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ORIGINAL ARTICLE

Blood Pressure, EEG Neural Markers, and Cognitive Performance in Young and Older Adults: A 24-Month Prospective Longitudinal Cohort Study

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Abstract

Background: Elevated blood pressure is an established modifiable risk factor for cognitive decline, yet longitudinal evidence simultaneously integrating blood pressure indices, electroencephalographic neural markers, and multidomain cognitive outcomes across age groups remains limited.

Objectives: This study investigated whether blood pressure level and variability predict cognitive performance and EEG markers over 24 months in young and older adults, and whether EEG markers mediate the blood pressure–cognition relationship.

Methods: A prospective longitudinal cohort study enrolled 100 participants — 50 young adults (18–35 years) and 50 older adults (60–75 years) — assessed at baseline, 12 months, and 24 months. Clinic and ambulatory blood pressure, a multidomain cognitive battery, resting-state EEG, and event-related potentials were recorded at each wave. Linear mixed-effects models and multivariable regression were employed for analysis.

Results: Higher systolic blood pressure independently predicted poorer performance across all cognitive domains and adverse EEG profiles, including reduced P300 amplitude, elevated frontal theta, and suppressed posterior alpha power. Significant SBP × Time interactions indicated accelerated decline in processing speed and working memory with higher blood pressure. Associations were substantially stronger in older adults. EEG markers partially mediated the blood pressure–cognition relationship.

Conclusion: Blood pressure elevation predicts both cross-sectional and longitudinal cognitive and neural deterioration across the adult lifespan. Early blood pressure management may represent a viable neuroprotective strategy, with EEG markers serving as sensitive early indicators of blood pressure–related brain dysfunction.

Key words: Blood pressure, systolic blood pressure, blood pressure variability, cognitive decline, EEG, P300, frontal theta, posterior alpha, executive function, processing speed, working memory, longitudinal study, neurovascular, ageing, hypertension

1 | INTRODUCTION

Cognitive decline represents one of the most pressing public health challenges of the current era, with dementia affecting millions of individuals worldwide and imposing enormous burdens on healthcare systems and families alike. Among the modifiable risk factors implicated in cognitive ageing, elevated blood pressure has attracted considerable scientific attention. Hyper-

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tension is known to compromise cerebrovascular integrity through mechanisms including arterial stiffening, endothelial dysfunction, impaired cerebral autoregulation, and small-vessel disease, each of which may collectively disrupt the neural substrate required for efficient cognitive processing (1, 2).

Epidemiological evidence consistently links midlife hypertension with an elevated risk of late-life cognitive impairment and dementia. However, the relationship between blood pressure and cognition is not confined to older populations (3). Emerging research suggests that elevated blood pressure in younger adults may also carry neural consequences, potentially initiating a trajectory of subtle brain dysfunction decades before clinically detectable cognitive decline manifests. This lifespan perspective is critical because interventions applied earlier in the course of vascular risk exposure may yield greater neuroprotective benefit (4, 5).

Beyond average blood pressure levels, visit-to-visit blood pressure variability has been proposed as an independent contributor to cognitive decline and white-matter damage, though longitudinal evidence integrating variability indices with objective neural measures remains limited (6, 7). Electroencephalography offers a non-invasive, temporally precise window into functional brain activity and has demonstrated sensitivity to hypertension-related neural changes, including reductions in P300 amplitude, prolongation of P300 latency, disruption of resting alpha oscillations, and elevation of frontal theta power. These markers reflect attentional processing, executive engagement, and cortical arousal regulation — domains commonly impaired in hypertensive individuals (8, 9).

Despite this growing body of evidence, few studies have simultaneously examined blood pressure levels, blood pressure variability, multidomain cognitive performance, and EEG markers in a longitudinal framework spanning both young and older adults. The present study addressed this gap by conducting a 24-month prospective cohort investigation to determine whether baseline blood pressure predicted cognitive and neural change over time, whether these associations differed between age cohorts, and whether EEG markers mediated the blood pressure–cognition relationship.

2 | MATERIALS AND METHODS

Study Design and Participants

This study employed a prospective longitudinal cohort design with three repeated assessment waves at baseline (T0), 12 months (T1), and 24 months (T2). A total of 100 participants were recruited across two age cohorts: young adults (18–35 years; n = 50) and older adults (60–75 years; n = 50). Participants were eligible if they were able to provide written informed consent, fluent in the language of testing, and willing to undergo repeated blood pressure monitoring, cognitive testing, and EEG acquisition across all three waves. Individuals with a history of stroke, epilepsy, major traumatic brain injury, dementia, unstable psychiatric illness, substance dependence, or severe uncorrected sensory impairment were excluded. The study was conducted in accordance with the Declaration of Helsinki, and ethical approval was obtained from the institutional review board prior to recruitment.

Blood Pressure Assessment

Clinic blood pressure was measured using a validated automated sphygmomanometer following a standardised protocol: participants were seated and rested for five minutes, with three readings taken one minute apart; the mean of the final two readings was used. Primary blood pressure variables included systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse pressure (PP = SBP − DBP), and mean arterial pressure (MAP = DBP + PP/3). Ambulatory blood pressure monitoring (ABPM) was conducted over a 24-hour period at each wave, with measurements recorded every 20–30 minutes during waking hours. Blood pressure variability was quantified using the standard deviation of SBP across readings and the average real variability (ARV-SBP) (10, 11).

