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ORIGINAL ARTICLE

Dietary Pattern and Psychological Stress Are Not Associated with Gastro-oesophageal Reflux Symptoms in Female Preclinical Medical Students: A Precision-Focused Cross-Sectional Study

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ABSTRACT

Background: Gastro-oesophageal reflux disease is among the most prevalent chronic gastrointestinal disorders worldwide and its absolute burden continues to rise, yet studies of behavioural determinants in young adults report statistical significance without magnitude and almost never state the effect size their design could detect.

Objective: To estimate the association of dietary pattern and psychological stress with symptom-defined gastro-oesophageal reflux, and the precision with which those associations could be detected, in a restriction-based sample of female preclinical medical students.

Methods: This STROBE-compliant cross-sectional study used a restriction-based design to isolate behavioural exposures from adiposity and the other dominant reflux risk factors. Of 120 randomly selected female preclinical medical students at the Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, 104 responded (86.7%) and 87 met eligibility criteria fixing female sex, body mass index 18.5-24.9 kg/m^2, never-smoking, no alcohol intake and no gastro-active medication. Dietary pattern, stress and reflux symptoms were measured with validated Indonesian instruments and analysed with exact tests, Firth penalised logistic regression, Bayesian estimation, quantitative bias analysis and formal precision analysis.

Results: GERD-Q positivity was 12.6% (95% CI 7.2-21.2); 8.0% had a poor dietary pattern and 43.7% reported stress. Adjusted odds ratios were 3.35 (95% CI 0.55-16.12; p = 0.208) for poor dietary pattern and 1.10 (95% CI 0.31-3.78; p = 0.339) for stress; Bayes factors favouring the null ranged from 0.99 to 2.05 and from 1.78 to 4.90 across four priors. Minimum detectable odds ratios were 12.23 and 4.32. Correcting for outcome misclassification raised the dietary odds ratio to 6.61.

Conclusion: Neither exposure was associated with reflux symptoms; the study excludes only large effects, and about 459 participants would be needed to detect the observed dietary effect.

1. Introduction

Gastro-oesophageal reflux disease (GERD) is a chronic disorder of the upper gastrointestinal tract in which retrograde movement of gastric contents into the oesophagus produces troublesome symptoms, most characteristically heartburn and acid regurgitation. Successive analyses of the Global Burden of Disease 2021 dataset agree that GERD is among the most prevalent chronic gastrointestinal disorders worldwide, that its absolute burden has risen steadily since 1990, and that it is projected to continue rising to at least 2040, with the largest absolute case counts concentrated in populous middle-income countries of South and Southeast Asia, including Indonesia.[1-3] Beyond its gastrointestinal manifestations, reflux imposes a documented burden through impaired physical functioning, disturbed sleep and reduced quality of life,[4] and among Indonesian medical students it has been shown to impair learning concentration directly.[5] Identifying genuinely modifiable determinants is therefore a public-health priority as much as a clinical one.

This study was designed to answer a narrower and, we would argue, more tractable question than the one the existing literature poses. That literature asks whether dietary pattern and psychological stress are associated with reflux symptoms in unrestricted student samples. In such samples any observed association is confounded by body mass index, which affects both eating behaviour and reflux, and by sex, smoking and alcohol intake, which affect reflux directly. The present study instead restricts eligibility on all five of these variables, so that the question becomes: does dietary pattern or psychological stress have any measurable effect on reflux symptoms that is independent of adiposity and of the other dominant risk factors? That question cannot be answered by statistical adjustment in a small sample, and it has not previously been asked

in this population. The study's second contribution is to report, alongside every null result, the magnitude of effect the design was actually capable of detecting — a quantity absent from every comparable Indonesian study we identified.

The rationale for such a restriction rests on the observed hierarchy of reflux determinants. In a large population-based risk-factor study, obesity, smoking, alcohol intake and specific drug classes emerged as the dominant modifiable contributors.[6] A multivariable risk-scoring model developed and validated for GERD reproduces the same ordering, with anthropometric and smoking variables carrying the greatest weight.[7] Prospective evidence points the same way: in a large cohort of United States women, adherence to a composite antireflux lifestyle was associated with a substantially lower risk of reflux symptoms, but that composite was dominated by body weight and smoking rather than by diet alone.[8] Mendelian randomisation is the most decisive of all, showing robust causal signals for genetically predicted adiposity and smoking while dietary proxies remain weak and inconsistent.[9] If that hierarchy is correct, then removing adiposity and smoking by design should leave comparatively little for dietary pattern and psychological stress to explain — a prediction this study is able to test directly.

Both exposures nevertheless remain biologically plausible. Dietary pattern — a composite of meal regularity, meal frequency, meal timing, food composition and portion size — acts on gastric physiology directly, and experimental work shows that meal-induced heartburn identifies physiologically distinct reflux phenotypes rather than a single mechanism.[10] A systematic review of dietary and lifestyle factors finds the most consistent signals for meal timing, fat intake and portion size, with considerable heterogeneity elsewhere.[11] Psychological stress acts through the gut-brain axis, and the causal architecture here is genuinely bidirectional: Mendelian randomisation studies demonstrate that reflux disease raises the risk of anxiety and depression, and that major depressive disorder in turn raises the risk of reflux.[12,13] Observational work is consistent with this, with perceived stress associated with reflux symptoms in a countrywide Sri Lankan sample,[14] anxiety associated with reflux symptom burden in a prospective cohort,[15] and psychological comorbidity associated with impaired quality of life in population-based data.[4] Cross-sectional designs therefore cannot establish the direction of any stress-reflux association they detect.

Medical students are an informative population in which to test these exposures. Medical education imposes prolonged study hours, dense coursework and continuous assessment, a combination reliably associated with irregular eating, meal skipping, late-night food intake, shortened sleep and elevated psychological distress; pooled estimates place the prevalence of psychological distress among medical students high but heterogeneous,[16] and Southeast Asian health-science students show comparable figures.[17] Reflux symptoms are correspondingly common in this group, with nation-wide and single-institution surveys reporting substantial prevalence and demonstrable impairment of lifestyle and academic performance.[18-21] At the same time, students in health disciplines possess better nutritional knowledge than their peers, although that knowledge translates inconsistently into behaviour.[22] The Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, is a state medical school in the national capital whose preclinical curriculum spans the 2023 to 2025 cohorts examined here, with an annual intake of approximately 200 students; a substantial proportion live in boarding houses away from family food provision, an arrangement repeatedly linked in the Indonesian context to irregular meal timing and reliance on convenience foods.[23]

Indonesian evidence on this question is abundant but discordant. Studies at Universitas Baiturrahmah, ITSK RS dr. Soepraoen and Universitas Advent Indonesia report significant dietary associations,[24-26] whereas the Universitas Mataram study reports none;[27] comparable divergence exists for academic stress between the Universitas Ahmad Dahlan, Universitas Sebelas Maret and Universitas Batam samples.[28-30] Three features explain most of this heterogeneity and none is a genuine biological difference. Sample sizes are small and event counts smaller still, so estimates are dominated by sampling variation; populations differ markedly in the prevalence of the dominant risk factors, which are seldom restricted or adjusted for; and instruments and cut-points differ between studies even when they share a name. Against this background the specific aims of this study were: (i) to estimate the prevalence of symptom-defined GERD with an interval estimate; (ii) to quantify the association of dietary pattern and of psychological stress with GERD symptoms as adjusted odds ratios with 95% confidence intervals, in a population from which the dominant competing causes had been removed by eligibility; and (iii) to determine quantitatively what magnitude of association this design was able to detect, and to bound the systematic error in that determination, so that any null finding could be interpreted as evidence about precision rather than as a bare failure to reject the null hypothesis.

2. Methods

Study design, setting and period

This was an analytical observational study using a quantitative cross-sectional design, reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-sectional studies. A cross-sectional approach was selected because both exposures and the outcome were measured simultaneously at a single point of contact, which permits estimation of

prevalence and of exposure-outcome association without follow-up, at the cost of any temporal ordering.

The study was conducted at the Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, Jakarta, Indonesia. Data were collected between 6 October and 21 November 2025, in the middle third of the odd semester of the 2025/2026 academic year. This window was chosen deliberately: it falls after the settling-in period of the new academic year and before the end-of-semester examination block, so that the measured stress distribution reflects routine academic load rather than an examination peak. Readers comparing these findings with studies conducted during examination periods should bear that difference in mind, since it would be expected to shift the stress distribution upward and possibly the reflux prevalence with it.

The instrument was delivered online through Google Forms. Institutional single sign-on was used to restrict access to enrolled students and to prevent duplicate submissions; responses were pseudonymised at the point of entry and the linking key was destroyed once eligibility screening was complete. The questionnaire was piloted with ten students from a cohort outside the study frame to check comprehension and completion time; no item was changed and the pilot responses were not included in the analysis.

Study population and eligibility criteria

The source population comprised all 618 preclinical medical students enrolled in the 2023, 2024 and 2025 cohorts of the undergraduate medical programme, of whom 372 were female and thus constituted the eligible sampling frame. The complete participant pathway, from the source population to the final analytic sample and its outcome distribution, is shown in Figure 1. Inclusion criteria were active enrolment in one of these cohorts, female sex, age 18 to 23 years, body mass index between 18.5 and 24.9 kg/m^2, provision of written informed consent, and completion of all study questionnaires. Exclusion criteria were current or former smoking, current or previous alcohol consumption, and use of medications known to alter gastrointestinal physiology, specifically long-term acid-suppressive therapy, corticosteroids or high-dose non-steroidal anti-inflammatory drugs (collectively referred to hereafter as gastro-active medication). Height and weight were self-reported; self-reported body mass index is known to be modestly underestimated in young women, and the consequence for this study is considered in the Limitations.

These criteria constitute the study's principal strategy for confounder control and are stated as such. Male sex, excess body weight, smoking, alcohol intake and gastro-active medication are the exposures for which the evidence of a causal effect on reflux is strongest, in population-based risk-factor studies,[6] in validated multivariable risk models,[7] and in Mendelian randomisation analysis.[9] Restricting eligibility on all five removes them from the causal structure entirely rather than relying on statistical adjustment in a small sample, and it produces a homogeneous population in which any residual association with dietary pattern or stress must operate independently of the dominant pathway. The cost of this decision — a deliberate and accepted one — is that the findings apply to normal-weight, non-smoking, non-drinking young women and cannot be extrapolated beyond them.

