Review Article

The research progress on the association between dietary habits and esophageal cancer: a narrative review

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Objective: This article summarizes the research progress on the association of dietary habits with esophageal cancer (EC), with the aim of gaining a better understanding of lifestyle, dietary, and drug-related risk factors as well as protective factors of EC.

Background: EC is one of the most common malignant cancers and has a high mortality rate. Evidence has suggested that dietary habits are closely related to EC. Several researches refer to the issue of the relationship between dietary habits and EC, but rare of them summary systematically and comprehensively. It is of great importance to study the role of dietary habits in the etiology of EC for the prevention and treatment of this disease.

Methods: We searched the China National Knowledge Infrastructure (CNKI), PubMed, Medline and EMBASE databases to identify studies on the association of dietary habits with EC published between the databases' dates of inception and March 2021.

Conclusions: This comprehensive review summarizes the state-of-the-art research findings on the association between dietary habits and EC, with the literature we obtained revealing that dietary habits have been increasingly regarded as potential etiologies or protective factors of EC, which reinforce the importance of suitable planning of interventions to reduce the burden of this disease among the population.

Keywords: Esophageal cancer (EC); dietary habits; food; risk factor

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Introduction

Esophageal cancer (EC) is one of the most threatening malignant tumors, ranking seventh and fifth of all cancers for mortality and mortality, respectively (1). In 2015, approximately 245,700 new cases of EC were reported in China and 188,100 lives were lost; the incidence rate and mortality rate of EC were 17.87/10 and 13.68/10 13.68/10, respectively, placing it fourth among malignant tumors (2). According to the latest research report, there were 234,624 new cases of EC in China in 2017, and 212,586 people died due to the disease (3). However, the early symptoms of EC are not typical; consequently, in most cases, the patient is diagnosed in the advanced stage, and the disease progresses rapidly, resulting in a poor prognosis. There are 2 main histological subtypes of EC: esophageal squamous cell carcinoma (ESCC) and esophageal adenocarcinoma (EAC), which account for about 88% and 12% of cases, respectively.

Significant geographical differences have been reported in the incidence of EC around the world. The incidence rate is highest in East Asia, East Africa, South Africa, and

Northern Europe, while Central America has the lowest incidence rate (4). In China, a high incidence of EC has been recorded in the Taihang Mountains area, Dabie Mountains area, Yanting County in Sichuan province, and Linxian County in Henan province, indicating that the influence of environmental and dietary habits on EC is worthy of our attention.

Epidemiological studies have confirmed that diet and nutrition are important factors in the risk of EC (5). Several researches refer to the issue of the relationship between dietary habits and EC, but rare of them summary systematically and comprehensively (6). Therefore, this paper reviews the literature on the relationship between dietary habits and EC, with the aim of improving the understanding of the etiologies of this disease, in order to inform and strengthen efforts to prevent and treat it.

We present the following article in accordance with the Narrative Review reporting checklist (available at https://dx.doi.org/10.21037/apm-21-1467).

Methods

We searched PubMed, EMBASE, Medline, and China National Knowledge Infrastructure (CNKI) to identify studies on the relationship between dietary habits and EC risk published between the databases' dates of inception and March 2021. The search terms included "food/foods," "diet/dietary," and "lifestyle/lifestyles" in combination with "esophageal/oesophageal/esophagus/oesophagus".

The selection criteria were as follows: (I) studies including patients who were diagnosed with EC; and (II) original articles, narrative reviews, systematic reviews, and meta-analyses. The language of the studies included in the analysis was limited to English or Chinese.