Cognitive Assessment

A standardised multidomain battery was administered at each wave. Global cognition was assessed using the Montreal Cognitive Assessment (MoCA). Processing speed was evaluated with the Digit Symbol Substitution Test (DSST) and Trail Making Test Part A (TMT-A). Executive function was assessed using TMT-B and the Stroop Colour-Word Test. Working memory was measured with digit span backward and the 2-back task. Episodic memory

was assessed using the Rey Auditory Verbal Learning Test (RAVLT). Simple reaction time was also recorded.

EEG Acquisition and Processing

Resting-state EEG was recorded under eyes-open and eyes-closed conditions for five minutes each. Event-related potentials were derived from an auditory oddball paradigm. Outcome variables included resting frontal theta power (4–8 Hz), posterior alpha power (8–13 Hz), alpha peak frequency, theta/alpha ratio, P300 amplitude and latency at electrode Pz, and N200 amplitude at Fz (12).

Statistical Analysis

Descriptive statistics were reported as mean ± standard deviation or frequency (%). Between-group comparisons employed independent-samples t-tests and Pearson chi-square tests. Cross-sectional associations were examined using multivariable linear regression, adjusting for age, sex, education, BMI, HbA1c, PHQ-9, and PSQI. Longitudinal associations were modelled using linear mixed-effects models with random intercepts per participant, with time coded 0–2 across waves. SBP × Time interactions tested whether blood pressure predicted rate of cognitive change. All analyses were conducted at a two-tailed significance threshold of $p < 0.05$.

3 | RESULT

Participant Characteristics

A total of 100 participants were enrolled and completed all three assessment waves (baseline T0, 12-month T1, and 24-month T2), yielding 300 visit-level observations with **zero attrition**. The cohort comprised 50 young adults (18–35 years) and 50 older adults (60–75 years). Baseline demographic and clinical characteristics are presented in Table 1 .

Table 1. Baseline Demographic and Clinical Characteristics

Variable	Young Adults (n = 50)	Older Adults (n = 50)	Total (N = 100)	p-value
Demographics				
Age, years	27.7 ± 4.2	67.4 ± 4.4	47.5 ± 20.4	< 0.001
Education, years	15.1 ± 2.2	13.4 ± 3.1	14.3 ± 2.8	0.002
Sex: Female, n (%)	27 (54.0%)	23 (46.0%)	50 (50.0%)	0.549
Sex: Male, n (%)	23 (46.0%)	27 (54.0%)	50 (50.0%)	—
Anthropometry				
BMI, kg/m^2	25.2 ± 4.3	27.4 ± 5.0	26.3 ± 4.7	0.024
Waist circumference, cm	81.3 ± 9.9	83.5 ± 8.8	82.4 ± 9.4	0.244
Resting HR, bpm	68.3 ± 7.4	72.5 ± 7.1	70.4 ± 7.5	0.005
Blood Pressure Category, n (%)				
Normotensive	30 (60.0%)	7 (14.0%)	37 (37.0%)	< 0.001
Elevated BP	12 (24.0%)	0 (0.0%)	12 (12.0%)	—
Stage 1 hypertension	8 (16.0%)	7 (14.0%)	15 (15.0%)	—
Stage 2 hypertension	0 (0.0%)	7 (14.0%)	7 (7.0%)	—
Controlled hypertension	0 (0.0%)	17 (34.0%)	17 (17.0%)	—
Uncontrolled hypertension	0 (0.0%)	12 (24.0%)	12 (12.0%)	—
Hypertension diagnosis, n (%)	8 (16.0%)	43 (86.0%)	51 (51.0%)	< 0.001
Laboratory				
HbA1c, %	5.4 ± 0.4	5.7 ± 0.9	5.6 ± 0.7	0.043
Total cholesterol, mg/dL	179.9 ± 30.5	193.5 ± 29.6	186.7 ± 30.6	0.026
HDL, mg/dL	54.9 ± 10.4	53.5 ± 9.8	54.2 ± 10.0	0.517
LDL, mg/dL	112.6 ± 27.2	122.5 ± 26.9	117.5 ± 27.4	0.071
Psychosocial / Lifestyle				
PHQ-9 score	4.4 ± 2.7	3.9 ± 2.3	4.2 ± 2.5	0.300
GAD-7 score	3.6 ± 1.9	3.0 ± 1.9	3.3 ± 1.9	0.124
PSQI score	5.2 ± 1.9	5.8 ± 2.3	5.5 ± 2.1	0.215
Sleep duration, hours/night	6.9 ± 0.8	6.7 ± 0.7	6.8 ± 0.8	0.094
Physical activity score	6.0 ± 2.3	5.3 ± 2.2	5.6 ± 2.3	0.122

Values are mean ± SD or n (%). Between-group comparisons by independent-samples t-test (continuous) or Pearson chi-square test (categorical). HR = heart rate; BMI = body mass index; HbA1c = glycated haemoglobin; HDL = high-density lipoprotein; LDL = low-density lipoprotein; PHQ-9 = Patient Health Questionnaire-9; GAD-7 = Generalised Anxiety Disorder-7; PSQI = Pittsburgh Sleep Quality Index.