Sample size and sampling

The minimum sample size was calculated using the formula for the comparison of two independent proportions, $n = [Z_{1-\alpha/2}\sqrt{(2\bar{P}(1-\bar{P}))} + Z_{1-\beta}\sqrt{(P_1(1-P_1) + P_2(1-P_2))}]^2 / (P_1 - P_2)^2$, with a two-sided significance level α of 5% ($Z_{1-\alpha/2} = 1.96$) and a statistical power of 80% ($Z_{1-\beta} = 0.84$). For the dietary comparison, proportions of $P_1 = 0.47$ for GERD symptoms among students with a poor dietary pattern and $P_2 = 0.15$ among those with a good pattern were taken from the Indonesian literature from which the dietary instrument was also adapted.[25] These values give 32 participants per group, or 64 in total. The corresponding calculation for the stress comparison yielded a smaller requirement, so the dietary figure was retained. A further 10% was added for incomplete questionnaires and non-response, giving a minimum requirement of 70 participants.

Participants were selected by probability sampling using a simple random sampling technique applied to the eligible sampling frame, which gives every eligible student an equal selection probability and minimises selection bias. Randomisation was implemented by a pseudorandom number generator applied to the anonymised enrolment list by a researcher not involved in data collection. As detailed in Figure 1, 120 students were selected and invited, of whom 104 completed the instrument, giving a response rate of 86.7%; 17 respondents were excluded after screening — nine for a body mass index outside the eligible range, five for smoking or alcohol intake, two for gastro-active medication and one for an incomplete questionnaire — leaving 87 participants for analysis. No participant had a missing value on any exposure, covariate or outcome, so no imputation was required and all analyses are complete-case by construction.

Variables and measurement

The dependent variable was symptom-defined gastro-oesophageal reflux disease, measured with the Indonesian-language version of the Gastro-oesophageal Reflux Disease Questionnaire (GERD-Q), a validated six-item self-administered instrument covering reflux-related symptoms over the preceding seven days. Four items address positive reflux symptoms and two are reverse scored; each is scored on a four-point Likert scale, giving a total of 0 to 18. A score of 8 or more indicates a high probability of GERD and a score of 7 or less a low probability. The instrument was derived in symptomatic primary-care

attenders, where the cut-point of 8 achieved a sensitivity of approximately 65% and a specificity of approximately 71% against specialist diagnosis;[31] these operating characteristics are used directly in the bias analysis described below. The Indonesian version demonstrates corrected item-total correlation coefficients of 0.47 to 0.71 and a Cronbach alpha of 0.83,[32] and contemporary cross-cultural adaptations confirm that the six-item structure and the threshold of 8 transport across languages.[33] All reverse-scored items in all three instruments were independently re-checked for correct coding by a second investigator before analysis. The instruments and their scoring conventions are summarised in Table 1.

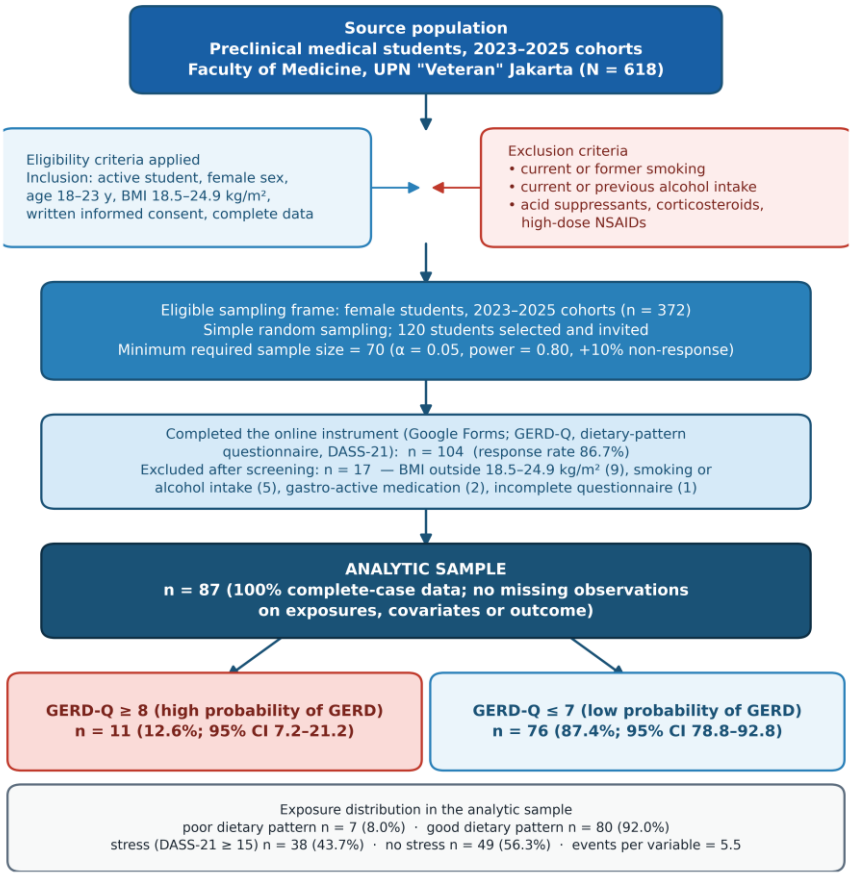

Figure 1. Participant flow through the study, reported according to the STROBE statement for cross-sectional studies. Of 618 preclinical medical students in the 2023-2025 cohorts, 372 female students formed the eligible sampling frame, 120 were randomly selected and invited, 104 responded (86.7%) and 17 were excluded after screening, leaving 87 participants with complete data on every variable. 11 participants (12.6%; 95% CI 7.2-21.2) screened positive on the GERD-Q at the threshold of 8. Restrictive eligibility fixed sex, body mass index, smoking status, alcohol intake and gastro-active medication use by design rather than by statistical adjustment.

The first independent variable was dietary pattern, assessed with a structured 26-item questionnaire adapted from previously validated Indonesian instruments.[25,26] The questionnaire evaluates four components of eating behaviour over the preceding seven days — meal regularity, meal frequency, food type and portion size — with responses on a four-point Likert scale and reverse scoring applied to negatively worded items. The total ranges from 0 to 78, with higher scores indicating healthier habits; participants scoring 39 or above were classified as having a good dietary pattern and those below 39 as having a poor dietary pattern. We note explicitly that 39 is the arithmetic midpoint of the scale and is a convention adopted from prior Indonesian work rather than a criterion-validated threshold. Validation studies report corrected item-total correlations of 0.365 to 0.602 and a Cronbach alpha of 0.884.[25]

The second independent variable was psychological stress, measured with the stress subscale of the Indonesian version of the Depression Anxiety Stress Scale-21 (DASS-21). The subscale comprises seven items scored from 0 ("did not apply to me at all") to 3 ("applied to me very much or most of the time") for the preceding week, with the total multiplied by two according to the standard scoring procedure. Scores of 0 to 14 were classified as no stress and 15 or above as stress.[34,35] The Indonesian version shows stress-subscale factor loadings of 0.45 to 0.78 and a Cronbach alpha of 0.85.[36] All three instruments referred to the same preceding seven-day window and were completed at a single sitting. Contextual variables were age in completed years, cohort year and residence (boarding house versus living with parents); these were recorded as descriptive characteristics of the sample and were not hypothesis-driven exposures.

Table 1. Measurement instruments, scoring and classification thresholds.

VARIABLE	INSTRUMENT	SCORE RANGE	THRESHOLD APPLIED	REPORTED PSYCHOMETRIC PROPERTIES
GERD symptoms (outcome)	GERD-Q, 6 items, 4-point Likert, 7-day recall	0-18	≥ 8 = high probability of GERD; ≤ 7 = low probability	Derivation sensitivity ≈ 65%, specificity ≈ 71%; Indonesian version corrected item-total r 0.47-0.71, Cronbach α 0.83
Dietary pattern (exposure 1)	Structured 26-item questionnaire covering meal regularity, meal frequency, food type and portion size	0-78	≥ 39 = good pattern; < 39 = poor pattern	Corrected item-total r 0.365-0.602; Cronbach α 0.884
Psychological stress (exposure 2)	DASS-21 stress subscale, 7 items, total multiplied by two	0-42	0-14 = no stress; ≥ 15 = stress	Indonesian version factor loadings 0.45-0.78; Cronbach α 0.85
Age	Self-reported completed years	18-23 y (eligibility)	Analysed continuous and dichotomised at 20 y	Not applicable
Body mass index	Self-reported height and weight	18.5-24.9 kg/m^2 (eligibility)	Fixed by restriction	Not applicable
Residence	Single item	Binary	Boarding house vs living with parents	Not applicable

GERD-Q = Gastroesophageal Reflux Disease Questionnaire; DASS-21 = Depression Anxiety Stress Scale-21. The 39-point dietary threshold is the arithmetic midpoint of the scale and is a convention adopted from prior Indonesian work rather than a criterion-validated cut-point. All three instruments referred to the same preceding seven-day window and were completed at a single sitting.

Statistical analysis

All analyses followed a pre-specified plan and were performed in Python 3.11 with NumPy 2.4.4 and SciPy 1.17.1. All tests were two-sided with α set at 0.05, all confidence intervals are two-sided at the 95% level, and all p values are reported to three decimal places. The random seed was fixed at 20260727 for every stochastic procedure. The complete analysis script is available with the data.

Inferential hierarchy. Dietary pattern and psychological stress were the two pre-specified co-primary exposures, meaning that each was tested against the outcome without any ordering between them and that the study was not designed to claim success on the basis of either alone. The analyses of residence, cohort year, age group and the composite "poor diet and/or stress" exposure were exploratory and post-hoc, and are labelled as such wherever they appear. Because six bivariate comparisons were performed, Benjamini-Hochberg and Holm-adjusted p values are reported in the footnote to Table 3.

