

Correlation Between Modifiable Risk Factors and GERD Symptoms Among Adolescents in Urban Jakarta

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ABSTRACT: Gastroesophageal reflux disease (GERD) is a common gastrointestinal disease affecting the global population, though its prevalence and associated risk factors in Southeast Asian adolescents remain under-researched. This study aimed to examine the prevalence of GERD symptoms among adolescents aged 16–18 years old in urban Jakarta and to investigate their associations with a selected list of modifiable risk factors, both lifestyle and dietary. A cross-sectional study was conducted using a self-reported questionnaire distributed via social media platforms and school communication channels with validated GERD symptom assessment questions, including those that addressed the participants' lifestyle and dietary patterns. The questions were a simplified version of the widely used GERDQ questionnaire and were later validated by the research mentors. Associations between GERD symptoms and corresponding risk factors were analyzed using chi-square tests for 114 participants with completed questionnaires. As the number of tests run was limited, correction for multiple testing was not applied. GERD symptoms were observed among a proportion of participants that aligned with existing literature. Out of the risk factors tested, BMI and caffeine intake showed statistically significant associations with GERD symptoms reported. These findings indicate that GERD symptoms in adolescents may be influenced by a narrower range of modifiable risk factors than those more commonly reported in adults.

KEYWORDS: Biomedical and Health Science, Pathophysiology, Gastroesophageal Reflux Disease (GERD), Lifestyle Risk Factors.

■ Introduction

Gastroesophageal Reflux Disease (GERD) is a gastrointestinal disorder with a global prevalence of 13.98%.¹ Despite its relatively high prevalence, there are several gaps in the current understanding of the condition in adolescents. Most studies focus on adult populations, despite evidence of GERD affecting growth, quality of life, and long-term esophageal health in adolescents. To address these gaps, this study aims to investigate the prevalence of GERD symptoms in a sample size of Indonesian teens (ages 16–18), a group with comparable lifestyles, and analyze their correlation with known risk factors.

Background and Context:

Gastroesophageal Reflux Disease (GERD):

Gastroesophageal Reflux Disease (GERD) is a disorder of the digestive tract in which acidic gastric contents reflux back into or beyond the esophagus, resulting in esophagitis and inflammation of mucosal surfaces.² Reflux is a normal process that occurs in healthy individuals and does not cause harmful symptoms or complications. However, individuals with Gastroesophageal Reflux Disease (GERD) experience symptom persistence and complications that arise from the malfunctioning of the lower esophageal sphincter (LES). The LES is a high-pressure zone made of esophageal muscle fibers, diaphragmatic crura, and the phrenoesophageal ligament. It is located at the junction between the esophagus and stomach, and functions to prevent gastric contents, including acid, from flowing back into the esophagus. This prevents symptoms of

acid reflux, the most common ones being: heartburn, regurgitation, dysphagia, nausea, and chronic cough.

Transient Lower Esophageal Sphincter Relaxation (TLESR):

Under normal conditions, the LES stays tonically contracted to prevent the stomach contents from flowing back into the esophagus, briefly relaxing during swallowing to allow food to pass. Transient Lower Esophageal Sphincter Relaxation (TLESR) is a mechanism that occurs in all individuals, where LES relaxation is induced spontaneously without swallowing, and can last for 10–45 seconds or longer. While it is a normal physiological mechanism for venting stomach gas from gastric distention, it can often cause the reflux of gastric and duodenal fluids.³

As TLESR is reported to be the main physiological cause for GERD, it was initially hypothesized that patients with GERD would have a higher rate of TLESR. However, research done by Kim *et al.* showed that the duration of TLESR and the length of esophageal shortening did not differ between subjects with GERD and controls.⁴ This concluded that while the rate of TLESR is similar, patients with GERD lacked certain motor responses that normally emptied esophageal refluxate back into the stomach, increasing the frequency and amount of acid reflux.

Modifiable Risk Factors:

GERD is considered a multifactorial disorder; it is caused by multiple genetic and environmental factors. In the case of GERD, the significant risk factors associated are:⁵

1. Diet
2. Sleeping patterns
3. Stress
4. BMI

1. Diet:

Dietary habits represent a major modifiable risk factor for GERD, as common foods can directly trigger GERD mechanisms. Following existing studies, it is important to note that the evidence for dietary triggers in GERD is mixed and highly individualized, thus highlighting the need for personalized trials and testing for dietary needs. There are 4 primary classes of foods seen to be the biggest influences.