Smoking and drinking

A large number of studies on the relationship between lifestyle or eating habits and EC have been carried out. Although the geographical factors and ethnic groups in these studies have differed, the research results show some commonalities. For instance, smoking and drinking have been confirmed to be closely related to EC in many studies (7-10). In China, heavy drinking and smoking are the main risk factors for EC (11), while in a Western study of ESCC, 80% of men and 40% of women had heavy drinking and smoking habits (12). A systematic analysis of 195 countries found that a large part of disability-adjusted life years caused by EC was attributable to smoking and drinking (13). A meta-analysis of smoking and EAC risk among white non-Hispanic ethnic groups found that the risk of EAC for smokers was nearly twice that of non-smokers [odds ratio (OR) =1.96, 95% confidence interval (CI): 1.64–2.34] and the degree of smoking was significantly correlated with long-term outcomes (14). In recent years, water pipe smoking has grown in popularity around the world. A systematic review of the effects of water pipe smoking on health revealed a significant correlation between water pipe smoking and EC (OR =3.63, CI: 1.39–9.44; I^2=94.49%) (15), and other studies have reached similar conclusions (16). Studies have found that smokers often also have the habit of heavy drinking. Pandeya *et al.* reported that more than 75% of ESCC cases in men were attributable to smoking and drinking a lot (12). EC appears to be a product of the combined effect of smoking and drinking. Steevens *et al.* found that smokers who consumed more than 15 g of alcohol per day had an 8-fold higher risk of developing ESCC than never-smokers who consumed less than 5 g of alcohol per day [risk ratio (RR) =8.05, 95% CI: 3.89–16.60] (17). Another case-control study identified a similar cumulative effect, with smokers who consumed more than 420 g of alcohol per week displaying a 20 times higher risk of developing ESCC than smokers who never drank (OR =21.87, 95% CI: 3.90–122.49) (18). Research has also shown tobacco specific nitrosamines and polycyclic aromatic hydrocarbons to be the main carcinogens in cigarettes (19,20).

Vitamins and minerals

Vitamins and minerals play the role of antioxidants, helping to reduce DNA mutations, and may thus lower the risk of EC. In 1961, Wynder and Bross reported that a diet rich in milk and vitamins might contribute to reducing the occurrence of EC; since then, the relationship between EC and diet has been widely investigated (21). A report by the World Health Organization on diet, nutrition, and the prevention of chronic diseases also pointed out that in developing countries, about 60% of EC cases are believed to be related to nutrients (7). There is limited evidence of benefits from vitamin and mineral supplementation for the prevention of cancer (22). In recent years, diet and nutrition have been evidenced as being important factors in EC risk, and some prominent data regarding the impact of antioxidant vitamins and minerals stems have been unearthed. From 1985 to 1991, 29,584 villagers aged

40–69 from Linxian, China, were given 1 of 4 vitamin and mineral combinations daily: factor A (retinol/zinc), factor B (riboflavin/nicotinic acid), factor C (vitamin C/molybdenum), factor D (selenium/vitamin E/β-carotene), or placebo. Ten years after the trial's end, follow-up revealed that among participants younger than 55 years old, the mortality of EC in factor D group was decreased by 17%, compared to those who did not receive factor D. While among participants older than 55 years old, the mortality of EC was increased by 14%, compared to those who did not (23). The team conducted another follow-up survey 25 years after the end of the trial, and the results showed that the protective effect of factor D on cancer lasted for 10 years (24), with selenium playing a major protective role (25-27). The results showed that factor B had a significant protective effect on EC, whereas factor C and factor D increased the risk of EC in people over 55 years old (24). It has been reported that poor nutritional intake carries a higher risk of EC, but dietary supplementation of selenium, beta carotene, and vitamin E can reduce cancer mortality by 13% (28-30). Meanwhile, low levels of selenium and zinc have been associated with EC in some studies (25,31). Bollschweiler *et al.* reported that low intake of vitamins C and E was correlated with an increased risk of EC (32). Additionally, intake of fiber, b-carotene, folate, vitamin B6, and vitamin C were found to be inversely associated with both types of EC, while cholesterol, animal protein, and vitamin B12 showed direct associations (33,34).

Fruits and vegetables

The antioxidant properties of fruits and vegetables aid in reducing oxidative stress and inflammation. A number of studies have confirmed that the intake of fresh fruits and vegetables is closely related to EC (34-37). A systematic review of 57 cohort studies by WCRF/AICR showed that fruits and vegetables have protective effects against EC (36). The risk of EAC was found to be inversely proportional to vegetable intake [RR =0.89 (100 g/day), 95% CI: 0.80–0.99], and that of ESCC was inversely proportional to fruit intake [RR =0.84 (100 g/D), 95% CI: 0.75–0.94]. Another meta-analysis revealed a negative correlation between fruit and vegetable intake and the risk of ESCC (38). These results suggest that the anti-cancer components such as vitamins, dietary fiber, and carotene in fresh fruits and vegetables may inhibit the occurrence of EC.