Descriptive and bivariate analysis. Categorical variables were summarised as counts and percentages with 95% Wilson score confidence intervals, which have substantially better coverage properties than Wald intervals and cannot violate the parameter boundaries. Risk differences use Newcombe hybrid-score intervals, risk ratios use the Katz log method, and adjusted odds ratios use profile penalised-likelihood intervals; each was selected as the interval method appropriate to its estimand. Bivariate associations were tested with the χ^2 test of independence, replaced by the Fisher exact test whenever any expected cell count fell below five. Because the conditional Fisher test is conservative under this design, the Barnard and Boschloo unconditional exact tests were computed in parallel as generally more powerful alternatives, and a Monte Carlo permutation test with 200,000 relabellings was run as a distribution-free check. The conditional maximum-likelihood odds ratio with its exact interval is the primary estimator because it requires no arbitrary continuity correction; the Haldane-Anscombe estimator, which adds 0.5 to each cell, is reported alongside it as a sensitivity analysis and differs materially only where one cell contains two events.

Multivariable analysis. Firth penalised logistic regression was used. With 11 events and one exposure stratum containing seven participants, ordinary maximum likelihood is biased away from the null and may not converge; penalising the score function by the Jeffreys prior removes first-order small-sample bias and guarantees finite estimates.[37] Firth estimates were obtained by modified score equations solved by iteratively reweighted least squares with step halving; confidence intervals were obtained by profiling the penalised likelihood with Brent root-finding, and p values by penalised likelihood-ratio tests rather than Wald statistics. Model 1, specified in advance, contained both pre-specified exposures simultaneously, giving 5.5 events per variable. Model 2 additionally adjusted for age, residence and cohort at 2.2 events per variable and is presented solely to demonstrate the stability of the Model 1 point estimates under further adjustment; it is not offered as an estimate in its own right. Both ratios fall below contemporary sample-size recommendations, and the penalised estimates should accordingly be read as

shrunken descriptions rather than as reliable adjusted effects. No stepwise or p-value-based variable-screening rule was used at any stage.

Bayesian analysis and equivalence. A Bayesian logistic model was fitted by random-walk Metropolis sampling with a multivariate normal proposal whose covariance was set to twice the Firth information-based covariance scaled by the square root of the parameter count (200,000 iterations, 40,000 burn-in, thinning of 10, 16,000 retained draws, acceptance rate 40%). Bayes factors were derived by the Savage-Dickey density ratio at a log odds ratio of zero. Because Bayes factors are sensitive to the prior on the alternative, four priors were used: Cauchy(0, 2.5) as the default, and Cauchy(0, 1), Normal(0, 1) and Normal(0, 2.5) as sensitivity analyses; conclusions are stated in terms of the resulting range. A region of practical equivalence spanning odds ratios of 0.80 to 1.25 was pre-specified, and two one-sided tests were applied to the risk difference. The equivalence margin of +/-10 percentage points was set at approximately the smallest difference that would justify a targeted campus health-promotion programme against a baseline risk of 12%; it corresponds to an odds ratio of roughly 2 and is deliberately permissive, so failure to demonstrate equivalence within it is a strong statement about the study's imprecision.

Precision analysis. The minimum odds ratio detectable with 80% power at the observed stratum sizes, and the sample size that would be required to detect effects of specified magnitude, were computed by the two-proportion arcsine transformation at $\alpha = 0.05$; these are approximations conditional on that choice and would differ modestly under an exact-test formulation. Power was additionally computed at pre-specified odds ratios of 2.0 and 3.0. Power computed at the observed effect size is reported for continuity with the existing literature but is not used for inference, because such a quantity is a deterministic function of the observed p value and therefore adds no information to it.

Quantitative bias analysis. Because the central claim of this study concerns the absence of an association, systematic error was bounded rather than merely acknowledged. Three analyses were performed. First, the specificity implied by the observed positivity was computed across a range of assumed true prevalences, to test whether the derivation operating characteristics of the GERD-Q[31] are plausible in this population. Second, a probabilistic bias analysis for non-differential outcome misclassification was run with 100,000 iterations, drawing sensitivity from a trapezoidal distribution over 0.55 to 0.90 (mode 0.65-0.80) and specificity over 0.85 to 0.99 (mode 0.92-0.97), applying the matrix correction to each two-by-two table and incorporating random error by binomial resampling of the cell counts. Third, E-values were computed for the observed estimates, for their upper confidence limits, and for a hypothetical true odds ratio of 2.0, in order to express how strong unmeasured confounding would have to be to have masked an effect of the magnitude the field regards as important. Non-parametric bootstrap resampling with 20,000 replications was used to verify the stability of every odds ratio, and 500 replications were used for the optimism correction of the c-statistic, in which the model was refitted within each bootstrap sample and evaluated both on that sample and on the original data.

Ethical considerations

This study was approved by the Research Ethics Committee of Universitas Pembangunan Nasional "Veteran" Jakarta, Indonesia (Ethical Approval No. 41/III/2026/KEP). All study procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki. Participation was entirely voluntary. Written informed consent was obtained electronically from every participant before any data were collected, and participants were informed of their right to decline participation or to withdraw at any time without consequence. All collected data were pseudonymised at the point of entry, anonymised on completion of eligibility screening, and treated confidentially throughout the research process to protect participant privacy.

3. Results

Participant characteristics

Of 618 preclinical medical students in the 2023 to 2025 cohorts, 372 female students formed the eligible sampling frame, 120 were randomly selected and invited, and 104 completed the instrument, a response rate of 86.7%. Seventeen respondents were excluded after screening, leaving 87 participants in the analytic sample; the complete pathway, together with the outcome distribution and the exposure distribution of the analysed sample, is presented in Figure 1. No observation was missing on any exposure, covariate or outcome.

The demographic profile of the sample, with an interval estimate for every proportion, is detailed in Table 2. Participants were aged 18 to 22 years, with a mean of 19.6 years (SD 1.1); as Table 2 shows, the most frequent single age was 19 years (26 students, 29.9%; 95% CI 21.3-40.2), followed by 20 years (25, 28.7%; 95% CI 20.3-39.0). The 2023 cohort contributed the largest group (35 students, 40.2%; 95% CI 30.6-50.7), followed by the 2025 cohort (29, 33.3%) and the 2024 cohort (23, 26.4%). Boarding houses accommodated 55 students (63.2%; 95% CI 52.7-72.6), while 32 (36.8%; 95% CI 27.4-47.3) lived with their parents. By eligibility, and as recorded in the corresponding rows of Table 2, all participants were female, had a body mass index between 18.5 and 24.9 kg/m^2, had never smoked, reported no alcohol intake and used no gastro-active medication.

The exposure and outcome distributions are given in the lower panel of Table 2. Most participants were

classified as having a good dietary pattern (80 students, 92.0%; 95% CI 84.3-96.0), leaving 7 (8.0%; 95% CI 4.0-15.7) in the poor-dietary-pattern stratum. Stress was more evenly distributed: 38 students (43.7%; 95% CI 33.7-54.1) scored 15 or above on the DASS-21 stress subscale and 49 (56.3%; 95% CI 45.9-66.3) did not. GERD-Q positivity, defined as a total score of 8 or more, was recorded in 11 students, giving a prevalence of 12.6% (95% CI 7.2-21.2). Because the analysis plan pre-specified the dichotomised form of all three instruments described in Table 1, continuous score distributions and domain-level dietary analyses were not undertaken; this is addressed in the Limitations.

Table 2. Characteristics of the study participants (n = 87).

CHARACTERISTIC	N	%	95% WILSON CI (%)
Total participants analysed	87	100.0	—
Age (years)			
18	17	19.5	12.6-29.1
19	26	29.9	21.3-40.2
20	25	28.7	20.3-39.0
21	16	18.4	11.6-27.8
22	3	3.4	1.2-9.7
Mean (SD)	19.6 (1.1)	—	19.4-19.9
Cohort year			
2023	35	40.2	30.6-50.7
2024	23	26.4	18.3-36.6
2025	29	33.3	24.3-43.8
Residence			
Boarding house	55	63.2	52.7-72.6
Living with parents	32	36.8	27.4-47.3
Fixed by eligibility criteria			
Female sex	87	100.0	95.8-100.0
Body mass index 18.5-24.9 kg/m^2	87	100.0	95.8-100.0
Never-smoker	87	100.0	95.8-100.0
No alcohol intake	87	100.0	95.8-100.0
No gastro-active medication	87	100.0	95.8-100.0
Dietary pattern			
Good (score ≥ 39)	80	92.0	84.3-96.0
Poor (score < 39)	7	8.0	4.0-15.7
Psychological stress (DASS-21 stress subscale)			
No stress (0-14)	49	56.3	45.9-66.3
Stress (≥ 15)	38	43.7	33.7-54.1
GERD symptoms (GERD-Q)			
No GERD (score ≤ 7)	76	87.4	78.8-92.8
GERD (score ≥ 8)	11	12.6	7.2-21.2

SD = standard deviation; DASS-21 = Depression Anxiety Stress Scale-21; GERD-Q = Gastroesophageal Reflux Disease Questionnaire. Wilson score intervals are reported because they have substantially better coverage properties than Wald intervals at proportions close to 0 and 1. Rows under "Fixed by eligibility criteria" are constant by design and represent confounder control by restriction rather than observed characteristics. Source: primary data, Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, 2026.

Bivariate associations

The association of each exposure with GERD symptoms, with the complete effect-size panel, is detailed in Table 3, and the corresponding stratum prevalences with their confidence intervals are displayed in Figure 2. Among the 80 students with a good dietary pattern, 9 (11.3%; 95% CI 6.0-20.0) screened positive; among the 7 with a poor dietary pattern, 2 did (28.6%; 95% CI 8.2-64.1). The minimum expected cell count was 0.89, so the Fisher exact test was applied and returned p = 0.214, as reported in Table 3. The unconditional exact alternatives agreed (Barnard p = 0.210; Boschloo p = 0.280), as did the permutation test (p = 0.216). The primary conditional maximum-likelihood odds ratio was 3.10 (95% exact CI 0.26-22.78); the Haldane-Anscombe sensitivity estimate was 3.42 (95% CI 0.66-17.66). The risk ratio was 2.66 (95% CI 0.81-8.75) and the risk difference 17.3 percentage points (95% CI -4.8 to 53.2). Cramer V was 0.142 and Cohen h was 0.444. The width of the interval for the poor-dietary-pattern stratum in Figure 2, which spans 8.2-64.1%, is the visual counterpart of these numbers.

