



# Development of scores assessing the refluxogenic potential of diet of patients with laryngopharyngeal reflux

Jerome R. Lechien<sup>1,2,3,4</sup> · Francois Bobin<sup>1,5</sup> · Francois Mouawad<sup>1,6</sup> · Karol Zelenik<sup>1,7</sup> · Christian Calvo-Henriquez<sup>1,8</sup> · Carlos M. Chiesa-Estomba<sup>1,9</sup> · Necati Enver<sup>1,10</sup> · Andrea Nacci<sup>1,11</sup> · Maria Rosaria Barillari<sup>1,12</sup> · Antonio Schindler<sup>1,13</sup> · Lise Crevier-Buchman<sup>1,14</sup> · Stéphane Hans<sup>1,14</sup> · Virginie Simeone<sup>1,14</sup> · Elzbieta Wlodarczyk<sup>1,15</sup> · Bernard Harmegnies<sup>1,3</sup> · Marc Remacle<sup>1,16</sup> · Alexandra Rodriguez<sup>1,4</sup> · Didier Dequanter<sup>1,4</sup> · Pierre Eisendrath<sup>1,17</sup> · Giovanni Dapri<sup>1,18</sup> · Camille Finck<sup>1,19</sup> · Petros Karkos<sup>1,20</sup> · Hillevi Pendleton<sup>1,21</sup> · Tareck Ayad<sup>1,22</sup> · Vinciane Muls<sup>1,17</sup> · Sven Saussez<sup>1,2,4</sup>

Received: 16 August 2019 / Accepted: 1 September 2019 / Published online: 12 September 2019  
© Springer-Verlag GmbH Germany, part of Springer Nature 2019

## Abstract

**Objective** To develop clinical tools assessing the refluxogenic potential of foods and beverages (F&B) consumed by patients with laryngopharyngeal reflux (LPR).

**Methods** European experts of the LPR Study group of the Young-Otolaryngologists of the International Federation of Otorhino-laryngological societies were invited to identify the components of Western European F&B that would be associated with the development of LPR. Based on the list generated by experts, four authors conducted a systematic review to identify the F&B involved in the development of esophageal sphincter and motility dysfunctions, both mechanisms involved in the development of gastroesophageal reflux disease and LPR. Regarding the F&B components and the characteristics identified as important in the development of reflux, experts developed three rational scores for the assessment of the refluxogenic potential of F&B, a dish, or the overall diet of the patient.

**Results** Twenty-six European experts participated to the study and identified the following components of F&B as important in the development of LPR: pH; lipid, carbohydrate, protein composition; fiber composition of vegetables; alcohol degree; caffeine/theine composition; and high osmolality of beverage. A total of 72 relevant studies have contributed to identifying the Western European F&B that are highly susceptible to be involved in the development of reflux. The F&B characteristics were considered for developing a Refluxogenic Diet Score (REDS), allowing a categorization of F&B into five categories ranging from 1 (low refluxogenic F&B) to 5 (high refluxogenic F&B). From REDS, experts developed the Refluxogenic Score of a Dish (RESDI) and the Global Refluxogenic Diet Score (GRES), which allow the assessment of the refluxogenic potential of dish and the overall diet of the LPR patient, respectively.

**Conclusion** REDS, RESDI and GRES are proposed as objective scores for assessing the refluxogenic potential of F&B composing a dish or the overall diet of LPR patients. Future studies are needed to study the correlation between these scores and the development of LPR according to impedance–pH study.

**Keywords** Reflux · Diet · Laryngitis · Laryngopharyngeal · Score · Symptoms · Food · Beverage

## Introduction

Laryngopharyngeal reflux (LPR) is an inflammatory condition of the upper aerodigestive tract tissues related to direct and indirect effect of gastric or duodenal content reflux, which induces morphological changes in the upper aerodigestive tract [1]. LPR symptoms concern 10–30% of patients visiting otolaryngological departments [2], and are associated with significant impairment of quality of life [3]. The

---

Vinciane Muls and Sven Saussez have equally contributed to this work and should be regarded as the joint last authors.

---

✉ Jerome R. Lechien  
Jerome.Lechien@umons.ac.be

Extended author information available on the last page of the article

etiopathological mechanisms underlying the development of LPR are poorly understood and would involve poor diet [4–6]; autonomic nerve dysfunction [7, 8]; lower (LES) and upper (UES) esophageal sphincter dysfunctions [9, 10]; and other unknown factors. The involvement of diet in the development of reflux has been mainly studied in patients with gastroesophageal reflux disease (GERD) through clinical and experimental studies. Overall, acid, fat, and low-protein foods, caffeine, alcohol and high-sugar beverages are suspected to be associated with impairments of the tonicity of LES (transient relaxation) or esophageal dysmotility, leading to abnormal acid exposure in the esophagus and GERD-related symptoms [11]. In otolaryngology, studies found that following a low-fat, high-protein and alkaline diet is associated with higher symptom resolution in patients with LPR symptoms or with recalcitrant symptoms to proton pump inhibitors (PPIs) [6, 12].

Currently, there is no tool that precisely assesses the refluxogenic potential of foods, beverages or the overall diet of reflux patients. Moreover, a significant proportion of otolaryngologists are still unaware about the foods and beverages (F&B) that the patient has to avoid [13]. The lack of a diet tool makes subjective and difficult both the physician's judgment and the post-treatment assessment of the respect of anti-reflux diet. Such tool could undeniably contribute to improve the management of LPR patients through a better identification of the diet factors that would be associated with LPR; providing more objective information to LPR patients.

The LPR Study Group of Young-Otolaryngologists of Otorhinolaryngological Societies (YO-IFOS) is composed of international experts in the management of LPR. The aim of this study is to develop clinical tools assessing the refluxogenic potential of foods, beverages, and overall diet of patients with LPR.

## Materials and methods

This study was realized following three steps:

1. The establishment of components of F&B that may be involved in the development of reflux (panel of experts and survey).
2. The realization of a systematic review about the impact of diet on reflux. On one hand, the review considered the studies investigating the impact of diet on clinical findings of LPR. On the other hand, the review considered the studies describing the role of foods, beverages (and their related components) in the development of reflux.
3. The establishment of Refluxogenic Diet Scores.

## Panel selection and survey

### Panel selection

Experts in LPR came from the LPR Study Group of YO-IFOS, which coordinates many studies on reflux. Initially, the research committee of YO-IFOS identified experts if they were actively working on LPR over the past few years. Since the diet is strongly related to the world area, we only considered European experts to establish scores considering Western European F&B.

### Survey

A modified Delphi technique [14] was used, asking experts to review and rate a list of potential components of F&B that may be involved in the dysfunction of esophageal sphincters and motility. This survey was conducted through Survey Monkey (San Mateo, CA, USA), allowing each participant to complete the survey only once each. The survey itself was developed in iterative fashion, with drafts revised by three certified otolaryngologists and one dietician.

### Systematic review

Based on the list generated by experts, four authors conducted the systematic review through Google Scholar, PubMed, and Scopus search to identify the potential foods, beverages, and their related components that may be involved in the development of esophageal sphincter and motility dysfunctions. Studies that have evaluated the impact of diet on the clinical evolution of LPR were also identified. The diagnostic of GERD or LPR should be based on symptoms  $\pm$  findings  $\pm$  objective examination (pH monitoring). Studies investigating the impact of diet on healthy subjects were also considered. They selected studies that had database abstracts, available full texts, books or other works referring to the condition. In addition, references were obtained from citations within the retrieved articles.

### Development of Refluxogenic Diet Scores

Based on the findings of the literature, the authors extracted the composition and the relevant characteristics of F&B from publications or scientific books. Four experts (2 otolaryngologists, 1 gastroenterologist, and 1 dietician) and one statistician developed a 'refluxogenic coefficient of severity' of the selected F&B, which served for the development of Refluxogenic Diet Scores.

Three diet scores has been developed for assessing (1) the refluxogenic potential of F&B (Refluxogenic Diet Score, REDS); (2) the Refluxogenic Score of a Dish (RESDI); and

(3) the refluxogenic potential of the overall diet of the patient (Global Refluxogenic Score; GRES). Foods, beverages, and greasy substances were distinctly treated due to their different biochemical composition.

## Results

### Survey

Twenty-six experts agreed to participate, coming from 10 countries (Belgium, France, Spain, Turkish, Italy, Poland, Greece, Luxembourg, Sweden, and Czech Republic) and 22 universities or hospitals. The mean years of practice of the panel was  $17.6 \pm 11.4$  (range 1–36 years). The experts identified the following components of F&B as important in the development of reflux (>50% converging opinion): alcohol degree; fat composition; pH; caffeine/theine composition; carbohydrate composition; the high osmolality of beverage (high-sugar beverages); protein composition; and water composition (Fig. 1).

### Systematic review

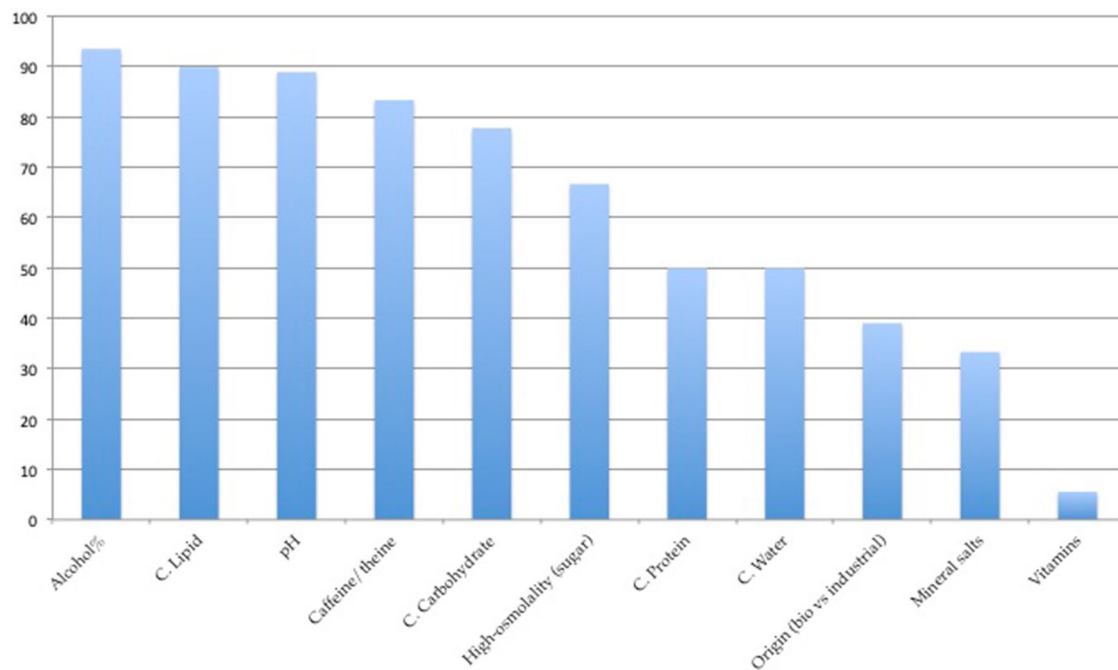
According to the survey results, the following keywords were used for the review: “food(s)”, “beverage(s)”; “pH”; “acidity”; “alkaline”; “composition”; “lipid”; “carbohydrate”; “protein”; “caffeine”; “tea”; “sugar”; and “alcohol”.

