# TITLE

## Age-related response speed deficits arise from specific impairments in sensory evidence accumulation rate

# AUTHORS/AFFILIATIONS

Méadhbh B. Brosnan[1,2,3,4] (meadhbh.brosnan@psy.ox.ac.uk)
Megan H. O'Neill[1] (mhoneill1@gmail.com)
Gerard M. Loughnane[5,6] (gerard.loughnane@ncirl.ie)
Daniel J. Pearce[1] (Daniel.Pearce@monash.edu)
Bryce Fleming[1] (bcfle1@student.monash.edu)
Trevor T.-J. Chong[1] (trevor.chong@monash.edu)
Anna C. Nobre[2,3,4] (kia.nobre@ohba.ox.ac.uk)
Redmond G. O Connell[1,6] (redmond.oconnell@tcd.ie)
Mark A. Bellgrove[1] (mark.bellgrove@monash.edu)

[1]Turner Institute for Brain and Mental Health and School of Psychological Sciences, Monash University, Melbourne, Victoria, 3800, Australia
[2]Department of Experimental Psychology, University of Oxford, Oxford, UK
[3]Oxford Centre for Human Brain Activity, University of Oxford, Oxford, UK
[4]Wellcome Centre for Integrative Neuroimaging, University of Oxford, Oxford, UK
[5]School of Business, National College of Ireland
[6]Trinity College Institute of Neuroscience and School of Psychology, Trinity College Dublin, Dublin, D02 PN40, Ireland

*Correspondence meadhbh.brosnan@psy.ox.ac.uk; mark.bellgrove@monash.edu

# ABSTRACT

Older adults exposed to enriched environments (EE) maintain relatively higher levels of cognitive function, even in the face of compromised markers of brain health. Response speed (RS) is often used as a simple proxy to measure the preservation of global cognitive function in older adults. However, it is unknown which specific sensory, decision, and/or motor processes provide the most specific indices of neurocognitive health. Here, using a simple decision task with electroencephalography (EEG), we found that the efficiency with which an individual accumulates sensory evidence was a critical determinant of the extent to which RS was preserved in older adults. Moreover, the mitigating influence of EE on age-related RS declines was most pronounced when evidence accumulation rates were shallowest. Our results suggest that EEG metrics of evidence accumulation may index neurocognitive vulnerability of the ageing brain.

Cognitive deficits occurring with healthy or pathological ageing catalyse a broad range of challenging consequences[1–5] and are marked by large inter-individual variability[6–8]. Robust evidence has emerged over the past three decades demonstrating a powerful positive influence of enriched environments (EE), such as education, leisure and work activities, on the preservation of cognitive function[9–15]. It has become increasingly apparent that older adults who have been exposed to EE can maintain high levels of cognitive function, despite objective evidence of compromised brain health, such as grey matter atrophy[17] in otherwise healthy individuals, and disease-related neuropathology[18] in clinical conditions including Alzheimer's[19], stroke[20], multiple sclerosis[21], and Huntington's Disease[22].

The speed with which older adults respond to sensory input – hereafter referred to as response speed – has been taken as a robust index of an individual's vulnerability to cognitive decline[23–28]. Compelling longitudinal work with 861 older participants has shown that response speed on simple decision tasks predicts a 50-60% increased risk of developing a full diagnosis of dementia over a four-year period, even when controlling for known risk factors for the disease (including age, cerebrovascular disease, genetic susceptibility, mood, cognition, and education[27], see also[25,26]). Moreover, emerging results from a large (*N*=2832) multicentre clinical trial using computerised speed-based cognitive training in older adults shows that training this capacity may lower the risk of developing clinical symptoms of dementia[29], adding to evidence that training response speed may causally improve neurocognitive health in older adults[30–33]. In further support of response speed as a meaningful proxy for healthy neurocognitive ageing, monozygotic twin pairs that have been exposed to greater levels of enrichment throughout life show relatively faster response speeds in later years[34].