Among the 49 students without stress, 6 (12.2%; 95% CI 5.7-24.2) screened positive; among the 38 with stress, 5 did (13.2%; 95% CI 5.8-27.3). As Table 3 records, the minimum expected cell count was 4.80 and the Fisher exact test returned p = 1.000, with concordant results from the unconditional tests (Barnard p = 0.972; Boschloo p = 0.915) and the permutation test (p = 1.000). The conditional maximum-likelihood odds ratio was 1.08 (95% exact CI 0.24-4.69) and the Haldane-Anscombe estimate 1.10 (95% CI 0.32-3.73); the risk ratio was 1.08 (95% CI 0.38-3.12) and the risk difference 0.9 percentage points (95% CI -13.2 to 16.5). Cramer V was 0.014 and Cohen h was 0.027.

None of the exploratory contextual comparisons listed in the lower part of Table 3 showed an association. GERD-Q positivity was 12.7% among boarding-house residents and 12.5% among those living with parents (odds ratio 0.98, 95% CI 0.28-3.44; p = 1.000), a comparison also displayed in the right-hand pair of Figure 2; 11.4% in the 2023 cohort versus 13.5% in the 2024-2025 cohorts (odds ratio 0.87, 95% CI 0.25-3.04; p = 1.000); and 13.6% at age 20 years or above versus 11.6% below 20 years (odds ratio 1.18, 95% CI 0.35-4.00; p = 1.000). These three variables were recorded as descriptive sample characteristics rather than as hypothesis-driven exposures, and the near-null estimates should be read accordingly. The post-hoc composite exposure, defined as carrying either a poor dietary pattern or stress or both, likewise showed no association (14.3% versus 11.1%; odds ratio 1.31, 95% CI 0.39-4.44; p = 0.753). Only 3 participants carried both exposures simultaneously, so no interaction term was estimable and no formal interaction test was attempted. After Benjamini-Hochberg adjustment across the six bivariate comparisons reported in Table 3, all adjusted p values were 1.000, as were all Holm-adjusted values (1.000).

Table 3. Association of dietary pattern, psychological stress and exploratory contextual variables with GERD symptoms.

EXPOSURE	GERD N/N (%)	NO GERD N/N (%)	OR (95% CI)[a]	RR (95% CI)	RISK DIFFERENCE, PP (95% CI)	CRAMÉR V	COHEN H	EXACT P [b]
Dietary pattern								
Good (reference)	9/80 (11.3)	71/80 (88.8)	1.00	1.00	0.0	—	—	—
Poor	2/7 (28.6)	5/7 (71.4)	3.10 (0.26-22.78)	2.66 (0.81-8.75)	17.3 (-4.8 to 53.2)	0.142	0.444	0.214
Psychological stress								
No stress (reference)	6/49 (12.2)	43/49 (87.8)	1.00	1.00	0.0	—	—	—
Stress	5/38 (13.2)	33/38 (86.8)	1.08 (0.24-4.69)	1.08 (0.38-3.12)	0.9 (-13.2 to 16.5)	0.014	0.027	1.000
Residence (exploratory)								
Living with parents (ref.)	4/32 (12.5)	28/32 (87.5)	1.00	1.00	0.0	—	—	—
Boarding house	7/55 (12.7)	48/55 (87.3)	1.02 (0.23-5.19)	0.98 (0.33-2.91)	0.2 (-16.6 to 13.8)	0.003	0.007	1.000
Cohort year (exploratory)								
2024-2025 (reference)	7/52 (13.5)	45/52 (86.5)	1.00	1.00	0.0	—	—	—
2023	4/35 (11.4)	31/35 (88.6)	0.83 (0.16-3.61)	0.88 (0.30-2.63)	-2.0 (-15.7 to 14.0)	0.030	-0.062	1.000
Age group (exploratory)								
< 20 years (reference)	5/43 (11.6)	38/43 (88.4)	1.00	1.00	0.0	—	—	—
≥ 20 years	6/44 (13.6)	38/44 (86.4)	1.20 (0.28-5.42)	1.16 (0.40-3.32)	2.0 (-12.7 to 16.6)	0.030	0.061	1.000
Composite exposure (post-hoc)								
Neither exposure (reference)	5/45 (11.1)	40/45 (88.9)	1.00	1.00	0.0	—	—	—
Poor diet and/or stress	6/42 (14.3)	36/42 (85.7)	1.31 (0.39-4.44)	1.26 (0.44-3.64)	3.2 (-11.3 to 18.1)	0.048	0.096	0.753

OR = odds ratio; RR = risk ratio; CI = confidence interval; pp = percentage points; ref. = reference. [a] The primary estimator is the conditional maximum-likelihood odds ratio with its exact interval, which requires no continuity correction; the Haldane-Anscombe sensitivity estimates were 3.42 (0.66-17.66) for poor dietary pattern and 1.10 (0.32-3.73) for stress. [b] Two-sided Fisher exact test. Unconditional exact alternatives agreed throughout (dietary pattern Barnard p = 0.210, Boschloo p = 0.280; stress Barnard p = 0.972, Boschloo p = 0.915), as did a 200,000-replication permutation test (p = 0.216 and p = 1.000). Minimum expected cell counts were 0.89 for dietary pattern and 4.80 for stress. Across the six comparisons every Benjamini-Hochberg adjusted p value was 1.000 and every Holm-adjusted value 1.000. Only 3 participants carried both exposures, so no interaction term was estimable. Source: primary data, Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, 2026.

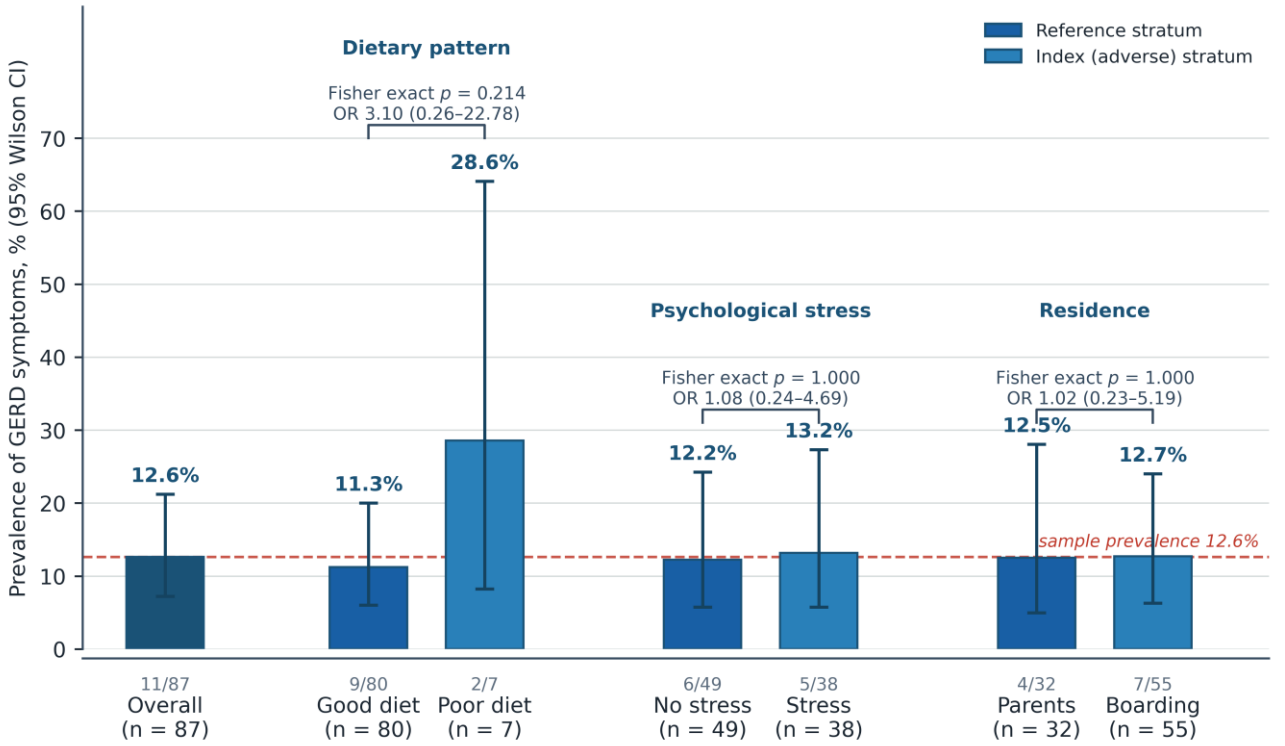

Figure 2. Prevalence of GERD symptoms by exposure stratum with 95% Wilson score confidence intervals, corresponding to the stratum rows of Table 3. Numbers below each bar give events over stratum size and the dashed line marks the whole-sample prevalence of 12.6%. The poor-dietary-pattern stratum contains 7 participants and its interval spans 8.2-64.1%, so the difference in point estimates (28.6% versus 11.3%) is statistically indistinguishable from no difference. p values are two-sided Fisher exact tests; the residence comparison is exploratory.

Multivariable analysis

Every estimator applied to the two co-primary exposures is displayed on a common logarithmic axis in Figure 3, and the numerical values are given in Table 4. In the pre-specified Model 1, which contained both exposures simultaneously at 5.5 events per variable, poor dietary pattern carried an adjusted odds ratio of 3.35 (95% profile-likelihood CI 0.55-16.12; penalised likelihood-ratio p = 0.208) and stress an adjusted odds ratio of 1.10 (95% CI 0.31-3.78; p = 0.339). Unpenalised maximum likelihood produced very similar estimates (3.16, 95% CI 0.53-18.75 and 1.09, 95% CI 0.30-3.94), indicating that small-sample bias was present but modest at this event count. In Model 2, presented in Table 4 only as a stability check, the estimates were essentially unchanged: 3.08 (95% CI 0.49-15.69; p = 0.241) for dietary pattern and 1.13 (95% CI 0.32-3.91; p = 0.329) for stress, with adjusted odds ratios for the contextual variables of 1.07 for boarding-house residence, 0.85 for the 2023 cohort and 1.10 per year of age, all with intervals crossing unity. Because those three contextual variables carried essentially no outcome information, their inclusion could not materially alter the exposure estimates, and the stability observed between the two models should not be read as independent evidence of robustness. The maximum variance inflation factor was 1.09.