A total of 708 relevant studies were identified. From them, 9 described the impact of diet on LPR disease (Table 1) [4, 5, 12, 15–20] and 63 focused on the relationship between the consumption of specific F&B and the development of esophageal dysmotility or sphincter dysfunctions or GERD-related symptoms or findings (i.e., esophagitis, hiatal hernia, etc.).

Two books of European dietician societies (Belgium: La Table de composition des Aliments and France: Ciquel) [21, 22] were used for the establishment of the composition (+pH) of F&B. Figure 2 summarized the different steps of the study (chart flow). Figure 3 summarizes the diet characteristics associated with the development of reflux regarding the literature research.

### Diet and LPR

A few studies specifically analyzed the impact of diet in LPR patients (Table 1). In the majority of studies, the anti-reflux diet is composed of alkaline diet/water and avoidance of fat, alcohol, and coffee/tea. The respect of anti-reflux diet is associated with better symptom improvement in LPR patients treated with PPIs in comparison with those treated with PPIs alone [5, 20]. Note that the effect of diet was assessed in association with some lifestyle changes, i.e., elevation of the head of the bed; avoidance of meal before sleep; and non-fasting eat in the majority of studies. The evidence level of studies is low and there is an important heterogeneity in the LPR diagnostic, content of diet and the outcomes used to assess the diet treatment efficacy [12]. The



**Fig. 1** The most important (%) components of foods/beverages associated with reflux regarding experts. C composition

**Table 1** Studies evaluating the impact of diet on laryngopharyngeal reflux

References	Design	EL	Patient characteristics	Diet and treatment	Outcomes	Results
Giacchi et al. 2000 [15]	Prospective Uncontrolled	C	N: 24 suspected LPR Gender (F/M): 0/24 Age: 48–80 years	GERD diet (4 m) Avoid: fat food, dairy products, coffee, alcohol, tartare meals and elevation of the head of the bed Drugs: PPIs or anti-H2	LPR symptoms	Significant impact of avoidance of food and liquid before sleep and elevation of the head of bed, but not food habits
Siupsinskiene 2003 [16]	Monocentric Prospective Controlled	B	N: 113 suspected LPR Gender (F/M): 87/26 Age: 40.4 years	Gr1: PPIs (5 weeks) Gr2: anti-reflux diet (5 weeks) Content of diet: N.A. Gr3: nothing	LPR symptoms LPR findings	LPR patients treated by PPIs had better improvement of those who only respected a diet
Koufman 2011 [12]	Prospective Uncontrolled	C	N: 20 LPR Gender (F/M): 8/12 Age: 54.3 years	Strict diet (2 weeks) Avoid high-acid, fat food, coffee, tea, alcohol and dairy products Drugs: –	LPR symptoms	Strict diet based on alkaline, low fat, high protein foods significantly improve clinical outcomes of patients with recalcitrant LPR
Hamdan et al. 2012 [17]	Prospective Uncontrolled	C	N: 22 healthy subjects Gender (F/M): 0/22 Age: 30.2 years	Diet (–): Fasting (daily drink/eat) vs non-fasting (drink/eat night) during the Ramadan	LPR symptoms LPR findings	LPR symptoms were significantly higher in fasting group than in non-fasting group
Chappity 2014 [18]	Prospective Controlled	B	Gr1: suspected LPR (N=117) Gr2: suspected LPR (N=117) Gender: 199 F, 115 M Age: 36.9 years	Gr1: PPIs and diet (12 weeks) Gr2: Diet (12 weeks) Content of diet: N.A.	LPR symptoms LPR findings	Patients treated with PPIs and diet had a better improvement of symptoms than patients who did not receive PPIs
Nanda 2016 [19]	Prospective Randomized Controlled	B	N: 200 suspected LPR Gender (F/M): 112/88 Age: 20–75 years	Diet (3 m) Gr1: PPIs and diet Gr2: PPIs without diet	LPR symptoms LPR findings	The improvement of symptoms was higher in patients treated by PPIs and diet
Zalvan et al. 2017 [4]	Retrospective Uncontrolled	C	Gr1: 85 suspected LPR Gr2: 99 suspected LPR Gender (F/M): 108/76 Age: 58 years	Gr1: PPIs (6 weeks) and anti-reflux diet Gr2: plant-based, alkaline water, Mediterranean-style diet and standard GERD precautions	LPR symptoms LPR findings	Symptom improvement was higher in group 2 than group 1
Yang et al. 2018 [20]	Retrospective Uncontrolled	C	Gr1: N: 105 Gr2: N: 81 Gender (F/M): 139/47 Age: 17–88 years	Gr1: diet: alkaline water, alcohol avoidance, smoking cessation, avoidance of meals before sleep High doses PPIs ± anti-H2 (2 weeks) Gr2: PPIs ± anti-H2 (2 weeks)	LPR symptoms Voice symptoms	The anti-reflux program yielded rapid and substantial results and it compared favorably with medication and behavioral modification alone
Lechien et al. 2018 (5)	Retrospective Uncontrolled	C	Gr1: N: 26 Gr2: N: 39 Gender (F/M): 34/31 Age: 52.4 years	Gr 1: alkaline, low fat, high protein, low raw green vegetable, low spicy, and low alcohol diet Drugs: PPIs Gr 2: PPIs	LPR symptoms LPR findings	Patients treated with PPIs and diet had better improvement of symptoms than those who did not respect diet

*anti-H2* anti-histamine 2, *F/M* female/male, *m* month(s), *GERD* gastroesophageal reflux disease, *LPR* laryngopharyngeal reflux, *N.A.* not available, *PPI(s)* proton pump inhibitor(s)

LPR diagnostic was based in pH monitoring in one study [12].

### Fat foods

The majority of clinical and experimental studies showed that the fat foods, including chocolates, decrease the LES pressure and increase the esophageal acid exposure [21, 23–26]. In addition, fat foods would increase the sensitivity of the esophagus to acid exposure [11]. Two studies reported a positive association between the consumption of fat foods, GERD and the occurrence of esophagitis [27, 28]. The results of these studies are, however, balanced by other, which failed to find similar association [29, 30]. The refluxogenic potential of fat would be related to the slow gastric emptying time (related to the lipid digestion) leading to a higher number of transient relaxation of LES [31]. There are limited studies investigating the refluxogenic potential of chocolate. Two studies [25, 32] reported that the ingestion of chocolate syrup was associated with a decrease of LES pressure and a greater esophageal acid exposure time. In summary, fat foods may be considered as refluxogenic foods and have to be avoided by reflux patients.

### High-osmolality beverages, coffee, and tea

High-osmolality beverages, such as sport beverages or fruit juices, are predictors of GERD symptoms [33–35]; while carbonated beverages decrease LES pressure [36]. Some studies supported that coffee (caffeine) ingestion induces heartburn in GERD patients [35, 37, 38], and decreases the LES pressure [39], while other did not find negative impact of coffee on esophageal function [40].

As for coffee, the majority of studies investigated the individual impact of tea in GERD, but not in LPR patients. The chronic consumption of tea increases the risk of erosive esophagitis [41], and GERD [34, 35, 38, 42–45]. Only Wei et al. did not corroborate these findings [46]. A recent meta-analysis investigating the association between the consumption of tea and the development of GERD reported that there will be several subgroups of tea drinkers; some developing GERD, other not [47]. In the same way, it has been supported that there are inter-individual differences in the caffeine (or theine) metabolisms, which may lead to controversial results. Thus, the gender [48] and the intake of contraceptives [49] are both factors modifying the metabolism of caffeine. In summary, coffee and tea are beverages that are still suspected to be associated with reflux.

### Alcohol

The association between alcohol and LES dysfunction is not yet formally demonstrated regarding the studies that find

