

Weekly Moderate Physical Activity and Blood Pressure Among Adults: A Cross-Sectional Analysis of Age, Sex, BMI, and Smoking

Abstract

This study examines the association between weekly minutes of moderate-intensity physical activity and blood pressure among 350 adults aged 25–74 years, adjusting for age, sex, body mass index (BMI), and smoking status. Because only 2 of 350 respondents (0.6%) were classified as hypertensive, the primary outcome is continuous systolic blood pressure (SBP; $n = 343$ with complete data), estimated with ordinary least squares and HC3 heteroskedasticity-robust standard errors; hypertension status is analysed as a secondary, exploratory outcome with Firth penalised logistic regression. Each additional standard deviation of moderate activity (about 52.6 minutes per week) was associated with 2.95 mmHg lower SBP (SE = 0.40; 95% CI -3.74 to -2.16 ; $p < .001$) in the fully adjusted model ($R^2 = .561$). The association was larger (4.09 mmHg) when BMI was omitted, consistent with partial overlap between activity and adiposity, and was unchanged by excluding outliers. For hypertension status, the odds ratio per SD of activity was 0.54 (95% CI 0.15–1.96) but rests on two cases and is inconclusive. Cross-sectional data cannot establish causal direction.

Keywords: physical activity; systolic blood pressure; hypertension; BMI; smoking; cross-sectional analysis; robust standard errors

1 Introduction

Elevated blood pressure is one of the most prevalent and consequential modifiable cardiovascular risk factors. Global estimates indicate that hypertension affects more than a billion adults [Mills et al. 2020; Poulter et al. 2015]. Systolic blood pressure (SBP), in particular, has been shown to carry a stronger and more consistent relationship with cardiovascular events and mortality than diastolic pressure, especially from middle age

onward [Kannel 2000; Port et al. 2000; Izzo et al. 2000]. Because pharmacological treatment is complicated by heterogeneous responses that depend on age, sex, and other patient characteristics [te Riet et al. 2015], lifestyle behaviours are recommended as a first-line strategy for prevention and control, and regular physical activity is among the most prominent of these [Charchar et al. 2023].

Public health guidelines typically recommend at least 150 minutes per week of moderate-to-vigorous physical activity, yet recent reviews note that this threshold may be an unnecessary barrier because health gains accrue below it as well [Warburton and Bredin 2017]. Moderate-intensity activity is of particular interest because it is the intensity most people can sustain, and observational evidence links it to lower risks of type 2 diabetes and mortality independently of vigorous activity [Jeon et al. 2007; Shiroma et al. 2014; Lee and Paffenbarger 2000]. Experimental syntheses likewise report that aerobic exercise lowers resting blood pressure [Pescatello et al. 2004]. What is less clear from cross-sectional population data is how strongly *minutes* of moderate activity track blood pressure once the main demographic and behavioural determinants of blood pressure—age, sex, adiposity, and smoking—are accounted for. Age is the dominant correlate of arterial stiffening and systolic pressure [Buford 2016]; excess weight raises blood pressure and clusters with inactivity [Chiang et al. 1969]; and smoking raises ambulatory pressure, although this is often masked in office measurements [Mann 1991].

A further complication concerns how blood pressure status is operationalised. Hypertension as a binary classification discards information and, in samples where it is rare, supports little inference. In our data only two of 350 respondents are classified as hypertensive, so we treat continuous SBP as the primary outcome and examine binary hypertension status as an explicitly secondary and exploratory analysis using penalised likelihood [Firth 1993].

This paper makes three contributions. First, it provides an adjusted estimate of the association between weekly moderate activity minutes and SBP expressed in clinically interpretable units. Second, it documents how much of that association is carried through or shared with BMI, by comparing models with and without BMI and by reporting an exploratory, bootstrap-based indirect effect. Third, it is transparent about the limits of inference for the binary hypertension outcome when events are extremely sparse, which is a common but seldom reported problem in applied cross-sectional work.

