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Original article

Elevated hypertension risk associated with higher dietary acid load: A systematic review and meta-analysis

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SUMMARY

Background & aims: The association between dietary acid load and hypertension risk is inconclusive. We conducted a systematic review and meta-analysis to summarize effect of dietary acid load on blood pressure.
Methods: A comprehensively search was performed in electronic databases including EMBASE, PubMed, Web of Science and Chinese National Knowledge Infrastructure. Summary ORs and their corresponding 95% CIs were computed assuming a randomized model or fixed model.
Results: Ten publications comprising 4 cohort and 6 cross-sectional studies were eligible for meta-analysis. There were 8 studies about potential renal acid load (PRAL) and 4 about net endogenous acid production (NEAP). Essential hypertension was statistically associated with higher PRAL (OR = 1.14, 95% CI = 1.02−1.17). Our findings also demonstrated a positive impact of higher PRAL on elevating both diastolic pressure (WMD = 0.96, 95% CI = 0.67−1.26) and systolic pressure (WMD = 1.57, 95% CI = 1.12−2.03). A 35% increased risk of hypertension associated with higher NEAP was identified (OR = 1.35, 95% CI = 1.03−1.78).
Conclusions: The current study suggests that dietary acid load might be potential risk factor of hypertension.

1. Introduction

Hypertension prevalence was 34.9% in adults (≥18 years) globally, continuing to be the primary contributor to the global burden of disease [1,2]. Even though a portion of individuals with hypertension raised their awareness of blood pressure, the situation is pessimistic as above 40% individuals receiving treatment did not control their blood pressure to a satisfied level [1,3]. The chronic disease is a synthesis of genetic and environmental effects and considerable of being associated with cardiovascular disease, stroke and etc. [4,5]. Dietary factors are considered to be in relation to blood pressure [6]. An appropriate consumption of dietary components is conducive to controlling or preventing hypertension.

According to previous studies, endogenous acid-base equilibria is potentially associated with the buffer capacity of the blood, renal and respiratory function [7,8]. Dietary acid load is perceived to have impact on endogenous acid-base equilibria [9,10]. Dietary components such as sulfate from protein and phosphorus as acid precursors contribute to acid load, while other components such as potassium, magnesium, and calcium as base precursors contribute to base load [11,12]. Heavy dietary acid load might cause chronic and mild metabolic acidosis, which potentially affect blood pressure toward kidneys and renin-angiotensin-aldosterone system [11]. Dietary acid load could be evaluated by two methods including the potential renal acid load [PRAL] and the net endogenous acid production [NEAP] [11,12]. PRAL computes endogenous acid load by dietary intake of protein, phosphorus, potassium, calcium, and magnesium, and NEAP by protein and potassium [11,12].

Recently, several observational studies suggest a positive association between dietary acid load and hypertension [13–15]. However, the association was towards to null according to some other studies [16,17], making an indeterminate conclusion. Thus, we perform a systematic review to quantitatively evaluate the risk of dietary acid load in relation to hypertension risk.

