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Review

Dietary Intake in Relation to the Risk of Reflux Disease: A Systematic Review

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ABSTRACT: Gastroesophageal reflux disease (GERD) is a chronic condition which has a high global prevalence. Dietary intake is considered to be a contributing factor for GERD. However, scientific evidence about the effect of diet on the risk of GERD is controversial. This systematic review was conducted to address this issue. A comprehensive structured search was performed using the MEDLINE, Scopus, and Web of Science databases up to August 2020, in accordance with the PRISMA statement. No restrictions were set in terms of language, time of publication, or study location. Study selection and data abstraction was conducted independently by two authors, and risk of bias was assessed using a modified Quality in Prognosis Studies Tool. Eligible studies evaluating the impact of food and dietary pattern on GERD were included in qualitative data synthesis. After excluding duplicate, irrelevant, and low quality studies, 25 studies were identified for inclusion: 5 case-control studies, 14 cross-sectional studies, and 6 prospective studies. This review indicates that high-fat diets, carbonated beverages, citrus products, and spicy, salty, and fried foods are associated with risk of GERD.

Keywords: diet, food, gastroesophageal reflux, systematic review

INTRODUCTION

Gastroesophageal reflux disease (GERD) is a common disorder that affects quality of life. GERD develops when reflux of stomach contents causes troublesome symptoms and long-term complications (Rajaie et al., 2020). The major symptoms of GERD include heartburn and regurgitation (Kahrilas, 2003), however, GERD can also manifest with atypical symptoms including epigastric pain, dyspepsia, nausea, bloating, and belching (Badillo and Francis, 2014). GERD pathogenesis involves esophagitis, hemorrhage, stricture, Barrett's esophagus, and adenocarcinoma (Rajaie et al., 2020). Moreover, GERD is independently associated with increased risk of cardiovascular diseases, including acute myocardial infarction (Lei et al., 2017). It is a global disease, with an estimated highest incidence in North America (18.1%～27.8%), followed by the Middle-East America (8.7%～33.1%), Europe (8.8%～25.9%), and East Asia (2.5%～7.8%) (Seremet et al., 2015).

GERD is a multifactorial disease influenced by both genetic predisposition and environmental factors. Diet (an environmental factor) has important roles in gastrointestinal and cardio metabolic disorders (Argyrou et al., 2018; Heshmati et al., 2019; Surdea-Blaga et al., 2019; Heshmati et al., 2020), and modifiable risk factors included long meal-to-sleep intervals, speed of eating, and scale and temperature of foods (Esmaillzadeh et al., 2013; Yuan et al., 2017). Intake of alcohol, chocolate, and high-fat meals reduces esophageal sphincter pressure and increases esophageal exposure to gastric juices (Kaltenbach et al., 2006). Some studies have reported that both the quality and quantity of carbohydrates in diet may be associated with GERD (Keshteli et al., 2017; Wu et al., 2018). However, current data are contradictory (Kim et al., 2014).

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Emerging data indicates that appropriate eating behaviors, i.e., healthy diets involving high intakes of fruits and whole grains (Wu et al., 2013), such as the Mediterranean diet (Mone et al., 2016), improves GERD symptoms. Therefore, improving diets can decrease the occurrence of GERD and should be considered a cost-effective strategy instead of pharmacotherapy.

Review studies have investigated predictors of GERD risk in terms of food related factors such as probiotics (Cheng and Ouwehand, 2020) and food components (Surdea-Blaga et al., 2019). However, to our knowledge, no systematic reviews have been conducted to assess the impact of diet on risk of reflux disease. We conducted a systematical review of articles investigating the association between food and dietary patterns with GERD.

MATERIALS AND METHODS

Search strategy

The literature search was conducted by two independent researchers using electronic databases, including the Web of Sciences, PubMed/MEDLINE, and Scopus, to identify relevant publications up to August 2020. This study was given ethical approval by the Ethics Committee of Research Council of Kermanshah University of Medical Sciences (Ethics Code: IR.KUMS.REC.1399.941).

We performed the systematic search using Medical Subject Headings (MeSH) along with non-MeSH keywords in the title and abstract as follows: "Diet" OR "Food" OR "Dietary Pattern" OR "Food Pattern" AND "Gastroesophageal Reflux" OR "Gastric Acid Reflux" OR "Gastroesophageal Reflux Disease" OR "GERD" OR "Esophageal Reflux" OR "Pyrosis" OR "Pyroses" OR "Heartburn" OR "Barrett's Esophagus". We did not consider any restrictions in terms of language, time of publication, and study location.

Inclusion and exclusion criteria

Eligible studies were performed on adults and evaluated all components of the dietary patterns and risk of reflux disease. In addition, we considered all observational studies, including cross-sectional, case-control, prospective, and retrospective studies. Interventional studies were not included in this study since the duration of exposure was short. Overall, 991 articles were identified during the initial search and duplicate studies (n=26) were removed. The remaining studies were screened based on topic and 833 irrelevant studies were excluded. Thereafter, 119 studies were reviewed in more detail, and 24 classed as irrelevant (including 17 that did not assess dietary pattern) and 52 that did not evaluate our outcome ("reflux diseases") were excluded. In addition, the qualification of 26 articles were evaluated and one study was excluded because of low quality. In total, 25 articles were eligible for inclusion in this review study (Fig. 1).