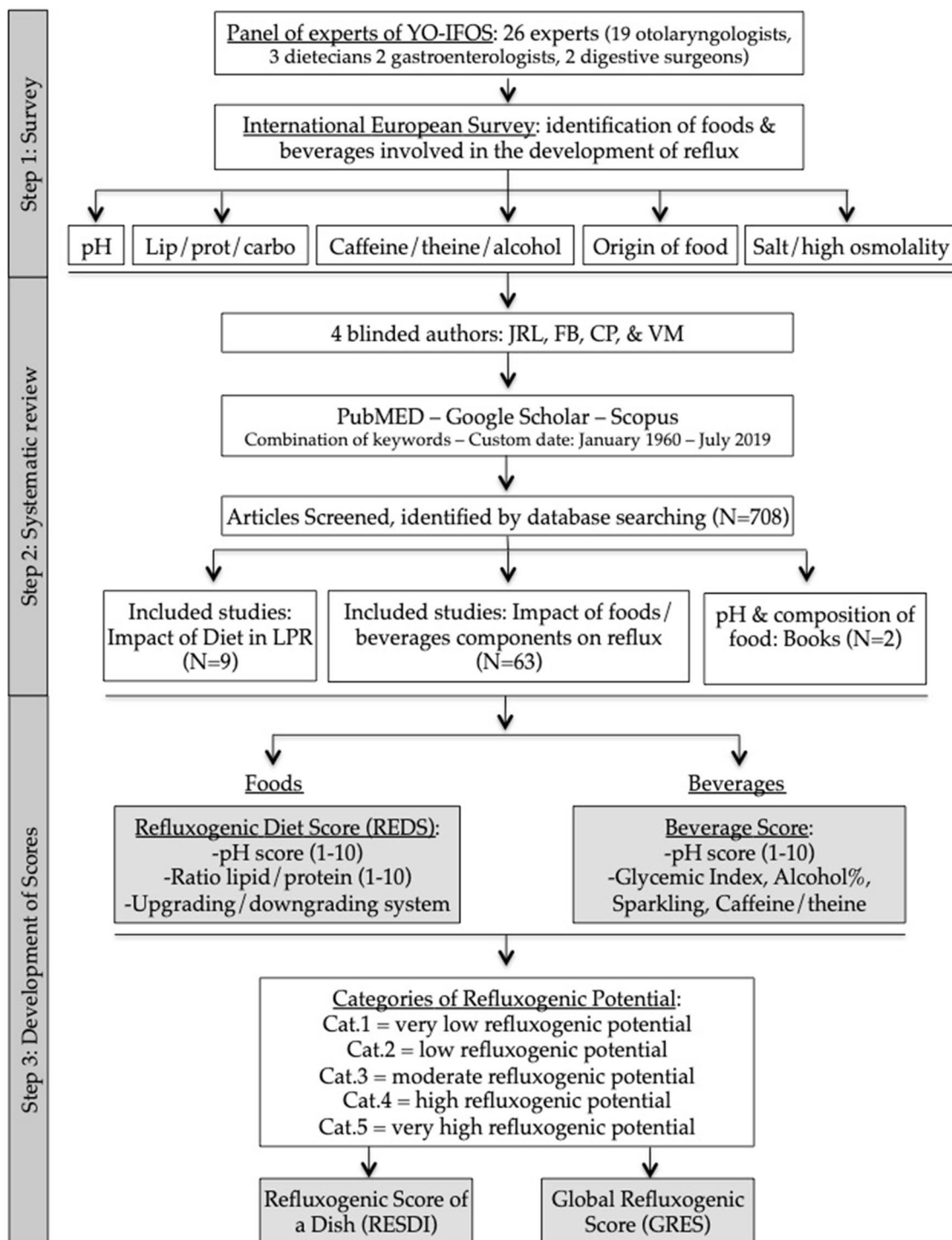
significant association [41, 45, 50–53] and those that did not find such association [54–57]. However, regarding the systematic review of Kaltenbach et al., alcohol appears to worsen the pH of gastric content, leading to GERD symptoms [11]. White wine would be worse than red wine, whereas beer would similarly lead to GERD symptoms than wine [58, 59]. Moreover, the alcohol intake would be associated with a reduced perception of esophageal acid reflux events [28]. Pathophysiologically, alcohol consumption may precipitate GERD by increasing acid secretion through gastrin stimulation, impairing LES pressure, esophageal motility and gastric emptying time [60]. Even a moderate consumption of alcohol would be associated with a decrease of esophageal pH in asymptomatic individuals with normal pH study measurements [61–64]. Although there are few controversial data, alcohol may be considered as risk factor of reflux.

### Spicy and fried foods

Fried and spicy foods have been suggested as two of the most common precipitating factors of GERD symptoms and LES insufficiency [65–67]. Precisely, chilli causes reflux-associated symptoms, including heartburn, chest discomfort, nausea, belching, abdominal discomfort and distension [68, 69]. Regarding some reports, chilli and spicy foods do not affect the overall esophageal motility but only alter the LES tonicity [70–72].

### Fruits and vegetables

Tomato, citrus fruits, onions and high-fiber vegetables are potential refluxogenic foods, involving many pathophysiological mechanisms such as increase of acidity or slow gastric emptying time (fiber composition) [54]. The consumption of tomato, or its based products [73] was higher in GERD patients than healthy controls [51, 74, 75]. Precisely, the two prominent organic acids present in tomato, i.e., citric and malic acids, are the most potent triggers of acid reflux in prone individuals and higher tomato consumers [76]. Citrus fruits are associated with the increase of heartburn [77, 78] and GERD [79], irrespective to the pH of citrus [37]. The high concentration of sugar in fruit juices consists of another component involved in the development of GERD [31]. Regarding the literature, onions may be a potent and long-lasting refluxogenic agent in heartburn patients [67], and may increase the number of reflux episodes and the esophageal acid exposure [67]. Similarly, mint is commonly thought to relax LES [11] although with controversial results [80].



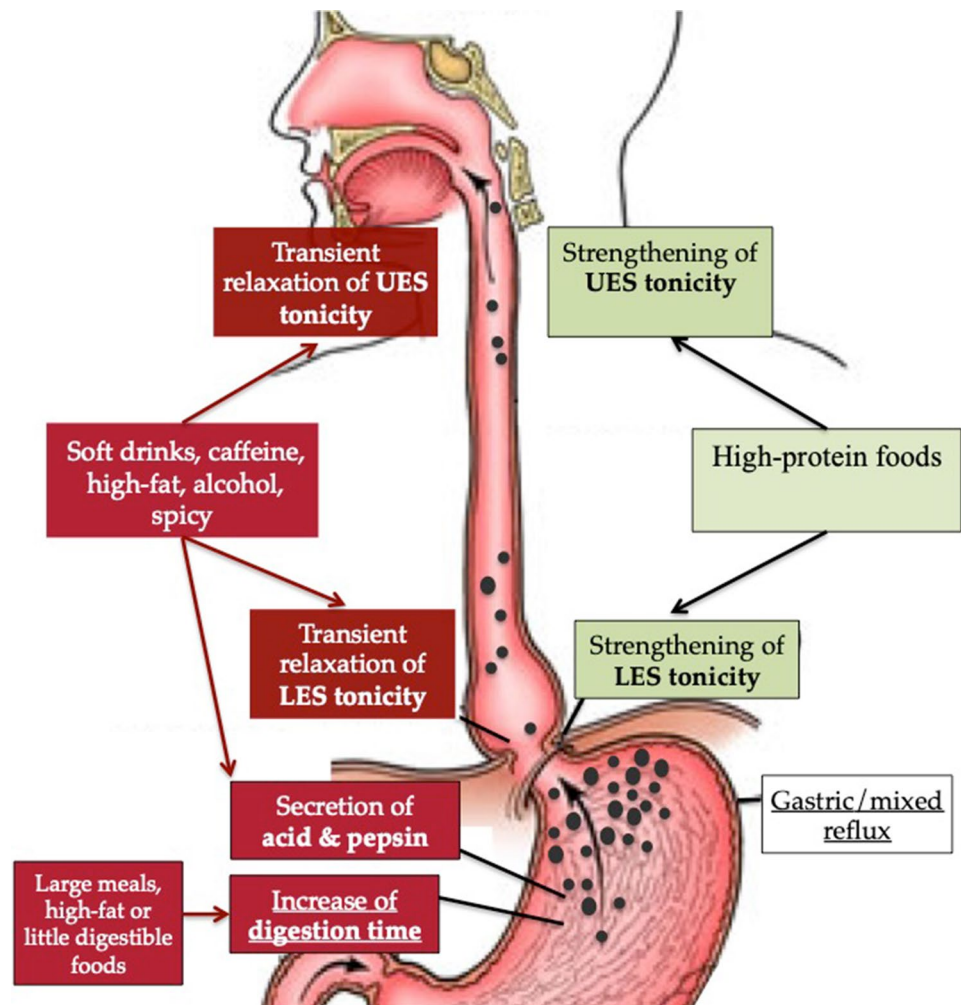
**Fig. 2** Chartflow of the study. *Cat.* Category, *LPR* laryngopharyngeal reflux, *Lip/prot/carbo* composition in lipids, proteins, and carbohydrates, *YO-IFOS* Young-Otolaryngologists of International Federation of Otorhinolaryngological Societies

### Other foods and factors

Additional foods or beverages have been identified as risk factors of GERD or esophageal dysfunction, including rice

cakes, ramen noodles, topokki, white bread, and extra-salt on regular meals [66, 81]. Sparkling beverages would be associated with GERD [5, 34, 82].

**Fig. 3** Summarize of the diet effect on gastroesophageal functioning. Foods that contribute to the effective gastroesophageal functioning are marked in green. Foods that alter the effective gastroesophageal functioning are marked in red



About the recommended foods, the high-protein foods would be associated with an increase of the LES tonicity, and may, therefore, be recommended [5, 12].

## Refluxogenic Diet Scores

### Refluxogenic Diet Score (REDS)

REDS was based on two subscores: the ‘pH score’ and the ‘composition score’.

The pH score has been created on the basis of a severity scale ranging from 0 (pH 10, very alkaline food/beverage) to 10 (pH 0, very acid food/beverage).

For each food, the experts identified protein, carbohydrate and lipid composition per 100 g. The ratio lipid/protein has been calculated for each food, leading to values ranging from 0 (very low-fat or very high-protein food) to 55 (very high-fat food); the latter being the food of our list with the maximal ratio value. Considering 0 as the minimal value and 55 as the maximal value, the authors transformed the values related to the ratio calculation to a composition score ranging

from 0 to 10 (10 being the previous 55 value). At most the food is rich in lipids and poor in proteins, at the most its composition score will be closed from 10. The final REDS per food consisted of the multiplication of the pH score and the composition score. REDS was theoretically ranged from 0 to 100. From the REDS values, five categories of foods have been established ranging from ‘very low refluxogenic food’ to ‘very high refluxogenic food’ (Table 2).

According to the literature, experts proposed to consider additional elements that are not considered in the pH or the composition score for the classification of the foods: the origin of the food [6, 12]; the fiber content and the cooking of vegetables [5]; and the spicy or aromatic herb composition [65–67]. The dietician or the physician may upgrade the category of a food if it is composed of many conservative agents (industrial food), most of them being acidifying agents. The consumption of raw high-fiber vegetables would be associated with a slow rate of gastric emptying that leads to an increased number of transient relaxation of LES, justifying the upgrade of the category. Moreover, some fibers may be irritative for the digestive tract. Thus, the addition

of spicy or aromatic herbs may also lead to an upgrade of the category. Table 3 describes the classification of foods into the five categories regarding the REDS values. Note that some greasy foods (oils) or spicy (herbs) are not water soluble and their acidity cannot be measured in terms of pH. They also did not contain enough proteins. For these reasons, they were classified on the basis of their refluxogenic potential regarding the literature.

