The Journal of Laryngology & Otology

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Dietary modification for laryngopharyngeal reflux: systematic review

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Review Article

Dr H G Choi takes responsibility for the integrity of the content of the paper

Cite this article: Min C, Park B, Sim S, Choi HG. Dietary modification for laryngopharyngeal reflux: systematic review. *J Laryngol Otol* 2019;**133**:80–86. https://doi.org/10.1017/S0022215118002256

Accepted: 13 September 2018
First published online: 16 January 2019

Key words:
Laryngopharyngeal Reflux; Diet Therapy; Human

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Abstract

Objective. This study aimed to determine the relationship between laryngopharyngeal reflux and dietary modification.

Methods. A systematic review was conducted. The data sources for the study were PubMed, Embase, Cochrane Library and Web of Science. Articles were independently extracted by two authors according to inclusion and exclusion criteria. The outcome focus was laryngopharyngeal reflux improvement through diet or dietary behaviour.

Results. Of the 372 studies identified, 7 met our inclusion criteria. In these seven studies, laryngopharyngeal reflux symptoms improved following dietary modifications. However, the studies did not present the independent effect of each dietary factor on laryngopharyngeal reflux. Moreover, only one of the seven studies had a randomised controlled study design.

Conclusion. The reference studies of dietary modification for laryngopharyngeal reflux patients are not sufficient to provide recommendations.

Introduction

Laryngopharyngeal reflux (LPR) results from the retrograde flow of stomach contents, and involves contact with the larynx and pharynx.[1] General symptoms of LPR include throat clearing, persistent cough, globus sensation and voice quality changes.[2,3] LPR has been implicated in various laryngeal diseases.[1] The Reflux Finding Score and the Reflux Symptom Index are used for diagnosing LPR.[4,5] Each checklist contains symptoms of LPR and reflux, respectively. Although 24-hour pH monitoring is recognised as the 'gold standard' for diagnosing gastroesophageal reflux disease, it is less reliable for detecting LPR.[6,7] According to one report, the prevalence of LPR is as high as 10 per cent of all otolaryngology referrals.[8]

The common treatment methods for LPR involve proton pump inhibitors (PPIs) and lifestyle modifications, including dietary and behavioural changes.[9] Review articles usually recommend dietary changes, such as avoiding fats, alcohol, acidic foods, caffeine, chocolate, spicy foods and late-night meals.[3,10] However, these recommendations are not sufficiently supported by LPR study findings, and were derived from studies of gastroesophageal reflux disease[11,12] and other symptoms such as hoarseness.[13] These articles were not based on the direct treatment of LPR. One study[9] was mainly cited as the reference for dietary recommendations for LPR.[10,14] This study has been cited 86 times in other studies. The study was performed as a randomised placebo-controlled trial with a PPI group and a placebo-controlled group. However, this study only compared LPR symptoms before and after treatment. Moreover, both groups performed the same lifestyle modifications, which included avoiding fatty meals, caffeine and alcohol. Therefore, this study could not support the independent effect of lifestyle modification on LPR.[9]

In light of the lack of evidence supporting the effects of dietary modifications on LPR, we conducted this systematic review to investigate the relevance of dietary recommendations for LPR patients.

Materials and methods

We searched the PubMed, Embase, Cochrane Library and Web of Science databases for relevant studies using the keywords: 'laryngopharyngeal reflux', and 'diet', 'fasting', 'coffee', 'caffeine', 'chocolate', 'alcohol', 'fat', 'spicy' or 'acidic'. A study was included if it met all of the following criteria: (1) diet or dietary behaviour for LPR treatment as a study purpose; (2) conducted among adults (aged 17 years or more); (3) a human-based study; (4) written in English language; and (5) published from 1991 to 2018. The last search was performed in January 2018.