Hot food

In some areas of Africa and China, people have the habit of eating hot food. Studies have reported that hot food and drink can increase the risk of EC (39,40). A case-control study in Kenya, which is a high-incidence country for ESCC, reported the OR of ESCC to be 3.7 (95% CI: 2.1–6.5) (41). A prospective study in China on the relationships of high-temperature tea drinking, alcohol and smoking with EC risk revealed an association between tea temperature and EC only in men. Compared with individuals who drank tea less than once a week, the hazard ratios (HRs) of those who drank warm tea, hot tea, and hot tea every day were 1.17 (95% CI: 0.91–1.50), 1.30 (95% CI: 1.05–1.50), and 55 (95% CI: 1.19–2.02) respectively. The risk of EC for people who drink hot tea is elevated by drinking or smoking at the same time. Compared with individuals who drank tea less than once a week and consumed less than 5 g of alcohol daily, those who drank hot tea every day and consumed alcohol more than 15 g of alcohol daily had the highest risk of EC (HR =5.00, 95% CI: 3.64–6.88), while drinking hot tea every day and smoking carried an HR of 2.03 (95% CI: 1.55–2.67) (42). Studies have also shown that a diet above 55–60 ℃ is associated with the occurrence of EC and gastric cancer (43). Furthermore, a study of 5,076 urban residents showed that drinking shallow well water elevated the risk of esophageal precancerous lesions compared with drinking tap water (OR =1.84, 95% CI: 1.18–2.89) (44). Although the molecular structure of dihydrogen and trihydroxy contained in tea polyphenols play a strong role in scavenging reactive oxygen species, which can reduce the risk of EC (39), drinking tea at high temperatures has the opposite effect. Heat injury of the esophageal mucosa caused by hot food may destroy its barrier protection function and stimulate inflammation. The destruction of the protective function of the barrier permits the invasion of carcinogens, and the release of N-nitroso compounds caused by inflammation also promotes the occurrence and development of EC.

Processed or pickled food

Another important risk factor for EC is a pickled diet, as the presence of some nitrosamines and polycyclic aromatic hydrocarbons may lead to carcinogenesis. There is a long history of pickled food consumption in China. Pickled foods include smoked or pickled poultry, meat, and fish products, bean products, vegetables, and fruits. They are extremely popular due to their unique taste, and each

region has its own signature pickled foods. However, they contain large quantities of nitrate and nitrite, and nitroso compounds are not only recognized animal carcinogens, but are also important pathogenic factors in some human tumors. Long-term consumption of food containing high levels of nitrate, which can cause various tumors, and available evidence supports a positive association between nitrite and nitrosamine intake and EC (45). In the past 2 decades, numerous studies have indicated that the intake of processed or pickled food might be associated with an increased risk of EC (46-53). For instance, Islami *et al.* reported that eating pickled food increased the risk of ESCC by 132% (50). One systematic review and meta-analysis explored the association of EC risk and a diet of processed food (including pickled food) or pickled food alone. It showed that the highest categories of processed food intake were associated with a 78% increase in the risk of EC compared with the lowest categories, and the results indicated that the combined OR/RR (95% CI) of studies comparing the highest and lowest categories was 2.10 (1.64–2.69) for pickled food. Subgroup analysis of the case-control studies indicated significant positive associations between EC risk and processed or pickled food intake (combined ORs: processed food: 1.93; 95% CI: 1.66–2.24, pickled food: 2.28; 95% CI: 1.93–2.70) (51).

Fried food

When fried at a high temperature, protein-rich foods can produce volatile and non-volatile carcinogens. Volatile carcinogens can include malondialdehyde, acetaldehyde, acrolein, and ethylene oxide, among others. Non-volatile carcinogens, such as heterocyclic amines, polycyclic aromatic hydrocarbons, and acrylamide, can also be detected on the surface of some fried food (52-54). A large number of studies have shown that long-term consumption of fried food increases the risk of EC (47,54,55). Galeone *et al.* conducted 2 case-control studies in Italy and Switzerland between 1992 and 1999 to investigate the relationship of fried foods with oropharyngeal cancer and EC. They found that the multivariate ORs for an increment of 1 portion of fried foods per week was 1.16 (95% CI: 1.08–1.26) for EC (54).