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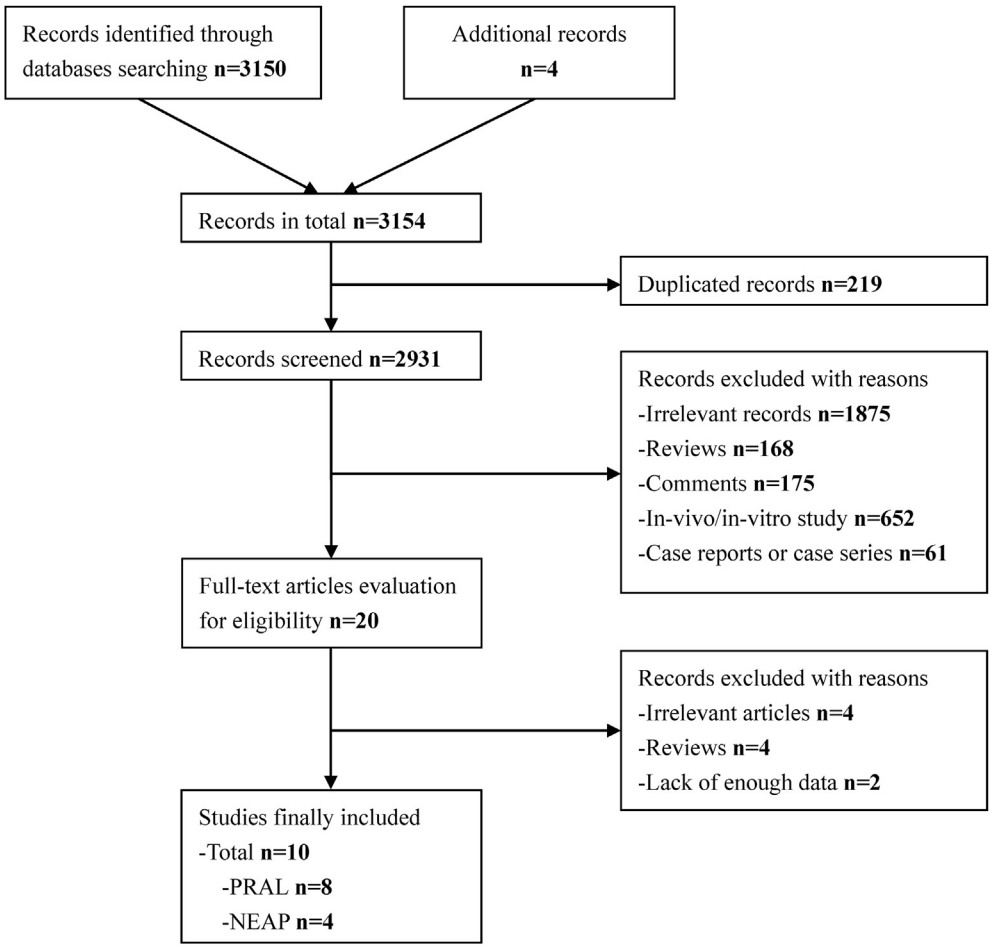

Fig. 1. Flow diagram of the selection of the studies.

2. Methods

2.1. Search strategy and study selection

We comprehensively searched relevant studies in EMBASE, PubMed, Web of Science and Chinese National Knowledge Infrastructure up to March 2018 without specifying limitation of publication date. Additionally, potential studies were identified from checking references of review or original articles. If there were several publications from the identical population, we only included the most comprehensive one. Combined keywords were adopted in this study as follow, related to dietary acid load (acid base equilibrium OR acid base imbalance OR acid ash OR alkaline ash OR acid base or acid load OR potential renal acid load OR PRAL OR net endogenous acid production OR NEAP) and outcome of hypertension (hypertension OR HTN OR blood pressure).

Cross-sectional, cohort studies, case–control studies and clinical studies detailing hypertension incidence, systolic pressure and diastolic pressure in relation to dietary acid load were eligible. Studies concerning duplicate publications, animals and toxicological studies, commentaries and editorials, case reports and case series, reviewed articles, or without sufficient data were excluded. Included studies reported potential renal acid load (PRAL) and/or net endogenous acid production (NEAP) for assessment of dietary acid load using the following formulas [11,12]:

$$\text{PRAL (mEq/d)} = 0.4888 \times \text{protein intake (g/d)} + 0.0366 \times \text{phosphorus (mg/d)} - 0.0205 \times \text{potassium (mg/d)} - 0.0125 \times \text{calcium (mg/d)} - 0.0263 \times \text{magnesium (mg/d)};$$

$$\text{Estimated NEAP (mEq/d)} = (54.5 \times \text{protein intake (g/d)} \div \text{potassium intake (mEq/d)}) - 10.2$$

2.2. Data extraction and quality assessment

This study was in accordance with Meta-analysis Of Observational Studies in Epidemiology (MOOSE) guidelines and preferred reporting items of PRISMA [18,19]. A standardized form with items including author, year, location, period, sample size, male percentage, age, confirmation of dietary acid load and blood pressure, effect size (OR, RR or HR) and adjustments was applied for information extraction. Two investigators independently extracted data from eligible studies into the standardized form.

