Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Association Between PHQ-9 (Patient Health Questionnaire-9) Depression Scores and Cardiometabolic Control in Older Primary Care Patients: A Retrospective Cohort Study

Jenna Phreaner

Jenna.phreaner@quinnipiac.edu

Quinnipiac University

Shyam Maisuria

Quinnipiac University

Amber Parson

Quinnipiac University

Ingrid Philibert

Quinnipiac University

Alayna Santarosa

Quinnipiac University

Julianne Sohn

Quinnipiac University

Reynaldo Zamora

Quinnipiac University

Traci Marquis-Eydman

Quinnipiac University

Research Article

Keywords: depression, PHQ-9, triglycerides, older adults, primary care, cardiometabolic health

Posted Date: November 13th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11543460/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

Depression is common among older adults and may affect cardiometabolic health through behavioral and biological pathways. The relationship between routinely collected Patient Health Questionnaire-9 (PHQ-9) scores and cardiometabolic measures in usual primary care is incompletely characterized. The primary objective is to evaluate associations between PHQ-9 scores and body mass index (BMI), blood pressure, hemoglobin A1c (HbA1c), and lipid measures among older adults receiving primary care.

Methods

This is a retrospective electronic health record cohort study including adults aged 65 years or older with at least 1 documented PHQ-9 assessment at affiliated primary care sites between January 1, 2021, and December 31, 2025. PHQ-9 score was modeled continuously. BMI and blood pressure were obtained from the PHQ-9-associated encounter; laboratory values were matched to PHQ-9 assessments within ± 30 days. Linear mixed-effects models accounted for repeated observations and adjusted for age, sex, race, ethnicity, smoking status, and season.

Results

Among 1,089 patients contributing 1,552 PHQ-9 observations, the median PHQ-9 score was 0.0 (interquartile range [IQR], 0.0–2.0); 85.6% of observations reflected none/minimal depressive symptoms. Each 1-point higher PHQ-9 score was associated with 1.64 mg/dL higher triglycerides (95% confidence interval [CI], 0.27 to 3.01; p = 0.019). This association persisted in analyses using log-transformed triglycerides and a ± 90-day laboratory-matching window. In the repeated-measures triglyceride subgroup, both within-patient PHQ-9 deviations and patient mean PHQ-9 scores were associated with higher triglyceride levels. PHQ-9 score was not significantly associated with BMI, systolic or diastolic blood pressure, HbA1c, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, or total cholesterol.

Conclusions

In this older primary care cohort, depressive symptom burden was not broadly associated with cardiometabolic measures but was consistently associated with triglyceride levels. Because the effect size was modest, depressive symptoms were predominantly none/minimal, and multiple outcomes were assessed without multiplicity adjustment. This finding should be considered hypothesis-generating.

Background

Depression is common among older adults and is associated with impaired quality of life, functional decline, and adverse physical health outcomes. In the United States, 14.2% of adults aged 65 years or older report a lifetime diagnosis of depression.1 Identifying depression in this population can be challenging because symptoms may overlap with chronic illness, multimorbidity, and functional impairment.2,3

Primary care is central to depression screening and longitudinal monitoring. The US Preventive Services Task Force recommends screening adults, including older adults, for depression and suicide risk when systems are available to ensure appropriate diagnosis, treatment, and follow-up.4 The Patient Health Questionnaire-9 (PHQ-9) is a brief, validated, and widely used measure of depressive symptom burden.5

Depression commonly coexists with obesity, diabetes, hypertension, and dyslipidemia.3,6–11 These conditions may be linked through behavioral pathways, including reduced physical activity, dietary changes, sleep disruption, and reduced adherence to treatment, as well as biological pathways involving inflammation, hypothalamic-pituitary-adrenal axis activation, and autonomic dysfunction.3,6–11 However, less is known about whether PHQ-9 scores collected during routine primary care visits correspond with concurrently measured cardiometabolic markers among older adults. Because older adults with chronic disease commonly undergo repeated office based and laboratory monitoring, electronic health record data provide an opportunity to evaluate PHQ-9 scores alongside contemporaneous measures of cardiometabolic control.

