ORIGINAL ARTICLE

Smoking Behaviors, Dietary Patterns, and Dyspepsia among Indonesian Adults in 2022

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ABSTRACT

Introduction: The global prevalence of dyspeptic syndrome among adults was estimated to be 1.8–57% in 2014, with an average of 20.8%. In a pilot survey involving 30 respondents, we found 21 acute respiratory infections, 16 dyspeptic syndromes, and 6 hypertension cases. Smoking was observed in 70% of the total respondents. This was the basis for conducting additional research in 2022 to examine the relationship between dyspepsia, smoking behaviors, and dietary patterns among adults in Krajan Hamlet, Malang, Indonesia.
Methods: This quantitative study used an analytical observational design with a cross-sectional approach. The sample consisted of 70 adults aged 18–65, selected by random sampling. The collected data were analyzed using the Chi-squared test ($p<0.05$).
Results: The prevalence rate for dyspepsia was 44.3%. Among them, 67.1% were smokers (both frequent and non-frequent), and 32.9% were non-smokers (including former and passive). The active smokers had a smoking duration of >2 years and smoked >10 cigarettes daily. A total of 11.4% of subjects exhibited eating habits of consuming trigger foods that put them at a high risk for dyspepsia. Specifically, 42.0% of subjects had an eating interval of >6 hours, while 87.1% had a meal frequency that led to a high risk of developing dyspepsia. There was a significant relationship between a high-risk diet and dyspeptic syndrome ($p=0.037$).
Conclusion: High-risk diets exhibited a relationship with the prevalence of dyspeptic syndrome. Early detection is crucial in an effort to prevent and reduce the incidence and complications of dyspeptic syndrome.

Keywords: Dyspepsia; tobacco use; dietary pattern; dyspepsia risk factors

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Highlights:
1. This study investigated the relationship between dyspepsia, smoking behaviors, and dietary patterns, which are often overlooked, particularly among those living in rural areas.
2. The findings of this study are anticipated to raise awareness regarding the detrimental impacts of smoking and unhealthy diets on dyspepsia.

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INTRODUCTION

Dyspepsia is a disorder that is often encountered by healthcare workers in their daily practice. There are various mechanisms that explain the pathophysiology of dyspepsia. One of which is an increase in gastric acid secretion, which causes stomach discomfort when exposed to the gastric mucosa (Setiadi et al., 2014). Several factors can influence the onset of dyspepsia, including psychological aspects, smoking behaviors, dietary choices, the use of steroids and non-steroid anti-inflammatory drugs (NSAIDs), and other medical conditions (Sidik, 2024). Cigarette smoking can increase the amount of gas in the stomach and damage the gastric mucosa, resulting in a condition known as dyspeptic syndrome (Purnamasari, 2017). Furthermore, irregular eating patterns and consumption of certain types of food can trigger increased stomach acid secretion. Excessive consumption of spicy and fatty foods will easily cause symptoms of dyspeptic syndrome because it slows stomach emptying, which can cause gastric acid reflux.

The prevalence of dyspepsia in Indonesia is high, affecting 40–50% of adults at the age of 40. This amounts to 10 million people, which makes up 6.5% of the total population. In 2020, it was estimated that the incidence of dyspepsia increased from 10 to 28 million, equivalent to 11.3% of the

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entire population in Indonesia (Syafriani, 2015). In the end of October 2022, we conducted a preliminary survey in the Krajan Hamlet, Sidodadi Village, Lawang District, Malang, Indonesia. Among the 30 respondents who participated in the survey, 16 had dyspeptic syndrome. Individuals who were smokers were found to account for 70% of the total respondents. We also found 21 acute respiratory infections and 6 hypertension cases among the respondents. The findings of the preliminary survey served as the basis for our present study. The general purpose of this study was to examine the relationship between dyspeptic syndrome and smoking behaviors as well as dietary patterns among adults residing in the hamlet. Additionally, the special purposes of this study were to analyze the characteristics of the research sample, determine the prevalence of dyspepsia, and analyze the distribution of smoking behaviors and dietary patterns in the sample that contribute to a higher risk of developing dyspepsia.

METHODS

This study employed quantitative research methods, namely an analytical observational design with a cross-sectional approach, to investigate the relationship between the incidence of dyspeptic syndrome and two different variables, i.e., smoking behaviors and eating patterns (Ranganathan & Aggarwal, 2019). The eating pattern variable consisted of eating interval, eating frequency, and the consumption of foods that are spicy and have high levels of acidity and fat. The analysis of the data was performed using the Chi-squared test to determine the relationship between dyspepsia, smoking behaviors, and eating patterns. IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA) was utilized to assist the analysis.