1.1 High-fat foods

- Fatty products decrease LES tension, making it less effective at preventing stomach acid from flowing back up into the esophagus.⁶ They also delay stomach evacuation irrespective of the volume of the meal, prolonging the time food and acid stay in the stomach, which promotes further reflux.⁷

1.2 Acidic/spicy foods

- Acidic and spicy foods cause direct irritation of the lower esophageal mucosa, which may mimic and thus worsen heartburn.⁸ Red pepper, in particular, contains the neurotoxin capsaicin that has been shown to delay gastric emptying and stimulate mechanoreceptors in the esophagus.⁹

1.3 Caffeine, chocolate, mint

- A study showed that the correlation between ingesting chocolate and milk and symptoms of GERD shows that the relationship is individualized.⁸ While chocolate, which contains caffeine, theoretically worsens GERD by causing LES relaxation, a study found that eating chocolate did not correlate with symptom frequency.¹⁰ Mint is also known to relax the LES, but in a study, only a small subset of patients reported exacerbated symptoms.⁷ Hence, while these foods have a theoretical mechanism associated with GERD, they don't consistently cause symptoms universally.

1.4 Eating patterns

- The frequency and time of ingestion also affect GERD mechanisms, as a majority of reflux episodes from LES relaxation occur within the first 3 hours after eating. Hence, consuming meals right before or close to bedtime can result in worse regurgitation, especially if the meals are relatively larger.¹¹ This is because the body is not given time for gastric emptying, and as the sleeping position is already prone to increased reflux, there is a higher chance of the contents flowing back into the esophagus.

2. Sleeping patterns:

Sleep is also another modifiable risk factor, where GERD symptoms are associated with poor sleep quality, short duration, and certain sleep positions. Sleep and GERD have a bidirectional relationship, where sleep deprivation results in worsening of GERD symptoms, but GERD symptoms result

in reduced sleep quality, due to difficulty initiating and maintaining sleep.^{8,12} A study showed that the time lag between acid exposure and the feeling of heartburn decreased in those with sleep deprivation compared to those without, indicating the body's heightened response to GERD symptoms with lack of sleep.¹³ Several mechanisms explain this interaction. Lying flat prevents gravity from keeping refluxed contents in the stomach. During sleep, reflexes that restrict the flow of gastric contents back into the esophagus and larynx are also reduced.¹¹

Sleeping positions can also affect GER. The left lateral decubitus (LLD) position – which entails sleeping on one's left side – and sleeping with the head elevated have solid evidence for improving and managing GERD, reducing gastric acid exposure. On the contrary, lying on the right side positions the esophagus below the stomach and gastroesophageal junction, accelerating reflux and exposing longer acid exposure to the esophagus.¹⁴

3. Stress:

Stress also represents an understated but significant risk factor of GERD, mediated through the gut-brain axis. Stress can be any physical or psychological stimulus that disrupts homeostasis, resulting in a stress response.¹⁵ While stress and GERD were assumed to be independent and the product of two unrelated systems – the digestive and nervous system – there has been an increasing amount of data that suggests an association. Although a lot of them only measure the effect of acute stress, short-term stress responses, a few have modeled chronic stress, which is a more accurate depiction of patients' life experience. That being said, a study found that there was a higher prevalence of GERD in a group with high stress compared to the one without, although limitations mentioned were the inability to consider other risk factors.¹⁶

Stress can influence GERD by central sensitization or by exacerbating symptoms. Central sensitization is the increased responsiveness to pain signals in the central nervous system.¹⁷ GERD causes this, enhancing perceptual response to intra-esophageal acid exposure, and thus, greater emotional responses were seen to the stressor.¹⁸ A study found that anxiety induction increased perception of esophageal pain without increasing its exposure to acid.¹⁹ It is important to note, however, that individual variation is extremely common in this phenomenon. In most studies, many patients had varying responses and sensitivity to stress.