Yet response speed is the outcome of multiple sensory, decisional, and motoric computations, and it remains unclear which of these neural processes account for the close association between response speed and neurocognitive health in older adults. Identifying the extent to which discrete neurophysiological processes predict response speed in older individuals would help to elucidate the causal mechanisms underpinning the association between cognitive capacity and the neurocognitive health of the ageing brain. Here, we investigate the hypothesis that the association between response speed and brain health in older adults may arise from deficits in the accumulation of sensory evidence during the formation of a perceptual decision (e.g.[41,47] but see also [41,48,49], for a recent review see[50]). The centro-parietal positivity (CPP) is an extracranial human EEG signal which exhibits the key characteristics of evidence accumulation signals observed using invasive electrophysiological recordings in animals[51,52]. Specifically, it rises with the strength of available sensory evidence and peaks prior to an individual's response. Moreover, the CPP is supramodal (occurring irrespective of the modality of sensory input), exhibits evidence accumulation dynamics across a multitude of perceptual tasks[53–57], and is separable from both early visual processing and motor preparatory activity, thereby reflecting a distinct intermediate neurophysiological process between early sensory analysis and the motor response[51,52]. Recent work on a perceptual decision-making (choice reaction time) task showed that the CPP build-up rates were shallower in older relative to younger adults[41]. However, the potential for CPP build-up rate to account for individual differences in response times in older adults remains unclear. In younger adults, the CPP has been repeatedly shown to capture individual variability in response speed[51,52,58–62]. Moreover, we have recently observed that younger individuals with enhanced structural (white matter macrostructural organisation of the superior longitudinal fasciculus) and functional connectivity (resting state functional MRI connectivity) within the dorsal FPN accumulate sensory evidence at a steeper rate, indexed via the build-up rate of the CPP which, in turn, facilitates faster response speed[62]. Converging evidence from neuroimaging[63], modelling work[64], and causal manipulation techniques[65,66] suggest that response speed in older adults is related to structural and functional properties of the fronto-parietal networks (FPN). This raises the possibility that the established relationship between the FPN and individual differences in

response speed in older adults[64–67] might arise from an individuals' capacity to accumulate sensory evidence.

In the current study EEG was used to isolate neural metrics indexing the discrete processing stages underpinning perceptual decisions while a group of older adults performed a variant of the random-dot motion detection task[69]. We tested the hypothesis that neural markers of sensory evidence accumulation (build-up rates of the CPP[51,52]) best capture individual variations in speeded target detections, over and above any influence of other neurophysiological processes contributing to the timing of response. These included early target selection (N2c latency and amplitude[70–73]), sensory evidence accumulation (CPP onset latency or starting point[62,68,70]), decision criterion, or bound (amplitude of the CPP[60]), and motor preparatory activity (response-aligned build-up rate, stimulus-aligned peak latency, and amplitude of beta band activity (13-30 Hz) in the hemisphere contralateral to the motor response[41,62]). In addition, we assessed the beneficial effects of EE using a well-validated assessment tool that investigates occupational, professional, and leisure engagements[74]. A lifetime of EE was hypothesised to offset age-related deficits in response speed (e.g.[34,75,76]). Critically, in keeping with the phenomenon of cognitive reserve (e.g.[9,17,18],see [19] for a review), we hypothesised that the beneficial influence of EE on age-related declines in response speeds would be moderated by shallower build-up rates of the CPP (i.e. would be most pronounced for individuals with relatively less efficient evidence accumulation).

# RESULTS

## Individuated neurophysiological metrics indexing visual response speed were isolated using a perceptual-decision making EEG task

Seventy two participants (41 older *M*=73 years, *SD*=5, range = 63-87 and *N*=31 younger *M*=24 years, *SD*=3, range = 18-28) performed a variant of the random-dot motion task[69] while 64-channel EEG was recorded to isolate eight distinct, previously validated neural metrics[62,68]: early target selection (N2c amplitude and latency[70], sensory evidence accumulation (CPP starting point (onset latency), build-up rate (slope), and decision bound (amplitude)[41,51,60], and motor preparation (left hemisphere beta (LHB) build-up rate (slope), timing (stimulus-aligned peak latency), and threshold (amplitude[41,51]), see Fig. 1[69].

