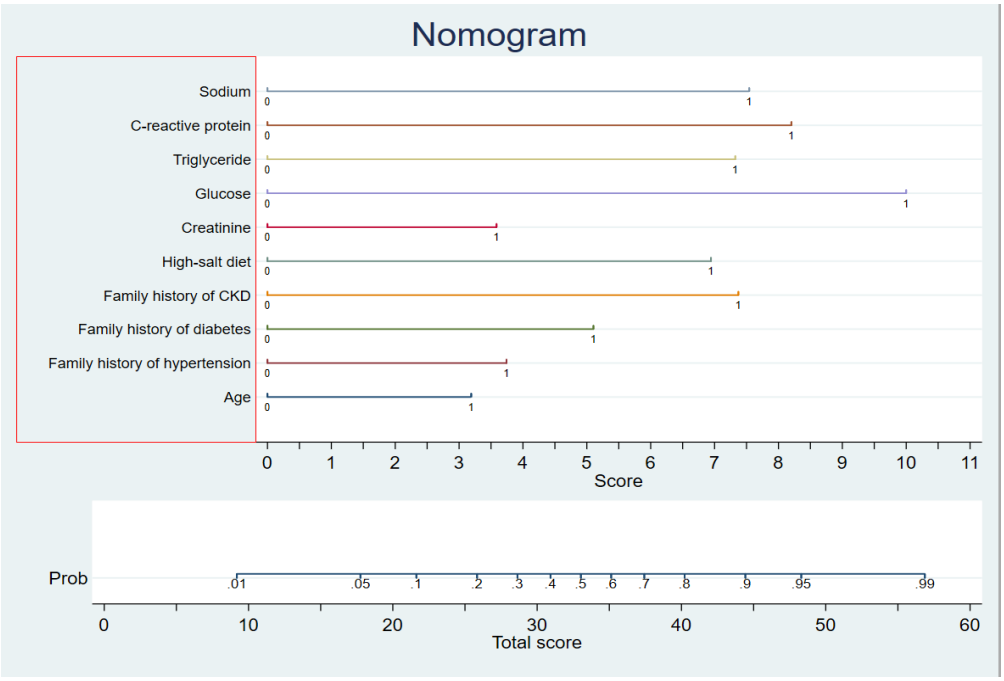


**Figure 2.** Nomogram of risk for CKD

**Table 3.** Independent variable assignment

| Variables | Evaluation | Variables | Evaluation |
|---|---|---|---|
| Age | ≤60 = 0, >60 = 1 | Uric acid | Normal = 0, Abnormal = 1 |
| Family history of hypertension | NO = 0, Yes = 1 | Glucose | Normal = 0, Abnormal = 1 |
| Family history of diabetes | NO = 0, Yes = 1 | Triglyceride | Normal = 0, Abnormal = 1 |
| Family history of CKD | NO = 0, Yes = 1 | Cholesterol | Normal = 0, Abnormal = 1 |
| High-salt diet | NO = 0, Yes = 1 | C-reactive protein | Normal = 0, Abnormal = 1 |
| Creatinine | Normal = 0, Abnormal = 1 | Sodium | Normal = 0, Abnormal = 1 |

## 3.3. Discrimination and calibration verification

Bootstrap method repeated sampling for 1,000 times, and internal verification C-index was 0.9225. The area under the curve values for the performance of the modeling and validation groups for the test nomogram were 0.9225 and 0.9466 and 0.9616, respectively [modeling set: Hosmer–Lemeshow test ($\chi2 = 2.237$, $P = 0.973$); verification set (1): Hosmer–Lemeshow test ($\chi2 = 5.380$, $P = 0.716$)]; verification set (2): Hosmer–Lemeshow test ($\chi2 = 6.752$, $P = 0.564$). The ROC curves are shown in **Figure 3**. The nomogram calibration curves predicting the risk of CKD showed good consistency between the modeling and verification group, the model has a good fitting degree (**Figure 4**).