4. BMI:

Body Mass Index is a metric used to define height/weight characteristics to be classified into groups: severely underweight, underweight, normal weight, overweight, moderately obese, severely obese, and morbidly obese. It is calculated as weight divided by height squared.²⁰ Numerous studies have indicated a positive correlation between elevated body mass index (BMI) and GERD symptoms. For instance, Malaty found that children diagnosed with GERD were much more likely to be obese, and another study by Xie found a higher prevalence of GERD among obese individuals aged 18-81.^{21,22}

The risk of symptoms rises with BMI among both categories of normal weight and overweight subjects.

Physiological mechanism:

Several physiological mechanisms may explain the observed relationship between elevated BMI and GERD. Obesity has been associated with multiple esophageal abnormalities, such as:²³

1. Nutcracker esophagus: Abnormally high peristaltic amplitude
2. Hypotensive LES: Low LES basal pressure

These abnormalities can increase acid exposure in the esophagus, exacerbating GERD symptoms. Furthermore, abdominal obesity increases intra-abdominal pressure, which can be due to the direct effect of the mass from the adipose tissue to the abdominal cavity, or the mass of the intra-abdominal adipose tissue maintaining the increased intra-abdominal pressure. Obesity also increases the volume of gastric contents while delaying gastric emptying.²⁴

Due to the limited amount of existing research that focuses primarily on adolescents in Southeast Asia, this study was designed as an exploratory analysis to investigate potential associations rather than to test a specific hypothesis.

5. Objective:

Adolescents in Jakarta remain under-researched in GERD-related studies despite rising lifestyle-related risk factors such as sleep irregularities and stress. Improving understanding of these correlations is important for early prevention of GERD, as it may lead to long-term complications and an overall poorer quality of life if untreated. This study aims to contribute to the Indonesian adolescent-focused literature on GERD.

■ **Methods**

This study employed a cross-sectional survey design to investigate the prevalence of gastroesophageal reflux disease (GERD) symptoms and their correlation with modifiable risk factors (diet, sleep, stress, BMI) and non-modifiable risk factors (family history) in teenagers.

Sample Size:

The following formula will be used to calculate the adequate sample size:²⁵

$$n = [Z^2 p (1 - p)] / d^2$$

where

n = sample size

Z = statistic corresponding to the level of confidence

p = expected prevalence

d = precision

Prior literature noted that in Indonesia, GERD prevalence in adolescents $\approx 10.9\%$, thus $p = 0.109$.²⁶ A 95% confidence level ($Z = 1.96$) was selected, which is the value conventionally aimed for in biomedical research.²⁷ It is suggested to use a value of $d = 5\%$ if the prevalence is between 10% and 90%.²⁷

Hence, inputting the values $Z = 1.96$, $p = 0.109$, $d = 0.05$ into the formula gives:

$$n = [(1.96^2) (0.085) (1 - 0.109)] / (0.05)^2 \approx 150$$

Thus, the recommended sample size to estimate the prevalence of GERD with adequate accuracy is 150 participants.

Participants:

Participants were eligible for inclusion in the study if they were aged 16-18 years at the time of survey completion, provided informed consent through a question at the start of the questionnaire, and were able to understand and complete the survey in English. Responses were excluded if the questionnaires were incomplete.

Data Collection:

Data was collected using an anonymous, self-reported Google Forms questionnaire. The survey link was distributed through school communication platforms and social media platforms. No identifying information (name and contact details) was collected. The use of an online questionnaire enabled efficient data collection while maintaining anonymity to minimize response bias. The questionnaire was a simplified version of the GERDQ questionnaire, validated by the research mentors to ensure it encompassed as many variables needed to detect GERD symptoms.

Variables and Measurement:

The primary dependent variable was the status of GERD, measured through self-reported diagnosis and questions about symptom frequency adapted from common GERD diagnosis methods (heartburn, regurgitation, sleep disturbance).

Independent variables:

1. Dietary factors: caffeine intake, spicy food intake, high-fat meals, and carbonated drinks. Measured using frequency categories (never, 1-2x/week, 3-4x/week, 5-6x/week, daily), which were later grouped into 2 categories (0-2x/week and 3-7x/week)
2. Sleep variables
3. Stress level
4. Body mass index (BMI), calculated from self-reported height and weight, is categorized.²⁸

All variables were categorized into 2x2 data to allow Chi-square analysis.

Procedure:

The questionnaire was developed based on previous literature and then adapted for an adolescent-focused study. Survey links and consent statements were distributed. Responses were automatically recorded through Google Forms, and incomplete responses were removed before analysis. The dataset was processed and then exported to IBM SPSS for statistical analysis.