2 Theoretical Background and Hypotheses

2.1 Physical activity and systolic blood pressure

Exercise physiology provides several plausible pathways from physical activity to lower blood pressure: improved endothelial function and nitric-oxide-mediated vasodilation, lower sympathetic tone and peripheral vascular resistance, and reduced adiposity [Pescatello et al. 2004; Warburton and Bredin 2017]. In the cardiovascular risk-factor framework that grew out of the Framingham tradition, blood pressure is a graded, continuous risk factor in which small differences in SBP translate into meaningful differences in cardiovascular risk [Kannel 2000; Port et al. 2000]. Consequently, a behavioural exposure that shifts the SBP distribution by a few mmHg is relevant even if it does not change the proportion classified as hypertensive. Lifestyle guidance consistently places physical activity among the behaviours expected to lower blood pressure in adults [Charchar et al. 2023]. Within a cross-sectional design, the corresponding testable implication is that adults who report more weekly moderate activity should have lower SBP than otherwise similar adults who report less, net of age, sex, BMI and smoking. We therefore expect a negative conditional association.

H1: Among adults, more weekly minutes of moderate physical activity are associated with lower systolic blood pressure, after adjusting for age, sex, BMI, and smoking status.

2.2 Physical activity and hypertension status

The same mechanisms imply that the probability of being classified as hypertensive should fall with activity. The clinical definition of hypertension is a threshold on SBP and diastolic blood pressure [Charchar et al. 2023], so any shift in the SBP distribution should reduce the proportion above the threshold. However, a threshold-based outcome is informative only if a reasonable number of respondents cross it; in a sample with very few hypertensive respondents, the statistical power to detect even a sizeable association is minimal. We therefore state the hypothesis in the same direction but flag it as secondary and exploratory, to be assessed with penalised (Firth) logistic regression, which reduces small-sample bias and handles separation [Firth 1993].

H2: Among adults, more weekly minutes of moderate physical activity are associated with lower odds of hypertension, after adjusting for age, sex, BMI, and smoking status (secondary, exploratory).

2.3 Covariates and the role of BMI

Age, sex, BMI, and smoking are included because they are established determinants of blood pressure that are also related to activity levels [Buford 2016; Chiang et al. 1969; Mann 1991]. BMI warrants special care: adiposity is a confounder if heavier individuals are less active and also have higher pressure, but it is also a plausible mediator if activity lowers BMI which in turn lowers pressure. Adjusting for BMI therefore targets a direct association, whereas omitting it yields a total association. We estimate both and describe the difference. Because the study was not designed as a mediation analysis, the BMI pathway is reported only as an exploratory supplement.

3 Data and Method

3.1 Sample and data source

The data are a respondent-level cross-section of 350 adults aged 25 to 74 years (mean 47.9, SD 14.2). No respondent was removed during cleaning: there were no all-missing rows, no duplicates, and no implausible values. Systolic BP is missing for 7 respondents (2.0%); these were not imputed and are excluded listwise from models that use SBP, giving an analytic sample of $n = 343$ for the primary analysis. Four rows (1.1%) were flagged as outliers using a $|z| > 3.5$ or Tukey $1.5 \times \text{IQR}$ rule—two for activity minutes (rows 171 and 174) and two for SBP (rows 222 and 275)—and retained in the main analysis; a sensitivity analysis excludes them. Sex is evenly split (175 men, 175 women), 34 respondents (9.7%) are current smokers, 104 (29.7%) are former smokers, and the rest are never smokers.

3.2 Measures

Weekly moderate physical activity. The focal predictor is weekly minutes of moderate-intensity physical activity (mean 261.4, SD 52.6, range 128–435). Activity is probably self-reported. For estimation it is standardised to a z-score, so coefficients represent the change in the outcome per one SD (52.6 minutes) of weekly activity.

Systolic blood pressure. The primary outcome is SBP in mmHg (mean 107.0, SD 10.5, range 95–145; $n = 343$). The distribution is mildly right-skewed (skewness 0.81) and the mean is low relative to general adult populations, which should be borne in mind when generalising.

Hypertension status. The secondary outcome is a binary indicator of hypertension. Only 2 respondents (0.6%) are classified as hypertensive; both have SBP of 141–145

mmHg. Because SBP is a component of the classification, it is not used as a covariate in hypertension models.

Controls. Age (years) and BMI (kg/m²; mean 26.9, SD 4.5, range 18.0–38.0) are standardised. Sex is a dummy variable (male = 1; reference female). Smoking is represented by two dummies, current and former smoker, with never-smoker as reference.