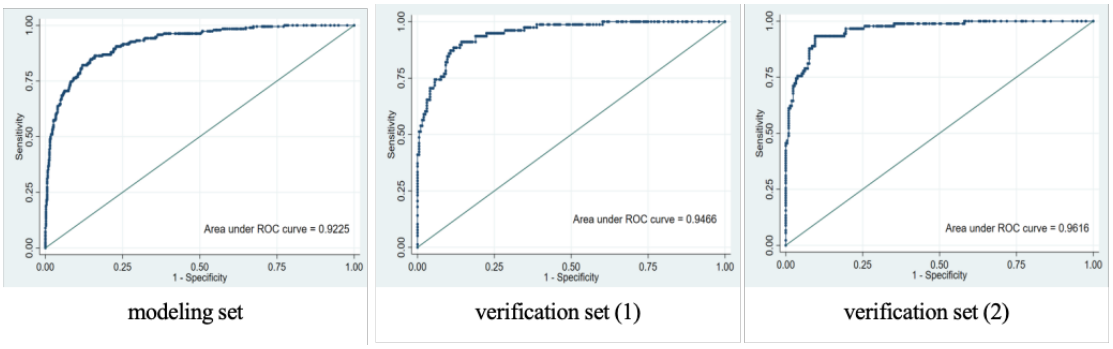


**Figure 3.** ROC curve

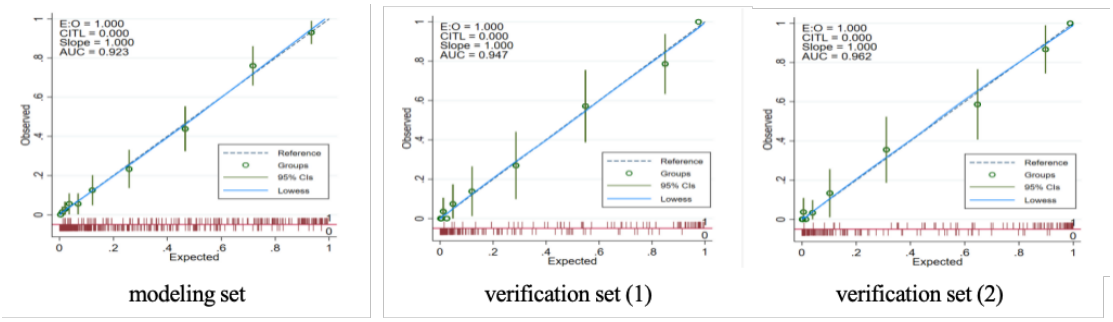


**Figure 4.** Calibration curve

## 3.4. Clinical application

The analysis of decision curves of the nomogram of the clinical application in the modeling and verification groups is shown in **Figure 5**. The DCA curve shows that the nomogram has a positive net benefit and a wide range of threshold probabilities.

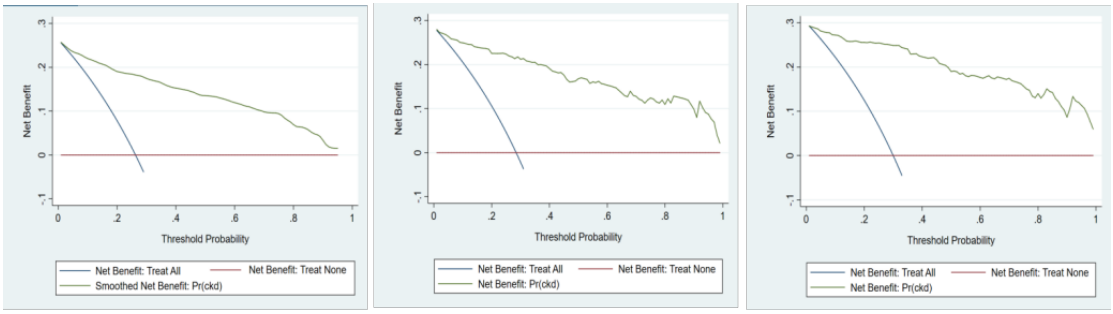


**Figure 5.** Decision curve

# 4. Discussion

## 4.1. Key findings

Our results illustrated the significant contributions of age, family history of hypertension, family history of diabetes, family history of CKD, high-salt diet, creatinine, glucose, triglyceride, C-reactive protein, and sodium to the risk of CKD. Using these variables, we have created an easy-to-use nomogram that includes ten variables, five of which are biochemical detection indicators, which are especially important for the prevention of CKD. Nomogram chart shows that the corresponding score of abnormal glucose is 10, which is the highest among the 10 risk factors, which is similar to the research result of Zhang *et al.* that diabetes is a high-risk factor for chronic kidney disease [19]. People with abnormal glucose are more prone to disorders of glucose and lipid metabolism, which suggests that we should strengthen the monitoring and intervention of abnormal glucose when abnormal glucose is found in the usual physical examination to reduce the risk of CKD development. The score corresponding to age is 3.25, which is the lowest among the 10 risk factors. Age is a non-modifiable risk factor for intervention. Even so, for non-modifiable variables, it is also beneficial for the public to be aware of CKD risk. That knowledge provides the public with the opportunity to voluntarily enhance early screening and promote secondary prevention [20]. Our research hopes to bring new hints to future model building, that is, biochemical detection factors can be considered in constructing CKD risk assessment models based on prospective studies.

