Cross-Sectional Study of Microalbuminuria and Subclinical Target-Organ Damage in Essential Hypertension

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ABSTRACT
Background: Microalbuminuria (MAU) is a well-established marker of endothelial dysfunction and early renal injury in essential hypertension. It reflects generalized vascular damage and may predict subclinical cardiac, vascular, and retinal involvement even before overt clinical complications arise. **Objectives:** To evaluate the prevalence of microalbuminuria in essential hypertension and its association with subclinical target-organ damage involving the heart, kidneys, vasculature, and retina. **Methods:** A hospital-based cross-sectional study was conducted among **140 adult patients** with essential hypertension. Demographic, clinical, and biochemical data were collected. Microalbuminuria was measured using the urinary albumin-to-creatinine ratio (UACR, 30-300 mg/g). Echocardiography was performed to determine the left ventricular mass index (LVMI), carotid intima-media thickness (CIMT) was assessed by B-mode ultrasonography, and fundoscopic examination was used to grade hypertensive retinopathy. Patients were classified by hypertension severity and blood-pressure control status. Statistical analysis included t-tests, chi-square tests, and Pearson/Spearman correlations; $p < 0.05$ was considered significant. **Results:** Microalbuminuria was present in **43.6%** of hypertensive subjects. Compared to those without MAU, affected patients were older (58.4 ± 9.7 vs 54.9 ± 9.1 years; $p = 0.031$), had higher LVMI (118.7 ± 21.5 vs 102.3 ± 18.9 g/m²; $p < 0.0001$), greater CIMT (0.86 ± 0.13 vs 0.78 ± 0.12 mm; $p = 0.0003$), and lower eGFR (74.1 ± 16.8 vs 82.7 ± 17.2 mL/min/1.73 m²; $p = 0.0035$). The prevalence of LVH, elevated CIMT, and hypertensive retinopathy was also significantly higher in the microalbuminuric group (all $p < 0.01$). UACR correlated positively with LVMI ($r = 0.38$), CIMT ($r = 0.34$), and retinopathy grade ($\rho = 0.29$), and negatively with eGFR ($r = -0.41$). Microalbuminuria prevalence and severity rose steadily with increasing hypertension stage and poor BP control. **Conclusion:** Microalbuminuria is highly prevalent in essential hypertension and closely linked with subclinical multi-organ damage. Its presence identifies patients at higher cardiovascular and renal risk who warrant intensified blood-pressure management and follow-up.
Keywords: Microalbuminuria; Essential Hypertension; Target-Organ Damage.
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INTRODUCTION

Essential hypertension, also known as primary hypertension, is a chronic elevation of blood pressure without an identifiable secondary cause. It affects approximately one-third of the adult population worldwide and is a leading contributor to global morbidity and mortality. Despite advances in pharmacological management, hypertension remains a major risk factor for cardiovascular, cerebrovascular, and renal diseases. The pathophysiology of hypertension involves a complex interplay of genetic, environmental, neurohumoral, and vascular mechanisms that contribute to endothelial dysfunction, increased systemic vascular resistance, and target-organ damage. Early identification of subclinical target-organ damage (TOD) in hypertensive individuals is crucial for effective risk stratification and prevention of long-term complications.[1]

Among the earliest detectable indicators of TOD, microalbuminuria has gained significant attention. It reflects minimal but abnormal urinary albumin excretion (typically 30-300 mg/day) and serves as a sensitive marker of generalized endothelial dysfunction and microvascular injury. The presence of microalbuminuria in hypertensive patients signifies not only renal involvement but also a systemic process affecting the vasculature of the heart and brain. Studies

have consistently demonstrated that hypertensive patients with microalbuminuria are at higher risk for left ventricular hypertrophy (LVH), increased carotid intima-media thickness (CIMT), and hypertensive retinopathy compared to those without microalbuminuria.[2]

Microalbuminuria is now recognized as an independent predictor of cardiovascular morbidity and mortality. Its detection in apparently uncomplicated hypertension indicates early renal damage and heightened cardiovascular risk even before overt proteinuria or structural organ injury develops. The underlying mechanism involves increased glomerular permeability secondary to endothelial dysfunction, inflammation, and oxidative stress. This leakage of albumin parallels similar vascular abnormalities in coronary and cerebral vessels, reflecting systemic vascular damage. The European Society of Hypertension (ESH) and European Society of Cardiology (ESC) guidelines therefore consider microalbuminuria an early marker of hypertensive target-organ damage requiring aggressive therapeutic intervention.[3]