Red meat

Red meat and processed meat contain large quantities of heme, heterocyclic amines, polycyclic aromatic hydrocarbons, and N-nitroso complexes, which can catalyze lipid peroxidation, damage cell DNA, and lead to carcinogenesis. A balanced human diet includes a reasonable amount of meat, since it provides valuable nutrients such as proteins and essential amino acids, vitamins, minerals, and other micronutrients. Cross *et al.* (56) conducted a large-sample cohort study, which confirmed that intake of red meat and processed meat was positively correlated with the occurrence of ESCC. Keszei *et al.* (57) also found that red meat and processed meat could significantly increase the risk of ESCC, but had no significant correlation with other types of EC. Salehi *et al.* (58) further confirmed that eating less red meat and processed meat, and eating more fish meat can lower the risk of ESCC. In their meta-analysis of 3 cohort and 7 case-control studies (59), Huang *et al.* observed a significant risk of EAC in individuals with a high intake of red meat (RR, 1.31; 95% CI, 1.05–1.64) and processed meat (RR; 1.41; 95% CI, 1.09–1.83). Similarly, Qu *et al.* and Choi *et al.* reported that the total consumption of red meat was significantly associated with both of EAC and ESCC (49,60).

Cereals and legumes

Cereals and legumes are the most important and largest plant group responsible for meeting nutritional requirements worldwide, and their consumption has been pointed out as being a protective factor in the prevention of EC (61-63). Existing research shows that rice contains vitamin E, which can reduce cholesterol and prevent some cancers. However, some studies have shown that consuming brown rice, corn, soybeans, and other coarse grains can also reduce the risk of cancer. Soybeans and other coarse cereals are rich in flavonoids, which can inhibit the proliferation of and induce G2/M cell-cycle arrest of EC cells *in vivo*; thus, they may play a role in the prevention of cancer. However, whether long-term consumption of coarse cereals can prevent EC awaits further discussion (64,65).

Drugs

Various common drugs can aid in reducing the risk of EC. Statins, which are among the most common drugs used to treat cardiovascular diseases, can promote cell apoptosis and inhibit tumor cell proliferation. Singh *et al.* reported that the incidence of EC in patients taking statins showed a significant reduction (of 28%) from that in patients not taking these drugs (66). A multicenter study in the

Netherlands found that the use of statins and non-steroidal anti-inflammatory drugs (NSAIDs) reduced the risk of Barrett's esophagus progressing to EC by 54% and 53%, respectively. The combination of NSAIDs with statins had a synergistic protective effect (HR =0.22, 95% CI: 0.06–0.85) (67). Further, the longer statins are used, the greater the risk reduction (68). A British cohort study found that the use of statins after diagnosis in patients with EAC can reduce both cancer-specific and all-cause mortality (69). Experiments have shown that statins exert antitumor effects, mainly by affecting 3-hydroxy-3-methylglutaryl-CoA reductase-dependent and non-dependent pathways (70). Further, statins have been proved to exert antitumor effects in EC cells by inhibiting the RAS, extracellular signal-regulated kinase (ERK), and Akt signaling pathways, as well as cyclooxygenase-2 (COX-2) (71-73). The above studies suggested that statins can play an active role in the prevention and treatment of EC.

A meta-analysis of observational studies showed that the use of proton pump inhibitors (PPIs) reduced the risk of progressing from Barrett's esophagus to highly atypical hyperplasia or EAC by 71% (74). A similar trend was observed in case-control studies of male veterans in the United States, and histamine H2 receptor antagonists were found to reduce the risk of Barrett's esophagus progressing to EAC. High-dose PPIs were observed to significantly reduce the risk of EAC (Omeprazole >40 mg/d, OR =0.11, 95% CI: 0.04–0.36); however, the incidence of EAC was not found to be associated with the dose response of PPI duration (75). Further, in a national case-control study in Denmark, PPIs did not play a protective role in the development of Barrett's esophagus (76). An Irish observational study reported that aspirin could significantly reduce the risk of Barrett's esophagus and EAC, and also uncovered a negative correlation of NSAIDs with the risk of Barrett's esophagus and EAC (77). Additionally, the use of benzodiazepine drugs, beta adrenoceptor agonists, anticholinergics, or nitroglycerin for more than 5 years increases the risk of EAC, because it reduces the tension of the esophageal sphincter (78). The reason for this is that a decrease in tension in the lower esophageal sphincter leads to the exposure of the distal esophagus to a strongly acidic environment, which promotes the occurrence of EC.

Limitations

The present review has some limitations. Firstly, this review paper is based on the authors' summary of the literature; thus, it may contain publication bias. Secondly, our paper did not discuss heterogeneity among the studies, which may have been influenced by the publication year, number of cases, race, geographic region, and other confounders, and may have resulted in bias and impacted on the conclusions.