The research population comprised the residents of Krajan Hamlet, Sidodadi Village, Lawang District, Malang, Indonesia. A total of 70 respondents within the ages of 18–60 were selected as the research sample by random sampling (Emerson, 2015). All of the selected individuals agreed to participate in the interviews as research respondents. We conducted this study from October 30 to November 5, 2022. The data were collected by medical students from Universitas Airlangga, Surabaya, Indonesia, who were stationed at the Murnajati Public Health Training Center (Latkesmas Murnajati), Malang, Indonesia. The medical students were under the guidance of academic supervisors from Universitas Airlangga as well as field supervisors from the Murnajati Public Health Training Center.

The instrument utilized in this study was a questionnaire that included inquiries relating to dyspepsia, smoking behaviors, and dietary patterns (Nkurunziza et al., 2016). The independent variables in this study were smoking behaviors and dietary patterns, while the dependent variable was dyspepsia. The questionnaire included inquiries about dietary patterns, specifically the eating frequency (i.e., less than or more than three times a day), eating interval (i.e., every six hours or more, or every four to five hours), and the consumption of spicy foods, coffee, fizzy drinks, fried foods, as well as cassavas, cabbages, and jackfruits.

The dietary patterns of the respondents were categorized as either low or high risk for dyspepsia. The respondents were considered to be at a high risk of developing dyspepsia if they consumed spicy foods, fried foods, cassavas, cabbages, jackfruits, coffee, and fizzy drinks more than three times a week. The questionnaire items pertaining to smoking behaviors consisted of smoking duration (i.e., two years or more, or less than two years) and cigarette consumption per day (i.e., less than ten cigarettes, or ten cigarettes or more). For the statistical analysis aimed at establishing the relationship between the dependent and independent variables, we used the Chi-squared test, where a value of $p<0.05$ was deemed significant (Pandis, 2016).

RESULTS

Table 1 presents the characteristics of the research sample, which consisted of adult individuals residing in Krajan Hamlet, Sidodadi Village, Lawang District, Malang, Indonesia. The table summarizes the distribution of dyspepsia incidence according to the respondents' age, sex, occupation, and educational attainment in the year 2022. The results of this study showed that 44.3% of the 70 respondents experienced dyspeptic syndrome. The respondents predominantly fell within the 36–45 age group (31.4%). Additionally, the majority of the respondents were female (88.6%), housewives (54.3%), and elementary school graduates (41.4%).

Table 1. Descriptive characteristics of the research sample

Characteristic	Dyspeptic Syndrome			
	Positive		Negative	
	n	%	n	%
Age (y.o)				
18-25	5	7.1	3	4.3
26-35	5	7.1	7	10.0
36-45	9	12.9	13	18.5
46-55	7	10.0	9	12.9
56-65	5	7.1	7	10.0
Total	31	44.3	39	55.7
Sex				
Male	6	8.6	2	11.4
Female	25	35.7	37	88.6
Total	31	44.3	39	100
Occupations				
Housewife	14	20	24	34.3
Self-employed	8	11.4	7	10
Farmer	1	1.4	3	4.3
Crafter	0	0	1	1.4
Unemployed	1	1.4	0	0
Laborer	0	0	3	4.3
Casual daily worker	4	5.8	0	0
Employee	3	4.3	1	1.4
Total	31	44.3	39	55.7
Educational attainment				
No school	4	5.7	4	5.7
Elementary school	11	15.7	18	25.7
Middle school	7	10	11	15.7
High school	8	11.4	14	20
College	1	1.4	0	0
Total	31	44.3	39	55.7

As shown in Table 2, the respondents' dietary patterns were specified as eating frequency, eating interval, and diets with a low or high risk of dyspepsia. This study found that 11.4% of the subjects had an eating frequency that presented a high risk for dyspepsia. Nearly half of the respondents (42.0%) had an eating interval of six hours or more. Furthermore, 87.1% of the respondents followed a diet that exposed them to the potential risk of developing dyspepsia.