## 4.2. Comparison with other studies

Compared with earlier studies, which focused on specific factors associated with CKD [20–22], we combined demographic, behavioral, and biochemical detection factors and found that the above 10 variables are related to high CKD risk. Among them, age, family history, and high-salt diet are basically consistent with the early reports [23]. In addition, family history of hypertension and diabetes is also a well-established risk factor for CKD [24]. We also found that people with a high-salt diet are prone to CKD. A high-salt diet can lead to fluid retention and excessive blood volume, which causes the risk of CKD, but its relationship with the progression of CKD is still unclear [25]. Some other studies have found that high-salt intake will inhibit the renin-angiotensin-aldosterone system (RAAS), thus reducing sodium reabsorption, helping to rebuild sodium and water homeostasis [26]. There are relatively few studies on biochemical detection indicators and the risk of CKD. We reported a hazard ratio of 2.235 (95% CI: 1.595–3.130) for the relation between creatinine and CKD risk, which is slightly higher than research by Chang *et al.* [27]. Research by Lin *et al.* found a high correlation between diabetes and CKD, but their research did not directly point out the relationship between glucose and CKD [28]. However, our research found that glucose has

a great correlation with the occurrence of CKD, which is of great significance to our research on the prevention of CKD. Experimental studies have shown that inflammatory cytokines can increase oxidative stress and trigger sympathetic nerve activity, thus increasing the risk of CKD [29]. Our findings point out that the risk of CKD in people with abnormal C-reactive protein is 4.861 times higher than that in people with normal C-reactive protein. The above findings have important practical significance for our early prevention of CKD. At the same time, our study has expanded its scope by providing data on biochemical detection factors.

### 4.3. Implications for clinicians and policymakers

A well-validated tool for predicting disease risk is important for appropriate clinical care, especially for primary prevention [30]. As part of the advances made in CKD prevention and management, the Robert prediction algorithm has been widely used to estimate CKD risk [31]. However, in some populations, including the Chinese, this algorithm would overestimate the risk of CKD [32]. This highlights the need to establish an effective CKD risk assessment tool in China. In addition, several recent studies have used nomograms to evaluate the risk of complications in CKD patients according to various test results, including the risk of fracture and cardiovascular disease in CKD patients [33,34]. These studies provide strong clinical evidence for the risk of other diseases caused by CKD patients, but they are ineffective in identifying and evaluating the factors that can change the risk of CKD, which is more important for primary prevention [35]. Our study, carried out in Shanghai and Jiangsu, China, identified ten variables based on a logistic regression model and constructed a nomogram risk prediction model for practical application. The validation results for the nomogram show that the model has good predictive ability and clinical application value. As these factors are readily available, our risk assessment model could potentially be widely accepted.

### 4.4. Strengths and limitations

A key strength of this study was our ability to simultaneously evaluate a wide range of predictors, including demographic, behavioral, and biochemical detection factors. In addition, previous studies have confirmed that substantial mitigation of CKD prevention could be achieved in clinical practice through improvements in modifiable risk factors [36,37]. Participants in our study were selected from three hospitals in two cities, Shanghai and Jiangsu, and we used the data from two other hospitals as the validation set, which makes our model more convincing. Although the construction effect of the prediction model is satisfactory, there are still some defects. The sample of this study only comes from three hospitals in two regions, and its application in the country or internationally requires the participation of participants from more regions. In addition, demographic, behavioral, and biochemical detection factors are numerous. Although we took 35 variables into account, that does not cover everything.

## 5. Conclusion

This analysis shows that age, family history of hypertension, family history of diabetes, family history of CKD, high-salt diet, creatinine, uric acid, glucose, triglyceride, cholesterol, C-reactive protein, and sodium are associated with high CKD risk. We developed a user-friendly nomogram that could potentially be of benefit to the public and to policymakers in formulating effective strategies for CKD risk assessment and primary prevention.

## Disclosure statement

The authors declare no conflict of interest.