Methodological quality of eligible studies was assessed by validated scales provided by Agency for Healthcare Research and Quality (AHRQ) [18]. Cross-sectional studies were assessed by a validated scale including 11 items. Case control and cohort studies were evaluated according to the Newcastle–Ottawa Quality

Table 1
Characteristic of eligible studies.

Study	Design	Country	Period	Sample size	Male/ %	Population	Age/year	Dietary Intake	DAL	Blood pressure measurement	Adjustments	Methodological quality
Tielemans et al., 2017	prospective cohort	Netherlands	2002 −2006	3411	0	pregnant women	31.4 ± 4.4	FFQ	PRAL/ NEAP	by Omron 907® automated digital oscillometric sphygmomanometer (OMRON Healthcare Europe B.V. Hoofddorp, the Netherlands)	maternal age, prepregnancy BMI, parity, educational level, household income, alcohol consumption during pregnancy and smoking behavior during pregnancy.	High
Chan et al., 2015	cohort	Hong Kong, China	2001 −2003	3956	50.1	older adults	≥65	FFQ	NEAP	by mercury sphygmomanometer (WA Baum Co. Inc., Copiague, NY, United States).	NR	Moderate
Engberink et al., 2012	population-based cohort	Netherlands	1990 −1999	2241	42.7	older adults	≥55	FFQ	PRAL	by random-zero sphygmomanometer	age, sex, BMI, smoking status, alcohol consumption, educational level, total energy, and fiber	High
Zhang et al., 2009	prospective cohort	America	1991 −2003	87,293	0	women	31−41	FFQ	NEAP	medical records	NR	Moderate
Krupp et al., 2018	cross-sectional	Germany	2008 −2011	7115	49.6	adults	18−79	FFQ	PRAL	by automated oscillometric device (Datascope Accutorr Plus, Mahwah, NJ, USA).	NR	Moderate
Murakami et al., 2017	cross-sectional	Japan	2012	15,618	42	adults	≥20	a 1-d semi-weighed household dietary record	PRAL	NR	age, smoking status, alcohol drinking, habitual exercise, and dietary reporting status. For systolic and diastolic blood pressure, total, HDL-, and LDL-cholesterol, and glycated hemoglobin, BMI.	Moderate
Han et al., 2016	cross-sectional	Korea	2008 −2011 and 2013	11,601	41.4	older adults	40−79	1 day face-to-face 24 h recall method	PRAL	by mercury sphygmomanometers (Baumanometer; W.A. Baum, Copiague, NY)	age, sex, exercise, family history of cardio- and cerebro-vascular disease, diabetes, hypertension, LDL cholesterol, eGFR, and urine pH	High
Akter et al., 2014	cross-sectional	Japan	2012 −2013	2028	89.2	adult workers	18−70	a validated brief 74 self-administered diet history questionnaire	PRAL/ NEAP	by an automated sphygmomanomete	age and sex, occupational physical activity, leisure-time physical activity, smoking, alcohol drinking, shift work, overtime work, parental history of hypertension, BMI, and sodium intake	High
Luis et al., 2014	cross-sectional	Sweden	1991 −1995 and 1997 −2001	673	100	older men	70−71	a 7-d dietary record	PRAL	by ABPM device Accutracker II (Suntech Medical Instruments)	age, energy intake, BMI, smoking status, physical activity, education level, sodium intake, alcohol intake, glomerular filtration rate, diabetes, cardiovascular disease, and hyperlipidemia.	High
Murakami et al., 2008	cross-sectional	Japan	2005 and 2006	1136	0	young women	18−22	a validated self-administered diet history questionnaire	PRAL	by automatic device (Omron model HEM-770A; Omron Health Care, Kyoto, Japan)	residential block, central, or south, size of residential area, survey year, current smoking, and physical activity.	High

Footprint: DAL = dietary acid load, FFQ = food frequency questionnaire, PRAL = potential renal acid load, NEAP = estimated net endogenous acid.