3.3 Model specification

The primary model for respondent i is

$$SBP_i = \beta_0 + \beta_1 PA_i + \beta_2 Age_i + \beta_3 BMI_i + \beta_4 Male_i + \beta_5 Current_i + \beta_6 Former_i + \varepsilon_i, \quad (1)$$

where PA, Age and BMI are standardised and H1 predicts $\beta_1 < 0$. The secondary model for hypertension status HTN _{i} is

$$\begin{aligned} \text{logit Pr}(\text{HTN}_i = 1) = & \gamma_0 + \gamma_1 PA_i + \gamma_2 Age_i + \gamma_3 BMI_i \\ & + \gamma_4 Male_i + \gamma_5 Current_i + \gamma_6 Former_i, \end{aligned} \quad (2)$$

with H2 predicting $\gamma_1 < 0$. Models are built stepwise: Model 1 contains the controls other than BMI (age, sex, smoking), Model 2 adds weekly activity, and Model 3 adds BMI. The ordering allows the contribution of activity to be read before and after BMI adjustment.

3.4 Estimation strategy

Equation (1) is estimated by OLS. A Breusch–Pagan test rejected homoskedasticity ($p < .001$), so all inference uses HC3 heteroskedasticity-robust standard errors [Long and Ervin 2000]. Residuals do not depart detectably from normality (residual normality test $p = .331$). Multicollinearity is assessed through variance inflation factors (VIF). The sample has no clustering structure, so cluster-robust errors are not needed, and no instruments are available; the analysis is therefore associational. The indirect effect of activity on SBP through BMI is estimated as the product of the activity→BMI and BMI→SBP paths with a 1,000-draw percentile bootstrap, and is explicitly exploratory. Equation (2) is estimated by Firth penalised-likelihood logistic regression, with inference from penalised likelihood-ratio tests and Wald intervals; with two events and six predictors (events per variable = 0.33) these results are descriptive only. Sensitivity analyses (i) exclude the four flagged rows, (ii) omit BMI, (iii) drop all controls, and (iv) add post-hoc activity×covariate interactions. The post-hoc interactions are not adjusted for multiplicity.

4 Empirical Results

4.1 Descriptive statistics and correlations

Table 1 reports descriptive statistics. Weekly moderate activity averages 261 minutes, well above the 150-minute guideline, and its minimum (128 minutes) is only slightly below it. The three continuous covariates show moderate dispersion and little skew. Table 2 reports pairwise Pearson correlations. SBP is negatively correlated with activity ($r = -.52$, $p < .001$) and positively with age ($r = .59$) and BMI ($r = .35$). Activity is negatively correlated with both age ($r = -.25$) and BMI ($r = -.36$), which is the pattern that makes adjustment necessary, whereas BMI and age are essentially uncorrelated ($r = -.05$). Correlations involving the binary hypertension indicator are unstable because it has only two positive cases, and should not be over-read.

Table 1 Descriptive statistics

Variable	<i>N</i>	Mean	SD	Min	Median	Max
Systolic BP (mmHg)	343	107.04	10.46	95	106.0	145
Weekly moderate PA (min)	350	261.43	52.56	128	265.0	435
Age (years)	350	47.93	14.16	25	47.0	74
BMI (kg/m ²)	350	26.94	4.47	18	26.4	38
Hypertension (0/1)	350	2 (0.6%)		0	–	1
Male sex	350	175 (50.0%)		0	–	1
Current smoker	350	34 (9.7%)		0	–	1
Former smoker	350	104 (29.7%)		0	–	1

Note. For binary variables the count and percentage of respondents coded 1 are shown. Systolic BP is missing for 7 of 350 respondents.

Table 2 Correlation matrix (pairwise Pearson r)

	1	2	3	4	5	6	7
1. Systolic BP	–						
2. Weekly moderate PA	-0.52***	–					
3. Age	0.59***	-0.25***	–				
4. BMI	0.35***	-0.36***	-0.05	–			
5. Male sex	0.00	0.05	-0.03	-0.02	–		
6. Current smoker	0.10	-0.03	0.08	0.08	0.06	–	
7. Former smoker	-0.17**	0.10	-0.09	-0.07	-0.06	-0.21***	–
8. Hypertension	0.26***	-0.09	0.12*	0.05	0.00	0.10	-0.05

Note. Pairwise deletion; $n = 343$ for correlations with SBP and $n = 350$ otherwise. * $p < .05$, ** $p < .01$, *** $p < .001$. Row 8 (hypertension) is included for completeness; with two cases its correlations are unstable.