## References


[1] Liu SY, Huang M, Chen R, et al., 2019, Comparison of Strategies for Setting Intervention Thresholds for Chinese Postmenopausal Women Using the FRAX Model. Endocrine, 65(1): 200–206.

[2] Abeysekera RA, Healy HG, Wang Z, et al., 2021, Heterogeneity in Patterns of Progression of Chronic Kidney Disease. Intern Med J, 51(2): 220–228.

[3] Stevens PE, Levin A, 2013, Evaluation and Management of Chronic Kidney Disease: Synopsis of the Kidney Disease: Improving Global Outcomes 2012 Clinical Practice Guideline. Ann Intern Med, 158(11): 825–830.

[4] Virani SS, Alonso A, Benjamin EJ, et al., 2020, Heart Disease and Stroke Statistics-2020 Update: A Report From the American Heart Association. Circulation, 141(9): e139–e596.

[5] Sung H, Ferlay J, Siegel RL, et al., 2021, Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA Cancer J Clin, 71(3): 209–249.

[6] Scott T, Ethier I, Hawley C, et al., 2021, Burden of Kidney Failure from Atheroembolic Disease and Association with Survival in People Receiving Dialysis in Australia and New Zealand: A Multi-Centre Registry Study. BMC Nephrol, 22(1): 401.

[7] Saad RA, Qutob HM, 2022, The Relationship Between Anemia and Obesity. Expert Rev Hematol, 15(10): 911–926.

[8] Wang L, Xu X, Zhang M, et al., 2023, Prevalence of Chronic Kidney Disease in China: Results From the Sixth China Chronic Disease and Risk Factor Surveillance. JAMA Intern Med, 183(4): 298–310.

[9] Haim-Pinhas H, Yoskovitz G, Lishner M, et al., 2022, Effect of Aspirin on Primary Prevention of Cardiovascular Disease and Mortality among Patients with Chronic Kidney Disease. Sci Rep, 12(1): 17788.

[10] Gallagher H, Dumbleton J, Maishman T, et al., 2022, Aspirin to Target Arterial Events in Chronic Kidney Disease (ATTACK): Study Protocol for a Multicentre, Prospective, Randomised, Open-Label, Blinded Endpoint, Parallel Group Trial of Low-Dose Aspirin vs. Standard Care for the Primary Prevention of Cardiovascular Disease in People with Chronic Kidney Disease. Trials, 23(1): 331.

[11] Li Y, Li D, Song Y, et al., 2020, Visit-to-Visit Variability in Blood Pressure and the Development of Chronic Kidney Disease in Treated General Hypertensive Patients. Nephrol Dial Transplant, 35(10): 1739–1746.

[12] Yang L, Li J, Wei W, et al., 2023, Blood Pressure Variability and the Progression of Chronic Kidney Disease: A Systematic Review and Meta-Analysis. J Gen Intern Med, 38(5): 1272–1281.

[13] Zhang L, Tang L, Chen S, et al., 2023, A Nomogram for Predicting the 4-Year Risk of Chronic Kidney Disease among Chinese Elderly Adults. Int Urol Nephrol, 55(6): 1609–1617.

[14] Wilson S, Mone P, Jankauskas SS, et al., 2021, Chronic Kidney Disease: Definition, Updated Epidemiology, Staging, and Mechanisms of Increased Cardiovascular Risk. J Clin Hypertens (Greenwich), 23(4): 831–834.

[15] Chatzipetrou V, Begin MJ, Hars M, et al., 2022, Sarcopenia in Chronic Kidney Disease: A Scoping Review of Prevalence, Risk Factors, Association with Outcomes, and Treatment. Calcif Tissue Int, 110(1): 1–31.

[16] Levine KE, Redmon JH, Elledge MF, et al., 2016, Quest to Identify Geochemical Risk Factors Associated with Chronic Kidney Disease of Unknown Etiology (CKDu) in an Endemic Region of Sri Lanka-A Multimedia Laboratory Analysis of Biological, Food, and Environmental Samples. Environ Monit Assess, 188(10): 548.

[17] Pry JM, Jackson W, Rupasinghe R, et al., 2021, A Pilot Case-Control Study Using a One Health Approach to Evaluate Behavioral, Environmental, and Occupational Risk Factors for Chronic Kidney Disease of Unknown Etiology in Sri

Lanka. One Health Outlook, 3: 4.

[18] Chang J, Hou W, Li Y, et al., 2022, Prevalence and Associated Factors of Cognitive Frailty in Older Patients with Chronic Kidney Disease: A Cross-Sectional Study. BMC Geriatr, 22(1): 681.

