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Metabolic risk factors, blood pressure and atherogenic indices of cardiovascular disease across different quartiles of dietary sodium to potassium ratio among young semi-professional athletes with overweight or obesity

Yisi Liu[1*], Xin Wu[1] and Liwei Sun[1]

Abstract

Background Previous studies have revealed the role of the dietary sodium-to-potassium ratio in predicting cardiovascular and total mortality. However, a limited number of studies have investigated the association between the dietary sodium-to-potassium ratio and individual biomarkers of metabolic disease in athletes. In this study, we aimed to compare metabolic, atherogenic, and anthropometric risk factors for cardiovascular disease across different quartiles of the dietary sodium-to-potassium ratio among young semi-professional athletes with overweight or obesity.

Methods In this cross-sectional study, 637 young semi-professional athletes, aged 20–40 years, were selected from active athletic and fitness clubs. Anthropometric measurements were performed, and fasting blood sugar, serum lipids, and lipoprotein (a) [Lp(a)] were measured. Atherogenic indices, including the atherogenic index of plasma (AIP), Castelli risk index (CRI)-I, CRI-II, and lipid tetrad index (LTI), were calculated.

Results Participants in the highest quartile of the dietary sodium-to-potassium ratio were younger and had a higher waist-to-hip ratio (WHR) compared to those in the lowest quartile. Additionally, those in the highest quartile showed significantly higher serum total cholesterol and Lp(a) concentrations after adjusting for confounding factors ($P < 0.05$).

Conclusion Our findings suggest that individuals in the higher quartiles of the dietary sodium-to-potassium ratio exhibit less favorable serum lipid profiles. Further longitudinal and interventional studies are needed to clarify the potential therapeutic role of reducing the dietary sodium-to-potassium ratio in managing cardiovascular disease risk factors.

Keywords Dietary sodium to potassium ratio, Metabolic risk factors, Serum lipids, Lipoprotein (a), Lipid atherogenic indices, Castelli risk index

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Introduction

Cardiovascular diseases (CVDs) are the leading cause of mortality worldwide, responsible for an estimated 17.9 million deaths annually, according to a WHO report [1]. Key risk factors for CVDs include high blood pressure, an unhealthy diet, elevated cholesterol levels, and obesity [2]. The World Heart Federation reported that the leading modifiable risk factor for CVD-related deaths in 2021 was high blood pressure, accounting for 10.8 million deaths globally. This was followed by elevated LDL cholesterol, high fasting blood glucose, and high body mass index, which contributed to over 3.8 million, 2.3 million, and 2 million deaths worldwide, respectively [2]. Behavioral factors, such as a sedentary lifestyle, unhealthy dietary habits, and tobacco and alcohol consumption, are among the major contributors to elevated blood pressure, obesity, and dyslipidemia [3].

Recent epidemiological studies have shown that athletes, despite their high levels of physical activity, are not immune to cardiovascular disease (CVD). Several factors contribute to the development of CVD in athletes, including genetic predisposition, unhealthy dietary habits, the use of supplements, and excessive exercise-induced stress on the cardiovascular system [4]. "A career in sports does not eliminate the risk of cardiovascular disease," as demonstrated by a recent systematic review and meta-analysis [5].

The Oxford Dictionary defines an athlete as a person who competes in one or more sports requiring physical strength, speed, power, or endurance [6], Semi-professional athletes, on the other hand, are individuals who participate in sports at a competitive level while balancing other professional commitments. These athletes typically receive some financial compensation for their sports involvement but do not rely on it as their primary source of income. Semi-professional athletes are characterized by their dual commitment to both athletics and professional careers, with diverse sports backgrounds such as soccer, cycling, and mountain biking. They are also highly engaged in training, measured in metabolic equivalents (MET-min/week), which vary according to the specific sport and training regimen [7–9].

Numerous studies have highlighted the importance of proper nutritional status for the physical performance and post-exercise recovery of semi-professional athletes [10–12]. However, the dietary intake of semi-professional athletes often falls short of sports nutrition recommendations [10, 11]. These athletes are also characterized by elevated levels of oxidative molecules, free radicals, and upregulation of antioxidant systems [13]. The increase in free radicals can reduce muscle force by inhibiting the sodium–potassium ATPase pump [14], suppressing sarcoplasmic reticulum calcium-ATPase activity [15], and decreasing myofibrillar calcium sensitivity [15]. Dietary sodium and potassium play crucial roles in regulating fluid balance and neuromuscular function, directly impacting athletic performance [16]. Maintaining an optimal sodium-to-potassium ratio can improve endurance, strength, and exercise capacity, allowing athletes to perform at their peak [17]. An appropriate sodium-to-potassium ratio, which enhances dietary acid load, is essential for preventing dehydration, heat-related illnesses, and maintaining cardiovascular health, especially in blood pressure regulation and the prevention of cardiovascular disease. While high sodium intake is linked to hypertension, endothelial dysfunction, and an increased risk of cardiovascular events, adequate potassium intake has antihypertensive effects, promoting vasodilation, sodium excretion, and arterial compliance [18, 19].

Previous studies have primarily focused on the role of dietary sodium and potassium in controlling hypertension and reducing the risk of cardiovascular disease (CVD). Numerous observational and interventional studies have demonstrated the negative impact of high dietary sodium intake on blood pressure and CVD [20–23]. In contrast, potassium intake has been shown to have beneficial effects on both blood pressure and hypertension management [22, 24].

The dietary sodium-to-potassium ratio is considered more important than the individual intake of these two elements. The ideal ratio is approximately 1:3, meaning potassium intake should ideally be around three times that of sodium. The World Health Organization (WHO) recommends a daily intake of less than 2000 mg of sodium and more than 3510 mg of potassium, with a sodium-to-potassium ratio of less than 1.0 being optimal for cardiovascular health [25]. However, due to low compliance with dietary guidelines and recent findings suggesting that a sodium-to-potassium ratio between 1.0 and 2.0 can also reduce the risk of cardiovascular disease, a ratio of less than 2.0 has been proposed as a more attainable suboptimal goal [26, 27].

Recent studies have examined the relationship between dietary sodium and potassium balance and cardiovascular disease (CVD) [28–30], highlighting the impact of the dietary sodium-to-potassium ratio on blood pressure and hypertension [29]. A 24-year follow-up study conducted in Japan found that the dietary sodium-to-potassium ratio was an independent and significant risk factor for all-cause mortality, as well as mortality from stroke and CVD, among 8283 men and women aged 30–79 years who had no prior history of hypertensive treatment, stroke, or acute myocardial infarction [30]. Another longitudinal study involving 2050 men and women aged 30–84 years, who were free of CVD at baseline, showed that a higher dietary sodium-to-potassium ratio was associated with an increased risk of CVD after a median follow-up of 10.6 years [hazard ratio (HR) = 1.99, 95% CI = 1.13–3.52] [31]. Conversely, some studies have reported no association between the dietary sodium-to-potassium ratio and all-cause or CVD mortality [23, 32] while others have found a positive association between the urinary sodium-to-potassium ratio and all-cause or CVD mortality [23].

Most studies on the health effects of the dietary sodium-to-potassium ratio have primarily focused on its association with blood pressure and have evaluated outcomes such as all-cause mortality, cardiovascular disease (CVD), and stroke. Few studies have examined the role of the sodium-to-potassium ratio in isolated metabolic risk factors, such as serum lipids, glycemic markers, or atherogenic indices. For example, a retrospective cross-sectional study using pooled data from the National Health and Nutrition Examination Survey (NHANES) found that a higher dietary sodium-to-potassium ratio was associated with increased odds of elevated HbA1c concentrations in men (OR: 1.56, 95% CI: 1.24–1.96). Additionally, higher odds of low HDL-cholesterol were observed in the highest quintiles of the dietary sodium-to-potassium ratio for both men (*P* for trend = 0.002) and women (*P* for trend < 0.001). However, no significant associations were found between the dietary sodium-to-potassium ratio and hypercholesterolemia in men, or high non-HDL-cholesterol levels in either gender [28].