Subclinical target-organ damage encompasses early, asymptomatic functional or structural abnormalities in the heart, kidneys, vasculature, or retina. Such damage typically occurs before the onset of clinical manifestations such as stroke, myocardial infarction, or renal failure. Commonly assessed indices of subclinical TOD include microalbuminuria (renal involvement), left ventricular hypertrophy on echocardiography (cardiac involvement), carotid intima-media thickening on ultrasound (vascular involvement), and fundoscopic evidence of hypertensive retinopathy (ocular involvement). Identification of these markers allows clinicians to re-classify a hypertensive patient's risk profile from "moderate" to "high" and tailor therapy accordingly.[4]

The kidneys are particularly vulnerable to hypertensive injury due to their high perfusion and intricate microvasculature. Chronic elevation of systemic blood pressure causes glomerular capillary hypertension, leading to endothelial damage, mesangial expansion, and progressive glomerulosclerosis. Microalbuminuria thus emerges as a sentinel sign of renal microvascular damage. Simultaneously, hypertensive cardiac remodeling results from sustained pressure overload leading to left ventricular hypertrophy. LVH, in turn, is a well-established risk factor for arrhythmias, heart failure, and sudden cardiac death. Similarly, thickening of the carotid intima-media layer, which can be measured non-invasively by B-mode ultrasonography, reflects early atherosclerosis and correlates with coronary artery disease.[5]

Aim

To evaluate the association between microalbuminuria and subclinical target-organ damage in patients with essential hypertension.

Objectives

1. To determine the prevalence of microalbuminuria among patients with essential hypertension.
2. To assess the correlation between microalbuminuria and subclinical cardiac, renal, and vascular target-organ damage.
3. To analyze the relationship between the severity of hypertension and the degree of microalbuminuria and organ involvement.

MATERIAL AND METHODOLOGY

Source of Data: The study population comprised adult patients diagnosed with essential hypertension attending the outpatient and inpatient departments of the Department of Medicine, a tertiary care teaching hospital.

Study Design: A hospital-based cross-sectional observational study.

Study Location: Department of Medicine, at tertiary care hospital.

Study Duration: The study was conducted over a period of 12 months, from July 24- June 25.

Sample Size: A total of 140 patients with essential hypertension were enrolled based on inclusion and exclusion criteria.

Inclusion Criteria:

1. Patients aged 30-70 years diagnosed with essential hypertension (systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg on at least two separate occasions).
2. Both newly diagnosed and known hypertensive patients on regular follow-up.
3. Patients providing informed written consent.

Exclusion Criteria:

1. Patients with secondary causes of hypertension (e.g., renal artery stenosis, endocrine disorders).
2. Patients with diabetes mellitus, chronic kidney disease (serum creatinine >1.5 mg/dL), or known cardiovascular disease.
3. Patients on nephrotoxic drugs or with urinary tract infections.
4. Pregnant women.

Procedure and Methodology:

All participants underwent a detailed clinical evaluation including demographic data, duration of hypertension, medication history, lifestyle habits, and anthropometric measurements. Blood pressure was measured in the sitting position using a calibrated sphygmomanometer after five minutes of rest, and the

average of three readings was recorded. Morning spot urine samples were collected for estimation of urinary albumin and creatinine. Microalbuminuria was assessed using the urinary albumin-to-creatinine ratio (UACR) measured by an immunoturbidimetric method. Microalbuminuria was defined as UACR between 30 and 300 mg/g. Blood samples were collected for routine investigations including fasting blood glucose, serum creatinine, lipid profile, and electrolytes. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI formula. Cardiac evaluation was performed by 2-dimensional echocardiography, and left ventricular mass index (LVMI) was calculated using the Devereux formula. LVH was defined according to the American Society of Echocardiography criteria (LVMI >115 g/m² in men and >95 g/m² in women). Vascular assessment included measurement of carotid intima-media thickness (CIMT) using high-resolution B-mode ultrasonography. Increased CIMT was defined as values >0.8 mm. Ophthalmologic examination was conducted by indirect ophthalmoscopy for detection and grading of hypertensive retinopathy using the Keith-Wagener-Barker classification.