Two authors (BP and HGC) independently searched and read the titles and abstracts, and extracted those articles that did not meet our inclusion criteria. We first screened the titles of the retrieved studies, and excluded those articles not related to diet or dietary behaviours associated with LPR. After reading the abstracts of the remaining articles,

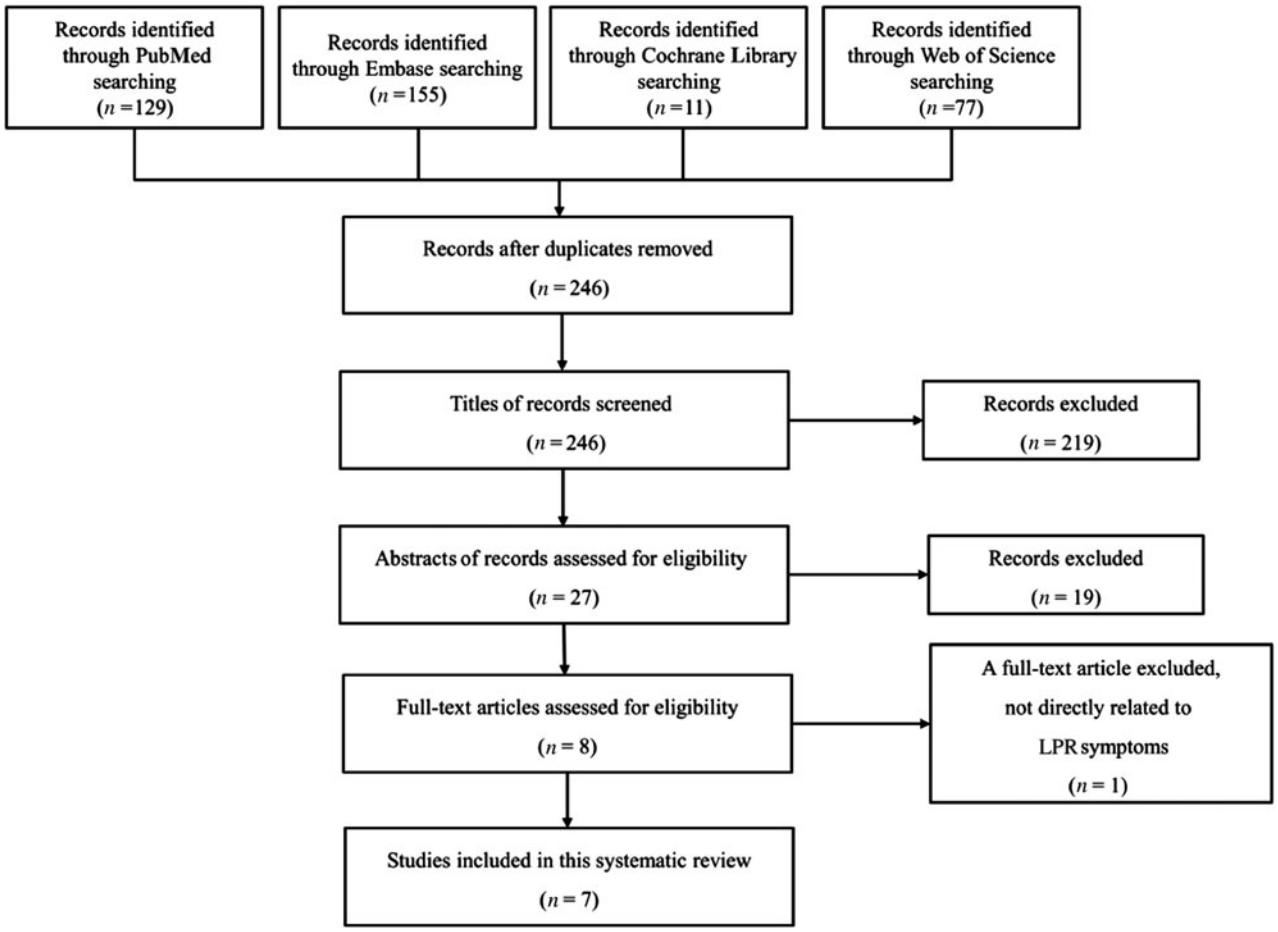

Fig. 1. Flow chart for study selection. LPR = laryngopharyngeal reflux

we then excluded those that did not meet our criteria. Finally, we selected the studies that matched our study purpose by reading the entire body of each article. The selected studies were summarised to report the relationship between LPR and diet or dietary behaviour.