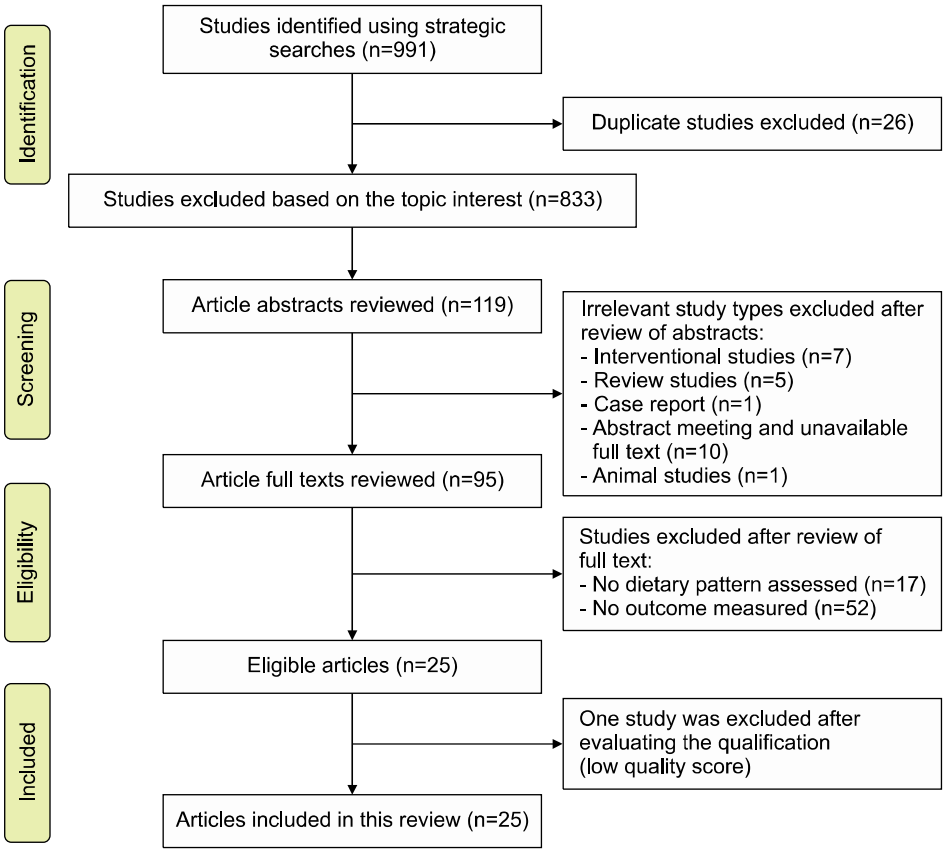

Fig. 1. Flow chart of the search and publication selection.

Quality assessment

Using all the data extracted, we scored the risk of bias of the selected studies on a six-point scale using a modified version of the Quality in Prognosis Studies (Hayden et al., 2006). Using this system, we assessed the quality of individual studies using the following criteria (one point per criterion): (I) study participation (the study sample represents the key characteristics of the population of interest sufficiently well to limit potential bias to the results); (II) study attrition (loss to follow-up is not associated with key characteristics); (III) prognostic factor measurement (prognostic factors of interest are measured in study participants in such a way that potential bias is limited); (IV) confounding measurement and account (outcomes of interest are measured in study participants in such a way that potential bias is limited); (V) outcome measurement (important potential confounders are appropriately accounted for, limiting potential bias with respect to the prognostic factor of interest); and (VI) analysis (the statistical analysis is appropriate for the design of the study, and limits the potential for invalid results). Studies with a score between 0 and 3 points were considered to be of low quality, while studies with a score >3 to 6 were considered to be of high quality.

Data extraction

Data extraction was performed independently by two researchers using a data collection checklist. Any disagreement has been discussed and resolved accordingly. For each article, the first author's name, publication year, study design (trial/prospective/cross-sectional/case control), sample size, study population demographics [age, sex, body mass index (BMI), and country], dietary assessment tools, dietary components, and outcomes (all reported data on association between GERD and dietary components) were extracted. All data are presented in the Results.

RESULTS

Study characteristics

In this literature review, we identified 25 eligible studies with quality scores from 3.5 to 6.0. As shown in Table 1, these studies included 5 case-control studies (Nandurkar et al., 2004; Murphy et al., 2010; Wu et al., 2013; Asl et al., 2015; Ebrahimi-Mameghani et al., 2017), 14 cross-sectional studies (El-Serag et al., 2005a; El-Serag et al., 2005b; Shapiro et al., 2007; Friedenberg et al., 2010; Kubo et al., 2014; Khodarahmi et al., 2016; Mone et al., 2016; Alkhathami et al., 2017; Eslami et al., 2017; Keshteli et al., 2017; Atta et al., 2019; Kim et al., 2019; Kariri et al., 2020; Rajaie et al., 2020), and 6 prospective studies (Ruhl and Everhart, 1999; Gutschow et al., 2005; Austin et al., 2006; Bhatia et al., 2011; López-Colombo et al., 2017; Wu et al., 2018). There were a total of 8 to 12,349 subjects per study. Studies were conducted in America (Ruhl and Everhart, 1999; Nandurkar et al., 2004; El-Serag et al., 2005a; El-Serag et al., 2005b; Austin et al., 2006; Shapiro et al., 2007; Friedenberg et al., 2010; Kubo et al., 2014; López-Colombo et al., 2017), Asia (Bhatia et al., 2011; Wu et al., 2013; Asl et al., 2015; Khodarahmi et al., 2016; Alkhathami et al., 2017; Ebrahimi-Mameghani et al., 2017; Eslami et al., 2017; Keshteli et al., 2017; Wu et al., 2018; Atta et al., 2019; Kim et al., 2019; Kariri et al., 2020; Rajaie et al., 2020), and Europe (Gutschow et al., 2005; Murphy et al., 2010; Mone et al., 2016). All studies included participates aged ≥18 years of age, and most assessed dietary intake using the food frequency questionnaire.