### Score for beverages

Since beverages are often not composed of fat (exception of milk), experts proposed to consider the pH as the main factor for the assessment of the refluxogenic potential of beverages (Table 2). Once the category is determined, regarding the literature findings, physician may upgrade the category score with regard to the glycemic index (which partly reflects the sugar concentration of the beverages;  $> 40 =$  upgrade); the alcohol degree ( $> 3\% =$  upgrade), and the composition of caffeine or theine (upgrade). Sparkling beverages may also be upgraded. Coffee and tea without caffeine and theine may be downgraded. Table 3 summarizes the categories of beverages.

### Absolute and average Refluxogenic Score of a Dish (RESDI)

Absolute RESDI consists of the addition of the categories of the F&B of a meal respecting the weight (quantity) of the foods/beverages (100 g of a food of category 5 + 200 g of a food of category 3:  $RESDI = (1 \times 5) + (2 \times 3) = 11$ ). Absolute RESDI (abRESDI) considers the size of the dish because at most the patient eats a large amount of food, at most the absolute RESDI is high. Average RESDI (avRESDI) is the mean category of the dish (set at 100 g). Thus, avRESDI is the abRESDI divided by the foods/beverages quantity. An example of RESDI calculation is available in Fig. 4.

### Global Refluxogenic Score (GRES)

GRES is dedicated to the assessment of the Global Refluxogenic Score of the daily life F&B consumed by the patient. Based on Tables 3 and 4, the patient selects the F&B that she/he have consumed over the previous 3 weeks and the physician may add the categories of the F&B to get a score. In case of daily consumption of a food, the physician has to multiply the category of the food by the number of day on which it was consumed. An example of GRES calculation is available in Fig. 4.

**Table 2** The categories related to the Refluxogenic Diet Score

Foods	Beverages
Category 1: very low refluxogenic potential REDS value: 0–0.125	pH value: $\geq 8.01$
Category 2: low refluxogenic potential REDS value: 0.125–0.250	pH value: 6.01–8.00
Category 3: moderate refluxogenic potential REDS value: 0.250–0.5	pH value: 4.01–6.00
Category 4: high refluxogenic potential REDS value: 0.5–2.00	pH value: 2.01–4.00
Category 5: very high refluxogenic potential REDS value: $\geq 2.00$	pH value: 0–2.00

The classification of beverages also depends on the glycemic index, the alcohol degree; the osmolality; and the composition of caffeine or theine

*REDS* refluxogenic diet score

## Discussion

Laryngopharyngeal reflux is a multifactorial disease in which anatomical and functional factors play a pathological role. The main pathophysiological mechanism consists of transient relaxations of LES and UES and the deposit of pepsin in the mucosa of the upper aerodigestive tract [83, 84]. The tonicity of LES and UES depends on many factors, including autonomic nerve function, mechanical factors, intrinsic factors and diet [85, 86]. In this study, based on evidences of the literature and expert opinion, we developed a clinical score allowing the rating and the classification of F&B regarding their refluxogenic potentials. From this score (REDS), two additional scores were developed, evaluating the refluxogenic potential of both a dish and the overall diet of LPR patients.

The pH and the ratio lipid/protein are considered as the most important factors associated with reflux. The impact of acid F&B is easily understood. At most the food is acid, at the most the gastric content is acid, as well as the gaseous droplets of proximal reflux episodes that contain pepsin and other gastroduodenal enzymes. The acidic environment is important for the pepsin activity and its related mucosa toxicity [87]. This fact is supported by the studies conducting on LPR patients treated with alkaline diet [4–6, 12, 20]. As presumed by our experts and confirmed by the studies, fat food is associated with a higher risk of reflux through the longer slow gastric emptying time and the related number of LES/(UES) transient relaxations. For these reasons, REDS was mainly based on two parameters: pH and lipid/protein ratio; proteins strengthening the sphincter tonicity. However, as reported by Newberry and Lynch [88], there is little doubt over the impact of high-fat, low-protein, and acidic foods on the development of both GERD and LPR; although



**Table 3** The Refluxogenic Diet Score of foods and their related categories

Very low reflux. foods	REDS	Cat	Low reflux. foods	REDS	Cat	Moderate reflux. foods	REDS	Cat	High reflux. foods	REDS	Cat	Very high reflux. foods	REDS	Cat
Artichoke	0.086	1	Aubergine	0.166	2	Apricot	0.391	3	Apple	0.534	4	Avocado	5.610	5
Asparagus <sup>a</sup>	0.072	1	Banana	0.227	2	Blueberry	0.472	3	Blackberries	0.640	4	Bacon	25.40	5
Baked spinach	0.025	1	Carrots	0.132	2	Boiled egg	0.348	3	Brie, Blue, bread cheeses	1.001	4	Butter	-	5
Beetroot	0.082	1	Cherry	0.243	2	Camembert	0.495	3	Cake	1.850	4	Candy or sweets	5.216	5
Broccoli	0.077	1	Chicken fillet	0.148	2	Cereals (corn flacks)	0.470	3	Cauliflower	0.596	4	Chocolate (dark)	4.171	5
Brussels sprout	0.030	1	Chilli	0.171	2	Courgettes	0.289	3	Cheddar	1.068	4	Chocolate (Milk)	3.787	5
Celery	0.101	1	Corn	0.244	2	Cucumber	0.274	3	Chocolate cookies	1.920	4	Chocolate (white)	4.543	5
Cooked mushrooms	0.103	1	Fat chicken	0.236	2	Dried plum	0.252	3	Cookies	1.695	4	Chocolate croissant	2.911	5
Crabs	0.088	1	Fennel	0.131	2	Duck (without skin and fat)	0.350	3	Cracker	0.952	4	Chocolate eclairs	2.079	5
Egg white	0.006	1	Ketchup <sup>b</sup>	0.166	2	Fat fish	0.368	3	Egg yolk	1.334	4	Croissant	2.860	5
Endive	0.014	1	Kidneys	0.192	2	Fig	0.267	3	Feta	1.501	4	Curry	2.985	5
Fresh and thin fish	0.058	1	Lamb	0.232	2	Fish oil (sardines, cods)	-	3	Fontina	0.946	4	French fries and frying	2.836	5
Garlic	0.035	1	Lamb chops or shoulder	0.201	2	Fish oil (herrings)	-	3	Goat cheese	1.061	4	Ice cream	3.364	5
Green beans	0.054	1	Leek	0.139	2	Fish sauce	0.428	3	Gouda	1.193	4	Macadamia nut	7.074	5
Green peas	0.095	1	Melon	0.189	2	Ginger	0.362	3	Ground meat	0.704	4	Mayonnaise	56.80	5
Green salad <sup>a</sup>	0.074	1	Oat	0.243	2	Grapefruit	0.392	3	Gruyere	0.992	4	Meat sauce (Bearnaise)	45.04	5
Honey	0.000	1	Onion <sup>a</sup>	0.129	2	Guava	0.376	3	Hard cheese, full-fat cheese	1.093	4	Meat sauce (Pepper)	3.839	5
Horse	0.076	1	Parsley	0.139	2	Lamb cutlets	0.462	3	Kiwi	0.540	4	Meat sauce (Roquefort)	3.060	5
Lentil	0.064	1	Pepper	0.186	2	Mandarin	0.478	3	Lychee	0.512	4	Milk (coco)	6.521	5
Low-fat cheese	0.003	1	Pork tenderloin	0.208	2	Milk (goat, semi-skimmed)	0.272	3	Mango	0.536	4	Nut, cashew, hazelnut	3.585	5
Milk (skimmed)	0.030	1	Rib steak	0.153	2	Milk (soja)	0.298	3	Meat sauce (mushroom)	1.116	4	Olive (black)	7.478	5
Mollusk	0.060	1	Ribs	0.246	2	Milk (semi-skimmed)	0.363	3	Milk (whole)	0.690	4	Oliver (green)	12.92	5
Pork roast	0.110	1	Rice (brown)	0.188	2	Mint	0.302	3	Mozzarella	1.025	4	Pasta sauce (carbonara)	2.071	5
Pumpkin	0.085	1	Rindless, fatless	0.131	2	Nectarine	0.292	3	Munster	1.223	4	Pasta sauce (pesto)	8.331	5
Red cabbage	0.046	1	Cooked ham	0.131	2	Olive oil	-	3	Mustard	1.839	4	Pesto	8.331	5
Rice (red)	0.121	1	Rye bread	0.166	2	Orange	0.381	3	Noodles	0.565	4	Potato chips	2.830	5
Rice (white)	0.089	1	Shallot <sup>a</sup>	0.201	2	Peach	0.361	3	Orange jam	0.623	4	Sauerkraut	5.696	5
Roast veal	0.090	1	Steak, fillet, striploin	0.208	2	Pear	0.364	3	Parmesan	0.836	4	Spicy <sup>d</sup>	0.000	5
Shrimps or lobster	0.033	1	Tofu	0.248	2	Pickle	0.270	3	Pasta sauce (Bolognese)	1.134	4			
Spaghettis (cooked)	0.060	1	Turnip	0.186	2	Plum	0.471	3	Pâté	1.612	4			
Sweet potato	0.073	1	Veal chop	0.181	2	Pork chops and shoulder	0.316	3	Peanut	1.618	4			

**Table 3** (continued)

Very low reflux. foods		Low reflux. foods		Moderate reflux. foods		High reflux. foods		Very high reflux. foods	
REDS	Cat	REDS	Cat	REDS	Cat	REDS	Cat	REDS	Cat
0.043	1	0.043	1	0.175	2	0.357	3	0.725	4
0.026	1	0.026	1	0.187	1	0.307	3	0.758	4
0.059	1	0.059	1	0.236	2	0.362	3	0.566	4
0.079	1	0.079	1			0.375	3	0.922	4
						0.290	3	1.030	4
						0.340	3	1.288	4
						0.000	3	1.177	4
						0.297	3	0.722	4
						0.255	3	1.942	4
						0.264	3	0.618	4
								1.538	4
								–	4
								0.674	4

Several foods may be upgraded or downgraded regarding to characteristics. <sup>a</sup>Raw vegetables are less digestible and may be associated with low gastric emptying time: in case of raw consumption, the food has to be upgraded for 1 category. Not for green salad, the addition of vinegar or vinaigrette upgrades the category

<sup>b</sup>In case of addition of spicy (for example, Spicy Ketchup), these foods have to be upgraded

<sup>c</sup>For sugar, only the pH and the glycemic index have been considered regarding the lack of fat

<sup>d</sup>Because spicy has no lipid and no pH, the authors based the classification of this food on the literature. If the patients only eat industrial foods (ready-made food), the foods may be upgraded regarding the acidifying potential of industrial conservative

some investigations of the role of specific foods or beverages (separately considered) have reported controversial results.