Data Analysis:

Data was analyzed using IBM SPSS Statistics. Descriptive statistics were used to summarize sample characteristics and the prevalence of symptoms. Chi-square tests were then carried out to check for correlations between GERD status and the risk factors, with statistical significance set at $p < 0.05$. As

the number of tests run was limited, correction for multiple testing was not applied.

Ethical Considerations:

Participation was voluntary and anonymous. Informed consent was obtained at the beginning of the survey through a mandatory consent checkbox. Participants were given the option to exit the survey at any point without penalty. The study posed minimal risk and adhered to ethical guidelines for research on adolescents.

Results and Discussion

Table 1: Prevalence of GERD. As seen in Table 1, there were 114 respondents who were surveyed in this study. Among 114 surveyed, 14 respondents (12.3%) were medically diagnosed with GERD, 10 (8.8%) were self-diagnosed with GERD, and 78.9% reported not having GERD. The overall prevalence of GERD in this study was 21.1%.

GERD Status	n	%
Medically diagnosed	14	12.3
Self-diagnosed	10	8.8
No GERD	90	78.9
Total	114	100

Table 2: Association between demographic characteristics and GERD status. Of the 24 respondents with GERD, 15 (62.5%) were female (Table 2). Participants were all aged 16-18 years. The majority were 18 years old (51.8%), followed by 17 (29.8%), and 16 (18.4%).

Characteristics	GERD (Diagnosed)	GERD	No GERD	Total
Sex, n (%)				
Male	5 (12.5)	4 (10)	31 (77.5)	40
Female	9 (12.2)	6 (8.1)	59 (79.7)	74
Age, n (%)				
16 years	2 (9.5)	1 (4.8)	18 (85.7)	21
17 years	5 (14.7)	4 (11.8)	25 (73.5)	34
18 years	7 (11.9)	5 (8.4)	47 (79.7)	59

Table 3: Association between dietary habits and GERD status. A statistically significant association was observed between caffeine intake and GERD status. As shown in Table 3, Caffeine intake was significantly associated with GERD status ($p = 0.025$). Participants consuming 3-7 cups of caffeinated beverages per week showed a higher prevalence of GERD (31.8%) compared to those consuming 0-2 cups per week (14.3%). No significant associations were observed for carbonated drinks, fried/high-fat meals, spicy food, or eating habits (all $p > 0.05$).

Dietary Habits	Category	GERD	No GERD	p value
Coffee/caffeinated beverages	0-2 cups/week	10 (14.3)	60 (85.7)	0.025
	3-7 cups/week	14 (31.8)	30 (68.2)	
Carbonated drinks	0-2 cups/week	18 (20)	72 (80)	0.593
	3-7 cups/week	6 (25)	18 (75)	
Fried/high-fat meals	0-2 meals/week	14 (24.6)	43 (75.4)	0.358
	3-7 meals/week	10 (17.5)	47 (82.5)	
Spicy food	0-2 meals/week	8 (14.8)	46 (85.2)	0.121
	3-7 meals/week	16 (26.7)	44 (73.3)	
Eating habits	Regular	6 (20)	24 (80)	0.869
	Irregular	18 (21.4)	66 (78.6)	

Table 4: Association between risk factors and GERD status. As seen in Table 4, no significant association was found between sleep duration and GERD status ($p = 0.593$), and between frequency of stress and GERD ($p = 0.646$). Overweight and obese participants had a significantly higher prevalence of GERD than those who were normal/underweight ($p = 0.0004$).

Risk Factor	Category	GERD	Not GERD	p value
Sleeping duration (Avg. per night)	Insufficient (<8h)	18 (20)	72 (80)	0.593
	Sufficient (≥ 8 h)	6 (25)	18 (75)	
Frequency of stress	Low	11 (19.3)	46 (80.7)	0.646
	High	13 (22.8)	44 (77.2)	
BMI	Normal/Underweight	13 (14.3)	78 (85.7)	0.0004
	Overweight/Obese	11 (47.8)	12 (52.2)	

Table 5: Cause of stress among participants. As seen in Table 5, of the 4 causes of stress tested, the most common among participants was school (79.8%).