4.2 Main regression

Table 3 presents the stepwise models. Model 1 includes age, sex and smoking; Model 2 adds weekly moderate activity; Model 3 adds BMI. In Model 2, one SD more weekly activity is associated with 4.09 mmHg lower SBP (HC3 SE = 0.40, $p < .001$), and R^2 rises from .359 to .501. In the full model (Model 3) the activity coefficient is -2.95 mmHg per SD (SE = 0.40; 95% CI -3.74 to -2.16 ; $p < .001$; standardised $\beta = -0.28$). Age ($B = 5.46$, SE = 0.44) and BMI ($B = 2.81$, SE = 0.44) are positively associated with SBP, both $p < .001$. Sex and smoking show no reliable association in the full model: male sex $B = 0.48$ (SE = 0.77, $p = .534$), current smoking $B = 0.62$ (SE = 1.41, $p = .661$), former smoking $B = -1.42$ (SE = 0.87, $p = .102$). The former-smoker coefficient is significant in Model 1 ($B = -2.44$, SE = 0.98, $p < .05$) but loses significance once activity and BMI are included, which is consistent with former smokers being somewhat more active (Table 2, $r = .10$). Hypothesis H1 is supported. The full model explains 56.1% of the variance (adjusted $R^2 = .553$; $F(6, 336) = 64.71$, $p < .001$). Activity adds $\Delta R^2 = .063$ beyond the other covariates (Cohen's $f^2 = 0.14$, a medium effect), and a post-hoc power calculation indicates that the sample can detect an f^2 of about 0.023 with 80% power. Figure 1 plots the Model 3 coefficients with 95% confidence intervals.

All VIFs are well below 5 (maximum 1.27 in Model 3; Table 3), so multicollinearity is not a concern, even though activity correlates $-.36$ with BMI.

Table 3 Stepwise OLS regressions of systolic blood pressure (mmHg)

	Model 1 Controls only	Model 2 + Activity	Model 3 + BMI (full)
Weekly moderate PA (per SD)	–	-4.09*** (0.40)	-2.95*** (0.40)
Age (per SD)	5.98*** (0.48)	5.00*** (0.45)	5.46*** (0.44)
BMI (per SD)	–	–	2.81*** (0.44)
Male sex	0.07 (0.92)	0.44 (0.81)	0.48 (0.77)
Current smoker	1.20 (1.75)	1.29 (1.48)	0.62 (1.41)
Former smoker	-2.44* (0.98)	-1.63 (0.90)	-1.42 (0.87)
Intercept	107.60*** (0.72)	107.16*** (0.61)	107.10*** (0.54)
<i>N</i>	343	343	343
<i>R</i> ²	0.359	0.501	0.561
Adjusted <i>R</i> ²	0.351	0.493	0.553
Robust <i>F</i>	43.57	65.13	64.71
<i>p</i> (model <i>F</i>)	<.001	<.001	<.001
Max VIF	1.06	1.08	1.27
VIF, weekly PA	–	1.08	1.27
VIF, BMI	–	–	1.20

Note. Unstandardised coefficients with HC3 heteroskedasticity-robust standard errors in parentheses. Weekly activity, age and BMI are z-scores (coefficients per 1 SD). Reference categories: female, never smoker. Complete cases, $n = 343$. * $p < .05$, ** $p < .01$, *** $p < .001$. *F* statistics are based on the robust covariance matrix. VIF values are from the corresponding design matrices.

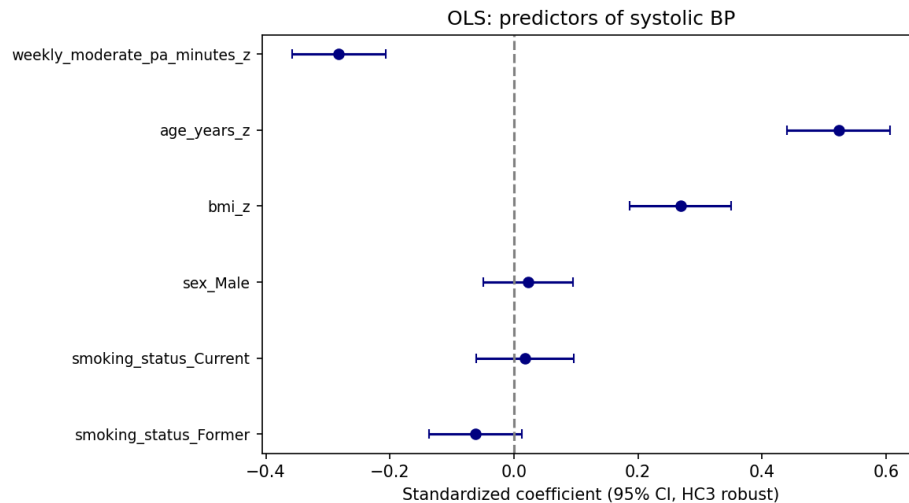


Figure 1 Regression coefficients (full model, Model 3) with 95% confidence intervals based on HC3 standard errors.