Another study conducted in Korea [33], found that individuals in the highest quartile of the dietary sodium-to-potassium ratio had a 19% higher odds of having metabolic syndrome compared to those in the lowest quartile (*P* for trend = 0.001). This study also reported a significant increase in the risk of elevated blood pressure and elevated blood sugar associated with a higher dietary sodium-to-potassium ratio (P for trend = 0.015 and 0.012, respectively). Similarly, a study in China found a positive association between the dietary sodium-to-potassium ratio and both high blood pressure and hypertriacylglycerolemia [34].

Obesity is an independent risk factor for cardiovascular disease, with established roles in increasing blood pressure, inducing dyslipidemia, and promoting atherogenesis [35, 36]. In a study by Geleijnse JM et al. [23], the urinary sodium-to-potassium ratio was associated with an increased risk of hypertension specifically in overweight individuals who were initially free of CVD (RR = 1.19, 95% CI: 1.02–1.39). Additionally, other studies have reported a positive association between the urinary sodium-to-potassium ratio and obesity [37, 38].

Increased dietary sodium intake is also associated with a higher risk of both central and general obesity; the prevalence of obesity among athletes appears to mirror that of the general population. One study found that adult athletes in the United States were at least 3.1 times more likely to be overweight or obese compared to their non-US counterparts [39]. Additionally, obesity rates in China have surged in recent decades, with more than half of the country's 1.4 billion population being overweight [40]. Projections suggest that by 2030, nearly 70% of the Chinese population could be overweight [41].

Due to the limited number of studies examining the role of the dietary sodium-to-potassium ratio in metabolic risk factors for cardiovascular disease, and the lack of research in the field of sports nutrition, this cross-sectional study aims to address this gap. For the first time, we compare metabolic risk factors—including serum lipids, glycemic markers, blood pressure, and atherogenic indices—across different quartiles of the dietary sodium-to-potassium ratio among young semi-professional athletes with overweight or obesity. The identification of semi-professional athletes in this study involved input from team managers, head coaches, and medical professionals.

Methods and materials

Study design, setting and participants

In this study, 637 young semi-professional athletes aged 20–40 years were selected from active athletic and fitness clubs in Xi'an, the capital of Shaanxi Province in Northwest China. The clubs were chosen through cluster random sampling from 11 districts in Xi'an, based on differences in socio-demographic levels. Within each district, athletic and fitness clubs were selected using simple random sampling. Participants were then enrolled through a convenience sampling procedure from these clubs. The inclusion criteria for participants were: aged 20–40 years, residing in Xi'an, having a healthy appearance, being overweight or obese (BMI 25.9 – 39.9 kg/m^2), and consenting to participate in the study (Table 1, Fig. 1). Athletes were included if they had engaged in consistent exercise for at least 3 years, training at least 3 days per week for a minimum of 90 min per session [42]. Individuals were excluded if they had a history of cardiovascular diseases, diabetes, cancer, liver or kidney problems, or if they used any medications affecting electrolyte homeostasis and body metabolism. Additionally,

Table 1 Frequency distribution of study participants by sport type

Sport type	N	Percentage
Basketball	80	12.55
Volleyball	102	16.01
Running	109	17.11
Wrestling	117	18.36
Swimming	69	10.83
Other sports (e.g. badminton, Ping-Pong, fitness, …)	55	8.63
Total	637	100

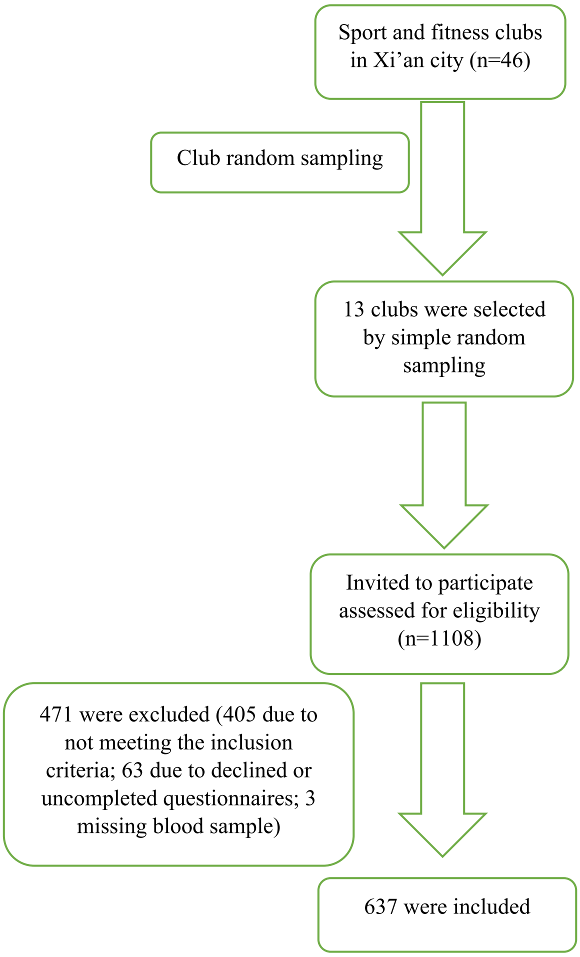

Fig. 1 Study flowchart

those using supplements that could alter weight or electrolyte levels were also excluded from the study.

Assessment of variables

Participants' general demographic information was collected using a questionnaire that included details about age, sex, personal and family medical history, and medication use. Physical activity was assessed using simplified Chinese-character version of the international physical activity questionnaire-long form (IPAQ) [43]. Height and weight were measured with a wall-mounted stadiometer and a Seca scale (Seca Co., Hamburg, Germany), with precision to the nearest 0.5 cm and 0.1 kg, respectively. Body mass index (BMI) was calculated as weight (kg) divided by height (m^2). Waist circumference was measured to the nearest 0.1 cm at the midpoint between the lowest point of the ribs and the iliac crest. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured using a calibrated mercury sphygmomanometer (Omron, Japan) on the same arm, following a minimum of 15 min of rest. The average of two readings was recorded. Biochemical variables, including serum lipids (e.g., triglycerides (TG), total cholesterol (TC), and high-density lipoprotein cholesterol (HDL-C)), fasting serum glucose, and serum lipoprotein (Lp) a, were measured after an overnight fast of 12 h. Serum samples were analyzed using an auto-analyzer (Alpha Classic E analyzer), and LDL-C was calculated using Friedewald's formula [44]. Dietary intake was assessed using a validated food frequency questionnaire (FFQ) for the Chinese population [45]. Participants provided information on the frequency and quantity of each food item consumed, which was used to evaluate their annual food intake. Food amounts were converted to grams using the Chinese Food Image Database and Sheppard's basic recommendations for portion sizes, cooking yields, and edible quantities [46]. The NUTRITIONIST IV software (N Squared Computing, California, USA) was used to analyze daily dietary consumption, including calories, macronutrients, micronutrients, and the mean intake of sodium (Na) and potassium (K) over the past year. The sodium-to-potassium ratio was then calculated.

In the current study, atherogenic indices of plasma were calculated as follows:

- Atherogenic Index of Plasma (AIP): Log (serum triglycerides divided by serum HDL cholesterol)
- Catelli Risk Index (CRI)-I**:** Serum total cholesterol divided by serum HDL cholesterol
- Catelli Risk Index (CRI)-II: Serum LDL cholesterol divided by serum HDL cholesterol
- Lipid Tetrad Index (LTI): Serum cholesterol × triglycerides × lipoprotein (Lp) (a) divided by serum HDL cholesterol [47, 48].