[19] Zhang X, Fang Y, Zou Z, et al., 2022, Risk Factors for Progression of CKD with and without Diabetes. J Diabetes Res, 2022: 9613062.

[20] Guo D, Wang H, Liu J, et al., 2022, Prediction of Chronic Kidney Disease After Orthotopic Liver Transplantation: Development and Validation of a Nomogram Model. BMC Nephrol, 23(1): 33.

[21] Lin Y, Shao H, Fonseca V, et al., 2023, A Prediction Model on Incident Chronic Kidney Disease among Individuals with Type 2 Diabetes in the United States. Diabetes Obes Metab, 2023.

[22] Shen H, van der Kleij R, van der Boog P, et al., 2020, Development and Evaluation of an eHealth Self-Management Intervention for Patients with Chronic Kidney Disease in China: Protocol for a Mixed-Method Hybrid Type 2 Trial. BMC Nephrol, 21(1): 495.

[23] Nerbass FB, Pecoits-Filho R, McIntyre NJ, et al., 2015, High Sodium Intake is Associated with Important Risk Factors in a Large Cohort of Chronic Kidney Disease Patients. Eur J Clin Nutr, 69(7): 786–790.

[24] Al-Shamsi S, Oulhaj A, Regmi D, et al., 2019, Use of Estimated Glomerular Filtration Rate to Predict Incident Chronic Kidney Disease in Patients at Risk of Cardiovascular Disease: A Retrospective Study. BMC Nephrol, 20(1): 325.

[25] Borrelli S, Provenzano M, Gagliardi I, et al., 2020, Sodium Intake and Chronic Kidney Disease. Int J Mol Sci, 21(13): 4744.

[26] Teo BW, Chan GC, Leo C, et al., 2021, Hypertension and Chronic Kidney Disease in Asian Populations. J Clin Hypertens (Greenwich), 23(3): 475–480.

[27] Chang HL, Wu CC, Lee SP, et al., 2019, A Predictive Model for Progression of CKD. Medicine (Baltimore), 98(26): e16186.

[28] Lin Y, Shao H, Fonseca V, et al., 2023, A Prediction Model of CKD Progression among Individuals with Type 2 Diabetes in the United States. J Diabetes Complications, 37(3): 108413.

[29] Hodson EM, Cooper TE, 2023, Altered Dietary Salt Intake for Preventing Diabetic Kidney Disease and Its Progression. Cochrane Database Syst Rev, 1(1): D6763.

[30] Lee SM, Kim SH, Yoon HJ, 2023, Prediction of Incident Chronic Kidney Disease in a Population with Normal Renal Function and Normo-Proteinuria. PLoS One, 18(5): e285102.

[31] Nelson RG, Grams ME, Ballew SH, et al., 2019, Development of Risk Prediction Equations for Incident Chronic Kidney Disease. JAMA, 322(21): 2104–2114.

[32] Suh SH, Song SH, Oh TR, et al., 2021, Association of Urinary Potassium Excretion with Blood Pressure Variability and Cardiovascular Outcomes in Patients with Pre-Dialysis Chronic Kidney Disease. Nutrients, 13(12): 4443.

[33] Li X, Lindholm B, 2022, Cardiovascular Risk Prediction in Chronic Kidney Disease. Am J Nephrol, 53(10): 730–739.

[34] Whitlock RH, Leslie WD, Shaw J, et al., 2019, The Fracture Risk Assessment Tool (FRAX(R)) Predicts Fracture Risk in Patients with Chronic Kidney Disease. Kidney Int, 95(2): 447–454.

[35] Pallikadavath S, Ashton L, Brunskill NJ, et al., 2022, Aspirin for the Primary Prevention of Cardiovascular Disease in Individuals with Chronic Kidney Disease: A Systematic Review and Meta-Analysis. Eur J Prev Cardiol, 28(17): 1953–1960.

[36] Roshanov PS, Garg AX, 2023, TIPS to Decide Whether to Prescribe Aspirin for the Primary Prevention of

Cardiovascular Events in Chronic Kidney Disease. Kidney Int, 103(2): 261–263.

[37] Major RW, Burton JO, 2021, “To Take or Not to Take an Aspirin?” The Age-Old Question of Cardiovascular Disease Primary Prevention for People with Chronic Kidney Disease. Kidney Int, 99(2): 308–310.

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