Secondary outcome: hypertension status

With two events, the Firth-penalised logistic model (Equation 2) yields an odds ratio of 0.54 per SD of activity (Wald 95% CI 0.15–1.96; penalised likelihood-ratio $p = .240$). The unadjusted estimate is 0.31 (95% CI 0.08–1.17; $p = .059$). Neither is statistically distinguishable from no association, and the intervals span more than an order of magnitude. Both cases have below-median weekly activity (201 and 200 minutes, about the 12th percentile of non-cases; non-case median 266), and they are aged 67 and 72, but Fisher’s exact test of hypertension by below/above-median activity gives $p = .499$ and a Mann–Whitney test of activity by hypertension gives $p = .066$. With events per variable of 0.33, H2 is neither supported nor refuted: the data are uninformative about it. The only covariate with a penalised-model Wald $p < .05$ is age (OR = 5.72 per SD, 95% CI 1.08–30.23), which should be treated with the same caution.

4.3 Mediation results

Mediation was not part of the research design, and BMI was entered as a covariate. Because BMI is theoretically both a confounder and a mediator of activity, we nevertheless report one exploratory decomposition. Without adjustment for age, sex, or smoking, the total association of activity with SBP is $c = -5.43$ mmHg per SD. Activity is associated with $a = -0.37$ SD lower BMI, and BMI is associated with $b = 1.92$ mmHg higher SBP per SD, controlling for activity. The indirect effect is $a \times b = -0.71$ mmHg (1,000-draw percentile bootstrap 95% CI -1.12 to -0.32), which excludes zero, with a direct effect $c' = -4.72$. The indirect path therefore represents roughly 13% of the total unadjusted association. This analysis was unplanned and unadjusted for the other covariates, and

the temporal ordering required for mediation cannot be established from cross-sectional data. A second, adjusted indication is the shrinkage of the activity coefficient from -4.09 (Model 2) to -2.95 (Model 3) when BMI is added.

4.4 Moderation results

No moderation was hypothesised, and the design file lists no moderators; accordingly, no simple-slopes plot is presented. As a post-hoc check we tested activity interactions with age, sex, and BMI. The activity $\times$ age interaction was -0.80 mmHg ($p = .045$; $\Delta R^2 = .005$), the activity $\times$ sex interaction was 0.63 ($p = .378$), and the activity $\times$ BMI interaction was -0.07 ($p = .836$). With three uncorrected tests, the age interaction would not survive a Bonferroni adjustment ($.045 \times 3 = .135$) and its incremental variance is only 0.5%; we treat it as a hypothesis for future work and not as a finding.

5 Robustness Checks

Table 4 compares the activity coefficient across specifications. Excluding the four flagged rows leaves the estimate virtually unchanged (-2.89 versus -2.95 mmHg per SD; $p < .001$). Omitting BMI increases the magnitude to -4.09 , and omitting all covariates increases it to -5.43 ; the pattern is as expected if age and BMI are both correlated with activity and with SBP. In every specification the 95% interval excludes zero. The binary-outcome estimates are included in the lower panel only for completeness and are not comparable in scale with the OLS rows (they are odds ratios).

Table 4 Robustness of the weekly moderate activity coefficient

Specification	<i>N</i>	<i>B</i> / OR	SE	95% CI	<i>p</i>	<i>R</i> ²
<i>Outcome: systolic BP (mmHg), OLS with HC3 SE</i>						
(a) Main model (complete cases)	343	-2.95	0.40	[-3.74, -2.16]	<.001	0.561
(b) Excluding 4 flagged rows	339	-2.89	0.41	[-3.70, -2.07]	<.001	0.554
(c) BMI omitted (total association)	343	-4.09	0.40	[-4.88, -3.31]	<.001	0.501
(d) Excluding flagged rows, BMI omitted	339	-4.01	0.41	[-4.82, -3.20]	<.001	0.492
(e) Unadjusted	343	-5.43	0.46	[-6.33, -4.54]	<.001	0.269
<i>Outcome: hypertension, odds ratio per SD of activity</i>						
(f) Firth logistic, adjusted	350	0.54	0.66 [†]	[0.15, 1.96]	.240 [‡]	–
(g) Firth logistic, unadjusted	350	0.31	0.68 [†]	[0.08, 1.17]	.059 [‡]	–

Note. Coefficients are per 1 SD of weekly moderate activity. Rows (a)–(d) include age, sex and smoking; (a)–(b) also include BMI; (e) has no covariates. Flagged rows: 171, 174, 222, 275. [†]Standard error on the log-odds scale. [‡]Penalised likelihood-ratio *p*-value; based on two events, so inference is unreliable.