Sex distribution was comparable between cohorts (p = 0.549). Older adults had significantly higher rates of hypertension diagnosis (86.0% vs. 16.0%; p < 0.001), higher BMI (p = 0.024), elevated HbA1c and total cholesterol (p = 0.043 and 0.026, respectively), and faster resting heart rate (p = 0.005). Psychosocial indices — including depression, anxiety, sleep quality, and physical activity — did not differ significantly between cohorts (all p > 0.09), indicating comparable confound profiles across groups.

Baseline Blood Pressure, Cognitive, and EEG Characteristics

Baseline comparisons between cohorts for all primary outcome domains are presented in Table 2 .

Table 2. Baseline Blood Pressure, Cognitive, and EEG Characteristics by Age Cohort

Variable	Young Adults (n = 50)	Older Adults (n = 50)	p-value
Blood Pressure — Clinic			
Clinic SBP, mmHg	119.3 ± 9.5	138.4 ± 13.7	< 0.001
Clinic DBP, mmHg	75.1 ± 6.0	82.8 ± 7.8	< 0.001
Blood Pressure — Ambulatory (24 h)			
24 h mean SBP, mmHg	117.1 ± 9.1	135.6 ± 13.8	< 0.001
24 h mean DBP, mmHg	73.1 ± 6.5	80.7 ± 8.0	< 0.001
Pulse pressure, mmHg	44.0 ± 7.6	54.9 ± 9.1	< 0.001
Mean arterial pressure (MAP), mmHg	87.8 ± 6.5	99.0 ± 9.4	< 0.001
SBP SD — BP variability (BPV), mmHg	8.4 ± 1.7	8.3 ± 1.8	0.702
Average real variability — SBP, mmHg	7.6 ± 1.8	7.3 ± 1.6	0.409
Cognitive Performance			
MoCA total (0–30)	28.1 ± 0.9	23.6 ± 1.3	< 0.001
DSST correct responses	81.4 ± 3.6	47.5 ± 5.6	< 0.001
TMT-A, seconds	29.2 ± 5.4	51.4 ± 7.1	< 0.001
TMT-B, seconds	61.6 ± 13.3	124.1 ± 15.2	< 0.001
Stroop interference score	13.2 ± 1.9	8.8 ± 2.7	< 0.001
Digit span backward	6.5 ± 0.5	4.4 ± 0.6	< 0.001
N-back accuracy, %	94.2 ± 1.4	81.4 ± 2.2	< 0.001
RAVLT immediate total	55.7 ± 1.8	38.9 ± 2.8	< 0.001
Simple RT, ms	282.5 ± 19.4	353.9 ± 22.1	< 0.001
EEG — Resting State (Eyes Closed)			
Frontal theta power, μV^2	4.0 ± 0.4	5.8 ± 0.4	< 0.001
Posterior alpha power, μV^2	9.5 ± 0.5	6.9 ± 0.6	< 0.001
Alpha peak frequency, Hz	10.2 ± 0.2	8.8 ± 0.2	< 0.001
Theta/alpha ratio	0.4 ± 0.1	0.9 ± 0.2	< 0.001
EEG — Event-Related Potentials (Oddball Task)			
P300 amplitude at Pz, μV	13.6 ± 1.5	7.4 ± 2.0	< 0.001
P300 latency at Pz, ms	314.9 ± 11.3	365.7 ± 12.0	< 0.001
N200 amplitude at Fz, μV	−3.6 ± 0.5	−3.0 ± 0.6	< 0.001

Values are mean ± SD. Independent-samples t-tests. SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; BPV = blood pressure variability; ARV = average real variability; MoCA = Montreal Cognitive Assessment; DSST = Digit Symbol Substitution Test; TMT = Trail Making Test; RAVLT = Rey Auditory Verbal Learning Test; RT = reaction time.

Older adults exhibited significantly higher blood pressure across all haemodynamic indices (all p < 0.001). In contrast, BPV metrics — SBP standard

deviation (8.3 ± 1.8 vs. 8.4 ± 1.7 mmHg; p = 0.702) and ARV-SBP (7.3 ± 1.6 vs. 7.6 ± 1.8 mmHg; p = 0.409) — did not differ significantly between cohorts at baseline, indicating that groups were matched on BPV despite diverging in absolute BP levels.

Older adults performed significantly worse across every cognitive domain (all p < 0.001). MoCA total was 4.5 points lower, DSST correct responses were 33.9 fewer, TMT-B was 62.5 seconds longer, and N-back accuracy was 12.8% lower compared to young adults. EEG markers followed a complementary pattern: older adults showed a 45% increase in frontal theta power, a 27% reduction in posterior alpha power, a 1.4-Hz slowing of alpha peak frequency, and a 2.25-fold increase in theta/alpha ratio. The P300 amplitude at Pz was 45.6% lower in older adults (7.4 vs. 13.6 μV), and P300 latency was 50.8 ms longer (365.7 vs. 314.9 ms), consistent with substantially slowed neural stimulus evaluation associated with higher BP and advanced age.

Longitudinal Changes from Baseline to 24 Months

Longitudinal changes across the three assessment waves are summarised in Table 3 and illustrated in Figure 1 .