Sample Processing: Urine samples were centrifuged, and albumin concentration was measured by automated immunoturbidimetric assay using standardized kits. Creatinine was estimated by the Jaffe's kinetic method. All assays were performed in the central biochemistry laboratory following internal and external quality control protocols.

Statistical Methods: Data were entered into Microsoft Excel and analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as proportions or percentages. Comparisons between groups (with and without microalbuminuria) were made using the independent sample *t*-test for continuous variables and the chi-square test for categorical variables. Correlations between microalbuminuria and markers of subclinical organ damage (LVMI, CIMT, retinopathy grade) were evaluated using Pearson's or Spearman's correlation coefficients as appropriate. A *p*-value <0.05 was considered statistically significant.

Data Collection: Data were collected using a pre-designed proforma that included patient identification details, demographic variables, clinical history, blood pressure readings, laboratory findings, echocardiographic data, carotid ultrasonography measurements, and fundoscopic findings. Each participant was assigned a unique study code to ensure confidentiality.

OBSERVATION AND RESULTS

Table 1: Association between microalbuminuria and subclinical target-organ damage (N = 140)

Variable	Microalbuminuria Present (n=61)	Microalbuminuria Absent (n=79)	Test of significance	Effect size (95% CI)	p-value
Age (years), Mean ± SD	58.4 ± 9.7	54.9 ± 9.1	Welch t(≈125)=2.17	Mean diff = 3.5 yrs (0.35 to 6.65)	0.031
LVMI (g/m²), Mean ± SD	118.7 ± 21.5	102.3 ± 18.9	Welch t(≈120)=4.71	Mean diff = 16.4 (9.58 to 23.22)	<0.0001
CIMT (mm), Mean ± SD	0.86 ± 0.13	0.78 ± 0.12	Welch t(≈124)=3.73	Mean diff = 0.080 (0.038 to 0.122)	0.0003
eGFR (mL/min/1.73 m²), Mean ± SD	74.1 ± 16.8	82.7 ± 17.2	Welch t(≈131)=−2.97	Mean diff = −8.6 (−14.27 to −2.93)	0.0035

LVH present, n (%)	29 (47.5%)	18 (22.8%)	$\chi^2(1)=9.46$	RD = +24.7% (+9.2% to +40.2%)	0.0021
Elevated CIMT (>0.8 mm), n (%)	34 (55.7%)	21 (26.6%)	$\chi^2(1)=12.27$	RD = +29.2% (+13.3% to +45.0%)	0.0005
Any hypertensive retinopathy, n (%)	22 (36.1%)	13 (16.5%)	$\chi^2(1)=7.06$	RD = +19.6% (+5.0% to +34.2%)	0.0079

Table 1, Among the 140 patients with essential hypertension, 61 (43.6%) exhibited microalbuminuria. Those with microalbuminuria were significantly older than those without (58.4 ± 9.7 vs 54.9 ± 9.1 years; *p = 0.031*), suggesting that advancing age contributes to vascular endothelial injury and renal microvascular dysfunction. Mean left-ventricular mass index (LVMI) was notably higher in the microalbuminuric group (118.7 ± 21.5 g/m²) compared to the normoalbuminuric group (102.3 ± 18.9 g/m²), a mean difference of 16.4 g/m² (95% CI 9.6-23.2; *p < 0.0001*). Likewise, carotid intima-media thickness (CIMT) was greater among those with microalbuminuria (0.86 ± 0.13 mm vs 0.78 ± 0.12 mm; *p = 0.0003*), indicating early atherosclerotic change. Estimated glomerular filtration rate (eGFR) was significantly lower in the microalbuminuric group (74.1 ± 16.8 mL/min/1.73 m² vs 82.7 ± 17.2 mL/min/1.73 m²; *p = 0.0035*), supporting subclinical renal involvement. The prevalence of left-ventricular hypertrophy (LVH) was approximately twice as high in the microalbuminuric group (47.5%) as in those without microalbuminuria (22.8%) (χ^2 = 9.46, *p = 0.0021*). Similarly, elevated CIMT > 0.8 mm was seen in 55.7% versus 26.6% (χ^2 = 12.27, *p = 0.0005*). Fundoscopic evidence of hypertensive retinopathy occurred in 36.1% of the microalbuminuric group compared with 16.5% in the normoalbuminuric group (χ^2 = 7.06, *p = 0.0079*). These findings collectively indicate that microalbuminuria is strongly associated with early and simultaneous involvement of cardiac, renal, vascular, and retinal target organs, highlighting its value as a sensitive marker of systemic endothelial damage.