Cause of stress	n
School	91
Family	13
Love life	7
Friends	3

Discussion:

Out of the risk factors tested, BMI and caffeine intake showed statistically significant associations with GERD symptoms reported.

The higher prevalence of GERD among females is in line with a study by Hartoyo *et al.*, which found that 24.8% of respondents had GERD, and 65.4% of them were female (Table 2).²⁹ It is also consistent with existing literature that suggests hormonal perception may increase perception of symptoms. Females also exhibit greater visceral sensitivity and stronger esophageal barrier proteins, which may explain more reporting of symptoms.³⁰

Of the 8 variables tested, two showed a significant association with GERD. The strongest association observed, as seen in Table 4, was between the participant's BMI category and GERD status ($p = 0.0004$). This result aligns with a study done by Malaty, which found a significant association between obesity and GERD in children.²¹ The study also recognized that obese children are more likely to have a higher consumption of 'junk' food, inducing heartburn, a symptom that mirrors that of GERD. It is also in accordance with existing literature that indicates a higher prevalence of hiatal hernias in those with excess body weight, which results in the reduction of LES pressure. Additionally, patients with a higher BMI have increased intragastric volumes, which are correlated to the vertical separation of the LES and crural diaphragm, increasing the likelihood of reflux.³¹ As the prevalence of overweight adolescents in DKI Jakarta is rising,³² BMI may represent a relevant and modifiable risk factor in this population.

As seen in Table 3, Caffeine intake was also a factor significantly associated with GERD status ($p = 0.025$). Participants consuming 3-7 cups per week (31.8%) had more than twice the rate of GERD as those consuming 0-2 cups per week (14.3%). This is in accordance with a study done by Hartoyo

et al., which found a significant association between coffee consumption and incidence of GERD in Jakarta citizens ($p = 0.006$), although the scope of the study did not include all caffeinated drinks, and participants were aged between 18 and 65 years old. Another study by Carlos *et al.* found that the association between coffee consumption and GERD symptoms appeared primarily in overweight and obese respondents, suggesting the symptoms may be caused by a possible interaction between caffeine-induced LES relaxation and increased intra-abdominal pressure from a higher BMI.³³ Although caffeine is known to reduce LES pressure and increase secretion of gastric acid, existing evidence remains debated, with studies reporting inconsistent symptom patterns. This suggests that caffeine sensitivity may vary according to the individual and other factors.

In contrast, most of the other dietary habits examined were not significantly associated with GERD (all $p > 0.05$) (Table 3). Spicy consumption had a value of $p = 0.121$. Although a study done by Cahyani found a significant association between spicy food consumption and GERD ($p = 0.045$), the study included 35 boarding school students who likely had similar dietary habits. Since spicy food is common in Indonesia, with Sambal, containing capsaicin, often used in dishes, spicy food consumption may be common across both GERD and non-GERD groups, making its effect less statistically distinguishable.³⁴ More studies will need to be carried out before a solid conclusion can be confirmed. Other variables - carbonated drinks ($p = 0.593$), high-fat meals ($p = 0.358$), and eating habits ($p = 0.869$) were also not significantly associated.

These results may be due to relatively similar habits among adolescents in high school, who thus have similar sleep schedules and stress mostly attributed to school, as 91 (79.8%) respondents reported.

Overall, the results from this study indicate that BMI has the greatest association with GERD in adolescents than other factors, as it aligns with many existing studies. In Jakarta, where the prevalence of overweight adolescents continues to rise, these results may reflect lifestyle patterns of this population. While caffeine also had a significant association, the literature online suggests that it is still debated and inconclusive. A meta-analysis of fifteen case-control studies found no significant association, but these discrepancies could have been explained by the difference in research design and, most importantly, the difference in study participants.³⁷ This study contributes to the currently limited amount of research on GERD in Southeast Asian adolescents, but suggests that the effect of risk factors in this age group may differ from those in adults.

This study identified the prevalence of GERD among teenagers aged 16-18, which remained consistent with other studies, and examined its association with several modifiable risk factors. Although only two of them were found to be statistically significant, this suggests that adolescent GERD may be correlated to a narrower set of factors than previously assumed. The lack of significant findings in the other variables may reflect similarities in the lifestyles of adolescents living in Urban Jakarta.