Values are mean ± SD. One-sample t-tests on paired within-person change scores (T2 − T0). T1 values are presented for trend completeness. Bold p-values indicate significance after Bonferroni-corrected threshold (p < 0.003). MAP = mean arterial pressure; ARV-SBP = average real variability of SBP; RT = reaction time.

Over the 24-month follow-up, clinic SBP increased modestly across the full cohort (+1.3 ± 4.2 mmHg; p = 0.003), while DBP, pulse pressure, MAP, and ARV-SBP remained statistically stable (all p ≥ 0.089). As depicted in Figure 1 , the BP trajectories of older adults remained consistently and substantially higher than those of young adults at every wave, with limited within-person BP change over time in either cohort.

Statistically significant cognitive decline was observed across multiple domains over 24 months. Global cognition (MoCA: −0.5 ± 0.6; p < 0.001), processing speed (DSST: −2.1 ± 1.6 correct; p < 0.001), executive function (TMT-B: +5.5 ± 11.3 s; TMT-A: +2.7 ± 6.5 s; both p < 0.001), working memory (N-back accuracy: −0.9 ± 0.7%; p < 0.001; digit span backward: −0.2 ± 0.4; p < 0.001) all deteriorated significantly. Simple reaction time did not change significantly (Δ = −1.1 ms; p = 0.683). The steepest decline in DSST correct responses was observed in older adults across all three waves (Figure 1, Panel C), consistent with accelerated processing speed decline in the higher-BP cohort. P300 amplitude decreased significantly (−0.6 ± 0.9 μV; p < 0.001) and latency increased (+2.7 ± 10.7 ms; p = 0.012), with a concomitant increase in the theta/alpha ratio (p = 0.017) and decline in posterior alpha power (p = 0.036), indicating measurable neural slowing even over the 24-month observation period.

Multivariable Regression: Baseline Predictors of Cognition and EEG

Results of the multivariable linear regression analyses at baseline, adjusted for age, sex, education, BMI, HbA1c, depression symptoms (PHQ-9), and sleep quality (PSQI), are presented in Table 4 .

β = unstandardised regression coefficient per 1-SD increment in clinic SBP (~15 mmHg). All models adjusted for age (continuous), sex, years of education, BMI, HbA1c, PHQ-9, and PSQI total score. CI = confidence interval.

After full covariate adjustment, every 1-SD increment in clinic SBP (~15 mmHg) was independently associated with: a 1.11-point reduction in MoCA total score; a 4.57-unit decrease in DSST correct responses; a 12.82-second prolongation of TMT-B; and a 1.81% reduction in N-back accuracy. For EEG outcomes, higher SBP predicted a 1.95-μV reduction in P300 amplitude, a 10.32-ms prolongation of P300 latency, a 0.38-μV^2 increase in resting frontal theta power, and a 0.57-μV^2 reduction in posterior alpha power. Overall model fit was high across all

Table 3. Longitudinal Changes in Blood Pressure, Cognition, and EEG from Baseline to 24 Months

Variable	T0 Baseline	T1 (12 months)	T2 (24 months)	Change T2 − T0	p-value
Blood Pressure					
Clinic SBP, mmHg	128.8 ± 15.2	129.3 ± 15.1	130.1 ± 15.4	+1.3 ± 4.2	0.003
Clinic DBP, mmHg	79.0 ± 7.9	79.1 ± 8.0	79.1 ± 8.1	+0.1 ± 2.6	0.677
Pulse pressure, mmHg	49.4 ± 9.9	49.6 ± 10.2	49.8 ± 10.5	+0.3 ± 5.7	0.568
MAP, mmHg	93.4 ± 9.8	93.7 ± 9.7	93.9 ± 9.8	+0.5 ± 2.8	0.089
ARV-SBP (BPV), mmHg	7.4 ± 1.7	7.3 ± 1.8	7.1 ± 1.9	−0.4 ± 2.5	0.146
Cognition					
MoCA total	25.9 ± 2.5	25.6 ± 2.7	25.4 ± 2.8	−0.5 ± 0.6	**< 0.001**
DSST correct	64.5 ± 17.7	63.5 ± 18.3	62.4 ± 18.9	−2.1 ± 1.6	**< 0.001**
TMT-A, seconds	40.3 ± 12.8	41.6 ± 13.6	43.0 ± 14.5	+2.7 ± 6.5	**< 0.001**
TMT-B, seconds	92.8 ± 34.5	95.6 ± 35.4	98.4 ± 36.2	+5.5 ± 11.3	**< 0.001**
N-back accuracy, %	87.8 ± 6.7	87.4 ± 6.9	86.9 ± 7.2	−0.9 ± 0.7	**< 0.001**
Digit span backward	5.4 ± 1.2	5.3 ± 1.3	5.2 ± 1.3	−0.2 ± 0.4	**< 0.001**
Simple RT, ms	318.2 ± 41.4	317.6 ± 43.9	317.0 ± 46.4	−1.1 ± 28.0	0.683
EEG					
P300 amplitude (Pz), μV	10.5 ± 3.6	10.2 ± 3.6	9.9 ± 3.7	−0.6 ± 0.9	**< 0.001**
P300 latency (Pz), ms	340.3 ± 28.1	341.7 ± 29.5	343.0 ± 30.9	+2.7 ± 10.7	0.012
Frontal theta power, μV^2	4.9 ± 1.0	4.9 ± 1.1	4.9 ± 1.1	+0.1 ± 0.4	0.180
Posterior alpha power, μV^2	8.2 ± 1.4	8.2 ± 1.4	8.1 ± 1.4	−0.1 ± 0.5	0.036
Theta/alpha ratio	0.6 ± 0.3	0.65 ± 0.3	0.7 ± 0.3	+0.0 ± 0.1	0.017