We examined cross-sectional and longitudinal associations between routinely collected PHQ-9 scores and BMI, blood pressure, HbA1c, and lipid measures in older adults receiving primary care. We hypothesized that greater depressive symptom burden would be associated with poorer concurrent cardiometabolic control.

Methods

Study Design, and Population

We conducted a retrospective cohort study using de-identified electronic health record (EHR) data from Hartford Healthcare-affiliated primary care sites. Eligible observations occurred between January 1, 2021, and December 31, 2025. Patients were eligible if they were aged 65 years or older and had at least 1 documented PHQ-9 assessment. Patients with missing age or sex were excluded. Patients could contribute multiple PHQ-9 observations.

The from Hartford Healthcare Institutional Review Board determined that the study was exempt under Department of Health and Human Services regulations (IRB #[HHC-2026-0005]). Analyses used de-identified data.

Exposure

The primary exposure was PHQ-9 score, modeled continuously (range, 0–27; higher scores indicate greater depressive symptom burden). For descriptive analyses, scores were categorized as none/minimal (0–4), mild (5–9), moderate (10–14), moderately severe (15–19), and severe (20–27).

Prespecified sensitivity analyses used collapsed categories: none/minimal (0–4), mild (5–9), and moderate-to-severe (≥ 10). Exploratory analyses used binary thresholds of PHQ-9 ≥ 5 versus 0–4 and PHQ-9 ≥ 10 versus 0–9.

Cardiometabolic Outcomes

Outcomes were BMI, systolic blood pressure, diastolic blood pressure, HbA1c, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, total cholesterol, and triglycerides. BMI and blood pressure were obtained from the PHQ-9-associated encounter. For laboratory measures, the closest available value within 30 days before or after the PHQ-9 date was matched to each PHQ-9 observation.

For exploratory clinical-threshold analyses, outcomes were dichotomized as BMI ≥ 30 kg/m^2; systolic blood pressure ≥ 140 mmHg; diastolic blood pressure ≥ 90 mmHg; HbA1c ≥ 6.5%; LDL cholesterol ≥ 100 mg/dL; low HDL cholesterol, defined as < 40 mg/dL for men and < 50 mg/dL for women; total cholesterol ≥ 200 mg/dL; and triglycerides ≥ 150 mg/dL.

Covariates

Models adjusted for age, sex, race, ethnicity, smoking status, and season of PHQ-9 assessment. Season was categorized as winter (December–February), spring (March–May), summer (June–August), or fall (September–November). Race, ethnicity, and smoking status were analyzed as documented in the EHR, including unknown, refused, and not-asked categories.

Statistical Analysis

Patient characteristics were summarized using the earliest PHQ-9-associated observation per patient. PHQ-9 scores, covariates, and cardiometabolic measures were summarized at the observation level. Continuous variables are presented as medians with IQRs and categorical variables as frequencies and percentages.

Separate linear mixed-effects models evaluated associations between continuous PHQ-9 score and each cardiometabolic outcome. Models included a random intercept for patient to account for repeated observations and were adjusted for prespecified covariates. Results are reported as adjusted beta coefficients with 95% CIs.

For triglycerides, we conducted sensitivity analyses using log-transformed values and a broader ± 90-day laboratory-matching window. We also performed a repeated-measures analysis among patients with at least 2 PHQ-9 observations and paired triglyceride data. This model included the patient's mean PHQ-9 score and the within-patient deviation from that mean, allowing separation of between-patient and within-patient associations. Log-transformed triglyceride coefficients were back-transformed and expressed as percentage differences.

Additional exploratory analyses evaluated PHQ-9 severity categories and binary PHQ-9 thresholds in linear mixed-effects models. Mixed-effects logistic regression models assessed associations between PHQ-9 threshold groups and clinically meaningful cardiometabolic cutoffs. Missing data was not imputed. Analyses used complete cases for each outcome and model covariates. Statistical tests were 2-sided with $p < 0.05$ considered statistically significant. Because multiple outcomes and exploratory analyses were evaluated without adjustment for multiple comparisons, findings are interpreted descriptively.

Analyses were conducted in R version 4.5.2 using dplyr, gtsummary, lme4, and lmerTest.