Sample size and grouping variable

Sample size calculation for the current study was performed using the formula $n = \frac{(\mathrm{Za}/2)^2 \mathrm{p}(1-\mathrm{p})}{\mathrm{d}^2}$, where the prevalence of cardiovascular events with a higher dietary sodium-to-potassium ratio was estimated at 0.16 [49] with an error coefficient of $d = 0.04$ and a significance level of $\alpha = 0.05$. This calculation resulted in an approximate sample size of 579. Considering a 10% dropout rate, the final estimated sample size was 637 participants. The dietary sodium-to-potassium ratio among participants ranged from 0.25 to 3.88, and they were divided into four groups based on this ratio: low (< 0.78), medium (0.78–1.06), high (1.06–1.45), and very high (> 1.45).

Statistical analysis

Continuous and discrete variables are presented as mean ± SD or n (%), respectively. Data normality was assessed using the Kolmogorov–Smirnov test, and Levene's test was used to evaluate the equality of variances. Comparisons of continuous and discrete variables across quartiles of the dietary sodium-to-potassium ratio were conducted using one-way analysis of variance (ANOVA) with Tukey's post-hoc tests and chi-square tests, respectively. For dietary data with non-normal distribution, between-quartile comparisons were performed using the Kruskal–Wallis test. Trend analysis was conducted with polynomial linear contrast analysis. Adjustments for confounders were made using the General Linear Model (GLM) approach. Polynomial logistic regression analysis was performed in three models: Model I (crude), Model II (adjusted for age and sex), and Model III (adjusted for age, sex, BMI, physical activity, and energy intake) to evaluate the linear trend association between the dietary sodium-to-potassium ratio and biochemical outcomes. Partial correlation analysis was used to assess the linear association between dietary sodium and potassium intake and serum lipoprotein (Lp)(a). *P*-values less than 0.05 were considered significant. Data analysis was conducted using SPSS software (version 23; IBM Corp., Armonk, NY, USA).

Results

Individuals with a higher dietary sodium-to-potassium ratio were younger and had a higher waist-to-hip ratio (WHR) compared to those with a lower sodium-to-potassium ratio (Table 2; $P < 0.05$). No significant differences were observed in other demographic or anthropometric variables across the quartiles of dietary sodium-to-potassium ratio. Crude and energy-adjusted differences in dietary intake of various food groups are presented in Table 3. As indicated, significantly higher intakes of grains, organ meats, red meat, eggs, cholesterol, total fiber, and table salt were found in the higher quartiles compared to the lower quartiles of the sodium-to-potassium ratio ($P < 0.05$). Conversely, lower intakes of fish, low- and high-fat dairy products, fruits, vegetables, nuts, poultry, and sweets were noted in the highest versus lowest quartiles of the sodium-to-potassium ratio in both crude and energy-adjusted models ($P < 0.05$). Among beverages, those in the highest quartile had a higher intake of soft drinks and a lower intake of both low- and high-fat milk ($P < 0.001$). Table 4 presents a comparison of fasting blood sugar (FBS), metabolic, and atherogenic risk factors across quartiles of the dietary sodium-to-potassium ratio. Individuals in the second and third quartiles had significantly higher serum total cholesterol and Lp (a) concentrations compared to those in the other quartiles in both crude and adjusted models (adjusted for age, sex, BMI, energy intake, and physical activity; $P < 0.05$). Serum triglycerides (TG), the atherogenic index of plasma (AIP), and diastolic blood pressure (DBP) were higher in the second and first quartiles, respectively, compared to the other quartiles in the crude model. Systolic blood pressure (SBP) was higher in the first quartile compared to others in both crude and adjusted models. A significant trend was observed for serum TG, SBP, DBP,

Table 2 General demographic and anthropometric characteristics of study population across quartiles of dietary Na/K ratio

Variable	All participants (*n* = 637)		Quartils of dietary Na/K								*P* Value*
			Q_1 (*n* = 159)		Q_2 (*n* = 160)		Q_3 (*n* = 160)		Q_4 (*n* = 158)		
			(< 0.78)		(0.78 -1.06)		(1.06–1.45)		(> 1.45)		
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	
Age (y)	39.99	9.54	42.54	9.77	39.97	8.76	39.77	9.43	37.68	9.62	< 0.001 †
Gender (% Male)	302	47.4	65	40.9	82	51.3	91	56.9	64	40.5	0.792
BMI (kg/m^2)	33.49	5.23	33.12	5.21	33.28	4.57	33.52	6.61	34.03	4.21	0.431
WC (cm)	106.51	9.78	105.48	9.40	106.03	9.32	108.18	9.93	106.35	10.33	0.076
WHR	0.92	0.08	0.91	0.07	0.92	0.08	0.94	0.10	0.91	0.08	0.0049‡
FM (%)	35.28	9.78	36.18	10.63	33.60	9.77	34.75	10.12	36.56	8.65	0.069
FFM (%)	58.53	13.24	57.32	11.60	60.66	13.36	58.84	14.99	57.29	12.25	0.164
PA.MET	2099.80	3103.97	1856.40	2724.57	2447.54	3322.14	2405.45	3630.45	1683.08	2565.24	0.060

BMI Body mass index, *WC* Waist circumference, *WHR* Waist to hip ratio, *FM* Fat mass, *FFM* Fat free mass

* *P*-values derived from ANOVA following *Tuky's* post-hoc test

† , *Tuky's* test significant for all of the groups

‡ *Tuky's* test significant between third quartile and others

Bold values are statistically significant

Table 3 Dietary intake of energy, macronutrients and energy-adjusted intake of food groups across quartiles of dietary Na/K ratio