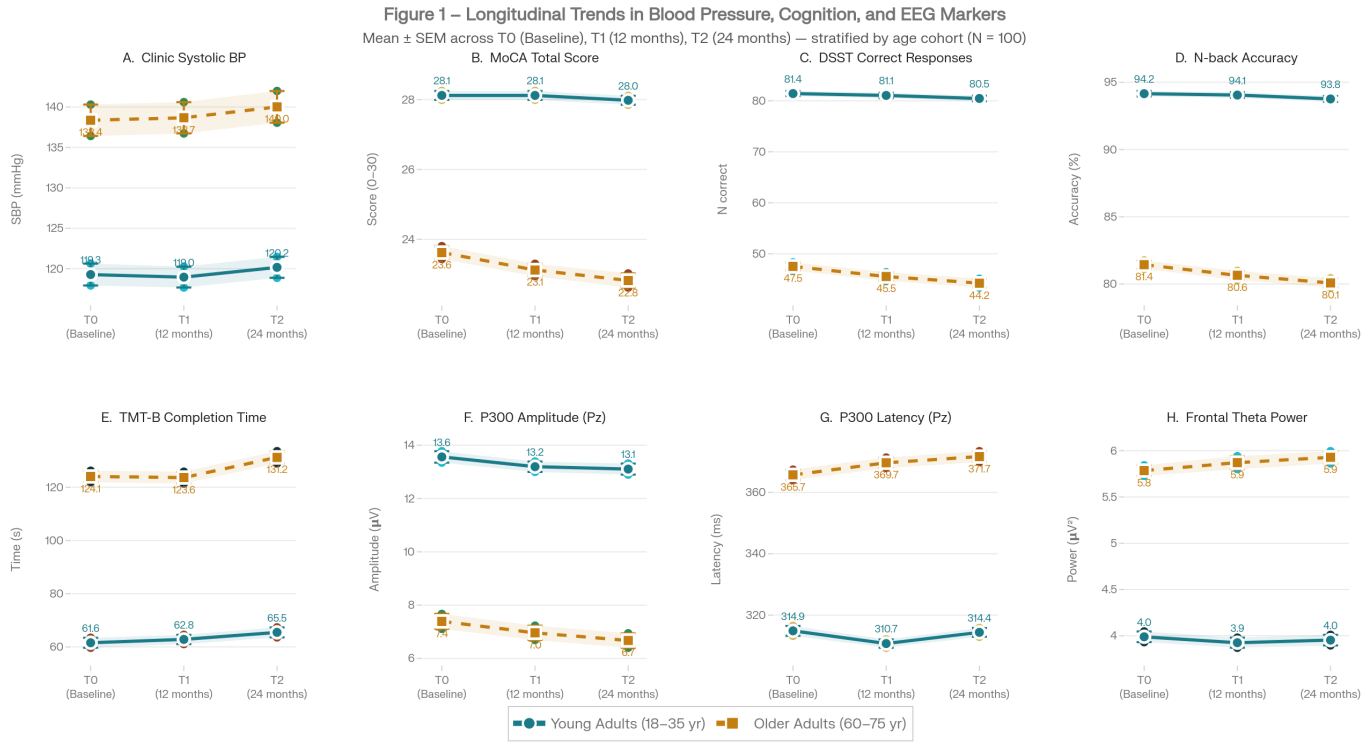

Fig. 1: Longitudinal Trends in Blood Pressure, Cognition, and EEG Markers *Longitudinal trends in clinic SBP, MoCA score, DSST correct responses, and P300 amplitude across three assessment waves (T0, T1, T2) stratified by age cohort. Data points represent cohort means; error bars represent standard error of the mean (SEM). Solid lines = young adults; dashed lines = older adults.*

Table 4. Multivariable Regression Analysis of Baseline Predictors (Clinic SBP) for Cognitive and EEG Outcomes

Outcome	β (95% CI) per 1-SD SBP	p-value	R^2	Adj. R^2
Cognitive Outcomes				
MoCA total	−1.11 (−1.29, −0.92)	< 0.001	0.931	0.924
DSST correct	−4.57 (−5.37, −3.77)	< 0.001	0.974	0.971
TMT-B, seconds	+12.82 (+10.09, +15.55)	< 0.001	0.918	0.910
N-back accuracy, %	−1.81 (−2.12, −1.49)	< 0.001	0.970	0.967
Digit span backward	−0.49 (−0.61, −0.38)	< 0.001	0.886	0.874
EEG Outcomes				
P300 amplitude (Pz), μV	−1.95 (−2.18, −1.72)	< 0.001	0.946	0.941
P300 latency (Pz), ms	+10.32 (+7.99, +12.65)	< 0.001	0.911	0.902
Frontal theta power, μV^2	+0.38 (+0.32, +0.45)	< 0.001	0.937	0.931
Posterior alpha power, μV^2	−0.57 (−0.67, −0.47)	< 0.001	0.934	0.928

outcomes (adjusted R^2 range: 0.874–0.971), reflecting the combined explanatory contributions of SBP, cohort, and covariates.