Model performance, reported in the second block of Table 4, was poor. The likelihood-ratio test for Model 1 gave χ^2 = 1.323 on 2 degrees of freedom (p = 0.516). The Nagelkerke pseudo-R^2 was 0.028; because pseudo-R^2 measures are not comparable to the coefficient of determination of ordinary least squares, this value should be read as indicating a very weak association between the two exposures and the outcome and not as a proportion of variance explained. The apparent c-statistic was 0.562 with a markedly asymmetric bootstrap interval of 0.513-0.766, itself an indicator of instability at 11 events; the optimism-corrected value of 0.486 falls below 0.5 and should be interpreted as evidence that the correction procedure is unstable at this sample size rather than as a substantive discrimination result. Discrimination is in any case not the relevant metric for an aetiological model and is reported only for completeness. The Hosmer-Lemeshow statistic was 5.770 (p = 0.056); with 11 events distributed across four groups this test has negligible power and is likewise uninformative about calibration.

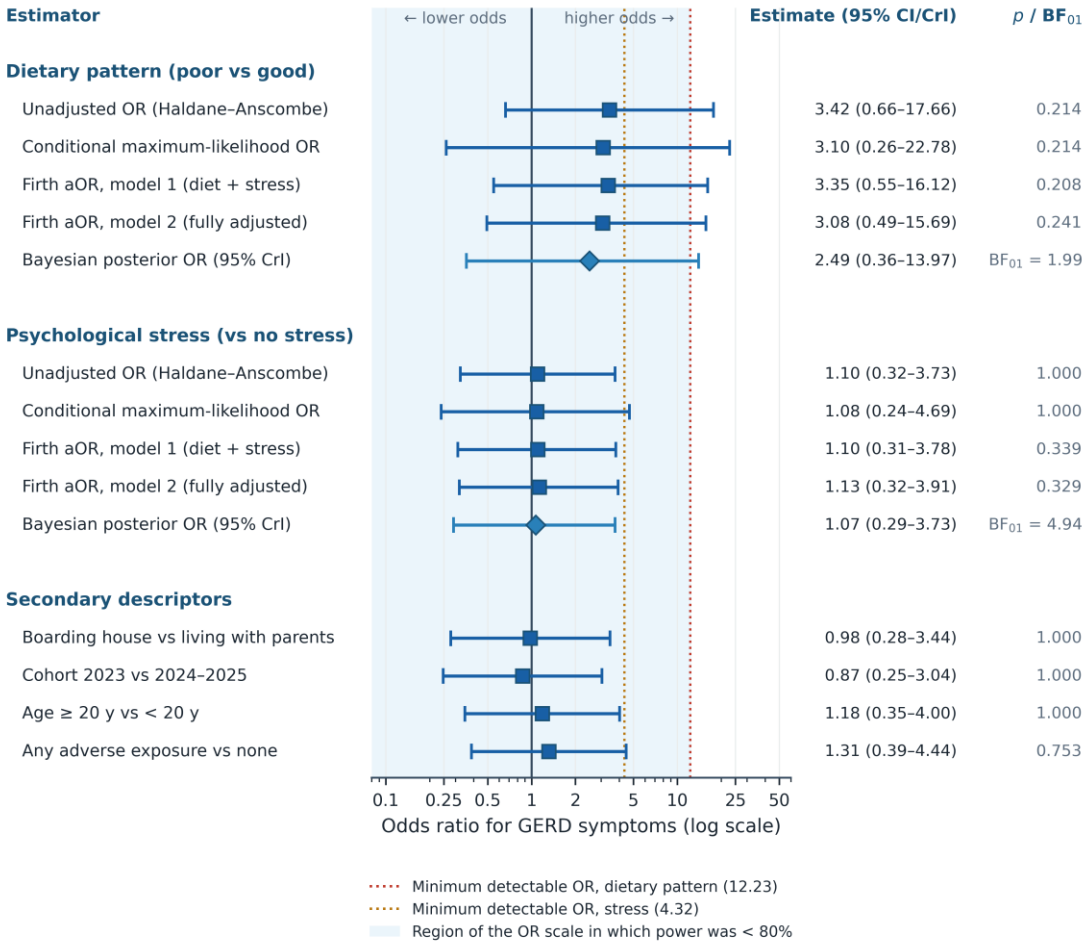

Figure 3. Composite forest plot of all estimators reported in Tables 3 and 4. For each exposure the unadjusted Haldane-Anscombe odds ratio, the primary conditional maximum-likelihood odds ratio with its exact interval, the Firth penalised adjusted odds ratios from the pre-specified and stability-check models, and the Bayesian posterior odds ratio with its credible interval are shown on a common logarithmic axis. The shaded band marks the region of the odds-ratio scale in which the study had less than 80% power and the dotted lines mark the minimum detectable odds ratio for each exposure (12.23 for dietary pattern and 4.32 for stress). Every interval crosses unity and every point estimate lies within the underpowered region — the two facts that must be read together when interpreting these null results.

Bayesian analysis, equivalence and prior sensitivity

Under the default Cauchy(0, 2.5) prior the posterior median odds ratio was 2.49 (95% credible interval 0.36-13.97) for poor dietary pattern and 1.07 (95% credible interval 0.29-3.73) for stress, with posterior probabilities of a positive association of 0.832 and 0.541 respectively; the two posterior distributions are drawn in panel A of Figure 4 and tabulated in the third block of Table 4. The Bayes factor favouring the null was 1.99 for dietary pattern and 4.94 for stress. The posterior probability of falling within the pre-specified region of practical equivalence, shaded in Figure 4A, was 0.113 for dietary pattern and 0.283 for stress.

Prior sensitivity analysis, detailed in Table 4, materially qualifies the stress result. Across the four priors examined, the Bayes factor favouring the null ranged from 0.99 to 2.05 for dietary pattern and from 1.78 to 4.90 for stress. Under the two sceptical priors the stress Bayes factor fell below the conventional threshold of 3, to 2.40 under Cauchy(0, 1.0) and 1.78 under Normal(0, 1.0). The posterior medians were considerably more stable, ranging from 1.76 to 2.61 for dietary pattern and from 1.05 to 1.07 for stress. The evidential claim that can be sustained is therefore that the data provide weak-to-moderate evidence against a strong stress effect, the strength depending on how much prior weight is placed on large effects, and that the data are close to uninformative about dietary pattern under every prior examined.

Two one-sided tests on the risk difference with a ±10-percentage-point margin gave p = 0.663 for dietary pattern and p = 0.104 for stress, as recorded in Table 4; neither reached the 0.05 threshold, so practical equivalence cannot be asserted for either exposure even at this permissive margin.

Precision, achieved power and quantitative bias analysis

The precision analysis is summarised in the final blocks of Table 4 and drawn as a power curve in panel B of Figure 4. The minimum odds ratio detectable with 80% power at α = 0.05 was 12.23 for dietary pattern, corresponding to a GERD prevalence of 60.8% in the poor-dietary-pattern stratum, and 4.32 for stress,

corresponding to a prevalence of 37.6% among stressed students; both thresholds are marked by dotted lines in Figure 4B. At pre-specified odds ratios of scientific interest, marked by open circles in the same panel, power was 9% for dietary pattern and 22% for stress at an odds ratio of 2.0, and 19% and 52% respectively at an odds ratio of 3.0. To detect the observed dietary-pattern odds ratio with 80% power at the observed exposure ratio would require approximately 459 participants; the corresponding figure for the observed stress odds ratio is of the order of 30,000, a calculation that is inherently unstable because the observed odds ratio lies almost exactly at the null. Power computed at the observed effect sizes was 20.2% and 3.3%; these values are reported for continuity with the literature but are algebraically determined by the observed p values and carry no independent information. Bootstrap resampling with 20,000 replications reproduced the analytic intervals closely (dietary pattern median odds ratio 3.41, 95% CI 0.47-17.67; stress 1.09, 95% CI 0.27-4.07).

The bias analysis produced three findings, all reported in the last block of Table 4. First, the derivation specificity of the GERD-Q is not tenable in this population: at a specificity of 0.71, even a true prevalence of zero would generate an apparent prevalence of 29.0%, and a true prevalence of 10% would generate 32.6%, against the 12.6% actually observed. Working backwards, the observed positivity implies a specificity of 0.90 at a true prevalence of 5%, 0.93 at 10% and 0.97 at 15% — that is, the instrument performs substantially better in this low-risk population than its primary-care derivation statistics suggest, which is the expected direction of the spectrum effect. Second, the probabilistic bias analysis for non-differential outcome misclassification, incorporating both systematic and random error, gave a median corrected odds ratio of 6.61 (95% simulation interval 0.71-206) for dietary pattern, with 80.3% of the simulated distribution exceeding 2.0, and 1.17 (95% simulation interval 0.07-17.9) for stress, with 30.6% exceeding 2.0. The conventional analysis therefore understates the plausible dietary effect, although with correspondingly enormous uncertainty. Third, the E-value for the observed dietary odds ratio was 3.10 and for its upper confidence limit 7.87; for stress the corresponding values were 1.28 and 3.27. An unmeasured confounder would need to be associated with both exposure and outcome by a risk ratio of at least 2.18, above and beyond the measured variables, to have masked a true odds ratio of 2.0.

A selection-bias sensitivity analysis was performed for the 16 invited students who did not respond and the 17 excluded after screening. If those 33 non-participants had a GERD prevalence of 5%, the combined prevalence across all 120 invited students would be 10.0%; at 12.6% it would be 12.3%, at 25% it would be 16.9% and at 40% it would be 21.5%. The observed estimate is therefore robust to plausible differential participation but would be materially understated if symptomatic students had systematically declined, which is the opposite of the usual direction of concern in symptom surveys.