The controversial results of these studies are probably related to inter-individual differences in the mucosa sensitivity, food/beverage component metabolism (caffeine, etc.) and other unknown environmental factors. First, the esophageal mucosa sensitivity to acid food may vary from one patient to another and would depend on the composition of food. Thus, Shapiro et al. observed that the alcohol intake was associated with a reduced perception of esophageal acid reflux events in some patients [28]. The reduction of the perception of esophageal acid reflux events could be an important factor, biasing the clinical assessment of some patients in the detection of reflux and leading to unclear conclusions. As found for caffeine, the metabolism of some refluxogenic molecules would be different from one patient to another [48, 49], being an additional factor that could explain the controversial results of many studies. The origin of food is probably an environmental factor associated with a higher risk of reflux. This finding has been reported by the works of Koufman [6, 12] who stated that the industrial foods are often more acidic than the organic (bio) foods regarding the acidic potential of artificial preservative of the industrial foods (named E200 to E297). These findings are

strengthened by the authors who reported that the consumption of Mediterranean fresh products may be associated with a decreased risk of GERD [89]. Among the F&B of the same family, some component differences may exist, yielding to different impacts on the gastroesophageal function. This is the case for tea according to our pH analysis, which reported that, among the various types of tea, some has a low pH (blackberry tea) whereas other has neutral pH (green tea). Interestingly, the meta-analysis of Cao et al., who investigated the association between the consumption of tea and the development of GERD, showed that there are several subgroups of tea drinkers, some developing GERD and some other not [47]. Additionally to individual factors, the conclusion of this meta-analysis may highlight these component differences. If the pH or the food composition does not significantly change regarding the cooking, the cooking process may, however, have an impact on the fiber composition of many high-fiber vegetables (onions, shallots, etc.), which are more digestible. This point has been considered in REDS. A meal-related factor that would be involved in both GERD and LPR patients is the size of the meals [90]. Thus, a strength of RESDI and GRES is the consideration of the quantity of the F&B consumed during the meal and over the previous 3 weeks, respectively.

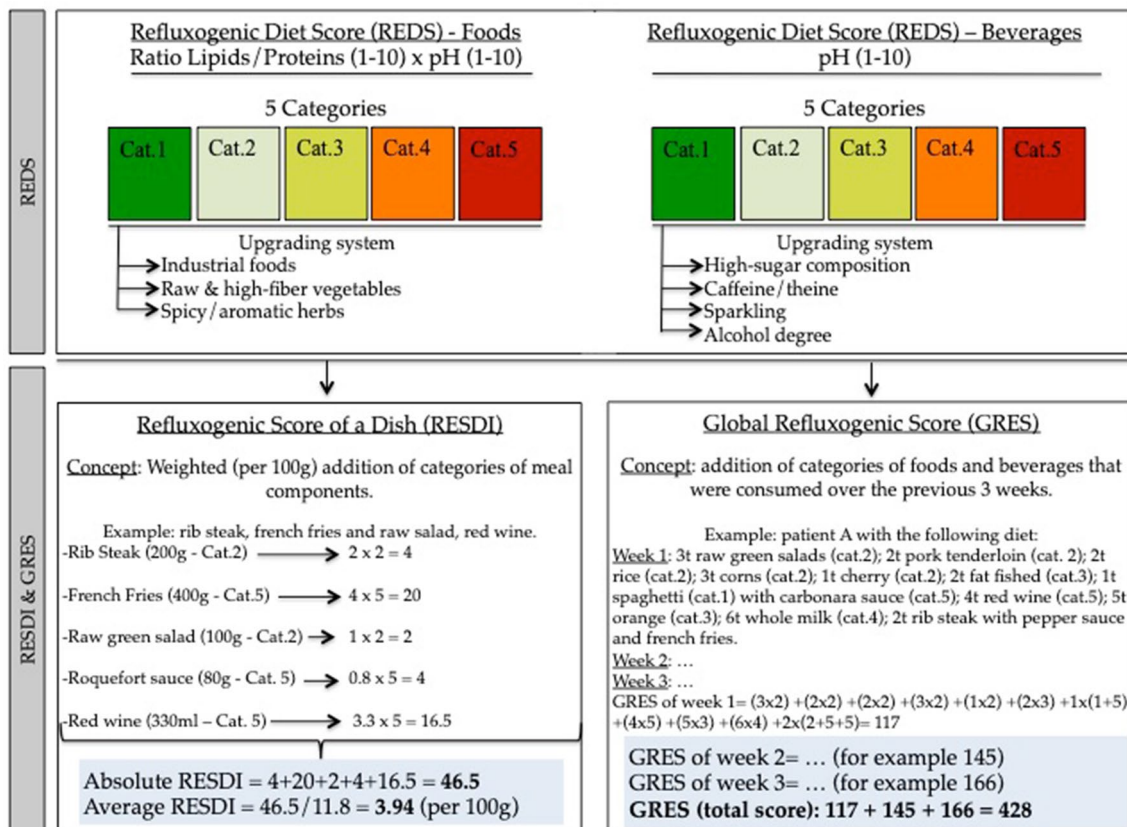


Fig. 4 Summary of Refluxogenic Diet Scores. *t* time, *4t* four times a week

**Table 4** The Refluxogenic Diet Score of beverages and their related categories

Juice, water and alcohol	pH	GI>40	Cat	UCat
Alcohol (strong and licor) <sup>a,c</sup>	4	+	3	5
Aloe vera	6.1	0	2	2
Apple juice	3.65	+	4	5
Beer <sup>b,c</sup>	4	+	3	5
Cacao (hot chocolate)	6.3	+	2	3
Chamomile	6.5	0	2	2
Chicory	5.95	0	3	3
Coffee <sup>d</sup>	5	0	3	4
Grapefruit juice	3.05	+	4	5
Lemon juice	2.3	+	4	5
Multifruit juice	3.8	+	4	5
Orange juice	3.5	+	4	5
Soda (sugar free) <sup>b</sup>	2.5	0	4	5
Soda (with sugar) <sup>b</sup>	2.5	+	4	5
Syrup (mint, lemon, grenadine)	2,15	+	4	5
Tea <sup>d</sup>	5	0	3	4
Tea (blackberry) <sup>d</sup>	2,5	0	4	5
Tea (black) <sup>d</sup>	5,3	0	3	4
Tea (green) <sup>d</sup>	7	0	2	3
Tea (lemon) <sup>d</sup>	2,9	0	4	5
Tomato juice	4,35	0	3	3
Water (sparkling) <sup>b</sup>	7	0	2	3
Water (still)	7	0	2	2
Water (alkaline)	8	0	1	1
Wine (red) <sup>c</sup>	4	0	4	5
Wine (rose) <sup>c</sup>	4	0	4	5
Wine (white) <sup>c</sup>	4	0	4	5

The classification of beverages depends on pH, <sup>a</sup>glycemic index (GI; high sugar-related osmolarity), <sup>b</sup>sparkling (upgrade), <sup>c</sup>the alcohol degree (>3% = upgrade) and the <sup>d</sup>presence or lack of caffeine or theine (<sup>d</sup>upgrade or downgrade)

GI glycemic index, *cat.* category at baseline, *ucat.* upgraded category. For hot chocolate, the category is upgraded in case of additional sugar

According to the above-mentioned factors, and because reflux results from the concurrence of many F&B, the experts of YO-IFOS want to emphasize about the importance to consider the diet in a holistic way, which may decrease the potential impact of the individual differences.

In this study, many additional factors that may be involved in the development of reflux, i.e., tobacco [39, 54, 72–74, 91–93] or exercise [94], has not been considered because our objective was to develop specifically diet scores. Other factors are still poorly investigated in LPR such as the heredity, which accounts for 31–43% of the likelihood of GERD [95, 96], whereas some GERD recommendations could be less important for LPR patients without GERD at the MII-pH [or without temporary pro-GERD habits (Ramadan, etc.)], such as avoidance meals before sleep and the elevation of

the head of the bed. Indeed, in LPR, there are less evidences about the usefulness of the elevation of the head of the bed because the majority of reflux episodes occur daytime and upright [3]. Future studies are needed to specifically investigate the impact of these recommendations in LPR patients in comparison with GERD patients.

The main theoretical weakness of these scores is the time to calculate each of them. For this reason, our team, which owns the intellectual properties of these scores, is developing a mobile phone application for the calculations of REDS, GRES, and RESDI.

## Conclusion and perspectives

The development of clinical tools assessing the refluxogenic potential of the diet of the LPR patients makes sense for improving the reflux management and opens up some opportunities for nonpharmacologic interventions in patients with mild LPR. The three scores developed in the present study are still theoretical and need future studies to be validated or correlated with impedance–pH monitoring or esophageal manometry findings. These studies should consider GERD, LPR patients and healthy individuals. The healthy population is particularly important for the establishment of pathological thresholds or categories for GRES. Since some pathophysiological differences exist between GERD and LPR, the consideration of these populations in a different way is important. The development of similar food and beverage categories is required for the use of REDS, RESDI and GRES in the other world regions which are characterized by local and specific diet.

**Acknowledgements** Vesale Grant and IRIS-Recherche Grant for the studies conducted in Brussels over the last year; these studies allowing Dr. Jerome Lechien to propose this consensus paper (Dr. Jerome R. Lechien owns the intellectual properties of diet scores).