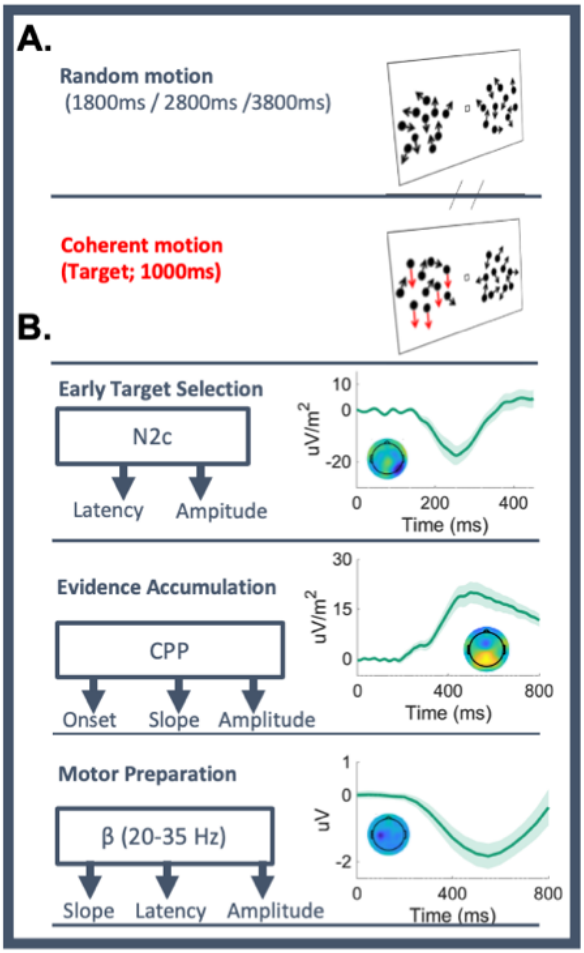



**Figure 1. The perceptual-decision making EEG task used to isolate individuated neurophysiological metrics indexing visual response speed**. **A.** During the random-dot motion detection task participants fixated centrally while two patches of randomly moving dots were presented peripherally (centred at 10° of visual angle either side and 4° visual angle below the fixation square) in both hemifields. During 'target' trials, 90% of the dots in one hemifield transitioned from random to coherent motion in either an upward or a downward direction. Targets remained on the screen for 1s, or until the participant pressed the button signalling the detection of coherent motion in either direction. If a fixation break occurred during a trial (either a blink or a gaze deviation >4° left or right of centre), the task halted (stationary dots) until fixation returned to the central fixation dot. Participant response speed was assessed via a right-hand button press for target detection (coherent motion in either upward or downward direction). **B.** Eight EEG metrics were isolated. The EEG waveforms and topoplots depicted are for visualisation of the relevant components only and are taken from an independent group of healthy younger adults (N=56[62]). For the response-aligned components, see Supplementary Fig. 3.

## Response Speed Measures Are Sensitive to both Age and EE

Behavioural analyses (Fig. 2A) indicated that this task was sensitive to age-related deficits in response speed. The older adults were markedly slower at responding, as evidenced by significantly slower response times (RTs) to the visual targets relative to the younger adults ($F_{1,70}$ =38.34, $p$<.001, partial $\eta^2$= 0.35, $BF_{10}$=287907.20; older *M*=593.33ms, *SD*=125.40; younger *M*=439.05ms, *SD*=67.87; Fig. 2A). Target detection accuracy was high for the overall sample 95.92% (*SD*=5.29, range 71-100%), but nonetheless the older adults were less accurate at detecting coherent motion than their younger peers

(older: $M$ = 94.40%, $SD$ = 6.3%; younger: $M$=97.90, $SD$=2.60%; $F_{1,70}$ = 8.12, $p$=.006, partial $\eta^2$ = 0.10, $BF_{10}$=7.17). Critically, the age-related declines in RTs remained significant even after covarying for differences in accuracy ($F_{2,69}$ = 27.16, $p$<.001, partial $\eta^2$= 0.44).

We next sought to verify previously reported associations between a lifetime of EE and response speed e.g.[34,75]. For this, we modelled RT from the random-dot motion task as a function of environmental enrichment using the Cognitive Reserve Index questionnaire (CRIq[74]) in the older adult cohort only. The CRIq is a previously validated semi-structured interview which assays levels of cognitive stimulation through the assessment of three domains of activity throughout an individual's lifetime: *Education, Work Activities,* and *Leisure Activities* (see methods for details). As the neuroprotective effects of EE are posited to accumulate over the course of a lifetime[77], we collected this information in the older cohort only. As expected, this model was statistically significant, and EE (the overall model) explained 20.5% of the variance of RT ($R^2_{adj}$ =.21, $F_{3,36}$=4.36, $p$=.01, partial $\eta^2$=.27). Consistent with previous work[34,75], this effect was driven by the CRI *Leisure* subscale, which accounted for independent variance in the modelling of RT (Standardized $\beta$=-.45, $t$=-3.13, $p$=.003, 95% CI [-4.98 -1.06]), such that older adults with greater exposure to enriched leisure activities exhibited faster visual response speeds (Fig. 2B). In contrast, neither CRI *Education* (Standardized $\beta$=.06, $t$=.41, $p$=.69, 95% CI [-2.73 4.10]) nor CRI *Work* (Standardized $\beta$=.31, $t$=1.94, $p$=.06, 95% CI [-.09 3.93]) accounted for independent variance in RT. In order to obtain accurate parameter estimates for the relationship between CRI *Leisure* and RT, not influenced by the non-informative signals, CRI *Leisure* was entered into a separate linear regression model. This model explained 13.2% of the variance (Cohen's $F^2$=.18) in RT (Standardized $\beta$=-.39, $t$=-2.63; $F_{(1,38)}$=6.93, $p$=.01, 95% CI [-4.61 -.60], partial $\eta^2$=.15, Fig. 3A).