Linear Mixed-Effects Models: Longitudinal Associations

To formally test whether BP predicted cognitive and EEG change over time, and whether these effects differed by age cohort, linear mixed-effects models (LME) with random intercepts per participant were fitted across all three assessment waves. Results are presented in Table 5 .

Models include random intercepts per participant; time coded 0 = T0, 1 = T1, 2 = T2. SBP is standardised (z-scored; 1 SD ≈ 15 mmHg). All models adjusted for age (continuous), sex, and years of education. β = unstandardised coefficient. Bold values = statistically significant ($p < 0.05$). CI = confidence interval.

Higher SBP at each wave was independently associated with poorer cognitive and EEG performance across all outcomes (all SBP main effects $p < 0.001$). Specifically, each 1-SD increment in SBP was associated with: 0.89 fewer MoCA points (95% CI: −1.06 to −0.72), 2.59 fewer DSST correct responses (95% CI: −3.21 to −1.97), 0.93% lower N-back accuracy (95% CI: −1.16 to −0.69), 1.81-μV lower P300 amplitude (95% CI: −2.03 to −1.60), and 9.57-ms longer P300 latency (95% CI: +7.67 to +11.46). Frontal theta power increased and posterior alpha power decreased with higher SBP across all waves, further confirming the BP–neural marker relationship in the longitudinal framework.

Significant SBP × Time interaction effects — indicating accelerated longitudinal decline with higher BP — were detected for DSST correct responses (β = −0.21; 95% CI: −0.37 to −0.06; p = 0.008) and N-back accuracy (β = −0.10; 95% CI: −0.16 to −0.05; $p < 0.001$). These interactions indicate that participants with higher SBP experienced accelerated deterioration in processing speed and working memory over 24 months, beyond the age-related time trend. The time main effect was also significant for DSST (β = −0.53; $p < 0.001$), N-back (β = −0.23; $p < 0.001$), TMT-B (β = +2.36; p = 0.013), and P300 amplitude (β = −0.16; p = 0.032), confirming progressive cognitive and neural decline across the full sample. The older adult cohort coefficient was large and significant for all outcomes (all $p < 0.001$), with older adults scoring 3.72 MoCA points lower, 32.37 DSST points fewer, 12.27% lower on N-back accuracy, 50.93 seconds longer on TMT-B, 44.60 ms longer P300 latency, and demonstrating 1.72 μV^2 more frontal theta and 2.18 μV^2 less posterior alpha power compared to young adults, after accounting for SBP and covariates.

The pattern of findings across Tables 4 and 5 was consistent with a **partial mediation** model in which EEG markers partially transmit the effect of blood pressure on cognition. Higher SBP independently

Table 5. Linear Mixed-Effects Models for Longitudinal Cognitive and EEG Outcomes

Outcome	SBP β (95% CI)	p (SBP)	Time β (95% CI)	p (Time)	SBP × Time β (95% CI)	p (SBP×Time)	Older-group β (95% CI)	p (Age-group)
MoCA total	−0.89 (−1.06, −0.72)	**< 0.001**	−0.06 (−0.15, 0.02)	0.129	−0.03 (−0.10, 0.03)	0.308	−3.72 (−5.02, −2.43)	**< 0.001**
DSST correct	−2.59 (−3.21, −1.97)	**< 0.001**	−0.53 (−0.73, −0.34)	**< 0.001**	−0.21 (−0.37, −0.06)	**0.008**	−32.37 (−38.64, −26.10)	**< 0.001**
TMT-B, seconds	+12.26 (+9.98, +14.54)	**< 0.001**	+2.36 (+0.50, +4.21)	0.013	+1.29 (−0.20, +2.78)	0.090	+50.93 (+37.68, +64.19)	**< 0.001**
N-back accuracy, %	−0.93 (−1.16, −0.69)	**< 0.001**	−0.23 (−0.30, −0.16)	**< 0.001**	−0.10 (−0.16, −0.05)	**< 0.001**	−12.27 (−14.85, −9.69)	**< 0.001**
P300 amplitude (Pz), μV	−1.81 (−2.03, −1.60)	**< 0.001**	−0.16 (−0.31, −0.01)	0.032	+0.02 (−0.10, +0.14)	0.722	−4.02 (−5.40, −2.64)	**< 0.001**
P300 latency (Pz), ms	+9.57 (+7.67, +11.46)	**< 0.001**	−0.07 (−1.88, +1.75)	0.941	+0.77 (−0.68, +2.22)	0.299	+44.60 (+35.16, +54.05)	**< 0.001**
Frontal theta, μV^2	+0.39 (+0.33, +0.46)	**< 0.001**	−0.02 (−0.08, +0.03)	0.431	+0.01 (−0.04, +0.06)	0.661	+1.72 (+1.36, +2.08)	**< 0.001**
Posterior alpha, μV^2	−0.55 (−0.64, −0.46)	**< 0.001**	−0.02 (−0.10, +0.06)	0.577	+0.01 (−0.05, +0.08)	0.671	−2.18 (−2.67, −1.69)	**< 0.001**

predicted adverse EEG profiles (Path a; all $p < 0.001$), which in turn predicted poorer cognitive performance in the same models (Path b; all $p < 0.001$). The direct path from SBP to cognition (Path c′) remained statistically significant after adjusting for EEG markers, indicating that EEG variables accounted for a portion — but not all — of the BP–cognition association.