Table 2: Prevalence of microalbuminuria and subgroup comparisons (N = 140)

Measure / Subgroup	**Microalbuminuria n/N (%)**	**Test of significance**	**Effect size (95% CI)**	**p-value**
Overall prevalence	**61/140 (43.6%)**	One-sample proportion (Wilson 95% CI)	95% CI = **35.6% to 51.8%**	-
Sex: Male (n=76)	37/76 (48.7%)	$\chi^2(1)=1.77$ (vs Female)	RD (Male-Female) = +11.2% (−5.2% to +27.5%)	0.184
Sex: Female (n=64)	24/64 (37.5%)	-	-	-
Duration <5 years (n=81)	28/81 (34.6%)	$\chi^2(1)=6.34$	RD (<5y − ≥5y) = −21.4% (−37.7% to −5.0%)	0.0118
Duration ≥5 years (n=59)	33/59 (55.9%)	-	-	-
BP controlled (n=58)	17/58 (29.3%)	$\chi^2(1)=8.19$	RD (Controlled − Uncontrolled) = −24.3%	0.0042

			(−40.3 % to −8.4%)	
BP uncontrolled (n=82)	44/82 (53.7%)	-	-	-

Notes: RD = difference in proportions between the two listed categories; 95% CIs are Wald; overall prevalence CI is Wilson.

Table 2, microalbuminuria was detected in 43.6% (95% CI 35.6-51.8) of the hypertensive cohort. Males had a higher, though statistically non-significant, prevalence compared to females (48.7% vs 37.5%; $\chi^2 = 1.77$, $p = 0.184$), indicating no strong sex predilection. Duration of hypertension significantly influenced the presence of microalbuminuria; patients with disease ≥ 5 years showed a higher prevalence (55.9%) compared to those with < 5 years (34.6%) ($\chi^2 = 6.34$, $p = 0.0118$), with an absolute risk difference of 21.4% (95% CI 5.0-37.7). Blood-pressure control status also showed a clear relationship: only 29.3% of controlled hypertensives had microalbuminuria versus 53.7% among uncontrolled individuals ($\chi^2 = 8.19$, $p = 0.0042$). This 24.3% difference (95% CI 8.4-40.3) underscores the protective effect of optimal BP control on renal microvasculature. Hence, the data reaffirm that duration and control of hypertension, rather than sex, are major determinants of microalbuminuria prevalence.

Table 3: Correlation between albuminuria (UACR) and subclinical organ-damage markers (N = 140)

Pair (UACR vs)	**Correlation (r/ρ)**	**95% CI for r (Fisher z)**	**Test statistic**	**p-value**
Left Ventricular Mass Index (LVMI)	r = **0.38**	**0.23 to 0.51**	t(138)=4.83	1.39×10^{-6}
Carotid Intima-Media Thickness (CIMT)	r = **0.34**	**0.18 to 0.48**	t(138)=4.25	2.17×10^{-5}
eGFR	r = **−0.41**	**−0.54 to −0.26**	t(138)=−5.28	1.29×10^{-7}
Retinopathy grade (0-3)*	ρ = **0.29**	**0.13 to 0.43**	t(138)=3.56	3.71×10^{-4}

*Spearman's ρ used for ordinal retinopathy grades; t statistic shown via large-sample approximation for significance.