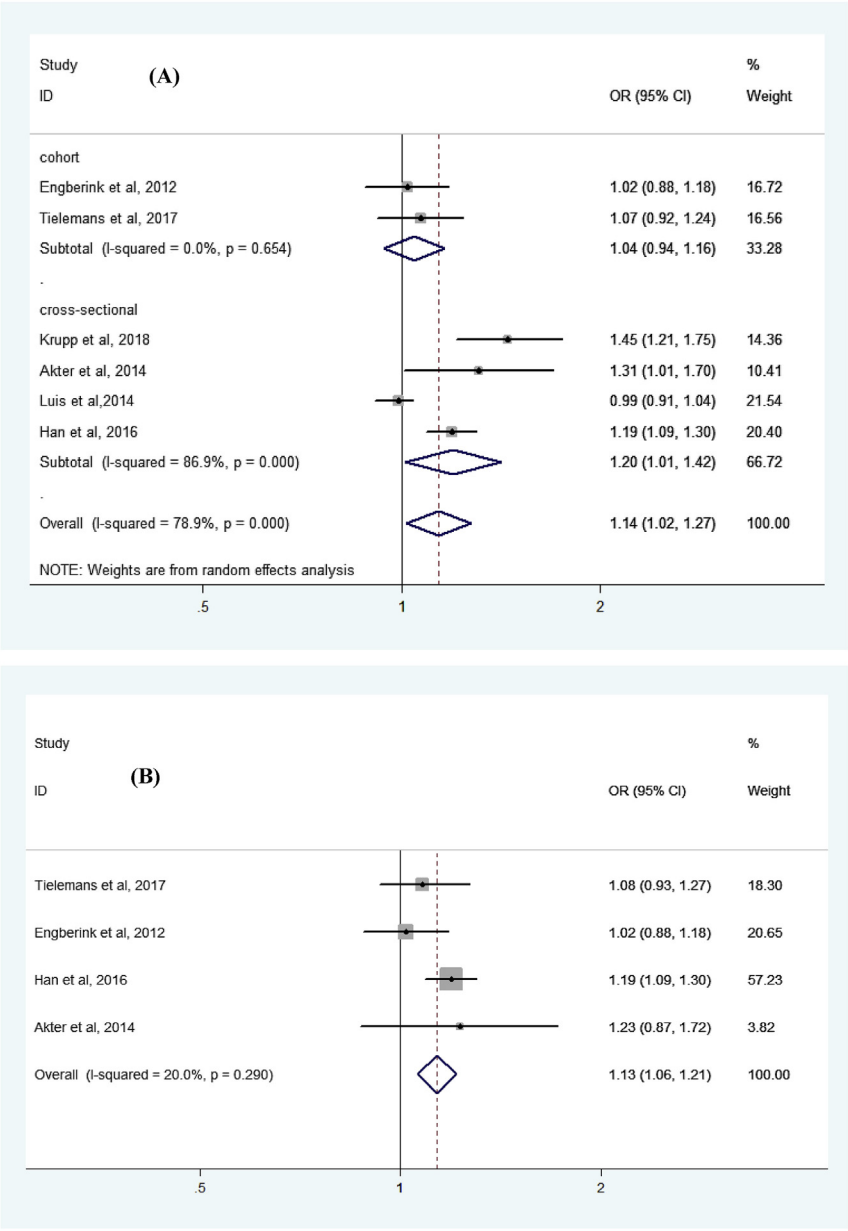

Fig. 2. Forest plot of hypertension risk with higher potential renal acid load exposure. (A) Demonstrates overall effect of higher potential renal acid load. (B) Demonstrates effect of pooling size effect with multiply adjustments.