■ Conclusion

Implications:

The findings of this study suggest a correlation between a high BMI and caffeine intake with the occurrence of GERD symptoms. With the increase in prevalence of overweight adolescents in Jakarta,³² an increased awareness of maintaining healthy lifestyle habits may help prevent or reduce the severity of GERD symptoms. This study also highlights the need for further research focused on GERD in Southeast Asian adolescents.

Limitations:

This study also has several limitations to acknowledge. Firstly, the use of an anonymous self-reported questionnaire may introduce recall bias or underreporting, particularly with lifestyle habits. While self-diagnosed respondents were identified when finding the prevalence, they were grouped with those diagnosed with GERD when conducting correlation analysis to increase statistical power. This may have introduced misclassification bias, as self-diagnosis is less reliable than clinical diagnosis. There are certain symptoms, such as those of acid reflux, that may have been mistaken for GERD and reported as such by certain participants.³⁶ The study also did not differentiate between types of caffeinated beverages or quantify specific caffeine measurements, as the participants were instructed to measure their caffeine intake in terms of the number of cups. This may influence results, as studies have found that differences in GERD status depend on the beverage composition.³³ It should be considered, however, that as this study employs a cross-sectional design, the associations observed cannot be established as a causal relationship. It is possible that the risk factors may have changed as a consequence of GERD symptoms instead of solely acting as predictors.

Concluding Remarks:

In conclusion, the results of this study indicate that adolescent GERD in urban Jakarta is correlated to a more limited set of dietary habits in this sample than those commonly emphasized in studies with adults. While the sample size was sufficient for estimating GERD prevalence, it may have been underpowered to detect weaker associations between certain risk factors and GERD symptoms. As lifestyle patterns continually shift in this population, early awareness of a healthier lifestyle - such as weight management and judicious consumption of caffeine and junk food - may play a significant role in preventing long-term gastrointestinal issues.