Conclusions

In conclusion, this comprehensive review has summarized the state-of-the-art research findings on the association between dietary habits and EC, which reinforce the importance of suitable planning of interventions to reduce the burden of this disease among the population. Large-scale public health interventions should be established, such as an indoor smoking ban, which has achieved a degree of success in some Western countries. It should be advocated to a healthy and well-balanced diet, including avoiding eating too fast, too hard, too thick and squatting habits, smoking and alcohol cession, and it is also feasible to supplement nutrients for people in high-incidence areas of EC, and to vigorously promote adequate intake of fresh fruits and vegetables and reduce the consumption of red meat. At present, the evidence level of most studies is low, and large-scale multicenter randomized controlled trials on EC, as the seventh most common malignant tumor in the world, are urgently needed to obtain more scientific evidence. Moreover, at the epidemiological level, more basic research is needed to clarify the relationship between lifestyle and dietary habits, drugs, and the pathogenesis of EC.

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Footnote

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Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://dx.doi.org/10.21037/apm-21-1467). The authors have no conflicts of interest to declare.

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References

1. Bray F, Ferlay J, Soerjomataram I, et al. Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2018;68:394-424.
2. Chen R, Zheng RS, Zhang SW, et al. Analysis of incidence and mortality of esophageal cancer in China, 2015. Zhonghua Yu Fang Yi Xue Za Zhi 2019;53:1094-7.
3. Yang S, Lin S, Li N, et al. Burden, trends, and risk factors of esophageal cancer in China from 1990 to 2017: an up-to-date overview and comparison with those in Japan and South Korea. J Hematol Oncol 2020;13:146.
4. Arnold M, Soerjomataram I, Ferlay J, et al. Global incidence of oesophageal cancer by histological subtype in 2012. Gut 2015;64:381-7.
5. Steinmetz KA, Potter JD. Vegetables, fruit, and cancer. I. Epidemiology. Cancer Causes Control 1991;2:325-57.
6. Palladino-Davis AG, Mendez BM, Fisichella PM, et al. Dietary habits and esophageal cancer. Dis Esophagus 2015;28:59-67.
7. Diet, nutrition and the prevention of chronic diseases. World Health Organ Tech Rep Ser 2003;916:i-viii, 1-149, backcover.
8. McCain RS, McManus DT, McQuaid S, et al. Alcohol intake, tobacco smoking, and esophageal adenocarcinoma survival: a molecular pathology epidemiology cohort study. Cancer Causes Control 2020;31:1-11.
9. Oze I, Charvat H, Matsuo K, et al. Revisit of an unanswered question by pooled analysis of eight cohort studies in Japan: Does cigarette smoking and alcohol drinking have interaction for the risk of esophageal cancer? Cancer Med 2019;8:6414-25.
10. Jin ZY, Wallar G, Zhou JY, et al. Consumption of garlic and its interactions with tobacco smoking and alcohol drinking on esophageal cancer in a Chinese population. Eur J Cancer Prev 2019;28:278-86.
11. Mmbaga EJ, Mushi BP, Deardorff K, et al. A Case-Control Study to Evaluate Environmental and Lifestyle Risk Factors for Esophageal Cancer in Tanzania. Cancer Epidemiol Biomarkers Prev 2021;30:305-16.
12. Pandeya N, Olsen CM, Whiteman DC. Sex differences in the proportion of esophageal squamous cell carcinoma cases attributable to tobacco smoking and alcohol consumption. Cancer Epidemiol 2013;37:579-84.
13. GBD 2017 Oesophageal Cancer Collaborators. The global, regional, and national burden of oesophageal cancer and its attributable risk factors in 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol Hepatol 2020;5:582-97.
14. Cook MB, Kamangar F, Whiteman DC, et al. Cigarette smoking and adenocarcinomas of the esophagus and esophagogastric junction: a pooled analysis from the international BEACON consortium. J Natl Cancer Inst 2010;102:1344-53.
15. Montazeri Z, Nyiraneza C, El-Katerji H, et al. Waterpipe smoking and cancer: systematic review and meta-analysis. Tob Control 2017;26:92-7.
16. Awan KH, Siddiqi K, Patil Sh, et al. Assessing the Effect of Waterpipe Smoking on Cancer Outcome - a Systematic Review of Current Evidence Asian Pac J Cancer Prev 2017;18:495-502.
17. Steevens J, Schouten LJ, Goldbohm RA, et al. Alcohol consumption, cigarette smoking and risk of subtypes of oesophageal and gastric cancer: a prospective cohort study. Gut 2010;59:39-48.
18. Pandeya N, Williams G, Green AC, Webb PM, Whiteman DC; Australian Cancer Study. Alcohol consumption and the risks of adenocarcinoma and squamous cell carcinoma of the esophagus. Gastroenterology 2009;136:1215-24, e1-2.
19. Helmfrid I, Ljunggren S, Nosratabadi R, et al. Exposure of metals and PAH through local foods and risk of cancer in a historically contaminated glassworks area. Environ Int 2019;131:104985.
20. Shahab L, Goniewicz ML, Blount BC, et al. Nicotine, Carcinogen, and Toxin Exposure in Long-Term E-Cigarette and Nicotine Replacement Therapy Users: A Cross-sectional Study. Ann Intern Med 2017;166:390-400.
21. WYNDER EL, BROSS IJ. A study of etiological factors