Variable	All participants		Quartils of dietary Na/K								*P* Value*	*P*-Value**	*P* for trend***
			Q_1 ($n = 159$)		Q_2 ($n = 160$)		Q_3 ($n = 160$)		Q_4 ($n = 158$)				
			(< 0.78)		(0.78 -1.06)		(1.06–1.45)		(> 1.45)				
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
Energy (kcal/d)	2928.48	1032.64	3033.15	1124.80	2982.89	1051.23	2834.66	929.25	2863.08	1012.63	0.258	-	0.073
Carbohydrate (%)	57.09	9.41	60.38	9.38	56.21	9.90	56.00	8.59	55.79	8.89	**< 0.001**	-	0.617
Protein (%)	12.77	2.56	13.39	2.48	13.12	2.71	12.95	2.34	11.66	2.36	**< 0.001**	-	0.685
Fat (%)	30.17	7.41	27.75	6.09	30.28	7.72	30.38	7.23	32.24	7.81	**< 0.001**	-	0.364
Cholesterol (mg/d)	282.53	192.89	273.22	130.09	307.97	235.07	308.28	239.40	241.13	130.44	**0.004**	**0.002**	0.152
Sodium (mg/d)	4528.64	2129.38	3288.42	1445.12	4015.10	1474.20	4717.21	1718.90	6066.53	2591.22	**< 0.001**	**< 0.001**	**< 0.001**
Potassium (mg/d)	4240.28	1972.11	5581.37	2369.12	4413.28	1597.25	3891.93	1484.59	3097.48	1419.38	**< 0.001**	**< 0.001**	**< 0.001**
Fiber (g/d)	62.35	38.72	57.30	29.08	56.28	30.84	63.48	43.76	72.18	46.25	**< 0.001**	**< 0.001**	**< 0.001**
Table salt (g/d)	3.49	3.63	1.78	2.02	2.56	1.95	3.26	1.91	6.31	5.41	**< 0.001**	**< 0.001**	**< 0.001**
Food groups													
Grains (g/d)	541.01	330.86	435.14	221.54	506.13	218.17	618.39	511.24	603.86	241.22	**< 0.001**	**< 0.001**	**< 0.001**
Organ Meat (g/d)	2.81	7.91	2.31	6.04	3.14	10.58	3.67	9.06	2.11	4.27	0.254	0.199	0.942
Red Meat (g/d)	20.54	21.92	20.53	19.00	24.65	31.78	22.43	16.65	19.53	15.86	**0.029**	**0.044**	0.066
Fish (g/d)	9.10	3.28	11.17	3.48	10.49	4.40	9.03	4.75	5.70	4.81	**0.001**	**0.001**	**< 0.001**
Low Fat Dairy (g/d)	227.08	199.37	232.40	249.53	246.14	175.84	205.11	162.63	133.69	146.36	**< 0.001**	**< 0.001**	**< 0.001**
High Fat Dairy (g/d)	104.16	139.71	163.95	195.61	108.78	130.38	79.20	100.79	64.98	85.91	**< 0.001**	**< 0.001**	**< 0.001**
Fruit (g/d)	564.55	510.80	948.63	656.36	598.48	482.22	420.75	281.65	290.01	247.62	**< 0.001**	**< 0.001**	**< 0.001**
Vegetables (g/d)	359.82	258.09	461.57	379.11	368.73	166.67	341.63	203.05	267.45	190.75	**< 0.001**	**< 0.001**	**< 0.001**
Nuts (g/d)	18.63	74.43	33.80	144.98	15.60	15.52	15.51	23.52	9.67	15.93	**0.024**	**0.035**	**0.004**
Beans (g/d)	53.14	56.00	61.32	65.17	54.25	47.14	48.71	43.94	48.34	64.16	0.133	0.416	0.026
Poultry (g/d)	26.72	26.02	29.45	28.45	28.49	26.59	28.20	26.93	20.69	20.69	**0.009**	**0.009**	**< 0.001**
Egg (g/d)	27.25	23.00	24.04	19.28	27.39	18.58	31.45	29.30	26.04	22.75	**0.031**	**0.006**	**0.249**
Sweets (g/d)	24.45	20.93	20.27	20.73	26.90	21.62	23.98	21.01	20.74	19.99	**0.037**	0.096	0.009
Beverages													
Tea (ml/d)	934.34	823.83	885.78	671.63	1009.91	981.30	981.02	757.83	826.85	850.19	0.314	0.319	0.728
Coffee (ml/d)	17.08	42.64	20.26	58.53	21.01	41.61	11.10	26.04	15.98	37.86	0.144	0.223	0.135
Fruit Juice (ml/d)	7.86	24.02	10.78	27.59	9.69	28.20	5.87	20.97	5.15	17.44	0.097	0.157	**0.016**
Low fat milk (ml/d)	88.79	110.99	131.35	128.90	101.80	121.68	77.52	94.06	45.55	73.36	**< 0.001**	**< 0.001**	**< 0.001**
High fat milk (ml/d)	34.19	68.16	45.75	83.74	39.54	75.89	28.07	57.76	23.69	48.09	**0.014**	**0.025**	**< 0.001**
Chocolate milk (ml/d)	10.99	33.35	13.00	32.51	15.71	49.13	6.58	19.68	8.72	23.83	0.065	0.134	0.063
Soft drinks (ml/d)	48.25	133.71	38.65	71.85	33.41	59.44	39.42	84.98	40.96	39.05	**0.004**	**0.002**	**0.005**

* *P*-values derived from one-way ANOVA or kruskall-wallis test if needed

** *P*-values derived from GLM after adjustment for total energy intake

Bold values are statistically significant

*** *P* for trend derived from polynomial linear contrasts analysis. Bold values are statistically significant

Lp(a), and AIP (P for trend). Table 5 illustrates the linear trend between the dietary sodium-to-potassium ratio and biochemical variables. Polynomial logistic regression revealed a significant positive association between serum LDL in the second quartile and between coronary risk indices (CRI-I and CRI-II) in the fourth quartile and the sodium-to-potassium ratio across all three models. Partial correlation analysis, adjusted for age, sex, BMI, energy intake, and physical activity, showed a significant positive correlation between dietary sodium ($r=0.428$, $P<0.001$), potassium intake ($r=0.305$, $P<0.001$), and serum Lp(a) concentrations (Fig. 2).

Discussion

In the current study, overweight or obese young semi-professional athletes in the second and third quartiles of the dietary sodium-to-potassium ratio had significantly higher serum total cholesterol and Lp(a) concentrations compared to those in the other quartiles in both crude and adjusted models ($P<0.05$). Additionally, systolic blood pressure (SBP) was higher in the first quartile compared to the other quartiles in both crude and adjusted models.

In the current study, overweight or obese young semi-professional athletes in the second and third quartiles of the dietary sodium-to-potassium ratio had significantly higher serum total cholesterol and Lp(a) concentrations compared to those in the other quartiles in both crude and adjusted models ($P<0.05$). Additionally, systolic blood pressure (SBP) was higher in the first quartile compared to the others in both crude and adjusted models.

Our participants with a higher dietary sodium-to-potassium ratio were younger and predominantly male. These findings align with previous studies. In the study by Okada E. et al. [28], an increase in dietary sodium-to-potassium ratio was associated with a reduction in age across higher quintiles for both men and women. Similarly, as in our study, Li X. et al. [34], found that the number of men was greater in the highest quartiles of the dietary sodium-to-potassium ratio. In the EPIC-Norfolk study of 24,963 participants (including 11,267 men and 13,696 women), men had significantly higher sodium and potassium intake, as well as a higher sodium-to-potassium ratio [50].

In our study, participants in the higher quartiles of dietary sodium-to-potassium ratio had significantly higher WHR, cholesterol, LDL, and Lp(a) levels compared to those in the lowest quartiles. Increased serum triglyceride levels were also observed in the crude model, but not in the adjusted model. Similar to our findings, previous studies have reported an increased risk of cardiometabolic factors, including higher waist circumference [33], hypertriglyceridemia [33, 34], increased obesity risk [30, 33, 51], elevated HbA1c [30, 33, 51], elevated HbA1c [28], and high blood pressure [33, 34] in individuals with higher dietary sodium-to-potassium ratios. However, some discrepancies exist. For example, several studies have found reduced cholesterol levels with increasing dietary sodium-to-potassium ratios [28, 30]. These differences may be due to variations in the demographic characteristics of the populations studied, the health status of participants, and differences in dietary assessment methods.

In our study, SBP decreased only in the higher quartiles of the dietary sodium-to-potassium ratio. The relationship between dietary sodium and potassium intake and blood pressure may be influenced by genetic, environmental factors, or both. Certain polymorphisms, such as those in the adiponectin gene [52], the sodium-bicarbonate co-transporter gene [53] and the renin–angiotensin–aldosterone system [54] along with other genetic variants [55] may contribute to salt sensitivity and potassium sensitivity of blood pressure, affecting susceptibility to salt sensitivity of blood pressure (SSBP).