Study outcomes

A total of nine studies examined the negative effects of reflux-triggering foods, including high-fat, spicy, fried, and citrus foods, carbonated beverages, and tea (Shapiro et al., 2007; Kubo et al., 2014; Asl et al., 2015; Alkhathami et al., 2017; Eslami et al., 2017; López-Colombo et al., 2017; Atta et al., 2019; Kim et al., 2019; Kariri et al., 2020). Other food components, such as saturated fatty acids (SFA), monounsaturated fatty acids, polyunsaturated fatty acids, cholesterol, smoky foods, salty foods, coffee, alcohol, chocolate, and dairies, did not contribute to risk of GERD (Nandurkar et al., 2004; El-Serag et al., 2005a; El-Serag et al., 2005b; Shapiro et al., 2007; Friedenberg et al., 2010; Murphy et al., 2010; Bhatia et al., 2011; Wu et al., 2013; Kubo et al., 2014; Asl et al., 2015; Alkhathami et al., 2017; Eslami et al., 2017; López-Colombo et al., 2017; Atta et al., 2019). The results of studies examining the role of dietary components in reflux disease are shown in Table 2.

Shapiro et al. (2007), Kubo et al. (2014), Asl et al. (2015), and Kim et al. (2019) observed that high-fat diets contributed to the risk of reflux disease. However, Ruhl and Everhart (1999) and Wu et al. (2013) did not observe any association between high-fat diets and reflux disease. On the other hand, Ruhl and Everhart (1999) reported that cholesterol may increase the risk of reflux disease, and El-Serag et al. (2005a; 2005b) found the relationship between dietary fat and reflux disease was non-significant after adjusting for BMI, energy intake, and demographic characteristics. Moreover, daily intakes of total fat, SFA, cholesterol, energy from dietary fat, and fat were significantly higher in subjects without GERD symptoms than those with GERD symptoms.

Fast food is a possible causal risk factor for reflux disease (Alkhathami et al., 2017; Kariri et al., 2020). Indeed, Kubo et al. (2014) and Atta et al. (2019) found a significant relationship between fried foods and symptoms of

Table 1. Important characteristics of the included studies

Reference	Study population/design/country	Age (yr)	BMI (kg/m^2)	Sample size	Dietary assessment tool	Dietary component	Quality score
El-Serag et al. (2005a)	Employees at the Houston VA Medical Center (VAMC)/ cross-sectional/USA	18~70	44.5±11.3	111 men 260 women	GERD FFQ	Fruits, vegetables, dairy, grain, and meat	6
El-Serag et al. (2005b)	Employees at the Houston VAMC/ cross-sectional/USA	18~70	44.9±11.0	111 men 261 women	GERDQ FFQ	Grains, dairy, fiber enrichment diet, fruits, vegetables, and alcohol	6
Khodarahmi et al. (2016)	Iranian adults/cross-sectional/Iran	>30	–	3,846	FFQ	Fast-food pattern, traditional pattern, and vegetarian pattern	6
Keshteli et al. (2017)	Non-academic members of staff of Isfahan University of Medical Sciences/cross-sectional/Iran	19~70	24.7	2,987	FFQ	Glycemic index and glycemic load	6
Rajaie et al. (2020)	General adult population/ cross-sectional/Iran	>18	25.1	2,046 men 2,587 women	FFQ	Pepper, curry, ginger, cinnamon, and turmeric	5.5
Wu et al. (2013)	Patients with reflux esophagitis/case-control/China	19~82	23.0±0.2	268 men 269 women	RDQ FFQ	Grains, potatoes, meat, fish and shrimp, eggs, dark-colored vegetables, light colored vegetables, fruits, nuts, beans and bean products, milk and dairy products, desserts, condiments, soft drinks, western-style fast food, and animal oils	5.5
Nandurkar et al. (2004)	Community subjects/case-control/USA	20~50	43.5±9.2	90 men 121 women	FFQ	Fiber, total fat, and coffee	5
Kubo et al. (2014)	Comparing 317 GERD patients to 182 asymptomatic population controls/cross-sectional/USA	20~80	28.9±5.2	334 men 155 women	FFQ	Soft drink, coffee, tea, bear, liquor, citrus fruits, tomato, fried food, and total fat	4.5
López-Colombo et al. (2017)	Primary care patients/cohort/Mexico	18~49	26.9±3.9	32 men 42 women	Lifestyle questionnaire	Coffee, citrus fruits, chocolate, spicy foods, and carbonated drinks	5
Eslami et al. (2017)	Patients with GERD/cross-sectional/Iran	20~60	BMI<30 219 (76.8) BMI≥30 66 (23.2)	156 men 349 women	Lifestyle questionnaire	Coffee, tea, citrus fruits, chocolate, fast food, spicy foods, and carbonated drinks	5
Friedenberg et al. (2010)	Living in the zip code immediately surrounding Temple University Hospital/cross sectional/USA	≥18	31.4±11.3	195 men 308 women	–	Coffee, fresh fruit, cooked vegetables, salad, soda, and fast food	5
Murphy et al. (2010)	Caucasian participants from throughout the island of Ireland/case-control/Northern Ireland	62±12	27.7±4.5	266 men	FFQ	Vitamin C, vitamin E, total carotenoids, zinc, copper, and selenium	6