Table 2. Distribution of dietary patterns among the respondents

	Dyspeptic Syndrome			
	Positive		Negative	
	n	%	n	%
Diets				
Low risk	12	17.1	26	37.2
High risk	19	27.1	13	18.6
Total	31	44.2	39	55.8
Eating interval				
≥6 hours	20	28.6	22	31.4
4–5 hours	11	15.7	17	24.3
Total	31	44.3	39	55.7
Eating frequency				
≥3 times	29	41.4	33	47.1
<3 times	2	2.8	6	8.6
Total	31	44.2	39	55.7

Table 3. Distribution of smoking behaviors among the respondents

Smooking behaviors	Dyspeptic Syndrome			
	Positive		Negative	
	n	%	n	%
Smoker (frequent and non-frequent)	24	34.3	23	32.9
Non-smoker (including former and passive)	7	10.0	16	22.8
Total	31	44.3	39	55.7

Table 4. Results from the analysis of the relationship between variables

	Dyspeptic Syndrome						P
	Positive		Negative		Total		
	n	%	n	%	n	%	
Eating interval							0.658
≥6 hours	20	28.6	22	31.4	42	55.7	
4–5 hours	11	15.7	17	24.3	28	44.3	
Total	31	44.3	39	55.7	70	100	
Eating frequency							0.287
≥3 times	29	41.4	33	47.1	62	55.7	
<3 times	2	2.8	6	8.6	8	44.3	
Total	31	44.2	39	55.7	70	100	
Diets							0.037
Low risk	12	17.1	26	37.2	38	54.3	
High risk	19	27.1	13	18.6	32	45.7	
Total	31	44.2	39	55.8	70	100	
Smoking behaviors							0.169
Smoker	24	34.3	23	32.9	47	67.1	
Non-smoker	7	10	16	22.8	23	32.9	
Total	31	44.3	39	55.7	70	100	

Table 3 displays the distribution of smoking behaviors among the respondents. Respondents who smoked either frequently or non-frequently were categorized as smokers. Specifically for frequent smokers, they had a smoking duration of more than two years and smoked over ten cigarettes per day. Those who did not engage in smoking behaviors, including both former smokers and individuals exposed to secondhand smoke, were defined as non-smokers. The respondents consisted of both smokers (67.1%) and non-smokers (32.9%). The data from this study showed that there were 24 (34.3%) smokers and 7 (10.0%) non-smokers among the respondents who experienced dyspeptic syndrome.

After mapping the distribution of dietary patterns and smoking behaviors among the respondents, we proceed to analyze the relationship between the dependent and independent variables (Table 4). The analysis revealed that dyspeptic syndrome was not significantly associated with eating frequency ($p = 0.287$), eating interval ($p = 0.658$), or smoking behaviors ($p = 0.169$). On the other hand, there was a statistically significant relationship between dyspeptic syndrome and a high-risk diet ($p = 0.037$).

DISCUSSION

According to the results of this study, diets had an impact on the incidence of dyspeptic syndrome. Individuals who followed a certain diet exhibited a high risk for dyspepsia. This diet consists of spicy foods, coffee, fizzy drinks, fried foods, and specific produce that might trigger indigestion and stomach gas, such as cabbages and jackfruits (Sari et al., 2021). Prior studies, such as the one conducted by Kefi et al. (2022), have provided evidence on the physiological and pathological processes by which certain foods and beverages might lead to dyspepsia. Caffeine contained in coffee induces several bitter taste-sensing type 2 receptors (TAS2Rs or T2Rs) in the mouth and also activates these receptors in the gastric fundus epithelial cells, leading to the secretion of gastric acid. Chemosensory mechanisms explain the involvement of taste receptors in regulating gastric acid secretion. These mechanisms include the excitation of intra-oral taste cells, which exert their effects through cephalic regulation in the gastrointestinal system. Additionally, these mechanisms require the release of gastrin from enteroendocrine cells and the regulation of gastric acid production by parietal cells. Gastrin hormone stimulates gastric acid secretion in the fundus. If there is excessive secretion, it can cause irritation of the stomach wall and other complaints in dyspepsia sufferers. Furthermore, coffee bean processing utilizes additional compounds that also play a role in increasing gastric acid secretion, such as chlorogenic acid (CQA), N-alkanoyl-5-hydroxytrymtamide (C5HT), N-methylpyridinium (NMP), chlorogenic acid lactone (CQL), and hydroxybenzene.

As indicated by the findings of this study, the consumption of spicy foods might contribute to the prevalence of dyspeptic syndrome among adults in Krajan Hamlet, Sidodadi Village, Malang, Indonesia. Prior research carried out by Feinle-Bisset & Azpiroz (2013) provided evidence regarding the ability of capsaicin to cause dyspepsia through visceral hypersensitivity. Capsaicin, as a vanilloid compound, stimulates the vanniloid receptor type 1 (VR1). The consumption of capsaicin can stimulate pain and burning sensations in the gastrointestinal tract through the expression of transient receptor potential vanilloid subtype 1 (TRPV1). Xiang et al. (2022) found that individuals with hypersensitivity exhibited higher levels of TRPV1 expression in comparison to those without the same condition. Dyspepsia symptoms arise through TRPV1 upregulation, which increases functional modification of VR1 and leads to hyperalgesia in the digestive tract. This results in visceral hypersensitivity due to the increased activity of TRPV1 nerve fibers. Spicy foods may cause specific symptoms, such as a feeling of fullness in the stomach and pain in the upper abdomen (Lee et al., 2016). A separate study conducted by Hammer et al. (2008) on patients with functional dyspepsia showed that their symptoms significantly worsened following the administration of capsaicin capsules, indicating a hypersensitivity to