Another reason for the reduced SBP in the highest quartiles of the dietary sodium-to-potassium ratio may be attributed to the higher intake of dietary fiber and grains in these quartiles. However, even after adjusting for dietary fiber, the difference in SBP across quartiles remained significant. The dietary sodium-to-potassium ratio in our study population ranged from 0.25 to 3.88, with most participants (89.3%) having a ratio below two, which falls within the optimal and suboptimal range recommended by the WHO [25]. Similar to our findings, a population-based study in northern China found no significant difference in SBP across quartiles of the dietary sodium-to-potassium ratio ($P=0.149$). However, in southern China, participants in the second and third quartiles had higher SBP compared to the other quartiles ($P=0.002$). The authors suggested that regional disparities in the association between dietary sodium-to-potassium ratio and blood pressure in China could be due to differences in dietary habits, cooking methods, social environments, and local foods between South and North China [51]; Our study was conducted in Xi'an, a city in north-central China, and our results align with those findings. Regional variations in the relationship between dietary sodium, potassium intake, and blood pressure are evident in other countries as well. The National Health and Nutrition Examination Survey (NHANES) III revealed higher sodium and lower potassium intake in the southern United States compared to other regions, where the prevalence of hypertension was also higher than in the northern United States [56]. In our study,

Table 4 Metabolic and atherogenic risk factors of study participants across quartiles of dietary Na/K ratio

Variable	All participants		Quartils of dietary Na/K								*P* Value*	*P* value**	*P* for trend***
			Q1 ($n=158$)		Q_2 ($n=160$)		Q_3 ($n=160$)		Q_4 ($n=158$)				
			(< 0.78)		(0.78 -1.06)		(1.06–1.45)		(> 1.45)				
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
FBS (mg/dl)	96.25	24.05	97.06	26.02	95.38	19.76	98.52	27.46	94.00	22.16	0.364	0.947	0.480
TC (mg/dl)	191.46	42.06	187.80	43.36	193.16	41.46	198.81	40.81	185.98	41.75	**0.028†**	**0.008**	0.944
TG (mg/dl)	138.34	84.95	142.69	83.12	150.08	105.41	148.42	71.90	121.99	73.24	**0.025‡**	0.445	**0.011**
HDL (mg/dl)	43.74	11.26	43.93	12.01	42.85	11.18	44.18	11.01	43.99	10.86	0.716	0.445	0.778
LDL (mg/dl)	122.73	37.93	121.31	38.88	123.08	35.59	127.94	38.44	118.54	38.46	0.159	0.097	0.758
SBP (mmHg)	120.55	16.19	124.89	14.34	118.76	18.56	119.16	13.97	118.88	16.18	**0.004** £	**0.023**	**0.006**
DBP (mmHg)	79.96	11.88	82.75	11.26	78.86	12.43	79.27	10.91	78.59	12.62	**0.017** £	0.136	**0.007**
Lp (a) (mg/dl)	20.15	7.80	14.36	2.13	16.12	3.60	26.35	7. 82	23.44	3.90	**< 0.001†**	**0.002**	**< 0.001**
AIP	3.54	2.89	3.60	2.90	4.04	3.75	3.46	2.24	3.08	2.38	**0.030**	0.302	**0.037**
CRI	4.63	1.52	4.53	1.50	4.78	1.54	4.72	1.41	4.47	1.60	0.213	0.756	0.738
CRII	2.98	1.26	2.96	1.28	3.05	1.16	3.05	1.21	2.87	1.38	0.554	0.938	0.653
LTI	14,876.73	2895.12	10,725.32	1894.45	13,479.69	2257.02	19,888.91	3494.05	15,354.39	1231.89	0.063	0.133	0.067

FBS Fasting blood sugar, *TC* Total cholesterol, *TG* Triglyceride, *HDL* High density lipoprotein cholesterol, *LDL* Low density lipoprotein cholesterol, *SBP* Systolic blood pressure, *DBP* Diastolic blood pressure, *Lp* Lipoprotein, *AIP* Atherogenic index of plasma, *CRI* Castelli risk index, *LTI* Lipid tetrad index

P-values derived from one-way ANOVA, following *Tuky's* post-hoc test

† *Tuky's* test significant between third quartile and others

‡ *Tuky's* test significant between second quartile and others.£,*Tuky's* test significant between first quartile and others

** *P*-values derived from GLM after adjustment for age, sex, BMI, physical activity total energy and dietary fiber intake

*** *P* for trend derived from polynomial linear contrasts analysis. Bold values are statistically significant we reported dietary intake of food groups and ingredients across quartiles of the dietary sodium-to-potassium ratio. Significantly higher intakes of grains, organ meat, red meat, eggs, cholesterol, total fiber, and table salt were observed in the higher quartiles compared to the lower quartiles in both crude and energy-adjusted models ($P<0.05$). Conversely, lower intakes of fish, low- and high-fat dairy products, fruits, vegetables, nuts, poultry, and sweets were noted in the highest versus lowest quartiles of the sodium-to-potassium ratio ($P<0.05$). To our

Table 5 The association between biochemical variables and dietary Na/K ratio using multinomial logistic regression model

Variable		**Q_1 ($n=159$) (>0.78)**	**Quartils of dietary Na/K**					
			Q_2 ($n=159$)		**Q_3 ($n=160$)**		**Q_4 ($n=158$)**	
			(0.78 -1.06)		**(1.06–1.45)**		**(> 1.45)**	
			OR (CI)	***P*-value**	**OR**	***P*-value**	**OR**	***P*-value**
FBC	Model-I	**1 REF**	0.996 (0.986–1.007)	0.464	0.997 (0.986–1.008)	0.588	0.997 (0.985–1.008)	0.552
	Model-II		0.997 (0.986–1.007)	0.523	0.998 (0.987–1.009)	0.690	0.999 (0.987–1.010)	0.825
	Model-III		0.996 (0.985–1.006)	0.415	0.997 (0.987–1.008)	0.619	0.998 (0.986–1.009)	0.692
TC	Model-I	**1 REF**	0.957 (0.908–1.008)	0.100	0.981 (0.903–1.065)	0.641	0.974 (0.903–1.050)	0.489
	Model-II		0.952 (0.902–1.006)	0.079	0.968 (0.895–1.048)	0.428	0.959 (0.891–1.033)	0.268
	Model-III		0.952 (0.902–1.005)	0.075	0.964 (0.895–1.038)	0.328	0.956 (0.888–1.028)	0.221
TG	Model-I	**1 REF**	0.995 (0985–1.006)	0.404	0.997 (0.981–1.110)	0.745	1.000 (0.982–1.017)	0.968
	Model-II		0.995 (0.984–1.006)	0.377	0.998 (0.982–1.015)	0.827	1.000 (0.983–1.017)	0.967
	Model-III		0.995 (0.984–1.007)	0.410	0.998 (0.983–1.014)	0.801	0.999 (0.982–1.016)	0.925
HDL	Model-I	**1 REF**	1.027 (0.956–1.103)	0.473	1.009 (0.918–1.110)	0.846	1.027 (0.940–1.122)	0.554
	Model-II		1.034 (0.960–1.112)	0.379	1.027 (0.936–1.127)	0.573	1.040 (0.951–1.136)	0.390
	Model-III		1.036 (0.963–1.115)	0.346	1.038 (0.949–1.135)	0.415	1.047 (0.959–1.144)	0.305
LDL	Model-I	**1 REF**	1.070 (1.008–1.136)	**0.027**	1.043 (0.956–1.138)	0.341	1.036 (0.956–1.122)	0.395
	Model-II		1.076 (1.012–1.145)	**0.020**	1.058 (0.972–1.151)	0.192	1.056 (0.976–1.143)	0.176
	Model-III		1.078 (1.013–1.147)	**0.018**	1.064 (0.982–1.152)	0.128	1.062 (0.982–1.149)	0.131
SBP	Model-I	**1 REF**	0.979 (0.955–1.003)	0.081	0.987 (0.954–1.003)	0.084	0.986 (0.961–1.012)	0.296
	Model-II		0.980 (0.956–1.005)	0.113	0.977 (0.951–1.002)	0.076	0.996 (0.969–1.023)	0.757
	Model-III		0.978 (0.954–1.003)	0.090	0.975 (0.949–1.001)	0.063	0.993 (0.967–1.021)	0.641
DBP	Model-I	**1 REF**	0.993 (0.961–1.026)	0.687	0.996 (0.963–1.030)	0.805	0.987 (0.953–1.022)	0.453
	Model-II		0.996 (0.963–1.029)	0.789	1.001 (0.967–1.036)	0.953	0.994 (0.959–1.030)	0.740
	Model-III		0.995 (0.962–1.028)	0.751	0.997 (0.963–1.032)	0.859	0.992 (0.957–1.029)	0.682
Lp (a)	Model-I	**1 REF**	1.008 (0.990–1.026)	0.372	1.016 (0.998–1.033)	0.079	1.020 (1.002–1.038)	**0.033**
	Model-II		1.008 (0.990–1.026)	0.368	1.015 (0.998–1.032)	0.092	1.021 (1.003–1.039)	**0.025**
	Model-III		1.011 (0.992–1.029)	0.260	1.022 (1.003–1.040)	**0.019**	1.025 (1.002–1.044)	**0.010**
AIP	Model-I	**1 REF**	1.034 (0.696–1.536)	0.869	0.844 (0.439–1.624)	0.612	0.748 (0.362–1.546)	0.433
	Model-II		1.027 (0.688–1.533)	0.897	0.789 (0.411–1.515)	0.477	0.709 (0.343–1.467)	0.354
	Model-III		1.027 (0.687–1.535)	0.897	0.794 (0.422–1.493)	0.474	0.727 (0.354–1.493)	0.386
CRI	Model-I	**1 REF**	1.611 (1.086–1.497)	**0.043**	1.006 (0.294–1.429)	0.195	1.566 (0.330–1.819)	0.177
	Model-II		1.396 (1.293–1.823)	**0.032**	1.052 (0.545–1.54)	0.103	1.830 (0.764–1.58)	0.070
	Model-III		1.575 (1.385–1.72)	**0.028**	1.603 (0.789–1.402)	0.068	1.909 (0.958–1.97)	0.053
CRII	Model-I	**1 REF**	0.031 (0.002–0.579)	**0.020**	0.053 (0.001–2.386)	0.130	0.069 (0.002–2.945)	0.163
	Model-II		0.023 (0.001–0.474)	**0.015**	0.028 (0.001–1.274)	0.066	0.025 (0.001–1.096)	0.056
	Model-III		0.023 (0.001–0.442)	**0.012**	0.024 (0.001–0.898)	**0.044**	0.020 (0.000–0.851	**0.041**
LTI	Model-I	**1 REF**	0.993 (0.961–1.026)	0.687	0.996 (0.963–1.030)	0.805	0.987 (0.953–1.022)	0.453
	Model-II		0.996 (0.963–1.029)	0.789	1.001 (0.967–1.036)	0.953	0.994 (0.959–1.030)	0.740
	Model-III		0.995 (0.962–1.028)	0.751	0.997 (0.963–1.032)	0.859	0.992 (0.957–1.029)	0.682