Assessment Scale (NOS) [19], with a higher score indicating a higher quality. Methodological qualities were divided into three levels, including poor, moderate and high.

2.3. Statistical analysis

With respect to summary size effects from individuals in the highest versus the lowest (reference) category, RRs and HRs were considered equivalent to ORs since a large portion of eligible studies were cross-sectional design. Summary ORs and their corresponding 95% CIs were computed assuming a randomized model or fixed model [20]. The randomized model was employed with material heterogeneity, otherwise the fixed model was applied. We used Q test and I^2 statistic to evaluated heterogeneity within included studies [21]. A p-value < 0.10 was set to as significant for Q test, indicating existence of heterogeneity. There were four levels of heterogeneity according I^2 value, with I^2 values $\leq$ 25%, 25–50%, 50–75% and >75% indicating no, mild, moderate and high heterogeneity, respectively [22]. A subgroup analysis was performed to evaluate the weight of dietary acid load on risk of hypertension with respect to gender, age and geographic region. We did a sensitivity analysis to assess robustness of the results, which recalculated summary ORs by omitting studies one by one. Egger's test [23], begger's test [24] and funnel plot [25] were conducted to evaluate publication bias. We did statistical analysis with STATA version 12.0 (StataCorp LP, College Station, TX, USA).

3. Results

3.1. Characteristics of included studies

A total of 3154 records were identified by electronic searching and references screening and 3144 were excluded with reasons demonstrated in flow chart (Fig. 1). Ten publications comprising 4

cohort [17,26–28] and 6 cross-sectional studies [13–16,29,30] were eligible for meta-analysis. According to methodological quality assessment by validated scales, 6 studies [13–17,26] were considered as high quality, 4 [27–30] were moderate, at least partly, inclined to reporting potential bias.

The characteristics of location, period, sample size, male percentage, age, confirmation of dietary acid load and blood pressure, effect size and adjustments varied materially among included studies (Table 1). These studies were conducted between 1990 and 2013. There were 3 studies from Japan [14,15,30], 2 from Netherlands [17,26], 1 from China [28], America [27], Germany [29], Korea [13] or Sweden [16], respectively. The 10 studies reported a sample size of 135,072 people and all participants were adults [4 concerning older adults [13,16,17,28], 3 concerning adults [14,15,29], 3 concerning women [26,27,30]. Among them, there were 5 studies collecting dietary intake by food frequency questionnaire (FFQ) [17,26–29], 2 were by dietary record [15,16], 2 were by self-administered diet history questionnaire [14,30], 1 was by face-to-face 24 h recall method [13]. Eight publications [13–17,26,29,30] reported PRAL for assessment of dietary acid load and four [14,26–28] reported NEAP.

3.2. Association between dietary acid load and hypertension

Essential hypertension was statistically associated with higher PRAL after pooling two cohort studies and four cross-sectional studies [13–17,26,29,30] (OR = 1.14, 95% CI = 1.02–1.17, I^2 = 79.8%) assuming a randomized model (Fig. 2). The positive association was also observed in summary result of cross-sectional studies (OR = 1.20, 95% CI = 1.01–1.42, I^2 = 86.9%) but prone inclined to be null in cohort studies (OR = 1.04, 95% CI = 0.94–1.16, I^2 = 0%). Significant result was identified in pooling size effect with multiply adjustments (OR = 1.13, 95% CI = 1.06–1.21, I^2 = 20.0%) (Fig. 2). Even though substantially high heterogeneity was found, it has different reductions in different stratifications (Table 2).

Result from statistical analysis demonstrated a positive impact of higher PRAL on elevating both diastolic pressure (weight mean difference, WMD = 0.96, 95% CI = 0.67–1.26, I^2 = 74.1%) and systolic pressure (WMD = 1.57, 95% CI = 1.12–2.03, I^2 = 57.0%) (Fig. 3).