Results

Patient Characteristics and Cardiometabolic Profile

The analytic cohort included 1,089 unique patients. Median age was 72.0 years (IQR, 69.0–78.0), and 636 patients (58.4%) were female (Table 1). Patients contributed 1,552 PHQ-9 observations. The median PHQ-9 score was 0.0 (IQR, 0.0–2.0), and 1,328 observations (85.6%) reflected none/minimal depressive symptoms.

Table 1

Characteristic	Overall, N = 1,089
Age, years, median (IQR)	72.0 (69.0–78.0)
Female sex, n (%)	636 (58.4)
Male sex, n (%)	453 (41.6)
White or Caucasian race, n (%)	496 (45.5)
Black or African American race, n (%)	266 (24.4)
Asian race, n (%)	96 (8.8)
Other race, n (%)	213 (19.6)
American Indian or Alaska Native race, n (%)	2 (0.2)
Native Hawaiian or Other Pacific Islander race, n (%)	1 (0.1)
Race refused or unknown, n (%)	15 (1.4)
Hispanic or Latino ethnicity, n (%)	292 (26.8)
Not Hispanic or Latino ethnicity, n (%)	783 (71.9)
Ethnicity refused or unknown, n (%)	14 (1.3)
Note: Characteristics are summarized at the patient level. For patients with repeated PHQ-9 assessments, the earliest PHQ-9-associated observation was used.	

Median BMI was 28.0 kg/m² (IQR, 25.1–32.1), median systolic blood pressure was 130.0 mmHg (IQR, 121.0–138.0), and median diastolic blood pressure was 76.0 mmHg (IQR, 68.0–80.0). Among observations with paired laboratory values, median HbA1c was 6.2% (IQR, 5.8–6.8), median LDL cholesterol was 90.0 mg/dL (IQR, 68.0–115.0), and median triglycerides were 94.0 mg/dL (IQR, 72.0–126.5) (Table 2).

Table 2
PHQ-9 and Temporally Paired Cardiometabolic Measures Across Observations

Measure	N	Value
PHQ-9 score, median (IQR)	1,552	0.0 (0.0–2.0)
None/minimal symptoms, PHQ-9 0–4, n (%)	1,552	1,328 (85.6)
Mild symptoms, PHQ-9 5–9, n (%)	1,552	155 (10.0)
Moderate symptoms, PHQ-9 10–14, n (%)	1,552	41 (2.6)
Moderately severe symptoms, PHQ-9 15–19, n (%)	1,552	17 (1.1)
Severe symptoms, PHQ-9 20–27, n (%)	1,552	11 (0.7)
BMI, kg/m², median (IQR)	1,546	28.0 (25.1–32.1)
Systolic blood pressure, mmHg, median (IQR)	1,543	130.0 (121.0–138.0)
Diastolic blood pressure, mmHg, median (IQR)	1,543	76.0 (68.0–80.0)
HbA1c, %, median (IQR)	577	6.2 (5.8–6.8)
LDL cholesterol, mg/dL, median (IQR)	655	90.0 (68.0–115.0)
HDL cholesterol, mg/dL, median (IQR)	671	56.0 (47.0–67.0)
Total cholesterol, mg/dL, median (IQR)	659	169.0 (141.0–197.0)
Triglycerides, mg/dL, median (IQR)	672	94.0 (72.0–126.5)

Note: Values are summarized at the PHQ-9-observation level. A patient could contribute more than 1 observation. BMI and blood pressure were obtained from the PHQ-9-associated encounter. Laboratory values were matched using the closest available result within 30 days before or after the PHQ-9 date.

Associations Between PHQ-9 Score and Cardiometabolic Measures

In adjusted mixed-effects models, triglycerides were the only cardiometabolic measure significantly associated with PHQ-9 score. Each 1-point higher PHQ-9 score was associated with 1.64 mg/dL higher triglycerides (95% CI, 0.27 to 3.01; p = 0.019). PHQ-9 score was not significantly associated with BMI, systolic or diastolic blood pressure, HbA1c, LDL cholesterol, HDL cholesterol, or total cholesterol (Table 3).