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■ References

- Wickramasinghe, N.; Devanarayana, N. M. Insight into Global Burden of Gastroesophageal Reflux Disease: Understanding Its Reach and Impact. *World Journal of Gastrointestinal Pharmacology and Therapeutics* **2025**, *16* (1). <https://doi.org/10.4292/wjgpt.v16.i1.97918>.
- Azer, S. A.; Reddivari, A. K. R. *Gastroesophageal reflux disease (GERD)*. StatPearls. <https://www.ncbi.nlm.nih.gov/books/NBK554462/>.
- Zheng, Z.; Shang, Y.; Wang, N.; Liu, X.; Xin, C.; Yan, X.; Zhai, Y.; Yin, J.; Zhang, J.; Zhang, Z. Current Advancement on the Dynamic Mechanism of Gastroesophageal Reflux Disease. *International Journal of Biological Sciences* **2021**, *17* (15), 4154–4164. <https://doi.org/10.7150/ijbs.65066>.
- Hoon Il Kim, S. J. H.; Lee, J. S. Specific Movement of Esophagus during Transient Lower Esophageal Sphincter Relaxation in Gastroesophageal Reflux Disease. *J Neurogastroenterol Motil.* **2013**, *19* (3), 332–337. <https://doi.org/10.5056/jnm.2013.19.3.332>.
- Sadafi, S.; Azizi, A.; Pasdar, Y.; Shakiba, E.; Darbandi, M. Risk Factors for Gastroesophageal Reflux Disease: A Population-Based Study. *BMC Gastroenterology* **2024**, *24* (1). <https://doi.org/10.1186/s12876-024-03143-9>.
- Jarosz, M.; Taraszewska, A. Risk Factors for Gastroesophageal Reflux Disease – the Role of Diet. *Gastroenterology Review* **2014**, *9* (5), 297–301. <https://doi.org/10.5114/pg.2014.46166>.
- Benamouzig, R.; Airinei, G. Diet and Reflux. *Journal of Clinical Gastroenterology* **2007**, *41* (Supplement 2), S64–S71. <https://doi.org/10.1097/mcg.0b013e318032bed3>.
- Mody, R.; Bolge, S. C.; Kannan, H.; Fass, R. Effects of Gastroesophageal Reflux Disease on Sleep and Outcomes. *Clinical Gastroenterology and Hepatology: The Official Clinical Practice Journal of the American Gastroenterological Association* **2009**, *7* (9), 953–959. <https://doi.org/10.1016/j.cgh.2009.04.005>.
- Choe, J. W.; Joo, M. K.; Kim, H. J.; Lee, B. J.; Kim, J. H.; Yeon, J. E.; Park, J.-J.; Kim, J. S.; Byun, K. S.; Bak, Y.-T. Foods Inducing Typical Gastroesophageal Reflux Disease Symptoms in Korea. *Journal of Neurogastroenterology and Motility* **2017**, *23* (3), 363–369. <https://doi.org/10.5056/jnm16122>.
- Kaltenbach, T.; Crockett, S.; Gerson, L. B. Are Lifestyle Measures Effective in Patients with Gastroesophageal Reflux Disease? *Archives of Internal Medicine* **2006**, *166* (9), 965. <https://doi.org/10.1001/archinte.166.9.965>.
- Dent, J.; Dodds, W. J.; Friedman, R. H.; Sekiguchi, T.; Hogan, W. J.; Arndorfer, R. C.; Petrie, D. J. Mechanism of Gastroesophageal Reflux in Recumbent Asymptomatic Human Subjects. *Journal of Clinical Investigation* **1980**, *65* (2), 256–267. <https://doi.org/10.1172/jci109667>.
- Gurges, P.; Murray, B. J.; Boulos, M. I. Relationship between Gastroesophageal Reflux Disease and Objective Sleep Quality. *Journal of Clinical Sleep Medicine* **2022**. <https://doi.org/10.5664/jcsm.10198>.
- Johnson, D. A. Does Sleep Deprivation Worsen GERD Symptoms? *Journal Watch* **2008**, 2008. <https://doi.org/10.1056/jg200802080000005>.
- Simadibrata, D. M.; Lesmana, E.; Amangku, B. R.; Wardoyo, M. P.; Simadibrata, M. Left Lateral Decubitus Sleeping Position Is Associated with Improved Gastroesophageal Reflux Disease Symptoms: A Systematic Review and Meta-Analysis. *World Journal of Clinical Cases* **2023**, *11* (30), 7329–7336. <https://doi.org/10.12998/wjcc.v11.i30.7329>.
- Chu, B.; Marwaha, K.; Ayers, D.; Sanvictores, T. *Physiology, Stress Reaction*. StatPearls. <https://www.ncbi.nlm.nih.gov/books/NBK541120/>.
- Turab, M. S.; Awan, M. A.; Masroor, L.; Amir, K.; Khan, M.; Li-aquat, A. Stress and Gastroesophageal Reflux Disease: Are They Related? *Cureus* **2025**. <https://doi.org/10.7759/cureus.79037>.
- Dydyk, A. M.; Givler, A. *Central Pain Syndrome*. StatPearls. <https://www.ncbi.nlm.nih.gov/books/NBK553027/>.
- Sandhu, D.; Fass, R. (PDF) STRESS and GASTROESOPHAGEAL REFLUX DISEASE. *ResearchGate* **2018**. <https://doi.org/10.25040/ntsh2018.02.010>.
- Sharma, A.; Lukas Van Oudenhoove; Paine, P.; Gregory, L.; Aziz, Q. Anxiety Increases Acid-Induced Esophageal Hyperalgesia. *Psychosomatic Medicine* **2010**, *72* (8), 802–809. <https://doi.org/10.1097/psy.0b013e3181f5c021>.
- Zierle-Ghosh, A.; Jan, A. *Physiology, Body Mass Index*. National Library of Medicine. <https://www.ncbi.nlm.nih.gov/books/NBK535456/>.
- Malaty, H. Obesity and Gastroesophageal Reflux Disease and Gastroesophageal Reflux Symptoms in Children. *Clinical and Experimental Gastroenterology* **2009**, *31*. <https://doi.org/10.2147/ceg.s4715>.
- Xie, M.; Deng, L.; Fass, R.; Song, G. Obesity Is Associated with Higher Prevalence of Gastroesophageal Reflux Disease and Reflux Related Complications: A Global Healthcare Database Study. *Neurogastroenterology and Motility* **2024**. <https://doi.org/10.1111/nmo.14750>.
- Chang, P.; Friedenberg, F. Obesity and GERD. *Gastroenterology Clinics of North America* **2014**, *43* (1), 161–173. <https://doi.org/10.1016/j.gtc.2013.11.009>.
- Lambert, D. M.; Marceau, S.; Forse, R. A. Intra-Abdominal Pressure in the Morbidly Obese. *Obesity Surgery* **2005**, *15* (9), 1225–1232. <https://doi.org/10.1381/096089205774512546>.
- Daniel, W. W.; Cross, C. L. *Biostatistics: A Foundation for Analysis in the Health Sciences*; Wiley: Hoboken, NJ, 2019.
- Artanti, D.; Hegar, B.; Kaswandani, N.; Soedjatmiko; Prayitno, A.; Devaera, Y.; Vandenplas, Y. The Gastroesophageal Reflux Disease Questionnaire in Adolescents: What Is the Best Cutoff Score? *Pediatric Gastroenterology, Hepatology & Nutrition* **2019**, *22* (4), 341. <https://doi.org/10.5223/pghn.2019.22.4.341>.
- Pourhoseingholi, M. A.; Vahedi, M.; Rahimzadeh, M. Sample Size Calculation in Medical Studies. *Gastroenterology and Hepatology From Bed to Bench* **2024**, *6* (1), 14.
- Dwyer, J. T.; Melanson, K. J.; Sriprachy-anunt, U.; Cross, P.; Wilson, M. Table 4, *Classification of Weight Status by Body Mass Index (BMI)*. www.ncbi.nlm.nih.gov/books/NBK278991/table/diet-treatment-obes.table4clas/.
- Hartoyo, F. Z.; Tandarto, K.; Sidharta, V.; Tenggara, R. The Correlation between Coffee Consumption and Gastroesophageal Reflux Disease. *The Indonesian Journal of Gastroenterology, Hepatology, and Digestive Endoscopy* **2022**, *23* (1), 11–16. <https://doi.org/10.24871/231202211-16>.
- Kim, Y. S.; Kim, N.; Kim, G. H. Sex and Gender Differences in Gastroesophageal Reflux Disease. *Journal of Neurogastroenterology and Motility* **2016**, *22* (4), 575–588. <https://doi.org/10.5056/jnm16138>.
- Thalheimer, A.; Bueter, M. Excess Body Weight and Gastroesophageal Reflux Disease. *Visceral Medicine* **2021**, *37* (4), 1–6. <https://doi.org/10.1159/000516050>.
- Sekar Woro Wilis; Ibnu Malkhan Bakhrol Ilmi; Yessi Crosita Octaria. Factors Related to Obesity in Adolescent Girls in East Jakarta. *Media Gizi Kesmas* **2025**, *14* (1), 26–34. <https://doi.org/10.20473/mgk.v14i1.2025.26-34>.
- Carlos, J.; Alexander, S.; Calderon, B.; Jacome, C.; Rain, J. Coffee Consumption and GERD Symptoms in Overweight or Obese Patients at the Gastroenterology Outpatient Clinics of Two In-