Bayesian Linear Regression analyses modelling RT as a function of each CRI subscale provided additional support for the results of the frequentist statistics (Supplementary Table 4). Any model including CRI *Leisure* indicated a Bayes Factor at least 2.9 times more in favour of $H_1$ than $H_0$ (Supplementary Table 4). In contrast, Bayes Factors for both CRI *Work* and CRI *Education* (independently and combined) provided anecdotal evidence for the null hypothesis (i.e., there was no evidence to suggest that these factors account for independent variance in RT; all three $BF_{10}$<.88 and >.03). This suggests that an individual's leisure engagements help to mitigate age-related declines in visual response speed. An exploratory analysis conducted to investigate which specific aspects of leisure activities may have contributed to this effect implicated using modern technology ($t_{39}$=-4.37, $p$<.001), engaging in social activities ($t_{26.88}$=-4.49, $p$<.001), and attending events such as conferences, exhibitions and concerts ($t_{32}$=-3.98, $p$<.001; Supplementary Fig. 3, for further information see Supplementary Results 1 and Supplementary Results 2).

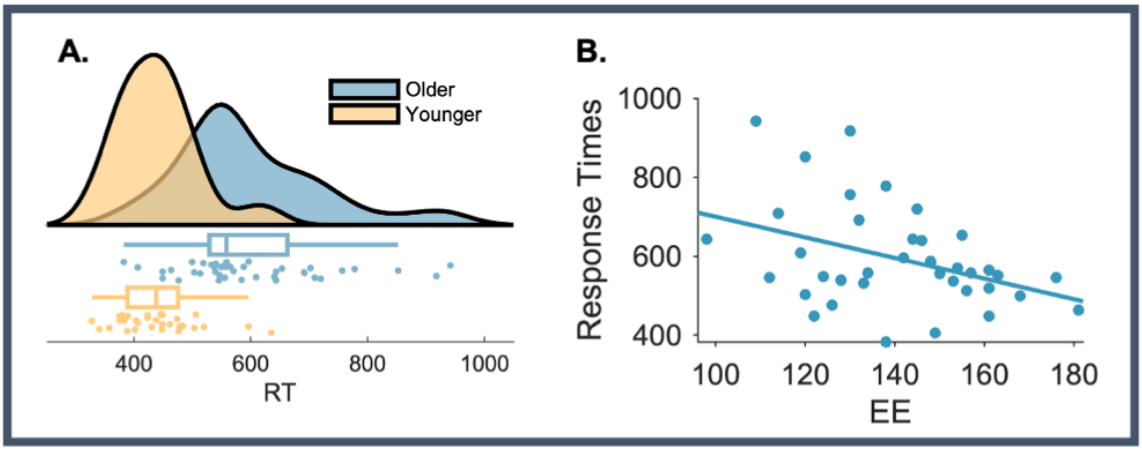


**Figure 2. Response Speed Measures on the Decision Task Sensitive to both Age and EE. A.** Healthy ageing was associated with markedly slower response times (RT) to perceptual targets, with large interindividual differences in response speed. During a variant of the random-dot motion task, older participants were, in general, slower to respond, relative to their younger peers, suggesting this measure was sensitive to age-related deficits in response speed. Each individual dot represents a participant (lower panel), and the distribution is captured by a violin plot for the two groups (upper panel). **B.** A lifetime of enriched environments (EE), captured by the composite score of Cognitive Reserve Index Questionnaire (CRIq[74]) varied according to individual differences in response speed in the older adults. This effect was driven by the Leisure subscale of the assessment which is visualised here as a function of RT.