## Compliance with ethical standards

**Conflict of interest** The authors have no conflicts of interest.

## References

1. Lechien JR, Akst LM, Hamdan AL et al (2019) Evaluation and management of laryngopharyngeal reflux disease: state of the art review. *Otolaryngol Neck Surg Off J Am Acad Otolaryngol Head Neck Surg* 160(5):762–782. <https://doi.org/10.1177/0194599819827488>
2. Koufman JA (1991) The otolaryngologic manifestations of gastroesophageal reflux disease (GERD): a clinical investigation of 225 patients using ambulatory 24-hour pH monitoring and an experimental investigation of the role of acid and pepsin in the development of laryngeal injury. *Laryngoscope* 101(4 Pt 2 Suppl 53):1–78

3. Lechien JR, Bobin F, Muls V et al (2019) Validity and reliability of the reflux symptom score. *Laryngoscope*. <https://doi.org/10.1002/lary.28017>
4. Zalvan CH, Hu S, Greenberg B, Geliebter J (2017) A comparison of alkaline water and Mediterranean diet vs proton pump inhibition for treatment of laryngopharyngeal reflux. *JAMA Otolaryngol Neck Surg* 143(10):1023. <https://doi.org/10.1001/jamaoto.2017.1454>
5. Lechien JR, Huet K, Khalife M et al (2019) Alkaline, protein, low-fat and low-acid diet in laryngopharyngeal reflux disease: our experience on 65 patients. *Clin Otolaryngol Off J ENT-UK Off J Neth Soc Oto-Rhino-Laryngol Cervico-Facial Surg* 44(3):379–384. <https://doi.org/10.1111/coa.13269>
6. Koufman JA, Johnston N (2012) Potential benefits of pH 8.8 alkaline drinking water as an adjunct in the treatment of reflux disease. *Ann Otol Rhinol Laryngol* 121(7):431–434. <https://doi.org/10.1177/000348941212100702>
7. Wang AM, Wang G, Huang N et al (2019) Association between laryngopharyngeal reflux disease and autonomic nerve dysfunction. *Eur Arch Oto-Rhino-Laryngol Off J Eur Fed Oto-Rhino-Laryngol Soc EUFOS Affil Ger Soc Oto-Rhino-Laryngol Neck Surg* 276(8):2283–2287. <https://doi.org/10.1007/s00405-019-05482-w>
8. Lechien JR, Nandhan Sampath Kumar R, Chiesa-Estomba CM (2019) Laryngopharyngeal reflux and autonomic nerve dysfunction: what about stress? *Eur Arch Oto-Rhino-Laryngol Off J Eur Fed Oto-Rhino-Laryngol Soc EUFOS Affil Ger Soc Oto-Rhino-Laryngol Neck Surg*. <https://doi.org/10.1007/s00405-019-05567-6>
9. Ding H, Duan Z, Yang D et al (2017) High-resolution manometry in patients with and without globus pharyngeus and/or symptoms of laryngopharyngeal reflux. *BMC Gastroenterol* 17(1):109. <https://doi.org/10.1186/s12876-017-0666-x>
10. Passaretti S, Mazzoleni G, Vailati C, Testoni PA (2016) Oropharyngeal acid reflux and motility abnormalities of the proximal esophagus. *World J Gastroenterol* 22(40):8991–8998. <https://doi.org/10.3748/wjg.v22.i40.8991>
11. Kaltenbach T, Crockett S, Gerson LB (2006) Are lifestyle measures effective in patients with gastroesophageal reflux disease? An evidence-based approach. *Arch Intern Med* 166(9):965–971. <https://doi.org/10.1001/archinte.166.9.965>
12. Koufman JA (2011) Low-acid diet for recalcitrant laryngopharyngeal reflux: Therapeutic benefits and their implications. *Ann Otol Rhinol Laryngol* 120(5):281–287. <https://doi.org/10.1177/00034894112000501>
13. Lechien JR, Mouawad F, Mortuaire G et al (2019) Awareness of European Otolaryngologists and General Practitioners toward laryngopharyngeal reflux. *Ann Otol Rhinol Laryngol*. <https://doi.org/10.1177/0003489419858090>
14. Fackrell K, Smith H, Colley V et al (2017) Core outcome domains for early phase clinical trials of sound-, psychology-, and pharmacology-based interventions to manage chronic subjective tinnitus in adults: the COMIT-ID study protocol for using a Delphi process and face-to-face meetings to establish consensus. *Trials* 18(1):388. <https://doi.org/10.1186/s13063-017-2123-0>
15. Giacchi RJ, Sullivan D, Rothstein SG (2000) Compliance with anti-reflux therapy in patients with otolaryngologic manifestations of gastroesophageal reflux disease. *Laryngoscope* 110(1):19–22. <https://doi.org/10.1097/00005537-200001000-00004>
16. Siupsinskiene N, Adamonis K (2003) Diagnostic test with omeprazole in patients with posterior laryngitis. *Med Kaunas Lith* 39(1):47–55
17. Hamdan A, Nassar J, Dowli A, Al Zagal Z, Sabri A (2012) Effect of fasting on laryngopharyngeal reflux disease in male subjects. *Eur Arch Oto-Rhino-Laryngol Off J Eur Fed Oto-Rhino-Laryngol Soc EUFOS Affil Ger Soc Oto-Rhino-Laryngol Neck Surg* 269(11):2361–2366. <https://doi.org/10.1007/s00405-012-2038-z>
18. Chappity P, Kumar R, Deka RC, Chokkalingam V, Saraya A, Sikka K (2014) Proton pump inhibitors versus solitary lifestyle modification in management of laryngopharyngeal reflux and evaluating who is at risk: scenario in a developing country. *Clin Med Insights Ear Nose Throat* 7:1–5. <https://doi.org/10.4137/CMENT.S13799>
19. Nanda MS (2016) Role of adjuvant lifestyle modifications in patients with laryngopharyngeal reflux disease in Hilly. *Int J Sci Study* 3(10):6
20. Yang J, Dehom S, Sanders S, Murry T, Krishna P, Crawley BK (2018) Treating laryngopharyngeal reflux: evaluation of an anti-reflux program with comparison to medications. *Am J Otolaryngol* 39(1):50–55. <https://doi.org/10.1016/j.amjoto.2017.10.014>
21. Ciqual Table de composition nutritionnelle des aliments. <https://ciqual.anses.fr/>. Accessed 30 July 2019
22. Amazon.fr, Table de composition des aliments, Etude Nutrinet Santé, Belgium. [https://www.amazon.fr/Table-composition-aliments-Etude-Nutrinet/dp/2717865373/ref=sr\\_1\\_1?hvadid=80814136925022&hvbm=be&hvdev=c&hvqmt=e&keywords=table+de+composition+des+aliments&qid=1564494275&s=gateway&sr=8-1](https://www.amazon.fr/Table-composition-aliments-Etude-Nutrinet/dp/2717865373/ref=sr_1_1?hvadid=80814136925022&hvbm=be&hvdev=c&hvqmt=e&keywords=table+de+composition+des+aliments&qid=1564494275&s=gateway&sr=8-1). Accessed 30 July 2019
23. Becker DJ, Sinclair J, Castell DO, Wu WC (1989) A comparison of high and low fat meals on postprandial esophageal acid exposure. *Am J Gastroenterol* 84(7):782–786
24. Hills JM, Aaronson PI (1991) The mechanism of action of peppermint oil on gastrointestinal smooth muscle. An analysis using patch clamp electrophysiology and isolated tissue pharmacology in rabbit and guinea pig. *Gastroenterology* 101(1):55–65. [https://doi.org/10.1016/0016-5085\(91\)90459-x](https://doi.org/10.1016/0016-5085(91)90459-x)
25. Murphy DW, Castell DO (1988) Chocolate and heartburn: Evidence of increased esophageal acid exposure after chocolate ingestion. *Am J Gastroenterol* 83(6):633–636
26. Nebel OT, Castell DO (1972) Lower esophageal sphincter pressure changes after food ingestion. *Gastroenterology* 63(5):778–783
27. El-Serag HB, Satia JA, Rabeneck L (2005) Dietary intake and the risk of gastro-oesophageal reflux disease: a cross sectional study in volunteers. *Gut* 54(1):11–17. <https://doi.org/10.1136/gut.2004.040337>
28. Shapiro M, Green C, Bautista JM et al (2007) Assessment of dietary nutrients that influence perception of intra-oesophageal acid reflux events in patients with gastro-oesophageal reflux disease. *Aliment Pharmacol Ther* 25(1):93–101. <https://doi.org/10.1111/j.1365-2036.2006.03170.x>
29. Pehl C, Waizenhoefer A, Wendl B, Schmidt T, Schepp W, Pfeiffer A (1999) Effect of low and high fat meals on lower esophageal sphincter motility and gastroesophageal reflux in healthy subjects. *Am J Gastroenterol* 94(5):1192–1196. <https://doi.org/10.1111/j.1572-0241.1999.01064.x>
30. Colombo P, Mangano M, Bianchi PA, Penagini R (2002) Effect of calories and fat on postprandial gastro-oesophageal reflux. *Scand J Gastroenterol* 37(1):3–5
31. Sutphen JL, Dillard VL (1989) Dietary caloric density and osmolality influence gastroesophageal reflux in infants. *Gastroenterology* 97(3):601–604. <https://doi.org/10.5555/uri:pii:001650859906306>
32. Wright LE, Castell DO (1975) The adverse effect of chocolate on lower esophageal sphincter pressure. *Am J Dig Dis* 20(8):703–707
33. Fass R, Quan SF, O'Connor GT, Ervin A, Iber C (2005) Predictors of heartburn during sleep in a large prospective cohort study. *Chest* 127(5):1658–1666. <https://doi.org/10.1378/chest.127.5.1658>
34. Alrashed AA, Aljammaz KI, Pathan A et al (2019) Prevalence and risk factors of gastroesophageal reflux disease among Shaqra University students, Saudi Arabia. *J Fam Med Prim Care* 8(2):462–467. [https://doi.org/10.4103/jfmpc.jfmpc\\_443\\_18](https://doi.org/10.4103/jfmpc.jfmpc_443_18)