Table 3
Adjusted Associations Between PHQ-9 Score and Cardiometabolic Measures

Outcome	Patients	Observations	Adjusted β per 1-point higher PHQ-9 (95% CI)	p Value
BMI, kg/m²	1,088	1,546	0.022 (− 0.019 to 0.062)	0.289
Systolic blood pressure, mmHg	1,084	1,543	−0.059 (− 0.257 to 0.139)	0.561
Diastolic blood pressure, mmHg	1,084	1,543	0.059 (− 0.068 to 0.186)	0.360
HbA1c, %	470	577	−0.003 (− 0.022 to 0.017)	0.779
LDL cholesterol, mg/dL	550	655	0.035 (− 0.876 to 0.946)	0.940
HDL cholesterol, mg/dL	563	671	−0.227 (− 0.570 to 0.116)	0.195
Total cholesterol, mg/dL	552	659	−0.126 (− 1.186 to 0.934)	0.816
Triglycerides, mg/dL	564	672	1.643 (0.272 to 3.013)	0.019

Note: Linear mixed-effects models included a random intercept for patient and were adjusted for age, sex, race, ethnicity, smoking status, and season of PHQ-9 assessment.

Repeated-Measures and Sensitivity Analyses

Among 399 patients with at least 2 PHQ-9 observations, 262 patients had 370 observations with paired triglyceride values available for the repeated-measures analysis. Each 1-point higher within-patient PHQ-9 deviation was associated with 2.82% higher triglycerides (95% CI, 0.50% to 5.20%; p = 0.018), and each 1-point higher patient mean PHQ-9 score was associated with 1.88% higher triglycerides (95% CI, 0.24% to 3.55%; p = 0.026) (Table 4).

Table 4
Repeated-Measures Analysis of Log-Transformed Triglycerides

PHQ-9 Term	Patients	Observations	Adjusted Difference in Triglycerides (95% CI)	p Value
Within-patient PHQ-9 deviation	262	370	2.82% higher (0.50% to 5.20%)	0.018
Patient mean PHQ-9 score	262	370	1.88% higher (0.24% to 3.55%)	0.026

Note: Restricted to patients with at least 2 PHQ-9 observations and paired triglyceride values. Models included a random intercept for patient and were adjusted for age, sex, race, ethnicity, smoking status, and season. Within-patient PHQ-9 deviation was calculated as visit-specific PHQ-9 score minus the patient's mean PHQ-9 score. Percent differences were calculated as (exp[β] − 1) × 100.

The triglyceride finding persisted after log transformation: each 1-point higher PHQ-9 score was associated with 1.37% higher triglycerides (95% CI, 0.25% to 2.51%; p = 0.016). Results were also unchanged using a ± 90-day laboratory-matching window (β = 1.64 mg/dL; 95% CI, 0.28 to 3.00; p = 0.018).

In category-based analyses, mild symptoms were associated with modestly higher BMI and diastolic blood pressure than none/minimal symptoms. Moderate-to-severe symptoms were associated with higher triglycerides in magnitude, but the estimate did not meet conventional statistical significance. Exploratory threshold analyses showed limited and inconsistent associations; detailed results are provided in the Supplementary Material.

Discussion

In this retrospective cohort of older adults receiving primary care, higher PHQ-9 scores were consistently associated with higher triglyceride levels but were not broadly associated with BMI, blood pressure, HbA1c, LDL cholesterol, HDL cholesterol, or total cholesterol. The triglyceride association persisted after log transformation and when laboratory values were matched within ± 90 days, suggesting that the result was not attributable to triglyceride skewness or the prespecified laboratory-matching interval. In the repeated-measures analysis, both higher-than-usual PHQ-9 scores within individuals and higher average PHQ-9 scores across individuals were associated with higher triglyceride levels.

The observed relationship between depressive symptoms and triglycerides is consistent with prior literature. A recent systematic review and meta-analysis reported higher triglyceride levels among patients with major depressive disorder than among healthy controls.10 Potential mechanisms include reduced physical activity, dietary changes, sleep disturbance, medication nonadherence, inflammation, neuroendocrine activation, and autonomic dysregulation.3,6–10 Our findings extend this literature to a real-world older primary care cohort in which symptom burden was measured through routinely Notably, the association was observed both across the overall cohort and within patients with repeated PHQ-9 assessments, suggesting that higher depressive symptom burden may track with higher triglyceride levels even within the same individual over time. However, the magnitude of the primary association was modest: each 1-point higher PHQ-9 score corresponded to approximately 1.64 mg/dL higher triglycerides. Although statistically significant and consistent across sensitivity analyses, the clinical significance of this Thus, the association should not be interpreted as evidence that an individual PHQ-9 score should trigger metabolic testing or altered lipid management. Rather, the result suggests a potential relationship between depressive symptom burden and triglyceride-related metabolic risk that merits confirmation in studies designed to establish temporal sequence and address confounding.