capsaicin. Figure 1 displays the physiological function and structure of TRPV1, a cation channel on the cell membrane located in nociceptive afferent neurons. This receptor works as a transducer not only for capsaicin but also for protons, ethanol, and intracellular lipid mediators.

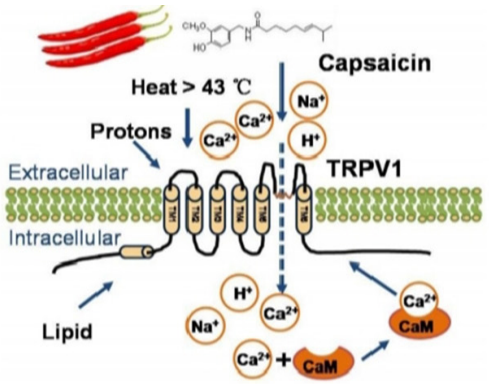

Figure 1. Physiological function and structure of transient receptor potential vanilloid subtype 1 (TRPV1) (Sun et al., 2016)

Some of the research subjects in this study had a diet that was considered to have a high risk of causing dyspepsia. They consumed fizzy drinks, also known as carbonated beverages, as part of their diets. Carbon dioxide (CO2) actually has an important role in the body by maintaining the acid-base balance, controlling respiration, and influencing heart and peripheral circulation. However, the situation may differ when it comes to the presence of CO2 in fizzy drinks. At standard pressure and temperature, approximately 1 mL (equivalent to 2 mg) of CO2 can dissolve in 1 mL of a solution with a neutral pH. When exposed to alkaline solutions, the gas changes into carbonate and bicarbonate. In these particular forms, any ingested CO2 resides in the intestines. In general, the daily intake of CO2 from foods or beverages is approximately equivalent to 1 g of sodium bicarbonate or carbonate. This amount represents only a small fraction of the compounds found in a regular diet. Fizzy drinks have demonstrated that they can interact with TRPV1 throughout the digestive tract. Stimulation caused by the consumption of fizzy drinks results in TRPV1-mediated effects, such as abdominal hyperalgesia. This sensitivity can also arise due to the presence of chemical compounds found in spicy foods, as stated in the previous paragraph (Tahara et al., 2010).

Fizzy drinks quickly pass through the duodenum, where CO2 is converted into bicarbonate. CO2 that has been dissolved is rapidly released in gas form. This was the reason why the dyspeptic subjects frequently complained of feeling bloated as a symptom. CO2 would be emitted through belching and as gas in the stomach, causing an increase in pressure and stimulating the gastric fundus. Distention of the gastric fundus might cause an increase in the relaxation of the transient lower esophageal sphincter. In a previous study conducted by Cuomo et al. (2009), it was demonstrated that CO2 induced rapid gastric distension, resulting in the inhibition of stomach movement for two to five minutes after belching. Typically, consuming 150–200 cc of fizzy drinks temporarily inhibits all peristaltic movements for a duration of two to three minutes. These data suggest that gastric stretching is related to motility. When carbonated liquids are consumed during or after eating, they tend to be digested in the upper part of the stomach. This may produce a rapid feeling of fullness if more than 300 mL is consumed. Carbonated beverages have been reported to decrease gastric emptying time, indicating a possible effect on gastric motility. On another note, incorporating carbonation into a beverage may not have a significant impact on gastric function or gastric discomfort. Pouderoux et al. (1997) found no difference in gastric emptying time or rapid feeling of fullness when comparing the consumption of 300 mL of carbonated beverages and mineral water after eating a 700-calorie meal. However, they observed an increased need to burp in subjects who consumed carbonated beverages. Gastric emptying time is also not affected by the consumption of non-carbonated sweetened beverages. It was found that healthy subjects who consumed 300 mL of these beverages did not experience any effect on gastric emptying time (Cuomo et al., 2009).