Table 1. Continued

Reference	Study population/design/country	Age (yr)	BMI (kg/m^2)	Sample size	Dietary assessment tool	Dietary component	Quality score
Asl et al. (2015)	Subjects with GERD symptoms/case-control/Iran	45±14	BMI<24 61 (29) BMI: 25~30 102 (48.6) BMI≥30 47 (22.4)	180 men 240 women	–	Smoky, salty, spicy foods, tea, soda, and coffee	3.5
Ruhl and Everhart (1999)	The first National Health and Nutrition Examination Survey (NHANES I)/cohort/USA	18.5	BMI<22 (3.52) BMI: 22.0~24.9 (5.02) BMI: 24.9~28.2 (6.18) BMI≥28.2 (8.42)	4,793 men 7,556 women	FFQ	High fat foods, and tea/coffee	6
Bhatia et al. (2011)	12 centers from different regions of India/prospective/India	38<	BMI<19 26 (10.6) BMI: 19.0~24.9 132 (53.9) BMI: 25~29.9 67 (27.3) BMI≥30 20 (8.2)	1,647 men 1,577 women	–	Vegetarian food, meat, fruits, and aerated drinks, tea/coffee, and spicy food	5
Mone et al. (2016)	Primary health-care centers in Tirana/cross-sectional/ the Netherlands	50.2±8.7	BMI<25 85 (23.8) BMI: 25~29.9 178 (49.9) BMI≥30 94 (26.3)	333 men 484 women	–	Non-Mediterranean diet	5
Alkhathami et al. (2017)	Community of Saudi Arabia/cross-sectional/ Saudi Arabia	–	BMI<18.5 32 (17.7) BMI: 18.5~24.9 201 (24.1) BMI: 24.9~30 182 (30.8) BMI≥30 172 (39.4)`1	2,043	GERD	Dinks, salty, spicy foods, pickles, fast food, fiber, greasy, and chocolate	3.5
Atta et al. (2019)	Medical students from Rabigh and Jeddah branches of university/cross-sectional/ Saudi Arabia	–	BMI<18.5 3 (11.1) BMI: 18.5~24.9 22 (19.5) BMI: 24.9~30 10 (28.6) BMI≥30 16 (72.7)	197 men	GERD	Coffee, tea, chocolate, energetic drinks, soft drinks, and fried food	3.5

Table 1. Continued 2

Reference	Study population/design/country	Age (yr)	BMI (kg/m^2)	Sample size	Dietary assessment tool	Dietary component	Quality score
Kim et al. (2019)	Patients who underwent an upper gastrointestinal endoscopy at the Health Promotion Center of the Gangnam Severance Hospital/cross-sectional/South Korea	48.9±11.6	–	4,622 men 3,596 women	–	Caffeinated drinks, spicy food, and fatty foods	6
Kariri et al. (2020)	Saudi population in Jazan/ cross sectional/Saudi Arabia	19～50	BMI<18.5 (26.2) BMI: 18.5～24.9 (29.9) BMI: 24.9～30 (33.6) BMI≥30 (35.3)	589 men 264 women	GERD	Fast food, spicy food, soft drinks, tea, and coffee	3.5
Shapiro et al. (2007)	Patients were recruited from Primary Care and Gastrointestinal Clinics/cross-sectional/USA	24±75	27.5±1.0	27 men 13 women	24-h dietary recalls	Alcohol, sea foods, MUFA, and PUFA	5
Gutschow et al. (2005)	Patients who presented to gastrointestinal function laboratory/ prospective/Germany	16～77	–	24 men 16 women	–	White diet	4
Austin et al. (2006)	Participants were recruited via the internal medicine and family Practice clinics at the University of North Carolina/prospective/USA	18～70	43.5±9.2	8 men	GSAS-ds	Low carbohydrates	3.5
Ebrahimi-Mameghani et al. (2017)	Patients referred to clinic of Tabriz University of Medical Sciences/case-control/Iran	21.9～48.2	–	57 men 160 women	3-day food record	High protein	5.5
Wu et al. (2018)	Patients with reflux disease/prospective crossover/Taiwan	52±12	24.3±3.8	7 men 5 women	GERD	High carbohydrate diet	5

Values are presented as mean±SD or number (%).
BMI, body mass index; GERD, gastroesophageal reflux disease; FFQ, food frequency questionnaire; RDQ, reflux diagnostic questionnaire; GSAS-ds, gastroesophageal reflux disease symptom assessment scale-distress subscale; -, not available.

Table 2. Summary of findings on the relationship of each dietary component with gastroesophageal reflux disease