The absence of significant associations with other cardiometabolic measures is also informative. PHQ-9 scores were highly concentrated in the none/minimal range, limiting the ability to detect differences across a broader symptom spectrum. In addition, blood pressure, HbA1c, BMI, and cholesterol measures reflect chronic disease severity, medication use, adherence, and longitudinal treatment management,

factors not fully captured in this analysis. Routine primary care screening may also identify a different population than studies enriched for major depression or poorly controlled cardiometabolic disease. These features may have reduced the ability to detect broader associations between depressive symptom burden and cardiometabolic control.

The sensitivity analyses provided additional support for the consistency of the triglyceride finding while highlighting the exploratory nature of the category-based results. The association between higher PHQ-9 score and higher triglyceride levels persisted after log-transformation of triglycerides and when the lab-matching window was broadened from ± 30 days to ± 90 days, suggesting the finding was not dependent on triglyceride skewness or the prespecified lab-matching interval. In analyses using collapsed PHQ-9 severity categories, mild depressive symptoms were associated with modestly higher BMI and diastolic blood pressure compared with no/minimal symptoms, while the moderate to severe group demonstrated a directionally higher triglyceride estimate that did not reach conventional statistically significance. These category-based findings should be interpreted cautiously because relatively few observations reflected moder to severe depressive symptoms, and multiple cardiometabolic outcomes were evaluated without adjustment for multiple comparisons. Nevertheless, the direction of the triglyceride association across continuous, log-transformed, severity-category, and expanded lab-window analyses supports the internal consistency of the primary finding.

Our findings suggest that routinely collected PHQ-9 scores may provide additional clinical context when assessing cardiometabolic health in older adults, particularly with respect to triglyceride-related metabolic risk. At the same time, the results do not support a broad claim that higher PHQ-9 scores are associated with worse control across all cardiometabolic markers. Because PHQ-9 scores are already routinely collected in the primary care setting, their potential relationship with metabolic measures may offer an opportunity for more integrated assessment of behavioral, mental, and cardiometabolic health. Of note, our findings do not establish a PHQ-9 threshold for intensified metabolic monitoring, demonstrate that PHQ-9 scores predict subsequent cardiometabolic deterioration, or show that interventions triggered by elevated PHQ-9 scores improve metabolic outcomes.

Our study has several strengths. We used real-world primary care EHR data, linked PHQ-9 assessments with temporally proximate clinical and laboratory measures, and used mixed-effects models to account for repeated observations. The triglyceride association was internally consistent across the primary, log-transformed, expanded-window, and repeated-measures analyses.

This study also has limitations. Its retrospective design precludes causal inference. Depressive symptoms were predominantly none/minimal, and few observations reflected moderate-to-severe symptoms. Laboratory data were available only when obtained during usual care, which may introduce selection bias. Fasting status was unavailable, which is particularly relevant to triglyceride interpretation. Important potential confounders, including depression treatment, medication use and adherence, cardiometabolic disease severity, comorbidity burden, diet, physical activity, alcohol use, and socioeconomic circumstances, were incompletely captured. Multiple outcomes and exploratory

analyses were evaluated without adjustment for multiple comparisons. Finally, our analysis was based on a cohort from a single institution in the Northeast, potentially limiting generalizability to other institutions or regions.

Prospective studies with temporally ordered assessments should evaluate whether changes in depressive symptom burden precede changes in triglycerides or other cardiometabolic measures. Future work should include medication data, fasting status, relevant behavioral factors, and more patients with clinically significant depressive symptoms.

In conclusion, Depressive symptom burden was not broadly associated with cardiometabolic control among older adults in primary care but was consistently associated with triglyceride levels. Given the modest effect size, predominance of none/minimal depressive symptoms, and lack of adjustment for multiple comparisons, this finding should be considered hypothesis-generating. Prospective studies are needed to clarify whether depressive symptom burden can meaningfully inform integrated mental health and cardiometabolic risk assessment in older adults.