in cancer of the esophagus. Cancer 1961;14:389-413.
22. Fortmann SP, Burda BU, Senger CA, et al. Vitamin and mineral supplements in the primary prevention of cardiovascular disease and cancer: An updated systematic evidence review for the U.S. Preventive Services Task Force. Ann Intern Med 2013;159:824-34.
23. Qiao YL, Dawsey SM, Kamangar F, et al. Total and cancer mortality after supplementation with vitamins and minerals: follow-up of the Linxian General Population Nutrition Intervention Trial. J Natl Cancer Inst 2009;101:507-18.
24. Wang SM, Taylor PR, Fan JH, et al. Effects of Nutrition Intervention on Total and Cancer Mortality: 25-Year Post-trial Follow-up of the 5.25-Year Linxian Nutrition Intervention Trial. J Natl Cancer Inst 2018;110:1229-38.
25. Mark SD, Qiao YL, Dawsey SM, et al. Prospective study of serum selenium levels and incident esophageal and gastric cancers. J Natl Cancer Inst 2000;92:1753-63.
26. Taylor PR, Qiao YL, Abnet CC, et al. Prospective study of serum vitamin E levels and esophageal and gastric cancers. J Natl Cancer Inst 2003;95:1414-6.
27. Abnet CC, Qiao YL, Dawsey SM, et al. Prospective study of serum retinol, beta-carotene, beta-cryptoxanthin, and lutein/zeaxanthin and esophageal and gastric cancers in China. Cancer Causes Control 2003;14:645-55.
28. Li B, Taylor PR, Li JY, et al. Linxian nutrition intervention trials. Design, methods, participant characteristics, and compliance. Ann Epidemiol 1993;3:577-85.
29. Li JY, Taylor PR, Li B, et al. Nutrition intervention trials in Linxian, China: multiple vitamin/mineral supplementation, cancer incidence, and disease-specific mortality among adults with esophageal dysplasia. J Natl Cancer Inst 1993;85:1492-8.
30. Blot WJ, Li JY, Taylor PR, et al. Nutrition intervention trials in Linxian, China: supplementation with specific vitamin/mineral combinations, cancer incidence, and disease-specific mortality in the general population. J Natl Cancer Inst 1993;85:1483-92.
31. Abnet CC, Lai B, Qiao YL, et al. Zinc concentration in esophageal biopsy specimens measured by x-ray fluorescence and esophageal cancer risk. J Natl Cancer Inst 2005;97:301-6.
32. Bollschweiler E, Wolfgarten E, Nowroth T, et al. Vitamin intake and risk of subtypes of esophageal cancer in Germany. J Cancer Res Clin Oncol 2002;128:575-80.
33. Mayne ST, Risch HA, Dubrow R, et al. Nutrient intake and risk of subtypes of esophageal and gastric cancer. Cancer Epidemiol Biomarkers Prev 2001;10:1055-62.
34. Steevens J, Schouten LJ, Goldbohm RA, et al. Vegetables and fruits consumption and risk of esophageal and gastric cancer subtypes in the Netherlands Cohort Study. Int J Cancer 2011;129:2681-93.
35. Vingeliene S, Chan DSM, Vieira AR, et al. An update of the WCRF/AICR systematic literature review and meta-analysis on dietary and anthropometric factors and esophageal cancer risk. Ann Oncol 2017;28:2409-19.
36. Vingeliene S, Chan DS, Aune D, et al. An update of the WCRF/AICR systematic literature review on esophageal and gastric cancers and citrus fruits intake. Cancer Causes Control 2016;27:837-51.
37. Jessri M, Rashidkhani B, Hajizadeh B, et al. Adherence to Mediterranean-style dietary pattern and risk of esophageal squamous cell carcinoma: a case-control study in Iran. J Am Coll Nutr 2012;31:338-51.
38. Liu J, Wang J, Leng Y, et al. Intake of fruit and vegetables and risk of esophageal squamous cell carcinoma: a meta-analysis of observational studies. Int J Cancer 2013;133:473-85.
39. Chen Y, Tong Y, Yang C, et al. Consumption of hot beverages and foods and the risk of esophageal cancer: a meta-analysis of observational studies. BMC Cancer 2015;15:449.
40. Andrici J, Eslick GD. Hot Food and Beverage Consumption and the Risk of Esophageal Cancer: A Meta-Analysis. Am J Prev Med 2015;49:952-60.
41. Middleton DR, Menya D, Kigen N, et al. Hot beverages and oesophageal cancer risk in western Kenya: Findings from the ESCCAPE case-control study. Int J Cancer 2019;144:2669-76.
42. Yu C, Tang H, Guo Y, et al. Hot Tea Consumption and Its Interactions With Alcohol and Tobacco Use on the Risk for Esophageal Cancer: A Population-Based Cohort Study. Ann Intern Med 2018;168:489-97.
43. Yi M, Wu X, Zhuang W, et al. Tea Consumption and Health Outcomes: Umbrella Review of Meta-Analyses of Observational Studies in Humans. Mol Nutr Food Res 2019;63:e1900389.
44. He L, He T, Farrar S, et al. Antioxidants Maintain Cellular Redox Homeostasis by Elimination of Reactive Oxygen Species. Cell Physiol Biochem 2017;44:532-53.
45. Jakszyn P, Gonzalez CA. Nitrosamine and related food intake and gastric and oesophageal cancer risk: a systematic review of the epidemiological evidence. World J Gastroenterol 2006;12:4296-303.
46. Sun Y, Zhang T, Wu W, et al. Risk Factors Associated with Precancerous Lesions of Esophageal Squamous Cell