After summarizing 3 cohort studies and 1 cross-sectional study [14,26–28], a 35% increased risk of hypertension associated with higher NEAP was identified (OR = 1.35, 95% CI = 1.03–1.78, I^2 = 91.8%) assuming a randomized model (Fig. 4).

Table 2
Results of subgroup analysis.

	n	Pooled OR	95%CI	P	I^2	Model
PRAL and hypertension	6	1.14	1.02–1.27	<0.05	78.9	Random
Location						
Europe	4	1.10	0.95–1.27	>0.05	79.7	Random
Asia	2	1.20	1.10–1.31	<0.05	78.9	Random
Gender						
Male	2	1.00	0.94–1.07	>0.05	4.6	Fixed
Female	2	1.01	0.90–1.13	>0.05	41.5	Fixed
Age						
Old adult	4	1.13	0.98–1.31	>0.05	86.1	Random
NEAP and hypertension	4	1.35	1.03–1.78	<0.05	91.8	Random
Location						
Asia	2	1.34	1.14–1.57	<0.05	91.8	Random
Other	2	1.35	0.83–2.21	>0.05	97	Random
Gender						
Female	2	1.35	0.83–2.21	>0.05	97	Random
	n	**WMD**	**95%CI**	**P**	**I^2**	**Model**
PRAL and DBP	5	0.10	0.03–0.17	<0.05	68.0	Random
Gender						
Male	2	0.86	0.17–1.54	<0.05	5.2	Fixed
Female	2	0.75	−0.59–1.31	>0.05	71	Random
Age						
Old adult	2	1.01	−0.37–2.39	>0.05	60.7	Random
PRAL and SBP	5	0.13	0.06–0.20	<0.05	70.7	Random
Gender						
Male	2	2.08	0.35–2.81	<0.05	64.6	Random
Female	2	1.14	0.40–1.89	<0.05	37.9	Fixed
Age						
Old adult	2	1.34	0.63–2.05	<0.05	0	Fixed

Footprint: PRAL = potential renal acid load, NEAP = estimated net endogenous acid, DBP = diastolic blood pressure, SBP = systolic blood pressure, WMD = weight mean difference.

3.3. Sensitivity analyses and publication bias

There was lack of obvious fluctuation after omitting any individual study, indicating stable results in our meta-analyses. No significant results were observed from Egger's test or begger's test (p-value >0.05) and funnel plots were symmetrical, suggesting absence of publication bias.

4. Discussion

In the current study, we collected the latest data on hypertension risk associated with dietary acid load. The methodological quality of eligible publications in this study was considered as moderate to high and the studies were conducted in seven separated countries across three continents. An elevated risk of hypertension was detected in those people with higher dietary acid load. There was a 14% increased risk of hypertension in relation to higher PRAL and a 35% in relation to higher NEAP based on pooling studies concerning adults. Therefore, an increasing intake of anti-PRAL and anti-NEAP dietary components such as potassium and magnesium and a decreasing intake of pro-PRAL and pro-NEAP dietary components such as protein intake and phosphorus, might exert an important role in reducing the risk of hypertension.

Multiple theories explain that elevated dietary acid load may increase blood pressure. Imbalance die might be involved in a chronic and low grade metabolic acidosis which is characterized with elevating proton load and lowering pH value in blood [31]. Metabolic acidosis was potentially associated with upgrade secreting cortisol, excreting calcium or inhibiting citrate excretion, which might alter salt sensitivity and indirectly increasing blood pressure [16,29]. Another mechanism involves increasing serum anion gap in metabolic acidosis. Previous studies demonstrated people have an elevated anion gap [9], indicating of an elevated blood pressure, nonetheless the possible underlying pathway is not understood.