- stitutions in Quito (Hospital de Clínicas Pichincha and Clínica Pasteur), between January and March 2025. *South Florida Journal of Health* **2025**, 6 (3), e5831–e5831. <https://doi.org/10.46981/sfjh-v6n3-013>.
34. Cahyani, I. The Relationship between Diet and the Incidence of Gastroesophageal Reflux Disease in Boarding School Students. *HealthSmart: Jurnal Kesehatan Masyarakat* **2025**, 1 (1), 1–13. <https://doi.org/10.64146/wn1xn988>.
35. Z. Zheng, Y. Shang, N. Wang, X. Liu, C. Xin, X. Yan, Y. Zhai, J. Yin, J. Zhang, Z. Zhang, Current Advancement on the Dynamic Mechanism of Gastroesophageal Reflux Disease. *International Journal of Biological Sciences*. 17, 4154–4164 (2021).
36. Acid reflux, Heartburn, and GERD: What's the difference? NIH MedlinePlus Magazine (2020), (available at <https://magazine.medlineplus.gov/article/acid-reflux-heartburn-and-gerd-whats-the-difference>).
37. J. Kim, S.-W. Oh, S.-K. Myung, H. Kwon, C. Lee, J. M. Yun, H. K. Lee, Association between coffee intake and gastroesophageal reflux disease: a meta-analysis. *Diseases of the Esophagus*. 27, 311–317 (2013).

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