Carcinoma: a Screening Study in a High Risk Chinese Population. J Cancer 2019;10:3284-90.
47. Bosetti C, La Vecchia C, Talamini R, et al. Food groups and risk of squamous cell esophageal cancer in northern Italy. Int J Cancer 2000;87:289-94.
48. Takezaki T, Gao CM, Wu JZ, et al. Dietary protective and risk factors for esophageal and stomach cancers in a low-epidemic area for stomach cancer in Jiangsu Province, China: comparison with those in a high-epidemic area. Jpn J Cancer Res 2001;92:1157-65.
49. Choi Y, Song S, Song Y, et al. Consumption of red and processed meat and esophageal cancer risk: meta-analysis. World J Gastroenterol 2013;19:1020-9.
50. Islami F, Ren JS, Taylor PR, et al. Pickled vegetables and the risk of oesophageal cancer: a meta-analysis. Br J Cancer 2009;101:1641-7.
51. Li K, Yu P. Food groups and risk of esophageal cancer in Chaoshan region of China: a high-risk area of esophageal cancer. Cancer Invest 2003;21:237-40.
52. Yan B, Zhang L, Shao Z. Consumption of processed and pickled food and esophageal cancer risk: A systematic review and meta-analysis. Bull Cancer 2018;105:992-1002.
53. Straif K, Baan R, Grosse Y, et al. Carcinogenicity of household solid fuel combustion and of high-temperature frying. Lancet Oncol 2006;7:977-8.
54. Galeone C, Pelucchi C, Talamini R, et al. Role of fried foods and oral/pharyngeal and oesophageal cancers. Br J Cancer 2005;92:2065-9.
55. Bosetti C, Talamini R, Levi F, et al. Fried foods: a risk factor for laryngeal cancer? Br J Cancer 2002;87:1230-3.
56. Cross AJ, Freedman ND, Ren J, et al. Meat consumption and risk of esophageal and gastric cancer in a large prospective study. Am J Gastroenterol 2011;106:432-42.
57. Keszei AP, Schouten LJ, Goldbohm RA, et al. Red and processed meat consumption and the risk of esophageal and gastric cancer subtypes in The Netherlands Cohort Study. Ann Oncol 2012;23:2319-26.
58. Salehi M, Moradi-Lakeh M, Salehi MH, et al. Meat, fish, and esophageal cancer risk: a systematic review and dose-response meta-analysis. Nutr Rev 2013;71:257-67.
59. Huang W, Han Y, Xu J, et al. Red and processed meat intake and risk of esophageal adenocarcinoma: a meta-analysis of observational studies. Cancer Causes Control 2013;24:193-201.
60. Qu X, Ben Q, Jiang Y. Consumption of red and processed meat and risk for esophageal squamous cell carcinoma based on a meta-analysis. Ann Epidemiol 2013;23:762-770.e1.
61. Bouchenak M, Lamri-Senhadji M. Nutritional quality of legumes, and their role in cardiometabolic risk prevention: a review. J Med Food 2013;16:185-98.
62. Makarem N, Nicholson JM, Bandera EV, et al. Consumption of whole grains and cereal fiber in relation to cancer risk: a systematic review of longitudinal studies. Nutr Rev 2016;74:353-73.
63. Skeie G, Braaten T, Olsen A, et al. Intake of whole grains and incidence of oesophageal cancer in the HELGA Cohort. Eur J Epidemiol 2016;31:405-14.