Results

We searched a total of 372 records (129 from PubMed, 155 from Embase, 11 from the Cochrane Library and 77 from Web of Science). After removing duplicates, there were 246 records remaining. We excluded 219 records after reading the titles. The excluded records were not related to diet or dietary behaviours associated with LPR in humans or were not written in English. Nineteen records were subsequently excluded after reading the abstracts. The excluded records were not related to our study purpose. One article was excluded after reading the entire body of text because the article focused on oesophagitis symptoms.[15] Hence, seven studies met our criteria (Figure 1).

A summary of the study findings is shown in Table 1.[16–22] We found one randomised controlled trial,[16] two retrospective studies comparing two groups,[17,18] three intervention studies which compared before and after treatment conditions within a single group of participants,[19–21] and one observational study that compared fasting and non-fasting within a single group of participants.[22] Only one study included participants who were not LPR patients.[22] This study examined the relationship between LPR and dietary behaviour. Three studies examined the effect of diet,[17,18,20] and the remaining three studies assessed both diet and dietary behaviour.[16,19,21] The evaluation of LPR or its symptoms was performed using the Reflux Symptom Index and Reflux Finding Score in three studies.[20–22] In the other studies, this was evaluated: using the Reflux Symptom Index, Reflux Finding Score, Voice Handicap Index-10, Cough Severity Index, Dyspnea Index and the 10-item Eating Assessment Tool;[18] using only the Reflux Symptom Index;[17] by assessing LPR clinical symptoms, endoscopic laryngeal signs and employing a Likert scale;[16] or using a separate questionnaire.[19] The studies instructed or advised the participants on diet or dietary behaviour treatments,[16–21] or only observed and evaluated LPR symptoms.[22] None of the studies provided the participants with food directly related to dietary treatment.

Yang *et al.* conducted a retrospective study comparing 105 LPR patients in an anti-reflux programme group and 81 LPR patients in an anti-reflux medication group.[18] Ninety-six patients (95 per cent) in the anti-reflux programme group reported subjective improvements in LPR symptoms after the treatment. Comparisons pre- and post-treatment for the anti-reflux programme group revealed significant improvements in Reflux Symptom Index and Cough Severity Index scores (Reflux Symptom Index = 19.74 *vs* 14.38, $p < 0.001$; Cough Severity Index = 9.58 *vs* 7.47, $p = 0.008$). Sixteen patients in the anti-reflux programme group, whose LPR symptoms failed to improve with medications, had significant improvements in Cough Severity Index and 10-item Eating Assessment Tool scores (Cough Severity Index = 14.73 *vs* 10.22, $p = 0.04$; 10-item Eating Assessment Tool = 8.45 *vs* 7.17, $p = 0.02$). The 37 patients in the anti-reflux programme

https://doi.org/10.1017/S0022215118002256 Published online by Cambridge University Press