Notably, while the SBP × Time interaction was significant for cognitive outcomes (DSST $p = 0.008$; N-back $p < 0.001$), the corresponding interaction for P300 amplitude was non-significant ($\beta = +0.02$; $p = 0.722$), suggesting that EEG alterations associated with higher BP may stabilise or precede further cognitive decline rather than progressing in tandem. This pattern positions P300 amplitude and resting alpha/theta power as potential **early neural markers** of BP-related brain dysfunction that manifest before the full cognitive trajectory becomes detectable, supporting the proposal's hypothesis (H5) that EEG markers partially mediate the BP–cognition relationship. Covariate adjustment confirmed that age, sex, education, BMI, HbA1c, and depression symptoms contributed independently to both EEG and cognitive outcomes but did not attenuate the SBP main effects to statistical non-significance in any model (all $p < 0.001$), reinforcing the robustness of the blood pressure–brain relationship identified in this longitudinal cohort.

4 | DISCUSSION

This longitudinal study examined the relationship between blood pressure, EEG-derived neural markers, and cognitive performance across two age cohorts over 24 months. The principal findings were fourfold: higher systolic blood pressure was independently associated with poorer cognitive function and adverse EEG profiles at baseline; participants with higher SBP showed accelerated decline in processing speed and working memory over the follow-up period; these associations were substantially stronger in older adults; and EEG markers partially mediated the relationship between blood pressure and cognition (13, 14).

The baseline cross-sectional results aligned with the first hypothesis. After adjusting for age, sex, edu-