Diets that posed a high risk for dyspepsia among the respondents in this study included foods with a high fat content, particularly fried foods. This high-fat diet might aggravate the symptoms of dyspeptic syndrome. Evidence has demonstrated that the absorption of fat in the duodenum causes increased sensitivity to gastric distension and worsening of symptoms in individuals with dyspeptic syndrome. The consumption of fat, which can delay gastric emptying, can cause disturbances in gastric motility and result in a feeling of fullness after eating in dyspeptic patients (Filipović et al., 2011; Khodarahm & Azadbakht, 2016). In addition, a study carried out by Feinle-bisset & Horowitz (2006) supports the findings of this study by demonstrating that fat has the ability to regulate upper digestive tract functions, such as slowing gastric emptying, stimulating pancreatobiliary secretion, and increasing gastric acid secretion. Dietary fat is considered the most potent nutrient for modulating intestinal motility. This is related to the regulation of gastric distension through intestinal hormones and the stimulation and secretion of gastrointestinal hormones such as cholecystokinin (CCK), glucagon-like peptide 1 (GLP-1), and polypeptide Y. These peptides can modulate gastric emptying and induce dyspepsia symptoms (Pilichiewicz et al., 2009).

The results of this study are corroborated by a meta-analysis performed by Fried & Feinle (2002), who revealed that the hormone CCK plays the main role in mediating the effects of lipids on gastrointestinal sensations. The study demonstrated that when gastric distension occurs, fat acts as the main trigger for dyspepsia symptoms such as nausea, bloating, pain, a feeling of fullness, and other related symptoms. The process of lipid regulation of gastrointestinal sensitivity is mediated by the release of CCK. Dexloxiglumide, a CCK-A receptor antagonist, can relieve complaints or symptoms caused by the absorption of fat in the duodenum and gastric distension. Therefore, apart from having an effect on gastrointestinal function, lipids are the main factor that plays a role in the pathophysiology of dyspepsia by releasing CCK (Feinle-bisset & Horowitz, 2006). The release of CCK is associated with the effects of long chain triglycerides (LCT), while medium-chain triglycerides (MCT) do not induce CCK release. The action of LCT is stronger than that of MCT in inducing dyspeptic symptoms such as a feeling of fullness, nausea, and suppression of hunger. Fatty acids produced from the breakdown of triglycerides affect gastrointestinal motility, hormone secretion, appetite suppression, and the initiation of symptomatic complaints (Fried & Feinle, 2002).

Studies conducted by Zhang et al. (2020) and Malagelada et al. (2017) have proven that high fiber consumption can increase bloating and affect digestive tract function. Dietary fiber is partially or completely fermented in the distal part of the small and large intestines. This fermentation results in the production of gases in the intestine, including carbon dioxide, hydrogen, and methane, which can cause bloating (Eswaran et al., 2013). In addition, the fermentation process creates an acidic environment that leads to inflammation in the intestines,

contributing to the occurrence of bloating. Dietary fiber can also interfere with gas transition and increase gas retention by reducing bolus thrust into the rectum (Gonlachanvit et al., 2004). Fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAPs) are short-chain fermentable carbohydrates that are often found in various foods such as onions, cabbages, and peas (Popa et al., 2022). A large number of Indonesian people, including those residing in Krajan Hamlet, follow diets that are rich in FODMAPs. This could perhaps contribute to the prevalence of dyspeptic syndrome among the respondents in this study. A diet high in FODMAPs can lead to malabsorption, resulting in intestinal fermentation that triggers gas formation and increases osmotic pressure. However, studies on the relationship between dietary fiber or vegetable consumption and dyspeptic syndrome have not yet been widely investigated. A recent study by Tabibian et al. (2021) showed that individuals with a high consumption of vegetables and fruits had a low risk of developing dyspeptic syndrome.

CONCLUSION

There was a significant relationship between diets with high risk components and the prevalence of dyspeptic syndrome among adults in Krajan Hamlet, Sidodadi Village, Malang, Indonesia. However, the incidence of dyspeptic syndrome was not significantly associated with eating frequency, eating interval, or smoking behaviors.

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CONFLICT OF INTEREST

All of the authors declare no conflicts of interest.

ETHICS CONSIDERATION

This study received ethical clearance from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia, under protocol No. 51/EC/KEPK/FKUA/2023 dated February 20, 2023.

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The authors did not receive any funding for conducting this study.

AUTHOR CONTRIBUTION

DA and SM contributed to the conception and design, analysis and interpretation of the data, drafting of the article, critical revision of the article for important intellectual content, and final approval of the article. MHN, ZZO, GSD, and NDPL contributed to the analysis and interpretation of the data, critical revision of the article for important intellectual content, and final approval of the article. ANF, ARP, PP, NTA, NAK, and ANW contributed to the provision of study materials or patients, statistical expertise, and the obtainment of funding. Additionally, SM contributed to the administrative, technical, or logistic support as well as the collection and assembly of the data.

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