Table 1. Summary of outcomes from each study

Study (year)	Gender (*n*)	Age range (years)	Characteristics of study participants	Study design	Study duration	Treatment method	Diet or dietary behaviour	Evaluation	Main outcomes
Yang *et al.* (2018)[18]	M, 47; F, 139	17–88	Diagnosed with primary LPR	Retrospective study, comparing anti-reflux programme (study group) with anti-reflux medication (control group)	2 weeks	– Anti-reflux programme group: prescribed 2-week induction diet, high-dose anti-reflux medications (PPI &/or H_2 blocker), alkaline water & behavioural modifications – Anti-reflux medication group: prescribed high-dose anti-reflux medications (PPI &/or H_2 blocker) & behavioural modifications	– Anti-reflux programme group: low-acid diet, with alcohol avoidance & eating no less than 3 h before lying down – Anti-reflux medication group: alcohol avoidance, & eating no less than 3 h before lying down	VHI-10, RSI, CSI, DI, EAT-10, RFS	– Anti-reflux programme group: RSI & CSI scores were significantly improved post-treatment – Anti-reflux medication group had significantly worse VHI scores post-treatment
Zalvan *et al.* (2017)[17]	M, 76; F, 108	18–93	Patients with LPR symptoms	Retrospective study of 2 cohorts	6 weeks	– Group 1: treated with PPI & standard reflux diet, & precautions – Group 2: given verbal & written instructions to replace all beverages with alkaline water & to eat plant-based Mediterranean-style diet, & treated with standard reflux precautions	– Group 1: instructed to avoid coffee, tea, chocolate, soda, greasy, fried, fatty & spicy foods, & alcohol – Group 2: instructed to replace all beverages with alkaline water, & to eat a 90–95% plant-based diet with less than 5–10% food from animal-based products	RSI	Mean reduction in RSI was higher in group 2 than group 1
Nanda (2016)[16]	M, 88; F, 112	20–75	Patients with LPR symptoms	Randomised controlled trial, comparing study group with control group	90 days	– Study group: asked to follow lifestyle modifications & given medical treatment (rabeprazole) – Control group: given medical treatment (rabeprazole), with no additional treatment	– Study group: avoided eating hot, spicy & oily food, avoided drinking alcohol & beverages like coffee & tea, avoided lying down ≤1 h after meals & going to sleep ≤2 h after dinner	LPR clinical symptoms, endoscopic laryngeal signs, Likert scale responses	Percentage improvement in LPR symptoms was higher in study group than control group

Hamdan *et al.* (2012)[22]	M, 22	20–48	Fasted during month of Ramadan, with no LPR-related history	Observational study, comparing fasting with non-fasting in a single group	1 day of fasting (>12 h) & non-fasting	Evaluated participants while fasting & not fasting	Fasting or non-fasting	RSI, RFS	Some LPR symptoms were significantly greater for fasting than non-fasting, but there was no significant increase in LPR
Koufman (2011)[20]	M, 12; F, 8	24–72	LPR patients who failed to improve with medical treatment	Intervention study, comparing pre- & post-treatment values in a single group	2 weeks	Instructed to eat only low acid reflux diet, with no additional therapy	Low-acid diet	RSI, RFS	LPR symptoms were significantly improved on low-acid diet
Naiboglu *et al.* (2011)[21]	M, 24; F, 26	43.60 ± 12.02*	Patients with LPR symptoms	Intervention study, comparing pre- & post-treatment values in a single group	12 weeks	Advised to have anti-reflux dietary modifications, & included medical treatment (lansoprazole)	At least 3 h interval between dinner & sleep, avoiding excess fat, chocolate & coffee	RSI, RFS	LPR symptoms were significantly improved by empirical anti-reflux treatment
Giacchi *et al.* (2000)[19]	M, 24	48–80	Patients with LPR symptoms	Intervention study, comparing pre- & post-treatment values in a single group	4–6 months	Provided with standardised GORD diet & behaviour modification form with medical therapy, either H_2 blocker (cimetidine) or omeprazole	Avoided lying down 2–3 h before bedtime, eating fatty food, & drinking coffee, alcoholic beverages, & milk products	Questionnaire asking whether symptoms had changed	Avoiding eating or drinking 2–3 h before bed time significantly improved LPR symptoms