OR Odds ratio, *CI* Confidence interval, *FBS* Fasting blood sugar, *TC* Total cholesterol, *TG* Triglyceride, *HDL* High density lipoprotein cholesterol, *LDL* Low density lipoprotein cholesterol, *SBP* Systolic blood pressure, *DBP* Diastolic blood pressure, *Lp* Lipoprotein, *AIP* Atherogenic index of plasma, *CRI* Castelli risk index, *LTI* Lipid tetrad index

Model I, crude; Model II, adjusted for sex and age; Model III, adjusted for age, sex, BMI, physical activity total energy and dietary fiber intake knowledge, only the study by Okada E. et al. has compared dietary intake of food groups across quintiles of the dietary sodium-to-potassium ratio. Similar to our findings, their study in Japan reported higher intakes of meat, eggs, and cereals, but lower intakes of fruits, vegetables, milk, and nuts in the higher versus lower quintiles of the dietary sodium-to-potassium ratio [28].

Lp(a), a novel marker of cardiovascular disease, is a low-density lipoprotein (LDL)-like particle bound to apolipoprotein (a). It plays a role in vessel calcification, vascular inflammation, and thrombosis [57]. Numerous epidemiological and genetic studies suggest a causal link between elevated Lp(a) concentrations and coronary artery disease [57–59], aortic valve stenosis [60]. Its harmful effects are likely due to its similarity to LDL and its structural resemblance to apolipoprotein (a) with plasminogen, which impairs thrombolysis [61].

Very few interventional studies have investigated the impact of therapeutic diets that reduce sodium intake on Lp(a) levels [62, 63]. In the current study, a higher dietary sodium-to-potassium ratio was associated with greater concentrations of Lp(a), independent of confounders. Additionally, there was a positive association between dietary sodium and potassium intake and serum Lp(a) levels, suggesting a potential therapeutic role for dietary salt restriction in managing Lp(a) concentrations. However, further research is needed to confirm these findings. According to our results, a higher dietary sodium-to-potassium ratio was associated with an increase in Lipid Turnover Index (LTI) at a near-significant level. LTI, a novel index of cardiovascular disease, reflects a mix of atherogenic and non-atherogenic lipid particles and is proposed as a model for global cardiovascular risk assessment [48].

In the current study, we used a semi-quantitative FFQ with an acceptable validity and reliability in China [45] with 109 food items. Previous questionnaires that was validated for athletes, included athletes of track and field, ice hockey, weightlifting, bicycle racing, judo, swimming, skiing, skating, table tennis, volleyball, basketball, baseball, lacrosse, and wrestling and had only 62 basic food items [64]. Similarly, another FFQ that was validated for Brazilian athletes contained 59 foods as predictors of the variance in nutrient intake [65]. These questionnaires included relatively limited number of food items and are not suitable for a comprehensive study. Our study population consisted from various sport fields and there is no validated FFQ to cover all of these sports in China. We must use a consistent tool for all of our participants to avoid any potential bias due to difference in food items.

Also, most large-sized athletes, including the present study participants, are categorized as unhealthy overweight or obesity based on their BMI [66]. Some of the athletes are completely metabolically healthy with daily dietary care or exercise habituation, in our study, the participants' biochemical variables including serum

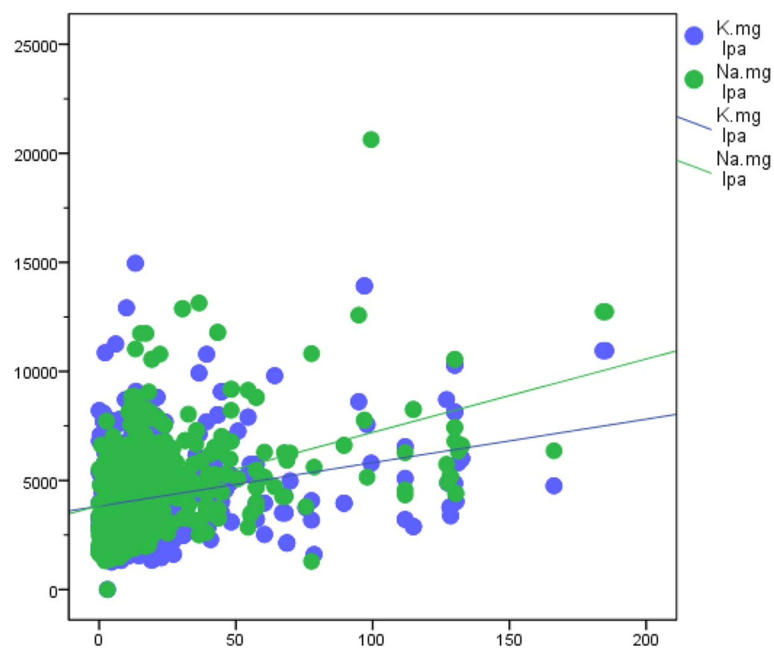

Fig. 2 The correlation between dietary sodium and potassium intake with serum Lp (**a**) concentrations among study population [(Na and Lp (**a**) correlation: $r=0.428$, $P<0.001$ and for K and Lp (**a**) correlation: $r=0.305$; $P<0.001$ using partial correlation analysis after adjustment for age, sex, BMI, energy intake and physical activity level)

lipids, blood pressure were optimal or near optimal range due to exercise effect. On the other hand most of same-sized sedentary have several healthy problems in general. Of course, some large-sized athletes have the risk of lifestyle diseases belong to their dietary life. Therefore, for the future studies comparing dietary sodium to potassium ratio and other variables between semi-professional athletes and sedentary individuals are suggested to better elucidate the effect of exercise on metabolic profile in obesity conditions.