Proton load from diet mainly depends on dietary protein and phosphorus intake, but dietary potassium, calcium and magnesium intake would counteract at least part of its production [11]. The potential renal acid load [PRAL] integrates effects of these factor on production of proton load depending on diet [11,14]. Our results supported that hypertension incidence was associated with higher PRAL (OR = 1.14, 95% CI = 1.02–1.17). Higher PRAL was also in relation to either elevated diastolic pressure (WMD = 0.96, 95% CI = 0.67–1.26) or elevated systolic pressure (WMD = 1.57, 95% CI = 1.12–2.03). Apart from proton load on increasing blood pressure, a low potassium intake in a high PRAL diet has its own direct elevating effect on blood pressure [16,29]. Potential mechanisms might be involved in exciting sympathetic nerve, simulating baroreceptor, producing renin and reducing renal natriuresis [31].

There were 4 included studies [13,16,17,28] concerning senior citizens and we separated effect of high dietary acid load on senior citizens. The association between PRAL and hypertension did not pronounce in this subgroup (Table 2). Previous study hinted that

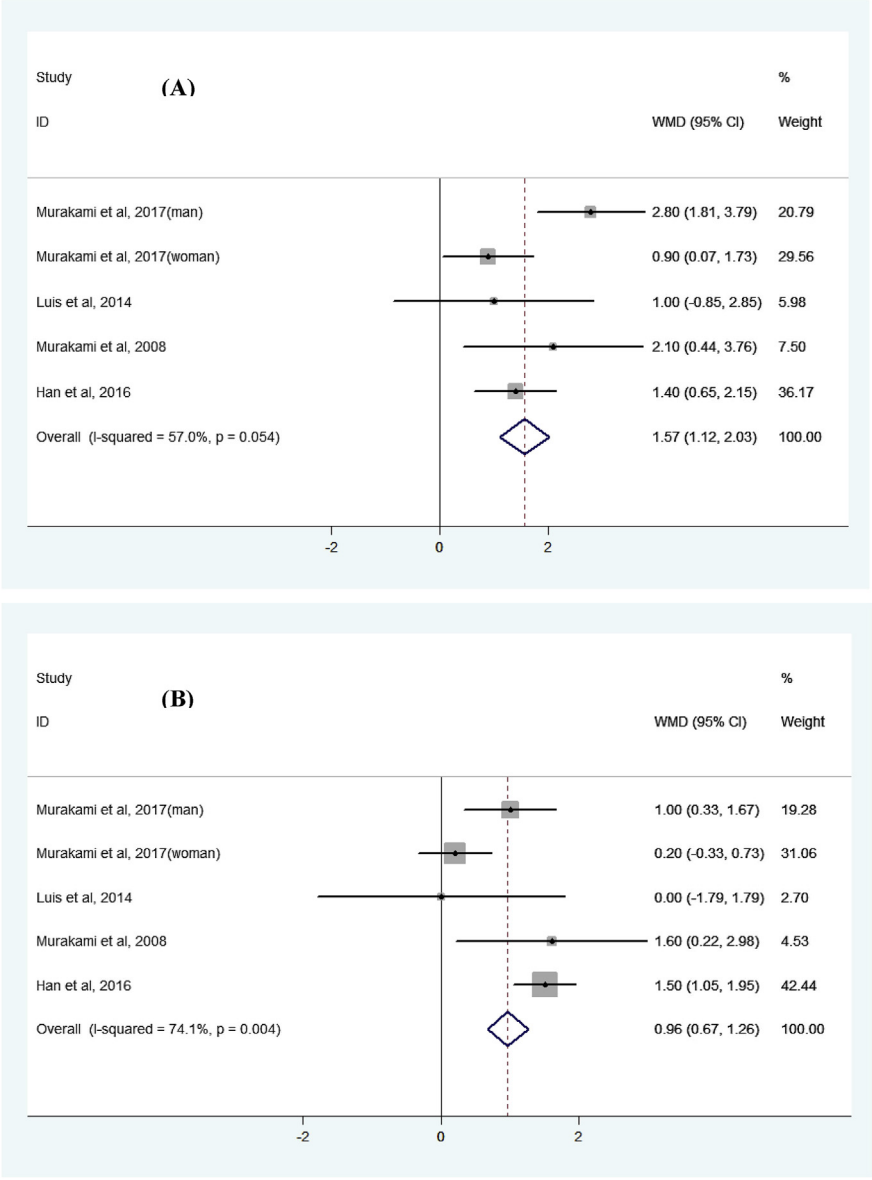

Fig. 3. Forest plot of elevating systolic pressure or diastolic pressure risk with higher potential renal acid load exposure. (A) Demonstrates elevating systolic pressure risk with higher potential renal acid load exposure. (B) Demonstrates diastolic pressure elevating risk with higher potential renal acid load exposure.