Table 3, Urinary albumin-to-creatinine ratio (UACR) correlated positively with most indices of target-organ damage. The relationship between UACR and left-ventricular mass index was moderate but highly significant ($r = 0.38$, 95% CI 0.23-0.51; $t(138)=4.83$; $p = 1.39\times10^{-6}$), suggesting that increasing albumin excretion parallels myocardial remodeling. A similar positive correlation existed between UACR and carotid intima-media thickness ($r = 0.34$, 95% CI 0.18-0.48; $p = 2.17\times10^{-5}$), reinforcing that microalbuminuria accompanies early atherosclerotic change. In contrast, a negative correlation was noted with eGFR ($r = -0.41$, 95% CI −0.54 to −0.26; $p = 1.29\times10^{-7}$), signifying declining renal function with rising albuminuria. The correlation between albuminuria and retinopathy grade was weaker but significant (Spearman ρ = 0.29, $p = 3.71\times10^{-4}$), indicating that patients with higher UACR values tended to exhibit more advanced retinal microvascular changes. Collectively, these findings confirm that microalbuminuria behaves as a continuous biomarker of diffuse vascular injury affecting multiple organs simultaneously.

Table 4: Hypertension severity vs degree of microalbuminuria and organ involvement (N = 140)

Severity Group	**n**	**UACR (mg/g) Mean ± SD**	**LVH present n (%)**	**Elevated CIMT n (%)**	**Retinopathy ≥ Grade 1 n (%)**
Stage 1	62	42 ± 18	14 (22.6%)	16 (25.8%)	10 (16.1%)
Stage 2	55	66 ± 34	23 (41.8%)	24 (43.6%)	19 (34.5%)
Stage 3 / Resistant	23	98 ± 41	15 (65.2%)	16 (69.6%)	13 (56.5%)

Table 4, When patients were stratified by hypertension severity, a progressive increase in UACR and prevalence of organ damage was observed. Mean UACR rose from 42 ± 18 mg/g in Stage 1 to 66 ± 34 mg/g in Stage 2 and 98 ± 41 mg/g in Stage 3 or

resistant hypertension. The differences were highly significant (ANOVA $F(2,137)=31.74$; $p < 0.0001$), with a mean gap of 56 mg/g (95% CI 39-73) between Stage 3 and Stage 1 groups. LVH prevalence increased correspondingly from 22.6% to 65.2% ($\chi^2 = 13.91$; $p = 0.0009$), as did elevated CIMT (25.8% → 69.6%; $\chi^2 = 13.88$; $p = 0.0009$) and retinopathy (16.1% → 56.5%; $\chi^2 = 13.93$; $p = 0.0009$).

DISCUSSION

Table 1 (microalbuminuria vs subclinical target-organ damage). In cohort, microalbuminuria (MAU) clustered with multi-bed target-organ damage (TOD). Patients with MAU were older by ~3.5 years (mean diff 3.5; 95% CI 0.35-6.65; p=0.031), and, more importantly, had substantially higher LVMI (mean diff 16.4 g/m²; 95% CI 9.6-23.2; p<0.0001), thicker carotid IMT (mean diff 0.08 mm; 95% CI 0.038-0.122; p=0.0003), and lower eGFR (mean diff −8.6 mL/min/1.73m²; 95% CI −14.27 to −2.93; p=0.0035). The excess prevalence of LVH (+24.7%; 95% CI +9.2% to +40.2%), elevated CIMT (+29.2%; 95% CI +13.3% to +45.0%), and hypertensive retinopathy (+19.6%; 95% CI +5.0% to +34.2%) in MAU mirrors the concept of microalbuminuria as a systemic endothelial injury signal rather than a purely renal phenomenon. These patterns align with Márquez DF et al.(2022)[6] (LIFE) who linked urinary albumin excretion to increased LV mass and adverse LV geometry in hypertensives, independent of brachial BP. Similar concordance is seen for vascular remodeling: Kanna D et al.(2020)[7] reported that hypertensive patients with MAU show higher CIMT and plaque burden than those without MAU, even after adjustment for standard risk factors [6]. The inverse relationship with eGFR is consistent with the early nephropathy spectrum described by Zangana SN.(2025)[8], wherein small increments in albumin excretion herald GFR decline and parallel macrovascular risk.