Dietary component	Reference	Finding
High fat diets	Asl et al. (2015)	Positive relationship between eating fatty foods and GERD (*P*-value: 0.0001)
	Ruhl and Everhart (1991)	No significant relationship of GERD with the fatty food quartiles (OR quartile 4 vs 1: 0.84; 95% CI: 0.65~1.07)
	Wu et al. (2013)	No significant relationship between eating fatty foods and GERD (OR: 1.24; 95% CI: 0.93~1.84)
	Kim et al. (2019)	Positive relationship between eating fatty foods and GERD (OR: 1.21; 95% CI: 1.08~1.35)
	Kubo et al. (2014)	A positive relationship between eating fatty food and GERD tertiles (OR tertiles 3 vs 1: 1.77; 95% CI: 1.07~2.93)
	Shapiro et al. (2007)	No significant relationship between eating fatty foods and GERD (OR: 2.1; 95% CI: 0.9~5.0).
SFA	Shapiro et al. (2007)	No significant relationship between eating SFA and GERD (OR: 1.4; 95% CI: 0.64~3.02).
	Wu et al. (2013)	No significant relationship between eating SFA and GERD (OR: 1.05; 95% CI: 0.81~1.47)
	El-Serag et al. (2005a) El-Serag et al. (2005b)	No significant relationship between eating SFA and GERD (*P*-value: 0.09; OR:1.71; 95% CI: 0.92~3.10)
MUFA	Shapiro et al. (2007)	No significant relationship between eating MUFA and GERD (OR: 1.2; 95% CI: 0.53~2.80).
PUFA	Shapiro et al. (2007)	No significant relationship between eating PUFA and GERD (OR: 0.8; 95% CI: 0.36~1.80).
Cholesterol	Shapiro et al. (2007)	No significant relationship between eating cholesterol and GERD (OR: 2.8; 95% CI: 1.2~6.5).
	Ruhl and Everhart (1991)	Positive relationship between eating cholesterol and GERD (OR quartile 4 vs 1: 1.37; 95% CI: 1.04~1.79)
	Wu et al. (2013)	No significant relationship between eatingcholesterol and GERD (OR: 0.96; 95% CI: 0.74~1.25)
	El-Serag et al. (2005a) El-Serag et al. (2005b)	No significant relationship between cholesterol and GERD (*P*-value: 0.28; OR:1.34; 95% CI: 1.02~1.76)
Fast food	Eslami et al. (2017)	No significant relationship between eating fast food and GERD (OR: 0.87; 95% CI: 0.42~1.82)
	Kariri et al. (2020)	Positive relationship between eating fast food and GERD (*P*-value: 0)
	Friedenberg et al. (2010)	No significant relationship between eating fast food and GERD (*P*-value: 0.49)
	Alkhathami et al. (2017)	Positive relationship between fast food and GERD (*P*-value: 0.001)
Spicy foods	Eslami et al. (2017)	No significant relationship between eating spicy food and GERD (OR: 0.97; 95% CI: 0.66~1.45)
	Alkhathami et al. (2017)	Positive relationship between spicy food and GERD (*P*-value: 0.023)
	Asl et al. (2015)	Positive relationship between spicy foods and GERD (*P*-value: 0.006)
	Bhatia et al. (2011)	No significant relationship between spicy food and GERD (*P*-value: 0.621; OR: 0.902; 95% CI: 0.621~1.355)
	Kariri et al. (2020)	Positive relationship between eating spicy food and GERD (*P*-value: 0)
	López-Colombo et al. (2017)	No significant relationship between eating spicy food and GERD (OR tertiles 3 vs 1: 4.06; 95% CI: 0.47~34.59)
Smoky food	Asl et al. (2015)	No significant relationship between smoky foods and GERD (*P*-value>0.05)
Fried food	Kubo et al. (2014)	Positive relationship between fried food and GERD (OR: 1.52; 95% CI: 0.947~2.45)
	Bhatia et al. (2011)	No significant relationship between eating fried food and GERD (OR: 1.148; 95% CI: 0.803~1.641)
	Atta et al. (2019)	Positive relationship between eating fried food and GERD (*P*-value: 0.02)
Salty foods	Alkhathami et al. (2017)	No significant relationship between eating salty foods and GERD (*P*-value: 0.353)
	Asl et al. (2015)	No significant relationship between eating salty foods and GERD (*P*-value>0.05)
	Wu et al. (2013)	A positive relationship between eating salty food and GERD (*P*-value<0.01; OR: 9.93; 95% CI: 5.33~18.49)
Fruits	El-Serag et al. (2005a) El-Serag et al. (2005b)	No significant relationship between eating fruits and GERD (*P*-value: 0.14; OR:0.77; 95% CI: 0.54~1.10)
	Wu et al. (2013)	Negative relationship between eating fruits and GERD (*P*-value<0.01; OR: 0.65; 95% CI: 0.51~0.83)
	Friedenberg et al. (2010)	No significant relationship between eating fruits and GERD (*P*-value: 0.42)
Citrus fruit	López-Colombo et al. (2017)	Positive relationship between eating citrus fruits and GERD (P-value: 0.01; OR: 14.76; 95% CI: 1.90~114.57)