In conclusion, our study reveals that a higher dietary sodium-to-potassium ratio is associated with increased central obesity, serum total cholesterol, and Lp (a) levels among young semi-professional athletes with overweight or obesity. Dietary interventions aimed at reducing the dietary sodium-to-potassium ratio are suggested as potential therapeutic strategies for reducing cardiovascular disease risk in this population.

The relatively large sample size and consideration of major traditional cardiovascular disease (CVD) confounders are strengths of the current study. Additionally, we accounted for table salt intake, a significant source of dietary sodium often overlooked in previous research [67]. However, the study has some limitations. We did not measure the urinary sodium-to-potassium ratio, which is a more precise indicator of dietary sodium and potassium intake. Nonetheless, previous studies have demonstrated a strong correlation between dietary sodium and potassium with urinary sodium and potassium concentrations [30, 68–71]; Furthermore, one study found that dietary sodium and potassium intake, rather than their urinary concentrations, are associated with cardiovascular events and all-cause mortality [23]. Additionally, the self-reported nature of our questionnaires may introduce potential biases in estimating food and nutrient intakes. Also, we did not ask the participants to report their training frequency or duration per a week. So, we did not have the quantitative data related data.

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Authors' contributions
The generating research hypothesis, supervision of project and a part of data analysis were performed by YL. YL was also involved in subjects' recruitment. XW and LS were involved in subject's recruitment, data analysis and writing the draft of manuscript. All of the authors read the final draft of the manuscript and agreed to its submission.

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Data availability
The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
All methods in the current research were performed in accordance with the declaration of Helsinki's guidelines and regulations. Written informed consent was obtained from all of the participants of the study. The protocol of the current study is approved by ethics committee of Department of Physical Education, Xi'an Aeronautical Institute, China (Project number: 2023003).

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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References

1. Organization, W.H. Fact sheets - Cardiovascular diseases (CVDs). 2020 accessed June 2020]; Available from: https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds).
2. Federation, W.H., World heart report 2023 confronting the world's number one killer W.H. Federation, Editor. 2023: Geneva, Switzerland.
3. Organization, W.H. Global atlas on cardiovascular disease prevention and control. 2011 accessed June 2020]; Available from: https://www.who.int/cardiovascular_diseases/publications/atlas_cvd/en/
4. Vasamreddy CR, et al. Cardiovascular disease in athletes. Clin Sports Med. 2004;23(3):455–71.
5. McHugh C, et al. A career in sport does not eliminate risk of cardiovascular disease: A systematic review and meta-analysis of the cardiovascular health of field-based athletes. J Sci Med Sport. 2020;23(9):792–9.
6. Oxford Learner's Dictionaries, Definition of athlete noun from the Oxford Advanced Learner's Dictionary. 2020.
7. Cepedal, M.Q., et al., Isokinetic thigh muscles strength in semi-professional athletics:: A one season prospective cohort study. Apunts: Medicina de l'esport, 2023. 58(220): p. 1–7.
8. Verborgt O, et al. Plate fixation of middle-third fractures of the clavicle in the semi-professional athlete. Acta Orthop Belg. 2005;71(1):17–21.
9. Tsarbou C, et al. Pre-Season ACL risk classification of professional and semi-professional football players, via a proof-of-concept test battery. Appl Sci. 2023;13(13):7780.
10. Chryssanthopoulos C, et al. Nutritional intake of semi-professional soccer players during a week in the competative season Serbian. J Sports Sci. 2009;3(1):19–27.
11. Jenner SL, et al. Dietary intakes of professional and semi-professional team sport athletes do not meet sport nutrition recommendations—a systematic literature review. Nutrients. 2019;11(5):1160.
12. Pérez-Castillo ÍM, et al. Biomarkers of post-match recovery in semi-professional and professional football (soccer). Front Physiol. 2023;14:1167449.
13. Mohr M, et al. Muscle damage, inflammatory, immune and performance responses to three football games in 1 week in competitive male players. Eur J Appl Physiol. 2016;116:179–93.
14. McKenna MJ, et al. N-acetylcysteine attenuates the decline in muscle Na+, K+-pump activity and delays fatigue during prolonged exercise in humans. J Physiol. 2006;576(1):279–88.
15. Gutiérrez-Martín Y, et al. Modulation of sarcoplasmic reticulum Ca2+-ATPase by chronic and acute exposure to peroxynitrite. Eur J Biochem. 2004;271(13):2647–57.
16. Abreu R, et al. Effects of dietary supplements on athletic performance in elite soccer players: a systematic review. J Int Soc Sports Nutr. 2023;20(1):2236060.
17. McCubbin AJ, Costa RJ. The impact of dietary sodium intake on sweat sodium concentration in response to endurance exercise: a systematic review. Int J Sports Sci. 2018;8(1):25–37.