4. Discussion

In a deliberately homogeneous sample of 87 female preclinical medical students at the Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, symptom-defined gastro-oesophageal reflux disease was present in 12.6% (95% CI 7.2-21.2), and neither dietary pattern nor psychological stress was associated with it. As summarised in Table 4 and displayed in Figure 3, poor dietary pattern carried an adjusted odds ratio of 3.35 (95% CI 0.55-16.12) and stress an adjusted odds ratio of 1.10 (95% CI 0.31-3.78). Those two intervals are the substance of the finding, and they are not equivalent. The dietary interval is compatible with anything from a moderate protective effect to a fifteen-fold increase in odds; the stress interval is narrow enough to make a strong effect unlikely but not a moderate one. The Bayes factors reflect this asymmetry, and their prior sensitivity constrains how strongly it can be stated: across the four priors listed in Table 4 the evidence against a stress effect ranged from 1.78 to 4.90, crossing the conventional threshold of 3 only under the more permissive priors, while the dietary evidence ranged from 0.99 to 2.05 and remained anecdotal throughout. The defensible claim is therefore that a strong independent effect of perceived stress is unlikely in this population, and that the data are close to uninformative about dietary pattern. Reporting these two nulls as though they carried equal evidential weight, which a p-value-only presentation would do, would misrepresent the data.

The observed prevalence deserves attention in its own right and not merely as context for the association analysis. A GERD-Q positivity of 12.6% in a population from which male sex, excess body weight, smoking, alcohol and gastro-active medication have all been excluded by the eligibility criteria shown in Figure 1 is, if anything, higher than one might have predicted, and it implies that a substantial baseline of reflux symptomatology in young Indonesian women is not attributable to the classical risk factors at all. It sits within the range reported for student populations elsewhere, where nation-wide and single-institution surveys have documented substantial symptom burden and measurable impairment of lifestyle and academic performance.[18-21] Three explanations merit consideration. The first is phenotype: experimental analysis of meal-induced heartburn shows that reflux symptoms arise from physiologically distinct phenotypes rather than a single acid-driven mechanism, and symptom questionnaires cannot separate them.[10]

Table 4. Multivariable, Bayesian, precision and quantitative bias analysis of the association between each exposure and GERD symptoms.

ANALYSIS	POOR DIETARY PATTERN	PSYCHOLOGICAL STRESS
Firth penalised logistic regression		
Model 1 — both exposures, adjusted OR (95% CI)	3.35 (0.55-16.12)	1.10 (0.31-3.78)
Model 1 — penalised likelihood-ratio p	0.208	0.339
Model 2 — stability check, adjusted OR (95% CI) [a]	3.08 (0.49-15.69)	1.13 (0.32-3.91)
Model 2 — penalised likelihood-ratio p	0.241	0.329
Unpenalised maximum-likelihood OR (95% CI)	3.16 (0.53-18.75)	1.09 (0.30-3.94)
Model performance (Model 1)		
Likelihood-ratio χ^2 (2 df), p	1.323, p = 0.516	
Nagelkerke pseudo-R^2 [b]	0.028	
Hosmer-Lemeshow χ^2 (2 df), p [c]	5.770, p = 0.056	
Apparent c-statistic (95% CI) [c]	0.562 (0.513-0.766)	
Optimism-corrected c-statistic [c]	0.486	
Events per variable	5.5 (Model 1); 2.2 (Model 2)	
Maximum variance inflation factor	1.09	
Bayesian analysis — prior sensitivity [d]		
Cauchy(0, 2.5), default: OR (95% CrI)	2.47 (0.36-13.52)	1.06 (0.29-3.71)
Cauchy(0, 2.5), default: BF_{01}	1.99	4.94
Cauchy(0, 1.0), sceptical: BF_{01}	1.28	2.40
Normal(0, 1.0), sceptical: BF_{01}	0.99	1.78
Normal(0, 2.5), weakly informative: BF_{01}	1.43	3.69
Range of BF_{01} across the four priors	0.99 to 2.05	1.78 to 4.90
Posterior probability that OR > 1 (default prior)	0.832	0.541
Posterior probability within ROPE (OR 0.80-1.25)	0.113	0.283
Evidential interpretation	Absence of evidence under every prior	Weak-to-moderate evidence against a strong effect
Equivalence testing		
Two one-sided tests, ±10 pp margin, p	0.663	0.104
Precision and power		
Stratum sizes (unexposed vs exposed)	80 vs 7	49 vs 38
Minimum detectable OR at 80% power	12.23	4.32
Corresponding prevalence in the exposed (%)	60.8	37.6
Power at a pre-specified OR of 2.0 (%)	9	22
Power at a pre-specified OR of 3.0 (%)	19	52
n required to detect the observed OR at 80% power	459	≈ 30,000 [e]
Power at the observed OR (%) [f]	20.2	3.3
Bootstrap median OR (95% CI), 20,000 replications	3.41 (0.47-17.67)	1.09 (0.27-4.07)
Quantitative bias analysis		
Apparent prevalence expected at specificity 0.71 and true prevalence 0	29.0%	
Specificity implied by the observed 12.6% positivity [g]	0.90 (true prevalence 5%) to 0.97 (15%)	
Misclassification-corrected OR (median, 95% simulation interval) [h]	6.61 (0.71-206)	1.17 (0.07-17.9)
Proportion of the corrected distribution above OR 2.0	80.3%	30.6%
E-value, observed estimate	3.10	1.28
E-value, upper confidence limit	7.87	3.27
E-value required to mask a true OR of 2.0	2.18	

OR = odds ratio; CI = confidence interval; CrI = credible interval; ROPE = region of practical equivalence; pp = percentage points; BF_{01} = Bayes factor favouring the null. Firth intervals are profile penalised-likelihood intervals and p values are penalised likelihood-ratio tests. [a] Model 2 additionally adjusted for age (continuous, centred), residence and cohort year; it is presented only to show the stability of the Model 1 point estimates and not as an estimate in its own right. [b] Pseudo-R^2 measures are not comparable to the ordinary least-squares coefficient of determination. [c] Discrimination and calibration statistics are reported for completeness only; the model is aetiological, not predictive, and both are unstable at 11 events. [d] Random-walk Metropolis, 200,000 iterations, 40,000 burn-in, thinning 10, 16,000 retained draws, acceptance rate 40%, seed 20260727; Bayes factors by the Savage-Dickey density ratio. [e] The calculation is unstable because the observed odds ratio lies almost exactly at the null; the figure is an order of magnitude. [f] Power at the observed effect is algebraically determined by the observed p value and is not used for inference. [g] Derived by inverting the apparent-prevalence equation at an assumed sensitivity of 0.65. [h] Probabilistic bias analysis for non-differential outcome misclassification, 100,000 iterations, trapezoidal priors on sensitivity (0.55-0.90, mode 0.65-0.80) and specificity (0.85-0.99, mode 0.92-0.97). Source: primary data, Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, 2026.

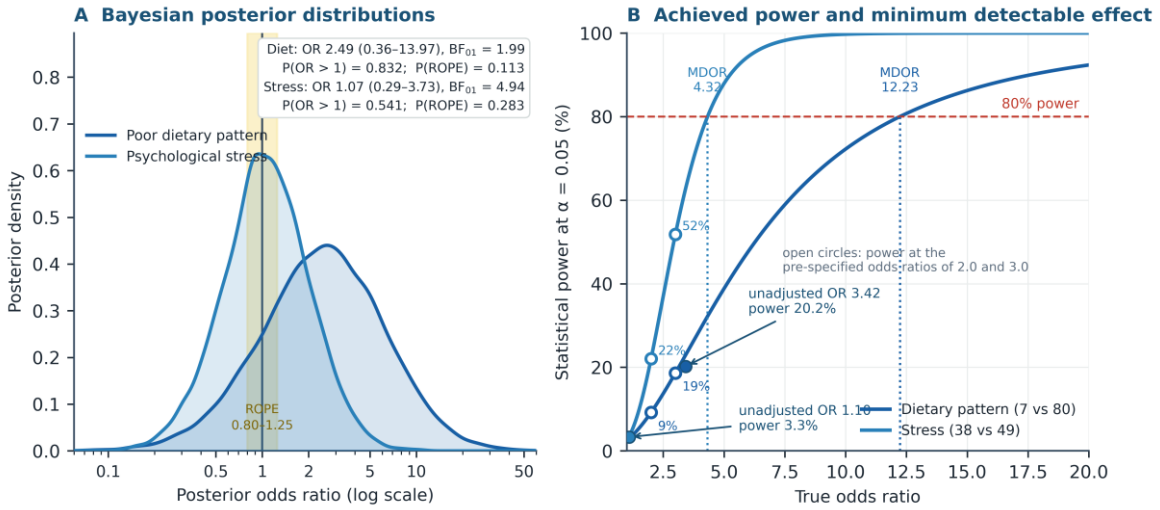

Figure 4. Bayesian evidence and study precision, corresponding to the third and fifth blocks of Table 4. **(A)** Posterior distributions of the odds ratio under the default weakly informative Cauchy(0, 2.5) priors (16,000 retained draws), with the pre-specified region of practical equivalence shaded. Across four priors the Bayes factor favouring the null ranged from 1.78 to 4.90 for stress and from 0.99 to 2.05 for dietary pattern, so the evidence against a strong stress effect is weak-to-moderate and prior-dependent, while the dietary data remain uninformative under every prior. **(B)** Statistical power as a function of the true odds ratio at the achieved stratum sizes. Filled circles mark the observed odds ratios, open circles the power at pre-specified odds ratios of 2.0 (9% and 22%) and 3.0 (19% and 52%), and dotted lines the minimum odds ratio detectable with 80% power.

The second is exposure specificity: meal-to-sleep interval and the consumption of spicy, acidic and carbonated items are prominent features of Indonesian student eating that the composite dietary instrument described in Table 1 captures only indirectly, and meal timing carries the strongest and most consistent support of any dietary exposure in reflux.[11] The third is psychological comorbidity beyond perceived stress: anxiety amplifies reflux symptom reporting in prospective follow-up[15] and is associated with impaired quality of life in population-based data,[4] and it was not measured here.