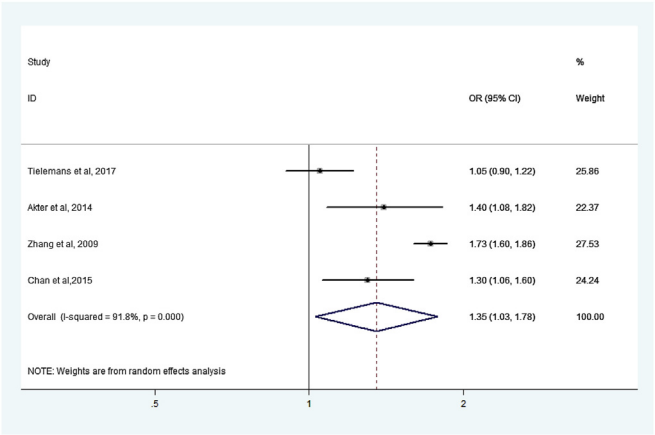

Fig. 4. Forest plot of hypertension risk with higher net endogenous acid production exposure.

blood pressure might seem to level off in the old [29,30]. Result from the current study supported that blood pressure might be less susceptible to dietary factors such as dietary acid load for those people with higher ages [32]. With respect to different genders, divergent results were identified. However, considering the limited eligible studies, the pooled effects on different genders is less conclusive. Further studies are needed to confirm the effect of dietary acid load in separated genders.

Net endogenous acid production (NEAP) considers protein and potassium as nutrients resource of dietary acid load [12]. Results from our findings also suggest an increased risk of hypertension associated with a high NEAP (OR = 1.35, 95% CI = 1.03–1.78). Compare with PRAL, effect of NEAP on hypertension seems to be more obvious. NEAP did not take into account absorption rates of protein and potassium, which might be the primary contributor to the distinction between effects of PRAL and NEAP on hypertension risk.

Several possible limitations should be discussed in this study. First of all, this study was based on cross-sectional or cohort design, which might carry possible bias, such as information bias. What's

more, dietary intake from included studies was evaluated by assessment tools of self-report, such as FFQ and 24-h recalls, which cannot avoid recall bias and misclassification of dietary exposure. Furthermore, there were material heterogeneity within studies summarizing effects of PRAL or NEAP on hypertension risk. One potential explanation accounting for this might be dietary divergence in different populations as well as various components in assessment questionnaires from separated studies. Another cause might stem from different study designs across these studies. In addition, dose–response effects were not conducted in this study as the eligible studies were limited.

Two aspects should be considered in future studies. Whether there was gender difference in hypertension risk associated with PRAL or NEAP should be confirmed. What is more, dose–response effects of dietary acid load on blood pressure should be uncovered and verified.

5. Conclusions

In summary, the current study suggests a material association between dietary acid load and hypertension risk. A higher PRAL or NEAP of dietary intake might be independent risk factors for elevating blood pressure. Further more comprehensive studies are warranted to elucidate the role of dietary acid load in hypertension risk of specific genders.

Conflict of interest

This is no financial conflict of interest or other type of conflict of interest.

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J.W.P: conception and design, revising and final approval of article. S.W.C.: conception and design, analysis plan, interpretation of data, drafting manuscript and final approval of article. Z.H.C.: data analysis and final approval of article. Y.H.L and P.W.: acquisition of data and final approval of article.

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