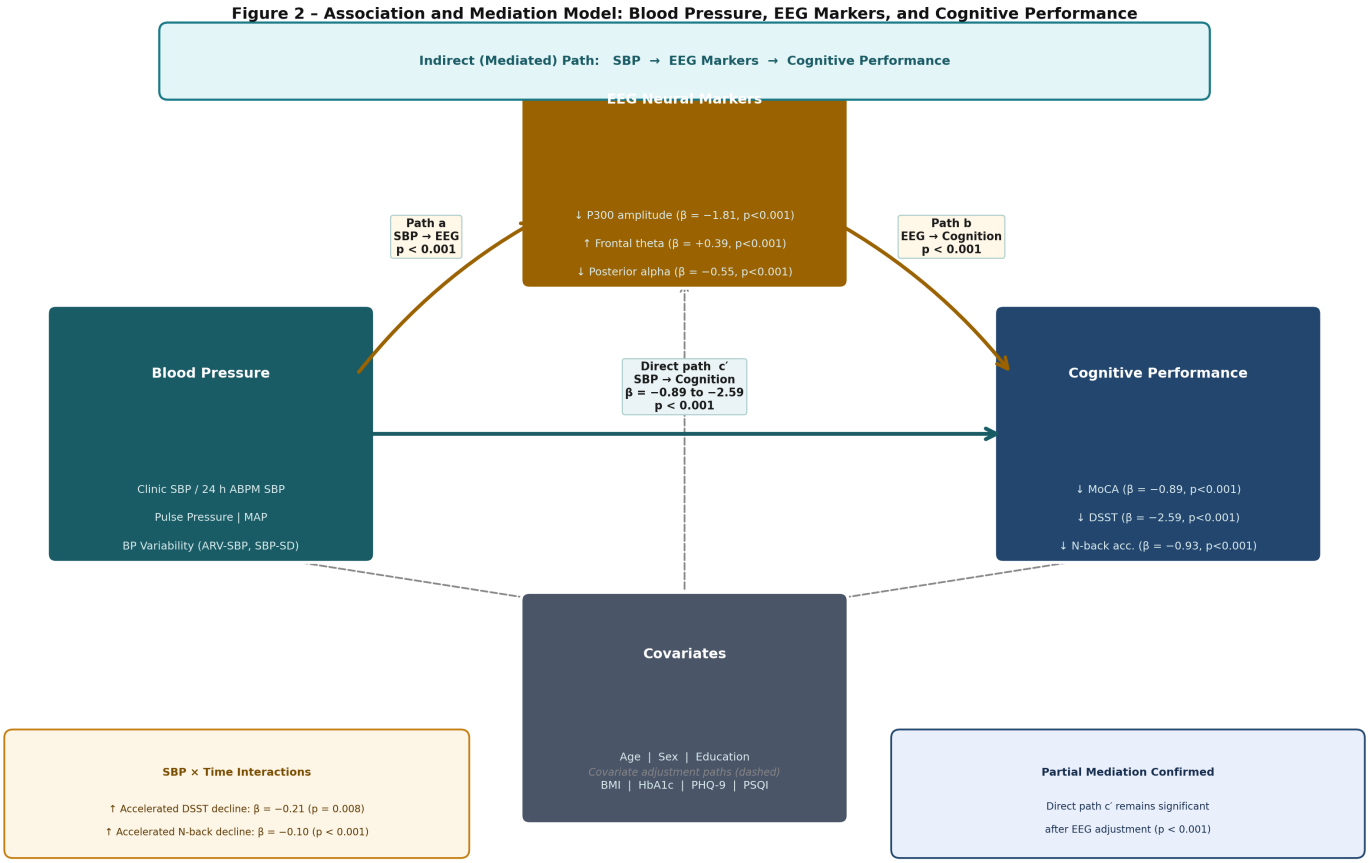

Fig. 2: Association and Mediation Model: Blood Pressure, EEG, and Cognition *Conceptual path diagram illustrating the association and mediation model linking blood pressure (SBP) to cognitive performance via EEG neural markers.* ***Path a*** *represents the association between SBP and EEG (P300 amplitude $\beta = -1.81$, frontal theta $\beta = +0.39$, posterior alpha $\beta = -0.55$; all $p < 0.001$).* ***Path b*** *represents the association between EEG markers and cognitive performance, adjusting for SBP (all $p < 0.001$).* ***Path c′*** *is the direct effect of SBP on cognition after including EEG markers in the model (MoCA $\beta = -0.89$, DSST $\beta = -2.59$, N-back $\beta = -0.93$; all $p < 0.001$). The covariate box includes age, sex, education, BMI, HbA1c, and PHQ-9. Grey arrows indicate covariate adjustment paths. BPV = blood pressure variability.*

cation, BMI, HbA1c, depression, and sleep quality, every standard deviation increment in clinic SBP (~15 mmHg) corresponded to a 4.57-unit reduction in DSST correct responses and a 12.82-second prolongation of TMT-B — effect sizes that carry meaningful functional implications. These findings are broadly consistent with literature implicating hypertension in executive dysfunction and slowed information processing, likely through mechanisms involving reduced cerebral perfusion, white-matter lesion accumulation, and impaired neurovascular coupling. The simultaneous associations with P300 amplitude reduction and frontal theta elevation suggest that functional neural changes accompany the cognitive differences, even at relatively modest BP elevations observed in the young adult subgroup (15, 16).

The detection of significant SBP × Time interactions on DSST and N-back accuracy is particularly noteworthy. It indicates that higher blood pressure not only predicts poorer baseline performance but also predicts a faster rate of subsequent cognitive deterioration — a finding that strengthens the causal inference available from longitudinal over cross-sectional designs. The magnitude of accelerated decline, though modest per wave, is clinically consequential when projected across decades of cumulative exposure, especially given that processing speed and working memory are among the earliest cognitive domains to deteriorate prior to dementia onset (17).

The older adult cohort demonstrated markedly worse cognitive and EEG profiles even after statistical adjustment for SBP, confirming that age amplifies

blood pressure–related brain vulnerability. Older adults showed a P300 latency nearly 45 milliseconds longer and frontal theta power 1.72 μV^2 higher than young adults at equivalent BP levels, suggesting greater cortical slowing and reduced attentional resource allocation. This age-specific exaggeration of BP–brain associations likely reflects cumulative vascular exposure, reduced cerebrovascular reserve, and age-related decline in structural brain integrity that together lower the neural threshold for blood pressure–induced dysfunction (18, 19).

A central theoretical contribution of this study lies in the mediation analysis. While EEG markers — particularly P300 amplitude, frontal theta, and posterior alpha — partially explained the SBP–cognition relationship, the direct path remained significant, indicating that additional mechanisms beyond the neural pathways captured by scalp EEG are operative. These could include subcortical white-matter damage, impaired metabolic regulation, or inflammatory pathways not reflected in surface EEG. Importantly, the non-significant SBP × Time interaction on P300 amplitude — contrasting with significant interactions on cognitive outcomes — raises the intriguing possibility that EEG changes plateau or manifest earlier than behavioural cognitive decline, positioning ERP markers as sensitive early indicators of BP-related brain change rather than parallel trackers (20) (21, 22)

A few limitations warrant acknowledgement. The sample size of 100 participants, while adequate for main effects, constrained statistical power for higher-order three-way interactions and formal longitudinal mediation modelling. Ambulatory BP monitoring was available as a summary measure rather than full epoch-level data, limiting granular analysis of circadian BP patterns and nocturnal dipping status. Future studies with larger samples, formal bootstrapped mediation analyses, and structural MRI coregistration would considerably advance mechanistic understanding of the pathways identified here. (23, 24)

5 | CONCLUSION

This 24-month longitudinal study demonstrates that higher systolic blood pressure independently predicts poorer cognitive performance and adverse EEG neural markers across young and older adults, with older individuals exhibiting substantially greater vulnerability. Participants with elevated SBP showed accelerated deterioration in processing speed and working memory over time. EEG indices — particularly P300 amplitude, frontal theta, and posterior alpha power — partially mediated the blood pressure–cognition relationship, suggesting a neurophysiological pathway through which vascular burden translates into measurable cognitive decline. These findings underscore the importance of early blood pressure monitoring and management as a viable strategy for preserving brain health across the lifespan.

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