The measurement properties of the instrument bear directly on this interpretation, and the arithmetic reported in Table 4 is informative. If the GERD-Q retained its derivation specificity of 0.71[31] in this population, then even a true prevalence of zero would have produced an apparent positivity of 29.0%, more than double the 12.6% observed. The observed value is compatible only with a considerably higher specificity here — approximately 0.90 at a true prevalence of 5%, rising to 0.97 at 15%. This is the classic spectrum effect: an instrument derived in symptomatic primary-care attenders performs better in an unselected low-risk population because the differential diagnosis is narrower, and contemporary cross-cultural adaptations confirm that the instrument's structure transports while its operating characteristics are population-dependent.[33] The finding is reassuring for the prevalence estimate. It does not, however, eliminate the problem for the association analysis, because even modest non-differential misclassification of a binary outcome biases the odds ratio towards the null. This is why the bias analysis matters: with plausible sensitivity and specificity distributions the corrected dietary odds ratio rose to 6.61, with 80.3% of the simulated distribution above 2.0. The conventional analysis, in other words, probably understates the dietary effect — although the corrected interval, running from 0.71-206, also demonstrates that correcting for one bias at this sample size buys very little certainty.

The null dietary finding is concordant with the Universitas Mataram study[27] and discordant with those from Universitas Baiturrahmah, ITSK RS dr. Soepraoen and Universitas Advent Indonesia,[24-26] a pattern set out study by study in Table 5. The most parsimonious explanation is not that these populations differ biologically but that the present study had almost no exposure contrast to work with. As Table 2 records, 92.0% of participants were classified as having a good dietary pattern, leaving a comparison group of 7. Restriction of range attenuates any association towards the null irrespective of the underlying truth and inflates the variance of the estimate simultaneously — the reason the confidence interval in Figure 3 spans a fifteen-fold range. Two features of the instrument compound this. Its threshold of 39 is the arithmetic midpoint of a 0-78 summated scale rather than a criterion-validated cut-point, so it classifies as "good" any respondent whose mean item response reaches the midpoint of the response scale, which is a low bar. And it aggregates four conceptually distinct domains into a single score, so that a favourable food-type and portion-size profile can offset genuinely unfavourable meal regularity and timing. The high proportion of favourable classification is therefore only partly explained by the documented advantage of medical students in nutritional knowledge and practice.[22]

Table 5. The present study in the context of comparable Indonesian and international evidence.

STUDY (YEAR)	SETTING	POPULATION	EXPOSURE EXAMINED	REPORTED RESULT	EFFECT SIZE WITH UNCERTAINTY REPORTED?
Present study (2026)	UPN "Veteran" Jakarta, Indonesia (n = 87)	Female preclinical medical students; normal BMI; never-smokers; no alcohol	Dietary pattern and psychological stress	No association for either exposure; GERD prevalence 12.6% (95% CI 7.2-21.2)	Yes — OR, RR, risk difference, 95% CI, Bayes factors, minimum detectable effect, bias analysis
Ardhan et al. (2022)	Universitas Mataram, Indonesia	Medical students	Dietary pattern	No significant association	No
Ilham et al. (2024)	Universitas Baiturrahmah, Indonesia	Medical students	Dietary pattern	Significant association	No
Alaya et al. (2025)	ITSK RS dr. Soepraoen Malang, Indonesia	Health-science students	Dietary pattern	Significant association	No
Lingga & Elon (2025)	Universitas Advent Indonesia	University students	Irregular eating pattern	Significant association	No
Nirwani et al. (2025)	Universitas Ahmad Dahlan, Indonesia	Medical students	Academic stress	Association reported	No
Ridho et al. (2025)	Universitas Sebelas Maret, Indonesia	Medical students	Academic stress and sleep quality	Association reported; sleep quality contributory	No
Sabisi & Kusumawardani (2026)	Universitas Batam, Indonesia	Clinical-phase medical students	Academic stress	Association reported	No
Baklola et al. (2023)	Nationwide, Egypt	Medical students	Multiple lifestyle factors	Irregular meals and distress associated with GERD	Partial
Essa et al. (2024)	Egypt	Undergraduate medical students	Multiple risk factors	Prevalence and risk factors quantified	Yes
Wickramasinghe et al. (2023)	Countrywide, Sri Lanka	General adult population	Perceived stress	Perceived stress associated with reflux symptoms	Yes
Mehta et al. (2021)	Nurses' Health Study II, USA	Adult women, prospective cohort	Composite antireflux lifestyle	Adherence associated with substantially lower risk; composite dominated by weight and smoking	Yes
Yuan & Larsson (2022)	Genetic consortia, Europe	Adults, Mendelian randomisation	Adiposity, smoking, dietary proxies	Causal signal for adiposity and smoking; dietary proxies weak	Yes

BMI = body mass index; OR = odds ratio; RR = risk ratio; CI = confidence interval. Sample sizes for the Indonesian comparators are omitted because they were not reported in a directly comparable form. The final column records whether the study published a quantitative effect estimate with a measure of uncertainty; the predominance of "No" among the Indonesian single-institution studies is the specific reporting gap this manuscript addresses, and it explains most of the apparent disagreement between them without invoking any biological difference between the populations studied.

The null stress finding is quantitatively better supported and warrants a different reading. The stress strata were nearly balanced at 38 versus 49, so restriction of range was not the limiting factor; the limiting factor was the event count. With 11 events, the minimum detectable odds ratio marked in Figure 4B was 4.32, meaning that this study could only have detected an effect in which stressed students had a reflux prevalence of 37.6% or more; power at a more realistic odds ratio of 2.0 was 22%. The observed prevalence difference reported in Table 3 was 0.9 percentage points. Countrywide evidence from Sri Lanka does report an association between perceived stress and reflux symptoms,[14] as do smaller South Asian[38] and Indonesian samples,[28-30] but those associations are of moderate magnitude, and moderate magnitudes are precisely what this design was unable to see. It is also relevant that data were collected in mid-semester rather than during an examination block, so the measured stress reflects routine rather than peak academic load; a study conducted during examinations might well observe both a higher stress prevalence and a stronger association.

Biologically, both null findings remain compatible with the established mechanisms summarised in Figure 5. Dietary exposures act through luminal and motor pathways — increased gastric acid secretion, reduced lower oesophageal sphincter resting pressure, more frequent transient sphincter relaxations, delayed gastric emptying and raised intragastric pressure — and meal-induced symptom provocation identifies these as physiologically separable phenotypes.[10,11] Psychological stress acts through the gut-brain axis, and the Mendelian randomisation evidence establishes that the relationship runs in both directions, with reflux disease raising the risk of anxiety and depression and

depression raising the risk of reflux.[12,13] Both pathways converge, as Figure 5 depicts, on the anti-reflux barrier and on afferent sensitisation. Critically, the magnitude of these effects is conditional on the state of that barrier. In a person with a competent barrier and normal intra-abdominal pressure — which describes a normal-weight young woman — the same dietary or psychological stimulus produces less reflux than in a person whose barrier is already compromised by central adiposity. This is an effect-measure-modification argument, and it generates a specific, testable prediction: the diet-reflux association should be weaker in normal-weight than in overweight populations, and any future study should therefore report the association stratified by body mass index rather than adjusted for it. The Mendelian randomisation and prospective cohort evidence, in which adiposity and smoking carry robust causal signals while dietary proxies do not,[8,9] is external corroboration of exactly this prediction, and the present study is its empirical complement: with the dominant pathway removed by eligibility, the residual behavioural exposures were too weak to detect at this scale.

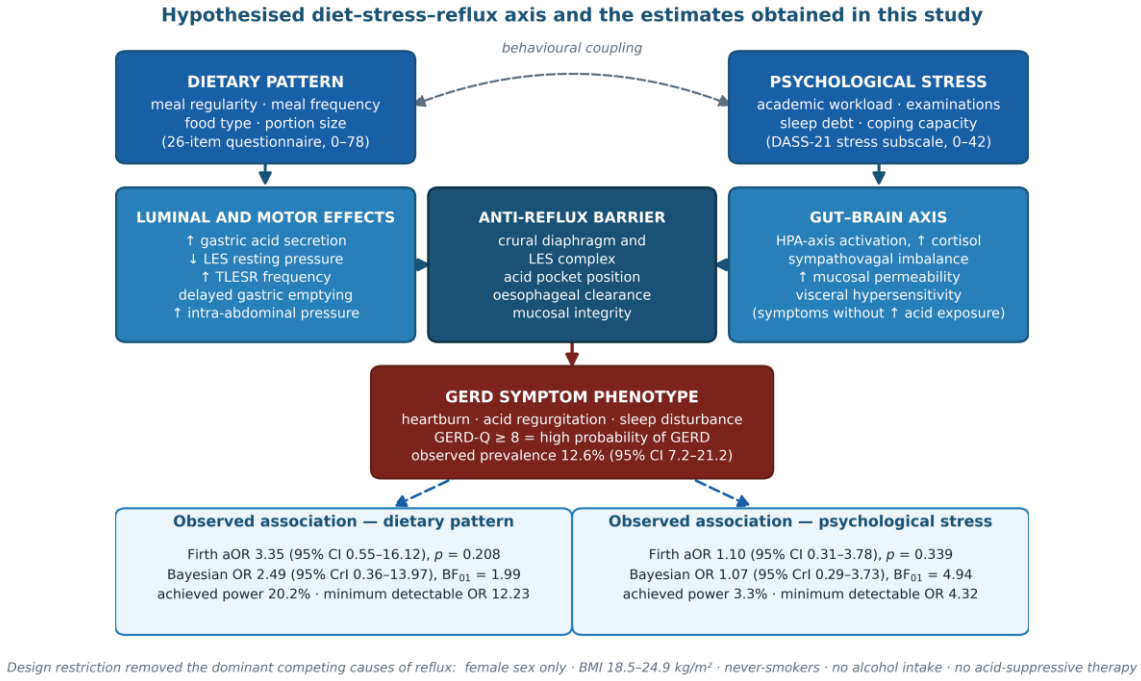

Figure 5. Hypothesised diet-stress-reflux axis annotated with the estimates obtained in this study. Dietary pattern acts principally through luminal and motor mechanisms and psychological stress principally through the gut-brain axis; both converge on the anti-reflux barrier and on the shared final pathway of increased mucosal permeability and afferent sensitisation to produce the GERD symptom phenotype. The lower panels give the adjusted, Bayesian and precision results actually observed, which are reported in full in Table 4. The footnote records that the dominant competing causes of reflux were removed by the eligibility criteria shown in Figure 1 rather than by statistical adjustment, which predicts attenuation of the behavioural effects in this population.