64. Martens S, Mithöfer A. Flavones and flavone synthases. Phytochemistry 2005;66:2399-407.
65. Neuhouser ML. Dietary flavonoids and cancer risk: evidence from human population studies. Nutr Cancer 2004;50:1-7.
66. Singh S, Singh AG, Singh PP, et al. Statins are associated with reduced risk of esophageal cancer, particularly in patients with Barrett's esophagus: a systematic review and meta-analysis. Clin Gastroenterol Hepatol 2013;11:620-9.
67. Kastelein F, Spaander MC, Biermann K, et al. Nonsteroidal anti-inflammatory drugs and statins have chemopreventative effects in patients with Barrett's esophagus. Gastroenterology 2011;141:2000-8; quiz e13-4.
68. Nguyen DM, Richardson P, El-Serag HB. Medications (NSAIDs, statins, proton pump inhibitors) and the risk of esophageal adenocarcinoma in patients with Barrett's esophagus. Gastroenterology 2010;138:2260-6.
69. Alexandre L, Clark AB, Bhutta HY, et al. Association Between Statin Use After Diagnosis of Esophageal Cancer and Survival: A Population-Based Cohort Study. Gastroenterology 2016;150:854-65.e1; quiz e16-7.
70. Demierre MF, Higgins PD, Gruber SB, et al. Statins and cancer prevention. Nat Rev Cancer 2005;5:930-42.
71. Ogunwobi OO, Beales IL. Statins inhibit proliferation and induce apoptosis in Barrett's esophageal adenocarcinoma cells. Am J Gastroenterol 2008;103:825-37.
72. Konturek PC, Burnat G, Hahn EG. Inhibition of Barret's adenocarcinoma cell growth by simvastatin: involvement of COX-2 and apoptosis-related proteins. J Physiol Pharmacol 2007;58 Suppl 3:141-8.
73. Ye F, Zhang GH, Guan BX, et al. Suppression of esophageal cancer cell growth using curcumin, (-)-epigallocatechin-3-gallate and lovastatin. World J Gastroenterol 2012;18:126-35.
74. Uhlenhopp DJ, Then EO, Sunkara T, et al. Epidemiology of esophageal cancer: update in global trends, etiology and risk factors. Clin J Gastroenterol 2020;13:1010-21.

75. Tan MC, El-Serag HB, Yu X, et al. Acid suppression medications reduce risk of oesophageal adenocarcinoma in Barrett's oesophagus: a nested case-control study in US male veterans. Aliment Pharmacol Ther 2018;48:469-77.
76. Hvid-Jensen F, Pedersen L, Funch-Jensen P, et al. Proton pump inhibitor use may not prevent high-grade dysplasia and oesophageal adenocarcinoma in Barrett's oesophagus: a nationwide study of 9883 patients. Aliment Pharmacol Ther 2014;39:984-91.
77. Anderson LA, Johnston BT, Watson RG, et al. Nonsteroidal anti-inflammatory drugs and the esophageal inflammation-metaplasia-adenocarcinoma sequence. Cancer Res 2006;66:4975-82.
78. Lagergren J, Bergström R, Adami HO, et al. Association between medications that relax the lower esophageal sphincter and risk for esophageal adenocarcinoma. Ann Intern Med 2000;133:165-75.

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