*Mean age ± standard deviation, in years. M = male; F = female; LPR = laryngopharyngeal reflux; PPI = proton pump inhibitor; h = hours; VHI-10 = Voice Handicap Index-10; RSI = Reflux Symptom Index; CSI = Cough Severity Index; DI = Dyspnea Index; EAT-10 = 10-item Eating Assessment Tool; RFS = Reflux Finding Score; GORD = gastroesophageal reflux disease

group who had cough had significant improvements in Reflux Symptom Index and Cough Severity Index scores (Reflux Symptom Index = 20.7 *vs* 16.42, $p = 0.001$; Cough Severity Index = 12.3 *vs* 8.2, $p = 0.005$). On the other hand, only 39 patients (48 per cent) of the anti-reflux medication group reported subjective improvements in LPR symptoms. Moreover, the Voice Handicap Index score in the anti-reflux medication group was significantly worsened post-treatment (9.93 *vs* 12.31, $p < 0.006$).[18]

Zalvan *et al.* conducted a retrospective study comparing two cohorts.[17] In group 1, 85 selected participants were treated with either esomeprazole or dexlansoprazole, and standard reflux diet and precautions, from 2010 to 2012. The precautions included avoiding coffee, tea, chocolate, soda, greasy, fried, fatty and spicy foods, and alcohol. In group 2, 99 selected participants were treated with alkaline water (pH > 8.0), a 90–95 per cent plant-based Mediterranean-style diet and standard reflux precautions, from 2013 to 2015. Comparisons pre- and post-treatment revealed a 6-point or greater reduction in Reflux Symptom Index scores in 54 per cent of group 1 and in 63 per cent of group 2, with no statistical difference. The reduction in Reflux Symptom Index was significantly greater in group 2 than group 1 (difference of 12.10, 95 per cent confidence interval = 1.53–22.68, $p < 0.05$).[17]

Nanda conducted a randomised controlled trial involving 200 LPR patients.[16] Following the 90-day study period, the 100 participants of the study group with lifestyle modifications had better and faster relief of clinical symptoms, with improvements in globus, hoarseness and chronic cough, compared with the 100 participants of the control group (35 per cent *vs* 42 per cent, 24 per cent *vs* 28 per cent, and 14 per cent *vs* 16 per cent, respectively). In addition, the frequencies of diagnostic laryngoscopy findings were lower in the study group compared with the control group after 90 days (laryngeal congestion or oedema, 25 per cent *vs* 33 per cent; posterior pharyngeal wall congestion, 11 per cent *vs* 15 per cent). Patient satisfaction was also greater in the study group than in the control group after 90 days (82 per cent *vs* 74 per cent).[16]

Hamdan *et al.* conducted an observational study of 22 healthy males to compare the percentage of LPR in patients who did or did not fast for over 12 hours.[22] According to the Reflux Symptom Index, the reflux symptoms of throat clearing, post-nasal drip and globus sensation were significantly higher in the fasting group than in the non-fasting group (68 per cent *vs* 64 per cent, 59 per cent *vs* 45 per cent, and 50 per cent *vs* 36 per cent, respectively; all $p < 0.05$). The frequency of the symptoms of thick endolaryngeal mucus, erythema or hyperaemia, and posterior commissure hypertrophy on the Reflux Finding Score were not different between the fasting and non-fasting groups (77 per cent *vs* 77 per cent, 68 per cent *vs* 77 per cent, and 64 per cent *vs* 55 per cent, respectively; all p values > 0.1). Moreover, the percentage of LPR patients was not different between the fasting and non-fasting groups (50 per cent and 31.8 per cent, respectively; $p = 0.361$).[22]

Koufman investigated whether a low-acid diet could improve LPR symptoms.[20] In that study, 20 LPR patients (12 males and 8 females) who failed to improve while receiving medical treatment were restricted to a low acid reflux diet. Compared with the pre-diet scores, the post-diet scores were significantly decreased (Reflux Symptom Index = 14.9 *vs* 8.6, and Reflux Finding Score = 12.0 *vs* 8.3; all $p < 0.05$).[20]

Naiboglu *et al.* demonstrated the effect of empirical anti-reflux treatment in 50 LPR patients (24 males and 26 females).[21] After treatment, both the Reflux Symptom Index and Reflux Finding Score were significantly decreased. The mean pre- and post-treatment Reflux Symptom Index scores were 21.42 and 12.88, respectively, and the mean pre- and post-treatment Reflux Finding Scores were 8.94 and 4.66, respectively (all p values < 0.001).[21]