35. Arivan R, Deepanjali S (2018) Prevalence and risk factors of gastro-oesophageal reflux disease among undergraduate medical students from a southern Indian medical school: a cross-sectional study. *BMC Res Notes* 11(1):448. <https://doi.org/10.1186/s13104-018-3569-1>
36. Hamoui N, Lord RV, Hagen JA, Theisen J, Demeester TR, Crookes PF (2006) Response of the lower esophageal sphincter to gastric distention by carbonated beverages. *J Gastrointest Surg Off J Soc Surg Aliment Tract* 10(6):870–877. <https://doi.org/10.1016/j.gassur.2005.11.010>
37. Price SF, Smithson KW, Castell DO (1978) Food sensitivity in reflux esophagitis. *Gastroenterology* 75(2):240–243
38. Vossoughinia H, Salari M, Mokhtari Amirmajidi E et al (2014) An epidemiological study of gastroesophageal reflux disease and related risk factors in urban population of Mashhad, Iran. *Iran Red Crescent Med J* 16(12):e15832. <https://doi.org/10.5812/ircmj.15832>
39. Thomas FB, Steinbaugh JT, Fromkes JJ, Mekhjian HS, Caldwell JH (1980) Inhibitory effect of coffee on lower esophageal sphincter pressure. *Gastroenterology* 79(6):1262–1266
40. Boekema PJ, Samsom M, Smout AJ (1999) Effect of coffee on gastro-oesophageal reflux in patients with reflux disease and healthy controls. *Eur J Gastroenterol Hepatol* 11(11):1271–1276
41. Chang C-H, Wu C-P, Wang J-D et al (2017) Alcohol and tea consumption are associated with asymptomatic erosive esophagitis in Taiwanese men. *PLoS ONE* 12(3):e0173230. <https://doi.org/10.1371/journal.pone.0173230>
42. Jarosz M, Taraszewska A (2014) Risk factors for gastroesophageal reflux disease: The role of diet. *Przegląd Gastroenterol* 9(5):297–301. <https://doi.org/10.5114/pg.2014.46166>
43. Lee S-W, Lee T-Y, Lien H-C, Yeh H-Z, Chang C-S, Ko C-W (2014) The risk factors and quality of life in patients with overlapping functional dyspepsia or peptic ulcer disease with gastroesophageal reflux disease. *Gut Liver* 8(2):160–164. <https://doi.org/10.5009/gnl.2014.8.2.160>
44. Lee S-W, Lee T-Y, Lien H-C, Yeh H-Z, Chang C-S, Ko C-W (2013) Comparison of risk factors and disease severity between old and young patients with gastroesophageal reflux disease. *Gastroenterol Res* 6(3):91–94. <https://doi.org/10.4021/gr549w>
45. Niu C-Y, Zhou Y-L, Yan R et al (2012) Incidence of gastroesophageal reflux disease in Uyghur and Han Chinese adults in Urumqi. *World J Gastroenterol* 18(48):7333–7340. <https://doi.org/10.3748/wjg.v18.i48.7333>
46. Wei T-Y, Hsueh P-H, Wen S-H, Chen C-L, Wang C-C (2019) The role of tea and coffee in the development of gastroesophageal reflux disease. *Ci Ji Yi Xue Za Zhi Tzu-Chi Med J* 31(3):169–176. [https://doi.org/10.4103/tcmj.tcmj\\_48\\_18](https://doi.org/10.4103/tcmj.tcmj_48_18)
47. Cao H, Huang X, Zhi X, Han C, Li L, Li Y (2019) Association between tea consumption and gastroesophageal reflux disease: a meta-analysis. *Medicine (Baltimore)* 98(4):e14173. <https://doi.org/10.1097/MD.00000000000014173>
48. Pollock BG, Wylie M, Stack JA et al (1999) Inhibition of caffeine metabolism by estrogen replacement therapy in postmenopausal women. *J Clin Pharmacol* 39(9):936–940
49. Abernethy DR, Todd EL (1985) Impairment of caffeine clearance by chronic use of low-dose oestrogen-containing oral contraceptives. *Eur J Clin Pharmacol* 28(4):425–428
50. Mohammed I, Nightingale P, Trudgill NJ (2005) Risk factors for gastro-oesophageal reflux disease symptoms: a community study. *Aliment Pharmacol Ther* 21(7):821–827. <https://doi.org/10.1111/j.1365-2036.2005.02426.x>
51. Wang J-H, Luo J-Y, Dong L, Gong J, Tong M (2004) Epidemiology of gastroesophageal reflux disease: a general population-based study in Xi'an of Northwest China. *World J Gastroenterol* 10(11):1647–1651. <https://doi.org/10.3748/wjg.v10.i11.1647>
52. O'Leary C, McCarthy J, Humphries M, Shanahan F, Quigley E (2003) The prophylactic use of a proton pump inhibitor before food and alcohol. *Aliment Pharmacol Ther* 17(5):683–686
53. Rosaida MS, Goh K-L (2004) Gastro-oesophageal reflux disease, reflux oesophagitis and non-erosive reflux disease in a multiracial Asian population: a prospective, endoscopy based study. *Eur J Gastroenterol Hepatol* 16(5):495–501
54. Nilsson M, Johnsen R, Ye W, Hveem K, Lagergren J (2004) Lifestyle related risk factors in the aetiology of gastro-oesophageal reflux. *Gut* 53(12):1730–1735. <https://doi.org/10.1136/gut.2004.043265>
55. Nilsson M, Johnsen R, Ye W, Hveem K, Lagergren J (2004) Prevalence of gastro-oesophageal reflux symptoms and the influence of age and sex. *Scand J Gastroenterol* 39(11):1040–1045. <https://doi.org/10.1080/00365520410003498>
56. Stanghellini V (1999) Relationship between upper gastrointestinal symptoms and lifestyle, psychosocial factors and comorbidity in the general population: results from the Domestic/International Gastroenterology Surveillance Study (DIGEST). *Scand J Gastroenterol Suppl* 231:29–37
57. Talley NJ, Zinsmeister AR, Schleck CD, Melton LJ (1994) Smoking, alcohol, and analgesics in dyspepsia and among dyspepsia subgroups: lack of an association in a community. *Gut* 35(5):619–624. <https://doi.org/10.1136/gut.35.5.619>
58. Pehl C, Wendl B, Pfeiffer A (2006) White wine and beer induce gastro-oesophageal reflux in patients with reflux disease. *Aliment Pharmacol Ther* 23(11):1581–1586. <https://doi.org/10.1111/j.1365-2036.2006.02922.x>
59. Pehl C, Pfeiffer A, Wendl B, Kaess H (1998) Different effects of white and red wine on lower esophageal sphincter pressure and gastroesophageal reflux. *Scand J Gastroenterol* 33(2):118–122
60. Bujanda L (2000) The effects of alcohol consumption upon the gastrointestinal tract. *Am J Gastroenterol* 95(12):3374–3382. <https://doi.org/10.1111/j.1572-0241.2000.03347.x>
61. Kaufman SE, Kaye MD (1978) Induction of gastro-oesophageal reflux by alcohol. *Gut* 19(4):336–338. <https://doi.org/10.1136/gut.19.4.336>
62. Vitale G, Cheadle WG, Patel B, Sadek SA, Michel ME, Cuschieri A (1987) The effect of alcohol on nocturnal gastroesophageal reflux. *JAMA* 258(15):2077–2079
63. Rubinstein E, Hauge C, Sommer P, Mortensen T (1993) Oesophageal and gastric potential difference and pH in healthy volunteers following intake of coca-cola, red wine, and alcohol. *Pharmacol Toxicol* 72(1):61–65
64. Grande L, Manterola C, Ros E, Lacima G, Pera C (1997) Effects of red wine on 24-hour esophageal pH and pressures in healthy volunteers. *Dig Dis Sci* 42(6):1189–1193. <https://doi.org/10.1023/a:1018893721735>
65. Nebel OT, Fornes MF, Castell DO (1976) Symptomatic gastroesophageal reflux: Incidence and precipitating factors. *Am J Dig Dis* 21(11):953–956
66. Choe JW, Joo MK, Kim HJ et al (2017) Foods inducing typical gastroesophageal reflux disease symptoms in Korea. *J Neurogastroenterol Motil* 23(3):363–369. <https://doi.org/10.5056/jnm16122>
67. Allen ML, Mellow MH, Robinson MG, Orr WC (1990) The effect of raw onions on acid reflux and reflux symptoms. *Am J Gastroenterol* 85(4):377–380
68. Kang JY, Tay HH, Guan R (1992) Chronic upper abdominal pain: site and radiation in various structural and functional disorders and the effect of various foods. *Gut* 33(6):743–748. <https://doi.org/10.1136/gut.33.6.743>
69. Lim LG, Tay H, Ho KY (2011) Curry induces acid reflux and symptoms in gastroesophageal reflux disease. *Dig Dis Sci* 56(12):3546–3550. <https://doi.org/10.1007/s10620-011-1799-3>
70. Yeoh KG, Ho KY, Guan R, Kang JY (1995) How does chili cause upper gastrointestinal symptoms? A correlation study with