Table 2. Continued

Dietary component	Reference	Finding
Citrus	Kubo et al. (2014)	Positive relationship between eating citrus and GERD (OR≥2 serving/wk vs. <2 serving/wk: 0.62; 95% CI: 0.41~0.94)
	Eslami et al. (2017)	Positive relationship between eating citrus and GERD (*P*-value: 0.08; OR: 2.22; 95% CI: 1.3~3.81)
	Alkhathami et al. (2017)	Positive relationship between eating citrus and GERD (*P*-value: 0)
Vegetables	El-Serag et al. (2005a) El-Serag et al. (2005b)	No significant relationship between eating vegetable and GERD (*P*-value: 0.47; OR: 0.9; 95% CI: 0.67~1.20)
	Wu et al. (2013)	No significant relationship between eating vegetables and GERD (*P*-value: 0.49; OR: 1.13; 95% CI: 0.80~1.58)
Non vegetarian diet	Bhatia et al. (2011)	Negative relationship between non-vegetarian diet and GERD (*P*-value<0.001; OR: 0.34; 95% CI: 0.211~0.545)
Cooked vegetable	Friedenberg et al. (2010)	No significant relationship between eating cooked vegetable and GERD (*P*-value: 0.55)
Salad	Friedenberg et al. (2010)	No significant relationship between eating salad and GERD (*P*-value: 0.21)
Fiber	El-Serag et al. (2005a) El-Serag et al. (2005b)	No significant relationship between eating fiber and GERD (*P*-value: 0.04; OR: 0.72; 95% CI: 0.53~0.90)
	Wu et al. (2013)	No significant relationship between eating fiber and GERD (*P*-value: 0.16; OR: 0.818; 95% CI: 0.619~1.080)
	Nandurkar et al. (2004)	No significant relationship between eating fiber and GERD (OR: 0.5; 0.95% CI: 0.1~2.1)
	Alkhathami et al. (2017)	Negative relationship between eating fiber and GERD (*P*-value: 0)
Tea	Asl et al. (2015)	No significant relationship between eating tea and GERD (*P*-value>0.05)
	Eslami et al. (2017)	No significant relationship between cooked tea and GERD (*P*-value: 0.54)
	Kariri et al. (2020)	Positive relationship between drinking tea and GERD (*P*-value: 0)
	Alkhathami et al. (2017)	Positive relationship between drinking tea and GERD (*P*-value: 0)
	Atta et al. (2019)	No significant relationship between drinking tea and GERD (*P*-value: 0.18)
	Bhatia et al. (2011)	No significant relationship between GERD and drinking tea (*P*-value: 0.309; OR: 0.659; 95% CI: 0.291~1.495)
	Kubo et al. (2014)	Positive relationship between drinking tea and GERD (OR≥1/d vs. none: 1.86; 95% CI: 1.02~3.40)
Alcohol	Kubo et al. (2014)	No significant relationship between drinking alcohol and GERD (OR≥1 drink/d vs. no drinks/d: 0.83; 95% CI: 0.46~1.48)
	El-Serag et al. (2005a) El-Serag et al. (2005b)	No significant relationship between alcohol and GERD (*P*-value: 0.82; OR: 0.99; 95% CI: 0.88~1.10)
	Murphy et al. (2010)	No significant relationship between drinking alcohol and GERD (*P*-value: 0.55)
	Friedenberg et al. (2010)	No significant relationship between drinking alcohol and GERD (*P*-value: 0.57)
	Kim et al. (2019)	Negative relationship between drinking alcohol and GERD (*P*-value: 0.027; OR: 1.121; 95% CI: 1.013~1.241)
	López-Colombo et al. (2017)	No significant relationship between drinking alcohol and GERD (*P*-value: 0.95; OR: 1.05; 95% CI: 0.15~7.29)
	Nandurkar et al. (2004)	No significant relationship between drinking alcohol and GERD (OR: 1; 95% CI: 0.3~3.5)
	Wu et al. (2013)	No significant relationship between drinking alcohol and GERD (*P*-value: 0.25; OR: 0.78; 95% CI: 0.5~1.2)
Carbonated beverages	Friedenberg et al. (2010)	No significant relationship between drinking carbonated beverages and GERD (*P*-value: 0.5)
	Asl et al. (2015)	Positive relationship between drinking carbonated beverages and GERD (*P*-value: 0.0001)
	Eslami et al. (2017)	No significant relationship between drinking carbonated beverages and GERD (OR: 0.56; 95% CI: 0.28~1.10)
	Atta et al. (2019)	Positive relationship between drinking carbonated beverages and GERD (*P*-value: 0.07)
	Kubo et al. (2014)	Positive relationship between drinking carbonated beverages and GERD (OR: 1.86; 95% CI: 1.16~2.97)
	López-Colombo et al. (2017)	No significant relationship between carbonated beverages and GERD (*P*-value: 0.26; OR: 4.24; 95% CI: 0.32~55.05)
	Bhatia et al. (2011)	No significant relationship between drinking carbonated beverages and GERD (*P*-value: 0.1218; OR: 2.021; 95% CI: 0.830~4.894)
	Kariri et al. (2020)	Positive relationship between drinking carbonated beverages and GERD (*P*-value: 0)
	Alkhathami et al. (2017)	Positive relationship between drinking carbonated beverages and GERD (*P*-value: 0)

Table 2. Continued 2

Dietary component	Reference	Finding
Coffee	Atta et al. (2019)	No significant relationship between drinking coffee and GERD (*P*-value: 0.18)
	Kariri et al. (2020)	Positive relationship between drinking coffee and GERD (*P*-value: 0)
	Nandurkar et al. (2004)	No significant relationship between drinking coffee and GERD (OR: 0.4; 95% CI: 1.0～1.4)
	Bhatia et al. (2011)	No significant relationship between drinking coffee and GERD (*P*-value: 0.3094; OR: 0.659; 95% CI: 0.291～1.495)
	Kubo et al. (2014)	No significant relationship between drinking coffee and GERD (OR≥2/d vs. none: 0.89; 95% CI: 0.52～1.51)
	Asl et al. (2015)	No significant relationship between drinking coffee and GERD (*P*-value>0.05)
	Eslami et al. (2017)	No significant relationship between drinking coffee and GERD (OR: 0.82; 95% CI: 0.29～2.30)
	Alkhathami et al. (2017)	Positive relationship between drinking coffee and GERD (*P*-value: 0)
	Friedenberg et al. (2010)	No significant relationship between drinking coffee and GERD (*P*-value: 0.22)
	López-Colombo et al. (2017)	No significant relationship between drinking coffee and GERD (*P*-value: 0.12; OR: 12.02; 95% CI: 0.51～278.12)
Antioxidants	Murphy et al. (2010)	No significant relationship between antioxidant tertiles and GERD (OR tertiles 3 vs. 1: 1.6; 95% CI: 0.86～2.98)
Chocolate	Eslami et al. (2017)	No significant relationship between eating chocolate and GERD (OR: 1.21; 95% CI: 0.80～1.82)
	López-Colombo et al. (2017)	No significant relationship between eating chocolate and GERD (*P*-value: 0.85; OR: 0.86; 95% CI: 0.18～4.06)
	Alkhathami et al. (2017)	Positive relationship between eating chocolate and GERD (*P*-value: 0.023)
	Atta et al. (2019)	No significant relationship between eating chocolate and GERD (*P*-value: 0.18)
Dairy	El-Serag et al. (2005a) El-Serag et al. (2005b)	No significant relationship between eating dairy products and GERD (*P*-value: 0.58; OR: 1.09; 95% CI: 0.79～1.50)
	Wu et al. (2013)	No significant relationship between eating vegetables and GERD (*P*-value: 0.06; OR: 1.2; 95% CI: 1.00～1.44)
Grain	El-Serag et al. (2005a) El-Serag et al. (2005b)	No significant relationship between eating grains and GERD (*P*-value: 0.82; OR: 1.05; 95% CI: 0.7～1.5)
	Wu et al. (2013)	Negative relationship between eating grains and GERD (*P*-value: 0.01; OR: 0.58; 95% CI: 0.39～0.85)

GERD, gastroesophageal reflux disease; OR, odds ratio; CI, confidence interval; SFA, saturated fattyacids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.