Giacchi *et al.* investigated compliance with anti-reflux therapy (standardised gastroesophageal reflux disease diet and behaviour modification), and examined its effect on 24 males with LPR disease.[19] Avoiding eating or drinking 2–3 hours before bedtime ($r = 0.48$, $p < 0.05$), and raising the head of the bed while sleeping ($r = 0.54$, $p < 0.05$), improved LPR symptoms. However, dietary changes did not improve LPR symptoms ($r \leq 0.21$, $p \geq 0.05$), and compliance with this treatment was lower than for other treatments ($r = 0.2674$).[19]

Discussion

We reviewed seven studies to determine the relevance of dietary modifications for LPR treatment. The studies revealed that dietary modifications, such as not fasting, avoiding eating or drinking 2–3 hours before bed, consuming low-acid drink and food including alkaline water and a plant-based Mediterranean-style diet, and a reduced consumption of fat, chocolate and coffee, improved LPR symptoms. However, the studies did not show the effect of each dietary factor. Furthermore, Giacchi *et al.* showed no change in LPR symptoms with dietary changes, such as avoiding fatty foods, coffee, cola, tea, alcoholic beverages or milk products, because of low compliance.[19] In a study by Naiboglu *et al.*, the treatment combined lifestyle modifications with medication use. The lifestyle modifications in this study included not only dietary changes, but also behavioural changes such as elevation of the head of the bed and not wearing tight clothes.[21] We could not determine which treatment factors affected LPR in that study. Similarly, the study groups in the investigations by Yang *et al.*[18] Zalvan *et al.*[17] and Nanda[16] were all treated with combined dietary modifications.[16–18] Each dietary factor should have been investigated individually to determine whether it affected LPR symptoms.

The seven reference studies do not provide adequate evidence for dietary recommendations for LPR because of limitations in their study designs. Although Nanda conducted a randomised controlled trial, the study did not show a statistical difference.[16] Both the Zalvan *et al.*[17] and Yang *et al.*[18] studies had limitations associated with retrospective studies. For example, the baseline conditions, such as the periods of treatment, were not the same across patients. Moreover, because Zalvan *et al.* utilised two cohorts with different treatment periods, each cohort potentially has different study conditions, and this could lead to information bias. Hamdan *et al.* recruited a small number of healthy individuals and found it difficult to examine LPR symptoms over a short period of time.[22] Moreover, this study could not adjust for other LPR-related factors because it was performed using an observational study design. As the Koufman study recruited only severe LPR patients, the effect of a low-acid diet for general LPR patients remains unknown.[20] Naiboglu *et al.* demonstrated the effect of empirical treatment for LPR.[21] However, the independent effect of dietary modification is unknown because this study used medication and other behavioural modifications in addition to dietary modification. In a study by Giacchi *et al.*, the LPR symptom evaluation questionnaire utilised had not been validated.[19]

https://doi.org/10.1017/S0022215118002256 Published online by Cambridge University Press

The common limitations of the seven studies are as follows. First, the findings of a number of studies on the relationship between LPR and dietary modification are not sufficient and do not provide support for current dietary recommendations. We were not able to assess the risk of bias because of the heterogeneity of the study designs. Furthermore, there was a lack of LPR patients in the studies and the absence of a definite tool for diagnosing LPR. The current studies were mostly evaluated using the Reflux Symptom Index and Reflux Finding Score. In order to demonstrate a clear relationship between LPR and diet, a gold standard for diagnosing LPR should be developed.

Second, only one of the seven studies was a randomised controlled trial;[16] four of the studies comprised only a small number of participants within a single group,[19–22] resulting in a non-representative study population. The randomised controlled trial study design is considered the highest level of evidence.[23] Trials that are conducted without controls do not allow determination of whether the outcomes are a result of coincidence or the effect of treatment. Moreover, if the participants do not represent the general population, the results may not be generalisable to other individuals. In other words, these limitations are a question of reproducibility. The effect of treatment should be observed consistently under the same conditions with a proper study design.