- esophageal mucosal sensitivity and esophageal motility. *J Clin Gastroenterol* 21(2):87–90
71. Milke P, Diaz A, Valdovinos MA, Moran S (2006) Gastroesophageal reflux in healthy subjects induced by two different species of chilli (*Capsicum annum*). *Dig Dis* 24(1–2):184–188. <https://doi.org/10.1159/000090323>
  72. Alsulobi AM, El-Fetoh NMA, Alenezi SGE et al (2017) Gastroesophageal reflux disease among population of Arar City. *North Saudi Arabia Electron Physician* 9(10):5499–5505. <https://doi.org/10.19082/5499>
  73. Kubo A, Block G, Quesenberry CP, Buffler P, Corley DA (2014) Dietary guideline adherence for gastroesophageal reflux disease. *BMC Gastroenterol* 14:144. <https://doi.org/10.1186/1471-230X-14-144>
  74. Richter JE (2000) Gastroesophageal reflux disease in the older patient: Presentation, treatment, and complications. *Am J Gastroenterol* 95(2):368–373. <https://doi.org/10.1111/j.1572-0241.2000.t01-1-01791.x>
  75. de Bortoli N, Guidi G, Martinucci I et al (2016) Voluntary and controlled weight loss can reduce symptoms and proton pump inhibitor use and dosage in patients with gastroesophageal reflux disease: a comparative study. *Dis Esophagus Off J Int Soc Dis Esophagus* 29(2):197–204. <https://doi.org/10.1111/dote.12319>
  76. Saleh K, Eid R, Haddad FG, Khalife-Saleh N, Kourie HR (2018) New developments in the management of head and neck cancer—impact of pembrolizumab. *Ther Clin Risk Manag* 14:295–303. <https://doi.org/10.2147/TCRM.S125059>
  77. López-Colombo A, Pacio-Quiterio MS, Jesús-Mejenes LY et al (2017) Risk factors associated with gastroesophageal reflux disease relapse in primary care patients successfully treated with a proton pump inhibitor. *Rev Gastroenterol Mex* 82(2):106–114. <https://doi.org/10.1016/j.rgmx.2016.09.001>
  78. Feldman M, Barnett C (1995) Relationships between the acidity and osmolality of popular beverages and reported postprandial heartburn. *Gastroenterology* 108(1):125–131. [https://doi.org/10.1016/0016-5085\(95\)90016-0](https://doi.org/10.1016/0016-5085(95)90016-0)
  79. Eslami O, Shahraki M, Bahari A, Shahraki T (2017) Dietary habits and obesity indices in patients with gastro-esophageal reflux disease: a comparative cross-sectional study. *BMC Gastroenterol* 17(1):132. <https://doi.org/10.1186/s12876-017-0699-1>
  80. Bulat R, Fachnie E, Chauhan U, Chen Y, Tougas G (1999) Lack of effect of spearmint on lower oesophageal sphincter function and acid reflux in healthy volunteers. *Aliment Pharmacol Ther* 13(6):805–812
  81. Yadegarfar G, Momenyan S, Khoobi M et al (2018) Iranian lifestyle factors affecting reflux disease among healthy people in Qom. *Electron Physician* 10(4):6718–6724. <https://doi.org/10.19082/6718>
  82. Artanti D, Hegar B, Kaswandani N et al (2019) The gastroesophageal reflux disease questionnaire in adolescents: what is the best cutoff score? *Pediatr Gastroenterol Hepatol Nutr* 22(4):341–349. <https://doi.org/10.5223/pghn.2019.22.4.341>
  83. Rees LEN, Pazmany L, Gutowska-Owsiak D et al (2008) The mucosal immune response to laryngopharyngeal reflux. *Am J Respir Crit Care Med* 177(11):1187–1193. <https://doi.org/10.1164/rccm.200706-895OC>
  84. Johnston N, Dettmar PW, Strugala V, Allen JE, Chan WW (2013) Laryngopharyngeal reflux and GERD. *Ann N Y Acad Sci* 1300:71–79. <https://doi.org/10.1111/nyas.12237>
  85. Crookes PF (2006) Physiology of reflux disease: role of the lower esophageal sphincter. *Surg Endosc* 20(Suppl 2):S462–466. <https://doi.org/10.1007/s00464-006-0039-y>
  86. Sidhu AS, Triadafilopoulos G (2008) Neuro-regulation of lower esophageal sphincter function as treatment for gastroesophageal reflux disease. *World J Gastroenterol* 14(7):985–990. <https://doi.org/10.3748/wjg.14.985>
  87. Johnston N, Dettmar PW, Bishwokarma B, Lively MO, Koufman JA (2007) Activity/stability of human pepsin: implications for reflux attributed laryngeal disease. *The Laryngoscope* 117(6):1036–1039. <https://doi.org/10.1097/MLG.0b013e31804154c3>
  88. Newberry C, Lynch K (2017) Can we use diet to effectively treat esophageal disease? A review of the current literature. *Curr Gastroenterol Rep* 19(8):38. <https://doi.org/10.1007/s11894-017-0578-5>
  89. Mone I, Kraja B, Bregu A et al (2016) Adherence to a predominantly Mediterranean diet decreases the risk of gastroesophageal reflux disease: a cross-sectional study in a South Eastern European population. *Dis Esophagus Off J Int Soc Dis Esophagus* 29(7):794–800. <https://doi.org/10.1111/dote.12384>
  90. Delgado-Aros S, Camilleri M, Cremonini F, Ferber I, Stephens D, Burton DD (2004) Contributions of gastric volumes and gastric emptying to meal size and postmeal symptoms in functional dyspepsia. *Gastroenterology* 127(6):1685–1694. <https://doi.org/10.1053/j.gastro.2004.09.006>
  91. Watanabe Y, Fujiwara Y, Shiba M et al (2003) Cigarette smoking and alcohol consumption associated with gastro-oesophageal reflux disease in Japanese men. *Scand J Gastroenterol* 38(8):807–811
  92. Tibbling L, Gibellino FM, Johansson KE (1995) Is mis-swallowing or smoking a cause of respiratory symptoms in patients with gastroesophageal reflux disease? *Dysphagia* 10(2):113–116
  93. Chattopadhyay DK, Greaney MG, Irvin TT (1977) Effect of cigarette smoking on the lower oesophageal sphincter. *Gut* 18(10):833–835. <https://doi.org/10.1136/gut.18.10.833>
  94. Pandolfino JE, Bianchi LK, Lee TJ, Hirano I, Kahrilas PJ (2004) Esophagogastric junction morphology predicts susceptibility to exercise-induced reflux. *Am J Gastroenterol* 99(8):1430–1436. <https://doi.org/10.1111/j.1572-0241.2004.30515.x>
  95. Cameron AJ, Lagergren J, Henriksson C, Nyren O, Locke GR, Pedersen NL (2002) Gastroesophageal reflux disease in monozygotic and dizygotic twins. *Gastroenterology* 122(1):55–59. <https://doi.org/10.1053/gast.2002.30301>
  96. Mohammed I, Cherkas LF, Riley SA, Spector TD, Trudgill NJ (2003) Genetic influences in gastro-oesophageal reflux disease: a twin study. *Gut* 52(8):1085–1089. <https://doi.org/10.1136/gut.52.8.1085>

**Publisher's Note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

## Affiliations

Jerome R. Lechien<sup>1,2,3,4</sup> · Francois Bobin<sup>1,5</sup> · Francois Mouawad<sup>1,6</sup> · Karol Zelenik<sup>1,7</sup> · Christian Calvo-Henriquez<sup>1,8</sup> · Carlos M. Chiesa-Estomba<sup>1,9</sup> · Necati Enver<sup>1,10</sup> · Andrea Nacci<sup>1,11</sup> · Maria Rosaria Barillari<sup>1,12</sup> · Antonio Schindler<sup>1,13</sup> · Lise Crevier-Buchman<sup>1,14</sup> · Stéphane Hans<sup>1,14</sup> · Virginie Simeone<sup>1,14</sup> · Elzbieta Wlodarczyk<sup>1,15</sup> · Bernard Harmegnies<sup>1,3</sup> · Marc Remacle<sup>1,16</sup> · Alexandra Rodriguez<sup>1,4</sup> · Didier Dequanter<sup>1,4</sup> · Pierre Eisendrath<sup>1,17</sup> · Giovanni Dapri<sup>1,18</sup> · Camille Finck<sup>1,19</sup> · Petros Karkos<sup>1,20</sup> · Hillevi Pendleton<sup>1,21</sup> · Tareck Ayad<sup>1,22</sup> · Vinciane Muls<sup>1,17</sup> · Sven Saussez<sup>1,2,4</sup>

<sup>1</sup> Laryngopharyngeal Reflux Study Group of Young-Otolaryngologists of the International Federations of Oto-Rhino-Laryngological Societies (YO-IFOS), Paris, France

<sup>2</sup> Laboratory of Anatomy and Cell Biology, Faculty of Medicine, UMONS Research Institute for Health Sciences and Technology, University of Mons (UMons), Avenue du Champ de mars, 6, 7000 Mons, Belgium

<sup>3</sup> Laboratory of Phonetics, Faculty of Psychology, Research Institute for Language Sciences and Technology, University of Mons (UMons), Mons, Belgium

<sup>4</sup> Department of Otorhinolaryngology and Head and Neck Surgery, CHU de Bruxelles, CHU Saint-Pierre, School of Medicine, Université Libre de Bruxelles, Brussels, Belgium

<sup>5</sup> Department of Otolaryngology, Polyclinique Elsan, Poitiers, France

<sup>6</sup> Department of Otorhinolaryngology and Head and Neck Surgery, CHU de Lille, Université de Lille, Lille, France

<sup>7</sup> Department of Otorhinolaryngology and Head and Neck Surgery, University Hospital Ostrava, Ostrava, Czech Republic

<sup>8</sup> Department of Otolaryngology, Hospital Complex of Santiago de Compostela, Santiago de Compostela, Spain

<sup>9</sup> Department of Otorhinolaryngology-Head and Neck Surgery, Hospital Universitario Donostia, San Sebastian, Spain

<sup>10</sup> Department of Otolaryngology, Pendik Training and Research Hospital, Marmara University, Istanbul, Turkey

<sup>11</sup> ENT Audiology and Phoniatic Unit, University of Pisa, Pisa, Italy

<sup>12</sup> Division of Phoniatics and Audiology, Department of Mental and Physical Health and Preventive Medicine, University of L. Vanvitelli, Naples, Italy

<sup>13</sup> Department of Biomedical and Clinical Sciences, Phoniatic Unit, L. Sacco Hospital, University of Milan, Milan, Italy

<sup>14</sup> Department of Otorhinolaryngology and Head and Neck Surgery, Foch Hospital, Paris, France

<sup>15</sup> World Hearing Center, Institute of Physiology and Pathology of Hearing, Kajetany, Warsaw, Poland

<sup>16</sup> Department of Otorhinolaryngology and Head and Neck Surgery, CH Luxembourg, Luxembourg, Belgium

<sup>17</sup> Department of Gastroenterology, CHU de Bruxelles, CHU Saint-Pierre, School of Medicine, Université Libre de Bruxelles, Brussels, Belgium

<sup>18</sup> Department of Gastrointestinal Surgery, European School of Laparoscopic Surgery, Saint-Pierre University Hospital, Université Libre de Bruxelles, Brussels, Belgium

<sup>19</sup> Department of Otorhinolaryngology and Head and Neck Surgery, CHU de Liège, Liège, Belgium

<sup>20</sup> Department of Otolaryngology-Head and Neck Surgery, Thessaloniki Medical School, Thessaloniki, Greece

<sup>21</sup> Division of Ear, Nose and Throat Diseases, Ellenbogen, Malmö, Sweden

<sup>22</sup> Division of Otolaryngology-Head and Neck Surgery, Centre Hospitalier de l'Université de Montréal, Montreal, QC, Canada