6 Discussion

6.1 Theoretical implications

The results are consistent with the exercise-physiology account in which more moderate activity goes together with lower resting SBP [Pescatello et al. 2004; Warburton and Bredin 2017]. The adjusted gap of about 3 mmHg per SD (roughly 52 minutes) is of a magnitude that is meaningful in the graded-risk perspective on SBP, in which small population-wide shifts are associated with large differences in cardiovascular outcomes [Kannel 2000; Port et al. 2000]. The comparison of Models 2 and 3 shows that about 28% of the BMI-unadjusted coefficient is absorbed by BMI, in line with the idea that adiposity is part of the pathway, and potentially part of the confounding structure, linking inactivity and blood pressure [Chiang et al. 1969]. The coefficient on age is the largest in standardised terms, a reminder that age remains the dominant determinant of SBP [Buford 2016]. The lack of an association for smoking is not evidence of no effect: office-type measurements are known to understate the pressor effect of smoking [Mann 1991], and the sample contains only 34 current smokers.

For the binary outcome, the analysis shows how rare events limit inference. That both hypertensive respondents fall in the lowest eighth of the activity distribution among non-cases is suggestive, and the odds ratio below 1 points in the expected direction, but two cases cannot distinguish a real association from chance. This is a reason to prefer continuous outcomes when hypertension is rare, and to report the sparsity openly.

6.2 Managerial and practical implications

For clinicians and public-health programme managers, the findings support the use of weekly moderate activity minutes as an easily communicated, trackable target in lifestyle programmes [Charchar et al. 2023]. Roughly an hour more moderate activity per week is associated with a 3-mmHg lower SBP in comparable adults, and the association appears across the whole observed range (128–435 minutes), not only below the 150-minute guideline, consistent with the view that the guideline should not be treated as a hard threshold [Warburton and Bredin 2017]. Programmes aimed at both activity and weight are likely to be complementary rather than redundant, given that activity retains an association after BMI is held constant. Targeting older adults, whose SBP is highest, is likely to produce the largest absolute benefits. We caution that these implications rely on an association and not on an intervention effect.

7 Conclusions

In this cross-sectional sample of 350 adults, weekly minutes of moderate physical activity were negatively and robustly associated with systolic blood pressure after adjusting for age, sex, BMI, and smoking (2.95 mmHg lower per SD; 95% CI 2.16–3.74), and the finding was stable to the exclusion of outliers and to alternative specifications. A substantial but incomplete share of the unadjusted association overlaps with BMI. Because only two respondents were hypertensive, the association with hypertension status could not be assessed with any confidence. The evidence supports H1 and is inconclusive for H2.

8 Limitations and Future Research

Several limitations apply. First, the design is cross-sectional, so reverse causality is possible: people with high blood pressure may change their activity, and the direction of association cannot be determined. Second, unmeasured confounders—diet and sodium, alcohol, family history, socioeconomic status, stress, sleep, and antihypertensive medication—may influence both activity and blood pressure. Third, activity is probably self-reported and subject to recall and social-desirability bias, which could attenuate or distort the estimate. Fourth, BMI is a potential mediator as well as a confounder; the model with BMI estimates a direct association and the model without it a total one, and the bootstrap mediation analysis was unplanned, did not adjust for other covariates, and is not causal. Fifth, the hypertension outcome has two cases, so H2 cannot be evaluated; systolic BP is itself part of the case definition. Sixth, seven SBP values are missing and were handled by complete-case analysis, which assumes data are missing at random. Seventh, the sample has a low average SBP (107 mmHg) and a narrow upper range, with no one above 145 mmHg, which limits generalisation to hypertensive populations, and the sampling frame is not described. Eighth, the post-hoc interaction tests were not corrected for multiplicity. Future work should use longitudinal or experimental designs, device-measured activity (accelerometry), samples enriched for hypertension, and a pre-specified mediation framework with temporal ordering.

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