Finally, the studies that examined dietary treatments did not provide sufficient details; for example, regarding the provision of food, or monitoring whether participants adhered to the guidelines. These studies only provided verbal or written instructions for participants. Without detailed treatment instructions, LPR patients cannot be treated in a practical manner. Moreover, participants' treatment compliance was unclear given the absence of monitoring by researchers.

Most of the evidence for dietary recommendations in treating LPR originates from inadequate studies with different patient groups, or from studies of gastroesophageal reflux disease or related symptoms.[11–13] Although the study by Steward *et al.*[9] was cited in some studies to support dietary recommendations for LPR,[10,14] we did not include this study for the following reasons. First, the study title did not include a keyword related to diet. Second, the lifestyle modification was not treated as an independent variable, but as an adjustment in that study. Finally, the lifestyle modifications included other behavioural changes, such as avoiding smoking and elevating the head of the bed. The study by Tsunoda *et al.*[12] was cited in eight other studies as mostly supporting dietary recommendations for LPR.[3,24] This study was also excluded from our review, for the following reasons. First, the study title was not found when we searched the data sources. Second, participants' symptoms were not caused by LPR but by gastroesophageal reflux disease. Third, the study participants included only five males investigated as a case series. Finally, the lifestyle modifications included behavioural modifications, such as avoiding smoking and elevating the upper body during sleep, in addition to dietary modifications.

Dietary recommendations for gastroesophageal reflux disease have been used for LPR treatment. However, gastroesophageal reflux disease guidelines should be cautiously considered for use as evidence for LPR lifestyle modifications. An adverse result was shown in gastroesophageal reflux disease patients in the study by Mardhiyah *et al.*[25] which is in contrast to our reference study by Hamdan *et al.*[22] Both studies involved fasting during Ramadan. Mardhiyah *et al.* suggested that fasting may help to reduce gastroesophageal reflux disease symptoms, which is inconsistent with the results of our review study. The gastroesophageal reflux disease guidelines for lifestyle modification treatment also lack evidence. According to these guidelines, lifestyle modifications, including dietary changes, are the initial treatments for gastroesophageal reflux disease.[26] However, a randomised trial of dietary changes had not been conducted when the guidelines were established. Instead, dietary changes were recommended based on small intervention studies.[27–31] Moreover, most of the references in recent review studies are cross-sectional studies, and few are prospective controlled trials.[32,33] Although LPR and gastroesophageal reflux disease are treated empirically at present, further randomised controlled trials are needed to support reliable evidence for dietary modifications.

Despite the existence of only a few reference studies, and bearing in mind their limitations, our review study is noteworthy. We identified and reviewed studies that directly show the relevance of dietary modifications for LPR treatment. Through this study, we can expect future studies developed from these reference studies, to enhance guidelines regarding the details of dietary modifications for LPR.

Conclusion

The evidence for current dietary recommendations for LPR mostly originate from references based on gastroesophageal reflux disease or related symptoms. Our study is the first systematic review to identify the relevance of dietary modifications for LPR treatment. Our selected studies suggest that dietary modifications might improve LPR. However, the studies are not sufficient to support dietary recommendations because they lack the appropriate study designs to independently show the relevance of dietary modifications for LPR treatment. The gastroesophageal reflux disease guidelines also lack evidence to support the LPR dietary recommendations. Moreover, different effects related to dietary changes have been shown in gastroesophageal reflux disease and LPR studies. Hence, longitudinal randomised controlled studies should be performed to determine the relevance of dietary modifications for LPR treatment, because the evidence supporting the current dietary recommendations is unclear.

Acknowledgements. This research was supported by Hallym University Research Fund and in part by a research grant (NRF-2015-R1D1A1A01060860) from the National Research Foundation of Korea.

Competing interests. None declared

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