Table 2 (prevalence and subgroups). The overall MAU prevalence of 43.6% (95% CI 35.6-51.8) is on the higher end of the global hypertensive spectrum (often 15-40%), but comparable to Indian tertiary-care series where higher cardiometabolic risk clustering is common. Duration of hypertension showed a graded relationship: ≥5 years had 55.9% MAU versus 34.6% when <5 years (RD −21.4%; p=0.0118), echoing longitudinal observations that cumulative pressure load and vascular aging drive albumin leakage. Chen Z et al.(2024)[9] Likewise, uncontrolled BP nearly doubled MAU prevalence (53.7% vs 29.3%; RD −24.3%; p=0.0042), reinforcing guideline statements (ESH/ESC 2023) that BP control is the most modifiable determinant of albuminuria regression and risk downgrading. The absence of a significant sex difference is typical of several hospital cohorts once BP and metabolic covariates are accounted for. Paz Landim M et al.(2021)[10]

Table 3 (continuous UACR correlations). Treating UACR as a continuum strengthened mechanistic inference. UACR correlated moderately with LVMI (r=0.38) and CIMT (r=0.34) and inversely with eGFR (r=−0.41), all with high statistical certainty ($p \leq 2\times10^{-5}$). These magnitudes are remarkably close to pooled effects in prior work: Liao Y et al.(2022)[11] summarized correlations between albuminuria and LV mass/arterial structure in the 0.25-0.45 range in essential hypertension, independent of clinic BP. The monotonic association with retinal grade (ρ=0.29; p<0.001) fits Vasan RS et al.(2022)[12], who reported higher odds of retinopathy per increasing albumin excretion stratum. Mechanistically, these cross-organ signals reflect shared pathways-RAAS activation, oxidative stress, and endothelial glycocalyx injury-linking glomerular permeability to concentric LV remodeling and intima-media hypertrophy.

Table 4 (severity staging, dose-response). A clear dose-response across hypertension stages for UACR (Stage 1: 42 ± 18; Stage 2: 66 ± 34; Stage 3/resistant: 98 ± 41 mg/g; ANOVA p<0.0001) and for organ damage (LVH 22.6%→65.2%; elevated CIMT 25.8%→69.6%; retinopathy 16.1%→56.5%; all p=0.0009). This staircase pattern matches the gradient reported in LIFE and other cohorts, where albuminuria tracks with ambulatory BP load, LV mass, and carotid atherosclerosis severity Bakhtar V et al.(2020)[13]. Importantly, contemporary guidelines formalize MAU as subclinical TOD that upgrades total cardiovascular risk even at modest BP elevations, thereby tightening treatment targets and supporting RAAS-blockade-centric strategies. In Indian datasets, Agarwal et al. similarly showed higher LVMI and worse diastolic function among hypertensives with MAU, underscoring regional generalizability Monzo L et al.(2021)[14].

CONCLUSION

In this cross-sectional study of 140 patients with essential hypertension, microalbuminuria was observed in nearly 44% of participants and was strongly associated with early indicators of cardiac, vascular, renal, and retinal target-organ damage. Patients with microalbuminuria exhibited significantly higher left ventricular mass index, greater carotid intima-media thickness, and a higher prevalence of left ventricular hypertrophy, retinopathy, and reduced glomerular filtration rate compared to those without albuminuria. The prevalence and magnitude of microalbuminuria increased proportionally with the duration and severity of hypertension and with poor blood-pressure control, reflecting cumulative

endothelial injury and systemic vascular dysfunction. These findings reinforce that microalbuminuria is not only an early marker of renal impairment but also a sensitive and inexpensive surrogate for generalized hypertensive organ damage. Routine screening for microalbuminuria in hypertensive patients can therefore serve as an important prognostic and therapeutic guide, enabling early risk stratification and timely institution of reno- and cardioprotective interventions.

LIMITATIONS

1. The study employed a cross-sectional design, which limits the ability to establish temporal or causal relationships between microalbuminuria and subsequent target-organ damage.
2. Being hospital-based, the study may over-represent patients with more advanced or poorly controlled hypertension, potentially limiting generalizability to the community.
3. Single spot urine samples were used for albumin estimation rather than serial 24-hcollections, which might introduce measurement variability due to transient albuminuria.
4. Confounding factors such as dietary sodium intake, nocturnal BP variation, and medication adherence were not fully controlled.
5. Echocardiographic and ultrasonographic assessments were operator-dependent, which could lead to minor inter-observer variations despite standardization.
6. Biomarkers of endothelial dysfunction and inflammatory mediators were not included, which might have provided mechanistic insights into the association observed.

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