Declarations

Declarations

Human Ethics and Consent to Participate declarations

This study was reviewed and approved by the Hartford HealthCare Institutional Review Board (HHC-IRB-2026-0005). The requirement for informed consent was waived by the Institutional Review Board due to the retrospective nature of the study.

Consent for publication

Not applicable

Funding

This research received no external funding.

Author Contribution

JP contributed to study design and methodology, IRB submission and approval, data collection, and manuscript writing and editing. SM, AP, AS, JS, and RZ contributed to study design and methodology and manuscript writing and editing. RZ additionally performed the statistical analysis. TME served as the principal investigator and contributed to study design and methodology, project supervision, and

manuscript writing and editing. IP contributed to manuscript writing and editing. All authors read and approved the final manuscript.

Acknowledgement

The authors thank the clinical and administrative teams at Hartford HealthCare Medical Group Primary Care for their support of this study and acknowledge the patients whose clinical data made this research possible. The authors also thank Emily Shearier, PhD, CCRP, for assistance with electronic health record data extraction.

Availability of data and materials

The datasets generated and/or analyzed during the current study are not publicly available due to patient privacy and confidentiality restrictions associated with electronic health record data but may be available from the corresponding author upon reasonable request and with appropriate institutional approval.

Competing Interests

The authors declare no conflicts of interest.

References

1. Lee B, Wang Y, Carlson SA, Greenlund KJ, Lu H, Liu Y, et al. National, state-level, and county-level prevalence estimates of adults aged ≥ 18 years self-reporting a lifetime diagnosis of depression—United States, 2020. MMWR Morb Mortal Wkly Rep. 2023;72(24):644–50. 10.15585/mmwr.mm7224a1.
2. World Health Organization. Mental health of older adults [Internet]. Geneva: World Health Organization. 2026 Available from: https://www.who.int/news-room/fact-sheets/detail/mental-health-of-older-adults/
3. Penninx BWJH, Milaneschi Y, Lamers F, Vogelzangs N. Understanding the somatic consequences of depression: biological mechanisms and the role of depression symptom profile. BMC Med. 2013;11:129. 10.1186/1741-7015-11-129.
4. US Preventive Services Task Force. Screening for depression and suicide risk in adults: US Preventive Services Task Force recommendation statement. JAMA. 2023;329(23):2057–67.
5. Park SC, Lee HY, Lee DW, Hahn SW, Park SH, Kim YJ, et al. Screening for depressive disorder in elderly patients with chronic physical diseases using the Patient Health Questionnaire-9. Psychiatry Investig. 2017;14(3):306–13. 10.4306/pi.2017.14.3.306.
6. Beverly EA, Gonzalez JS. The interconnected complexity of diabetes and depression. Diabetes Spectr. 2025;38(1):23–31. 10.2337/dsi24-0014.

7. Schmitt A, Bendig E, Baumeister H, Hermanns N, Kulzer B. Associations of depression and diabetes distress with self-management behavior and glycemic control. Health Psychol. 2021;40(2):113–24. 10.1037/hea0001037.
8. Luppino FS, de Wit LM, Bouvy PF, Stijnen T, Cuijpers P, Penninx BWJH, et al. Overweight, obesity, and depression: a systematic review and meta-analysis of longitudinal studies. Arch Gen Psychiatry. 2010;67(3):220–9. 10.1001/archgenpsychiatry.2010.2.
9. Inoue T. Depressive symptoms and the development of hypertension. Hypertens Res. 2024;47(11):3070–2. 10.1038/s41440-024-01856-8.
10. Xu DR, Gao X, Zhao LB, Liu SD, Tang G, Zhou CJ, et al. Association between triglyceride and depression: a systematic review and meta-analysis. PLoS ONE. 2024;19(10). 10.1371/journal.pone.0311625.
11. Frank P, Batty GD, Pentti J, Jokela M, Poole L, Ervasti J, et al. Association between depression and physical conditions requiring hospitalization. JAMA Psychiatry. 2023;80(7):690–9. 10.1001/jamapsychiatry.2023.0777.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Appendix.docx