For the Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, three practical implications follow, and they rest on different evidential bases which we distinguish explicitly. The first follows from this study's own prevalence data in Table 2: a reflux symptom prevalence of one in eight among female preclinical students with no classical risk factors justifies routine symptom screening at the student health service. Concretely, we propose administration of the GERD-Q described in Table 1 at the annual student health check, with a score of 8 or more triggering a structured consultation covering meal timing, portion size and nocturnal eating, and with alarm features — dysphagia, odynophagia, weight loss, anaemia, gastrointestinal bleeding — triggering referral, since reflux symptoms demonstrably impair learning concentration and academic performance in this population.[5,20] The second rests on external evidence rather than on our data: management should be phenotype-directed rather than uniform, because in this population a positive GERD-Q is as likely to reflect a hypersensitivity phenotype as acid exposure.[10] The third is a statement about what our data cannot support: these findings provide no evidence that single-exposure health-promotion campaigns targeting diet or stress in isolation would reduce reflux symptoms in this population, but the study is not powered to exclude a moderate benefit, as Figure 4B makes explicit, and should not be cited in support of any resource-allocation decision. Promoting regular eating and effective stress management remains justified on general health grounds, particularly given the clustering of modifiable lifestyle risk documented in Indonesian young adults.[23,39]

Placed in the Indonesian context, this study addresses a specific methodological weakness in the national

literature that the final column of Table 5 makes visible. Indonesian student studies of diet, stress and reflux are numerous, and they contradict one another with a regularity that biology cannot explain.[24-30] The recurring pattern is small samples, few events, no effect sizes, no confidence intervals and no power statement, with conclusions drawn directly from whether a p value fell below 0.05. With 11 events, a study of this size will declare "an association" or "no association" largely as a function of sampling noise. Publishing the minimum detectable effect alongside the null result makes the study interpretable and, more usefully, makes it poolable: a future meta-analysis can weight this estimate properly, which is not possible for a study that reports only a p value. Table 6 converts this into a concrete design specification. Detecting an odds ratio of 2.0 for dietary pattern at a 30% exposure prevalence and a 12% baseline risk requires approximately 574 participants, and an odds ratio of 2.5 requires approximately 306; for stress at a 45% exposure prevalence the corresponding figures in Table 6 are approximately 487 and 260. A coordinated study across six to twelve Indonesian medical faculties, using a common protocol, a common instrument version and a common cut-point, would reach those numbers comfortably within one academic year, and would additionally permit the body-mass-index-stratified analysis that the mechanistic argument above requires. That is a more productive use of national research effort than further single-institution surveys.

Table 6. Sample-size requirements for future studies of this question in Indonesian medical-student populations.

DESIGN SCENARIO	EXPOSURE PREVALENCE	BASELINE GERD RISK	TARGET OR	REQUIRED TOTAL N	FEASIBILITY COMMENT
Dietary pattern, balanced strata	30%	12%	1.5	1,854	Requires a national consortium or registry design
Dietary pattern, balanced strata	30%	12%	2.0	574	Feasible across six to eight Indonesian medical faculties
Dietary pattern, balanced strata	30%	12%	2.5	306	Feasible across three to four faculties
Dietary pattern, balanced strata	30%	12%	3.0	202	Feasible across two to three faculties
Stress, balanced strata	45%	12%	1.5	1,573	Requires a national consortium
Stress, balanced strata	45%	12%	2.0	487	Feasible across five to seven faculties
Stress, balanced strata	45%	12%	2.5	260	Feasible across three faculties
Stress, balanced strata	45%	12%	3.0	171	Feasible as a large two-centre study
Present study, dietary pattern	8.0%	11.3%	3.42 (observed)	459	Would require approximately five times the achieved sample
Present study, achieved design	8.0% / 43.7%	11.3% / 12.2%	12.23 / 4.32	87	Only implausibly large effects were detectable

OR = odds ratio; GERD = gastroesophageal reflux disease. All calculations use the two-proportion arcsine transformation at α = 0.05 (two-sided) with 80% power, at the stated exposure prevalence and baseline risk; an exact-test formulation would give modestly different figures. A study designed to the second row of this table would also be large enough to report the association stratified by body mass index, which the mechanistic argument in the Discussion identifies as the analysis most likely to resolve the current inconsistency in the Indonesian literature. This table is intended to be used as a design specification.

This study has four substantive strengths. The restriction-based design documented in Figure 1 removes the five dominant reflux risk factors by eligibility rather than by adjustment, which is a more reliable form of confounder control than statistical adjustment in a sample of this size, and it converts the research question into one about behavioural effects independent of adiposity. All three measurement instruments summarised in Table 1 are formally validated in Indonesian or in comparable adaptations, with published psychometric indices.[25,32,33,36] The response rate of 86.7% is high for an online survey and the analytic file was complete, so no assumption about missingness was required. Finally, the analytical strategy was matched to the data actually obtained rather than to the data that had been hoped for: exact unconditional tests instead of conditional ones, penalised likelihood instead of ordinary maximum likelihood,[37] Bayesian evidence measures with prior sensitivity instead of a dichotomised p value, quantitative bias analysis instead of a narrative acknowledgement of measurement error, and the explicit precision analysis of Figure 4B instead of a narrative acknowledgement of limited power.

Six limitations must temper interpretation, and for each we state the expected direction of bias. First, the cross-sectional design measures exposure and outcome simultaneously and cannot establish temporal ordering; this is a particular concern for stress, because Mendelian randomisation has established that reflux

disease itself raises the risk of anxiety and depression, so reverse causation would bias the observed association away from the null.[12,13] Second, the study is severely underpowered: 11 events, minimum detectable odds ratios of 12.23 and 4.32, and power of 9% and 22% at an odds ratio of 2.0 mean that only implausibly large effects could have been detected, and the 7-participant poor-dietary-pattern stratum makes the dietary estimate particularly fragile. Third, all three instruments are self-administered and retrospective over a seven-day window. Non-differential misclassification of the outcome biases the odds ratio towards the null, as the bias analysis in Table 4 quantifies; but because exposures and outcome were reported by the same respondent at the same sitting, common-method variance and symptom-driven recall could bias in the opposite direction, so the net direction of measurement bias is genuinely uncertain. Fourth, the outcome is symptom-defined rather than confirmed by endoscopy or pH-impedance monitoring, so hypersensitivity phenotypes cannot be distinguished from acid-driven disease.[10,31] Fifth, several determinants known to influence reflux were not measured — sleep quality, physical activity, anxiety, depression, caffeine and carbonated-beverage intake, and meal-to-sleep interval — and residual confounding from these would most plausibly bias towards the null by adding unexplained outcome variance; the E-value analysis in Table 4 indicates that a confounder would need an association of at least 2.18 with both exposure and outcome to have masked a true odds ratio of 2.0.[15,29] Sixth, analyses used the pre-specified dichotomised instrument scores defined in Table 1, so continuous-score and domain-level dietary analyses — which we now regard as the single most tractable improvement available to the next study — were not performed. A further consequence of the single-centre, female-only, normal-weight design is that these findings should not be extrapolated to men, to students with obesity, or to clinical-phase students whose stress profile differs,[30] and cannot exclude institution-specific effects operating elsewhere. Self-reported height and weight may also have admitted a small number of participants whose measured body mass index fell outside the intended range.

5. Conclusion

Among 87 female preclinical medical students at the Faculty of Medicine, Universitas Pembangunan Nasional "Veteran" Jakarta, the prevalence of symptom-defined gastro-oesophageal reflux disease was 12.6% (95% CI 7.2-21.2), and neither dietary pattern (adjusted odds ratio 3.35; 95% CI 0.55-16.12; p = 0.208) nor psychological stress (adjusted odds ratio 1.10; 95% CI 0.31-3.78; p = 0.339) was associated with it. The two nulls are not evidentially equivalent: across four priors the Bayes factor ranged from 1.78 to 4.90 for stress, making a strong stress effect unlikely, but from 0.99 to 2.05 for dietary pattern, leaving those data uninformative. With minimum detectable odds ratios of 12.23 and 4.32 this study excludes only large effects, and correcting for outcome misclassification raised the dietary odds ratio to 6.61 with an interval too wide to interpret. Reflux symptoms in one in eight students from whom every classical risk factor had been excluded nevertheless justify routine GERD-Q screening at the student health service, with individualised follow-up. Future work should be multicentre and adequately powered — approximately 574 participants would detect an odds ratio of 2.0 — should include men and a wider body-mass range with analyses stratified by body mass index, and should measure sleep, anxiety and meal-to-sleep interval alongside domain-level dietary pattern.

Declarations

Ethical approval

This study was approved by the Research Ethics Committee of Universitas Pembangunan Nasional "Veteran" Jakarta, Indonesia (Ethical Approval No. 41/III/2026/KEP). All study procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained electronically from all participants prior to data collection.

Author contributions

NFY served as the principal investigator and first author. She conceived and designed the study, collected and analysed the data, interpreted the findings, and prepared the initial manuscript. SW and WPJ served as research supervisors, providing guidance throughout the study, methodological input, critical feedback, and manuscript revision. AC served as the examiner, providing critical comments and scientific suggestions that contributed to the interpretation of the findings and improvement of the manuscript. All authors reviewed and approved the final version of the manuscript.

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Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this manuscript.

Data availability

The dataset generated and analysed during the current study and the complete analysis script, including the Firth regression, Metropolis sampler, permutation, bootstrap and probabilistic bias-analysis routines and the fixed random seed, are available from the corresponding author upon reasonable request.

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Generative AI disclosure
The authors declare that no generative artificial intelligence tools were used in the design, analysis, or writing of this manuscript.

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