GERD.

Keshteli et al. (2017) showed that foods with high-glycemic indexes increase the risk of uninvestigated heartburn [odd ratio (OR=1.75; 95% confidence interval (CI): 1.03, 2.97; *P*-value=0.04)] and uninvestigated chronic dyspepsia (OR=2.14; 95% CI: 1.04, 4.37; *P*-value=0.04) in men but not in women, even after adjusting for potential confounders, such as age, marital status, medications, education, sleeping, eating rate, and intake of spicy foods, cocoa, sugar-sweetened beverages, tea, coffee, energy, fat, fructose, and fiber. However, Kubo et al. (2014), Asl et al. (2015), Alkhathami et al. (2017), Atta et al. (2019), and Kariri et al. (2020) found a significant relationship between reflux disease and carbonated beverages, including soda, aerated, and soft drinks.

Wu et al. (2013) observed a negative relationship between intake of fruits and reflux disease, whereas other studies did not observe any significant association (Friedenberg et al., 2010; El-Serag et al., 2005a; El-Serag et al., 2005b). Kubo et al. (2014), Alkhathami et al. (2017), Eslami et al. (2017), and López-Colombo et al. (2017) found that citrus was associated with risk of GERD. Moreover, consumption of non-vegetarian foods was an independent predictor of GERD (Bhatia et al., 2011). However, El-Serag et al. (2005a; 2005b) and Wu et al. (2013) did not observe any association between vegetables and GERD.

Two studies reported that fast foods, including sausage, fried chicken, pizza, hamburgers, french fries, and doughnuts, were not significantly associated with reflux disease (Friedenberg et al., 2010; Eslami et al., 2017). Alkhathami et al. (2017) reported that the prevalence of GERD was higher in those who did not consume dietary fibers regularly.

DISCUSSION

To the best of our knowledge, this is the first systematic review to investigate the relationship between different foods and dietary patterns with the occurrence of GERD. Unlike previous review studies, the present study reviewed all previous observational studies. The results of this study showed a significant association between adherences to high-fat diets and increased the risk of GERD.

Consumption of large high-fat meals appears to accelerate development of GERD (Surdea-Blaga et al., 2019) by reducing lower esophageal sphincter (LES) pressure (Kumar and Katz, 2013; Kubo et al., 2014; Asl et al., 2015). Furthermore, large high-fat meals are correlated with increased acid exposure time in patients compared with low fat meals (Kahrilas et al., 2008; Kubo et al., 2010; Castillo et al., 2015; Ireland et al., 2016; Sethi and Richter, 2017). However, previous review studies in 2000 and 2009 investigating the pathogenic relationship between eating habits and occurrence of GERD were unable support the effect of dietary fat on incidence of GERD (Meining and Classen, 2000; Festi et al., 2009). In contrast to the results of the present study, Eslick and Talley (2009) emphasized the relationship between high cholesterol consumption and increased risk of GERD. The authors concluded that confounding factors such as BMI, energy and demographic variables were responsible for this result.

Decreased LES pressure is involved in progression of GERD in overweight individuals consuming a high-cholesterol diet. However, further studies are needed to investigate the independent association of dietary fat as a risk factor for GERD. Several studies reported that consuming a high-fat diet and food rich in cholesterol and SFA (El-Serag et al., 2005a; El-Serag et al., 2005b; Shapiro et al., 2007; Wu et al., 2013), and unsaturated fatty acids (Shapiro et al., 2007) was not associated with increased risk of GERD. Similarly, review studies investigating the pathogenic relationship between eating habits and occurrence of GERD in 2000 (Meining and Classen, 2000) and 2009 (Festi et al., 2009) did not support an effect of dietary fat on the incidence of GERD.

Consuming high-salt foods (Asl et al., 2015; Alkhathami et al., 2017), spicy foods (Bhatia et al., 2011; Eslami et al., 2017; López-Colombo et al., 2017), smoky foods (Asl et al., 2015), and fast foods (Friedenberg et al., 2010; Eslami et al., 2017) does not significantly increase the risk of GERD. However, several studies have emphasized the relationship between consumption of high-salt foods (Wu et al., 2013) and high-spice foods (Asl et al., 2015; Alkhathami et al., 2017; Kariri et al., 2020) in accelerating development of GERD. Furthermore, Wu et al. (2013) showed that increasing salt intake is only effective in reducing LES pressure, and alone cannot increase the risk of GERD. In addition, in Asian populations, consuming high-spice foods followed by the habit of lying down after eating increases the risk of GERD. Lying down after eating may be a major cause of this disorder since it reduces LES pressure and affects the reflux of gastric contents (Asl et al., 2015).