18. McCubbin AJ, Cox GR, Costa RJ. Sodium intake beliefs, information sources, and intended practices of endurance athletes before and during exercise. Int J Sport Nutr Exerc Metab. 2019;29(4):371–81.
19. Rash, C.L., et al., Nutrition-related knowledge, attitude, and dietary intake of college track athletes. Sport Journal, 2008. 11(1).
20. MacMahon S, et al. Blood pressure, stroke, and coronary heart disease: part 1, prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. The Lancet. 1990;335(8692):765–74.
21. Graudal NA, Galløe AM, Garred P. Effects of sodium restriction on blood pressure, renin, aldosterone, catecholamines, cholesterols, and triglyceride: a meta-analysis. JAMA. 1998;279(17):1383–91.
22. Grobbee, D., Electrolyte intake and human hypertension: sodium and potassium. Textbook of hypertension. Oxford: Blackwell Scientific Publications, 1994: p. 539–51.
23. Geleijnse JM, et al. Sodium and potassium intake and risk of cardiovascular events and all-cause mortality: the Rotterdam Study. Eur J Epidemiol. 2007;22:763–70.
24. Geleijnse JM, Kok FJ, Grobbee DE. Blood pressure response to changes in sodium and potassium intake: a metaregression analysis of randomised trials. J Hum Hypertens. 2003;17(7):471–80.
25. Organization, W.H., Guideline: sodium intake for adults and children. 2012: World Health Organization.
26. Cook NR, et al. Joint effects of sodium and potassium intake on subsequent cardiovascular disease: the Trials of Hypertension Prevention follow-up study. Arch Intern Med. 2009;169(1):32–40.
27. World Health Organization, Diet, nutrition, and the prevention of chronic diseases: report of a joint WHO/FAO expert consultation. , W.H. Organization, Editor. 2003.
28. Okada E, et al. Dietary sodium: potassium ratio and CVD risk factors among Japanese adults: a retrospective cross-sectional study of pooled data from the National Health and Nutrition Survey, 2003–2017. Br J Nutr. 2021;125(1):79–91.
29. Perez V, Chang ET. Sodium-to-potassium ratio and blood pressure, hypertension, and related factors. Adv Nutr. 2014;5(6):712–41.
30. Okayama A, et al. Dietary sodium-to-potassium ratio as a risk factor for stroke, cardiovascular disease and all-cause mortality in Japan: the NIPPON DATA80 cohort study. BMJ Open. 2016;6(7):e011632.
31. Mosallanezhad Z, et al. Dietary sodium to potassium ratio is an independent predictor of cardiovascular events: a longitudinal follow-up study. BMC Public Health. 2023;23(1):1–8.
32. Kwon Y-J, et al. Association between dietary sodium, potassium, and the sodium-to-potassium ratio and mortality: A 10-year analysis. Front Nutr. 2022;9:1053585.
33. Lee Y-S, Lee S-Y. The relationship between dietary sodium-to-potassium ratio and metabolic syndrome in Korean adults: using data from the Korean National Health and Nutrition Examination Survey 2013–2015. Korean J Health Promotion. 2020;20(3):116–24.
34. Li X, et al. Association of dietary sodium: potassium ratio with the metabolic syndrome in Chinese adults. Br J Nutr. 2018;120(6):612–8.
35. Kachur S, et al. Obesity and cardiovascular diseases. Minerva Med. 2017;108(3):212–28.
36. Bamba V, Rader DJ. Obesity and atherogenic dyslipidemia. Gastroenterology. 2007;132(6):2181–90.
37. Jain N, et al. Association of urinary sodium-to-potassium ratio with obesity in a multiethnic cohort. Am J Clin Nutr. 2014;99(5):992–8.
38. Rafie N, et al. 24-h urinary sodium to potassium ratio and its association with obesity in children and adolescents. Eur J Nutr. 2019;58:947–53.
39. Harris N, et al. Prevalence of obesity in International Special Olympic athletes as determined by body mass index. J Am Diet Assoc. 2003;103(2):235–7.
40. Zhu Z, Yin P. Overweight and obesity: The serious challenge faced by Chinese children and adolescents. J Glob Health. 2023;13:03036.
41. Bai R, et al. Projections of future life expectancy in China up to 2035: a modelling study. The Lancet Public Health. 2023;8(12):e915–22.
42. Hasanpouri A, et al. Nutritional knowledge, attitude, and practice of professional athletes in an Iranian population (a cross-sectional study). BMC Sports Sci Med Rehabil. 2023;15(1):164.
43. Ren YJ, et al. Validation of the simplified Chinese-character version of the international physical activity questionnaire-long form in urban community-dwelling adults: a cross-sectional study in Hangzhou China. Biomed Environ Sci. 2017;30(4):255–63.
44. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6):499–502.
45. Zhai L, et al. Reliability and validity of a semi-quantitative food frequency questionnaire: Dietary intake assessment among multi-ethnic populations in Northwest China. J Health Popul Nutr. 2023;42(1):111.
46. Li X, et al. Chinese food image database for eating and appetite studies. Nutrients. 2022;14(14):2916.
47. Acar, O., et al., Evaluation of pro-atherogenic lipid profile and high atherohenic indexes in patients with Behçet's disease: A case–control study. Journal of Cosmetic Dermatology, 2023.
48. Morais, C.A.d.S., S.H.V. Oliveira, and L.M. Lima, Lipid tetrad index (LTI) and lipid pentad index (LPI) in healthy subjects. Arquivos Brasileiros de Cardiologia, 2013. 100: p. 322–327.
49. Zwager, C., et al., Higher dietary Na/K ratio is associated with an increased risk of cardiovascular outdomes in the general population, but not in CKD patients Nephrology Dialysis Transplantation, 2023. 38(Supplement_1): p. gfad063c_5776.
50. Wouda RD, et al. Sex-specific associations between potassium intake, blood pressure, and cardiovascular outcomes: the EPIC-Norfolk study. Eur Heart J. 2022;43(30):2867–75.
51. Huang L, et al. Associations of dietary sodium, potassium, and sodium to potassium ratio with blood pressure—regional disparities in China. Nutrients. 2020;12(2):366–75.
52. Chu C, et al. Genetic variants in adiponectin and blood pressure responses to dietary sodium or potassium interventions: a family-based association study. J Hum Hypertens. 2016;30(9):563–70.
53. Carey RM, et al. Salt sensitivity of blood pressure is associated with polymorphisms in the sodium-bicarbonate cotransporter. Hypertension. 2012;60(5):1359–66.
54. Poch, E., et al., Molecular basis of salt sensitivity in human hypertension. Evaluation of renin-angiotensin-aldosterone system gene polymorphisms. Hypertension, 2001. 38(5): p. 1204–9.
55. Xie Y, et al. Candidate gene polymorphisms influence the susceptibility to salt sensitivity of blood pressure in a han Chinese population: risk factors as mediators. Front Genet. 2021;12:675230.
56. Hajjar I, Kotchen T. Regional variations of blood pressure in the United States are associated with regional variations in dietary intakes: the NHANES-III data. J Nutr. 2003;133(1):211–4.
57. Duarte Lau, F. and R.P. Giugliano, Lipoprotein(a) and its Significance in Cardiovascular Disease: A Review. JAMA Cardiol, 2022. 7(7): p. 760–769.
58. Huang YC, et al. Lipoprotein(a) is Associated with Cardiovascular Events in Low Risk Males: Results from a Health Checkup Cohort with Long-Term Follow-Up. Acta Cardiol Sin. 2022;38:381–90.
59. Guddeti RR, et al. Lipoprotein(a) and calcific aortic valve stenosis: A systematic review. Prog Cardiovasc Dis. 2020;63(4):496–502.
60. Kronenberg, F., et al., Lipoprotein(a) in atherosclerotic cardiovascular disease and aortic stenosis: a European Atherosclerosis Society consensus statement European Heart Journal, 2022. 43(39): p. 3925–3946.
61. Erqou S, et al. Lipoprotein(a) concentration and the risk of coronary heart disease, stroke, and nonvascular mortality. JAMA. 2009;302:412–23.
62. Haring B, et al. Healthy dietary interventions and lipoprotein (a) plasma levels: results from the Omni Heart Trial. PLoS ONE. 2014;9(12):e114859.
63. Matveyenko A, et al. Association of free-living diet composition with plasma lipoprotein(a) levels in healthy adults. Lipids Health Dis. 2023;22(1):144.
64. Ishikawa-Takata K, Okamoto K, Taguchi M. Development and validation of a food frequency questionnaire for Japanese athletes (FFQJA). J Int Soc Sports Nutr. 2021;18(1):34.
65. Godois ADM, et al. Development of a Food Frequency Questionnaire for Brazilian athletes. Nutr Diet. 2020;77(2):260–7.
66. Selden MA, et al. Cardiometabolic abnormalities in current National Football League players. Am J Cardiol. 2009;103(7):969–71.
67. Mosallanezhad Z, et al. Dietary sodium to potassium ratio is an independent predictor of cardiovascular events: a longitudinal follow-up study. BMC Public Health. 2023;23(1):705.
68. Goncalves C, Abreu S. Sodium and potassium intake and cardiovascular disease in older people: a systematic review. Nutrients. 2020;12(11):3447.

69. Bahadoran Z, et al. Monitoring population salt intake using casual urinary sodium: Tehran Lipid and Glucose Study. Nutr Metab. 2022;19(1):19.
70. Mirmiran P, et al. Urinary sodium-to-potassium ratio: a simple and useful indicator of diet quality in population-based studies. Eur J Med Res. 2021;26:1–8.
71. Mirmiran P, et al. Dietary sodium to potassium ratio and the incidence of chronic kidney disease in adults: a longitudinal follow-up study. Prev Nutr Food Sci. 2018;23(2):87.

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