Vegetables (cooked vegetables and salad) (El-Serag et al., 2005a; El-Serag et al., 2005b; Friedenberg et al., 2010; Wu et al., 2013), dietary fiber (Nandurkar et al., 2004; El-Serag et al., 2005a; El-Serag et al., 2005b; Wu et al., 2013), dairy products (El-Serag et al., 2005a; El-Serag et al., 2005b; Wu et al., 2013), and antioxidants (Murphy et al., 2010) were also not significantly associated with increased incidence of GERD. A previous review study investigating the relationship between diet and GERD showed that adherence to the Mediterranean diet (rich in vegetables, fiber, and antioxidants) (Winberg et al., 2012), could play a preventive role in GERD, especially

among patients with underlying diseases such as diabetes, heart disease, and cancer (Badillo and Francis, 2014; Newberry and Lynch, 2017). However, these results are inconsistent with those of the present study. Further studies are needed to assess the effectiveness of these food groups in preventing GERD in all individuals.

Consumption of high-caffeine products such as tea (Asl et al., 2015; Eslami et al., 2017), coffee (Nandurkar et al., 2004; Friedenberg et al., 2010; Kubo et al., 2014; Asl et al., 2015; Eslami et al., 2017; López-Colombo et al., 2017), and chocolate (Eslami et al., 2017; López-Colombo et al., 2017) are also not significantly associated with risk of developing GERD. Review studies investigating the relationship between lifestyle and GERD also did not support a role of high-caffeine sources in increasing the incidence of GERD (Meining and Classen, 2000; Kaltenbach et al., 2006; Vemulapalli, 2008). However, in 2014, a study suggested that tea consumption was associated with increased risk of GERD. These contradictory results may be due to differences between the types of tea consumed by the subjects in the study (Kubo et al., 2014). In addition, some studies have shown that coffee relaxes the LES, and increases percentage reflux time in the fasting state (Akbar and Howden, 2016).

Similarly to our results, other studies have not shown a significant relationship between alcohol consumption and increased risk of GERD (Festi et al., 2009; Kubo et al., 2010; Esmaillzadeh et al., 2013; Ireland et al., 2016). A further review study was conducted in parallel with our own, which showed that consumption of carbonated beverages increases the risk of GERD (Newberry and Lynch, 2017). Carbonated beverages may increase the likelihood of dysphagia reflexes by altering the acidity of the gastrointestinal tract, especially the stomach, and affecting digestion (intragastric residence time and inducing poor digestion) (Asl et al., 2015). Furthermore, these beverages contain high levels of acidity, added sugars and artificial sweeteners, and caffeine, which alter LES pressures and intraesophageal pH (Newberry and Lynch, 2017).

The results of the present study show that daily consumption of citrus increases risk of GERD (López-Colombo et al., 2017), which is consistent with the results of prior review studies (Meining and Classen, 2000; Kaltenbach et al., 2006; Sethi and Richter, 2017). These fruits increase the risk of GERD by reducing LES pressure or delayed gastric emptying (Eslami et al., 2017). A previous study conducted on adherence to dietary recommendations in GERD (Kubo et al., 2014) showed that frequent consumption of citrus fruits and juices of adjusted pH plays a preventive role in acid-sensitive individuals. Other compounds in citrus fruits in addition to acidity are also likely to play a preventive role. However, this study attributed infrequent citrus fruit intake to the strong recommendations on limiting these fruits in people predisposed to dysphagia, and did not provide any documented data linking this group of fruits to GERD (Kubo et al., 2014).

In the present study, a diet rich in fruits did not significantly effect on impact the risk of developing GERD. Nevertheless, results from previous review studies support the protective effect of fruits in reducing the incidence of reflux disorders (Wu et al., 2013; Badillo and Francis, 2014). Indeed, these studies attribute this positive effect to the high-fiber content in fruits (Badillo and Francis, 2014). Moreover, consumption of fruit juices increases pressure the gradient from the abdomen to the chest, resulting in GERD symptoms by increasing abdominal pressure or decreasing LES pressure (Fallah et al., 2020). Further studies are needed to investigate the relationship between different compounds in fruits and GERD.

In parallel with the current study, a review study was conducted in 2017 that showed whole grain consumption is significantly correlated with reduced incidence of GERD in people with underlying diseases such as diabetes, heart disease, and cancer (Badillo and Francis, 2014). Further studies are needed to evaluate the effectiveness of grain consumption in reducing the incidence of this disorder in all individuals. The inconsistencies between studies may arise from differences in the socio-demographic statuses of the study populations, sample sizes, and criteria used to diagnose reflux disease.

Review studies have several inherent limitations that should be considered. First, although the present study investigated the relationship between different food groups and GERD in adults of different races and in different geographical conditions by extracting data from observational studies, it was not possible to investigate different age groups due to data limitations. Hence, we recommend other researchers conduct systematic review of observational and clinical trial studies investigating the effect of diet on GERD in children. Second, there were significant heterogeneity between studies due to varying regimens, doses, duration, center settings, and populations. Hence, we could not determine the fundamental factors to explain the observed heterogeneity due to the limited number of studies and lack of information for pooling. Third, it is crucial to investigate grey literature as an important resource in systematic review in order to reduce publication bias, which was unfortunately neglected from the current study.

In this study, we concluded that diets rich in vegetables, fiber, antioxidants, and caffeine were not significantly associated with increased risk of dysphagia. However, consumption of citrus fruits, carbonate beverages, spicy, and fried food increases the risk of developing this disorder. Furthermore, we did not identify a specific diet that plays

an effective role in GERD. Other large-scale studies with robust study designs are needed to investigate the effect of different diets associated with this disorder in all age groups.

AVAILABILITY OF DATA AND MATERIALS

The datasets used and analyzed during the present study are available from the corresponding author on reasonable request.

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AUTHOR DISCLOSURE STATEMENT

The authors declare no conflict of interest.

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