## Individual differences in response speed are captured by sensory evidence accumulation rate

The analyses thus far have confirmed age-related differences in behavioural markers of response speed - a validated behavioural measure of cognitive resilience (see also Supplementary Results 4). We next sought to understand how each neural metric related to individual differences in response speed using a hierarchical regression model to isolate the contribution of each neural metric, over and above those which temporally preceded it.

To determine the explanatory power of the neurophysiological signals for predicting behaviour, over and above the known variance accounted for by older age, Age and the Age*RT interaction term were entered as nuisance variables (both centred to avoid multicollinearity) in the first step of the model. Unsurprisingly, both of these nuisance variables offered significant improvements in model fit, as compared with the intercept-only model ($R^2_{adj}$ =.50, $p$<.0005, Fig. 4). Neither N2c latency ($R^2_{adj}$ = .50, $p$=.77), N2c amplitude ($R^2_{adj}$ =.50, $p$=.21) nor CPP onset ($R^2_{adj}$ =.49, $p$=.87) offered any additional improvement in model fit.

CPP build-up rate significantly improved the model performance, accounting for an additional 12% of the variance ($R^2_{adj}$ =.61, $R^2_{change}$ =.12, $p$<.0005, Fig. 4, Table 1), such that steeper CPP slopes, indicative of a faster build-up rate of sensory evidence, were associated with faster response speeds. Adding CPP amplitude offered a further significant improvement in the model, such that individuals with lower CPP amplitudes showed faster RTs ($R^2_{adj}$ =.66, $R^2_{change}$ =.05, $p$=.002).

While LHB slope explained no additional variance in RT ($R^2_{adj}$ =.66, $R^2_{change}$ =0, $p$=.70), stimulus-aligned LHB peak latency significantly improved the fit, such that an earlier peak latency of this motor preparatory marker was associated with faster RT ($R^2_{adj}$ =.74, $R^2_{change}$ =.05, $p$=.002). Finally, adding LHB amplitude offered no significant improvement in the model ($R^2_{adj}$ =.71, $R^2_{change}$=0, $p$=.18; see Fig. 4 for the parameter estimates from this model). For additional analyses highlighting the validity of stimulus-aligned LHB latency as a marker of motor preparatory activity see Supplementary Results 4 and for detailed frequentist statistics from this modelling procedure see Supplementary Table 2.

In order to isolate the variables explaining independent variance in RT over and above that explained by other non-informative signals, age, the age*RT interaction, CPP build-up rate, CPP amplitude, and LHB peak latency were entered into a single separate linear regression model. When these five independent variables were included in the final model, they accounted for 71.4% of the variation in RT ($F_{5, 65}$=36.02, $p$<.0005; Cohen's $F^2$=2.85; Table 1). A post-hoc power analysis indicated that with 72 participants, 6 tested predictors (CPP build-up rate, CPP amplitude) and 2 control variables (Age and Age*RT interaction) 88.79% power was achieved (effect size $f^2$=.29, G*Power 3.1).

Finally, to further establish the utility of these signals as specifically sensitive to individual differences in ageing, we repeated this linear regression model (with CPP build-up rate, CPP amplitude, and LHB peak latency), just for the older cohort. This model accounted for 48.7% of the variation in RT ($F_{3, 37}$=13.66,

*p*<.0005) and CPP build-up rate (stand. β=-.66, *p*<.0005), CPP amplitude (stand. β =.27, *p*=.05) and LHB Latency (stand. β=-.31, *p*=.013) all accounted for independent variance in response speed.

**Table 1. Parameter estimates from the final linear regression model for reaction time (RT) as a function of the neurophysiological signals**

| Signal | Stand. *β* | *t* | *p* | *95% CI* |
|---|---|---|---|---|
| Age*RT | .26 | 3.80 | <.0005 | [.01, .02] |
| Age | .50 | 6.86 | <.0005 | [1.84, 3.35] |
| CPP build-up rate | -.45 | -5.16 | <.0005 | [-1139.40, -503.88] |
| CPP amplitude | .26 | 3.13 | .003 | [.94, 4.26] |
| LHB Latency | .24 | 3.39 | .001 | [.09, .36] |

*Note.* Age*RT, age, evidence accumulation (CPP) build-up rate, CPP amplitude, and LHB latency exerted partially independent influences on RT, together accounting for 71.4% of the variation (adjusted $R^2$ value) in RT. The absolute value of standardised (Stand.) *β* represents the importance of each predictor, independent of the unit of measurement. CI denotes confidence interval for *β*.

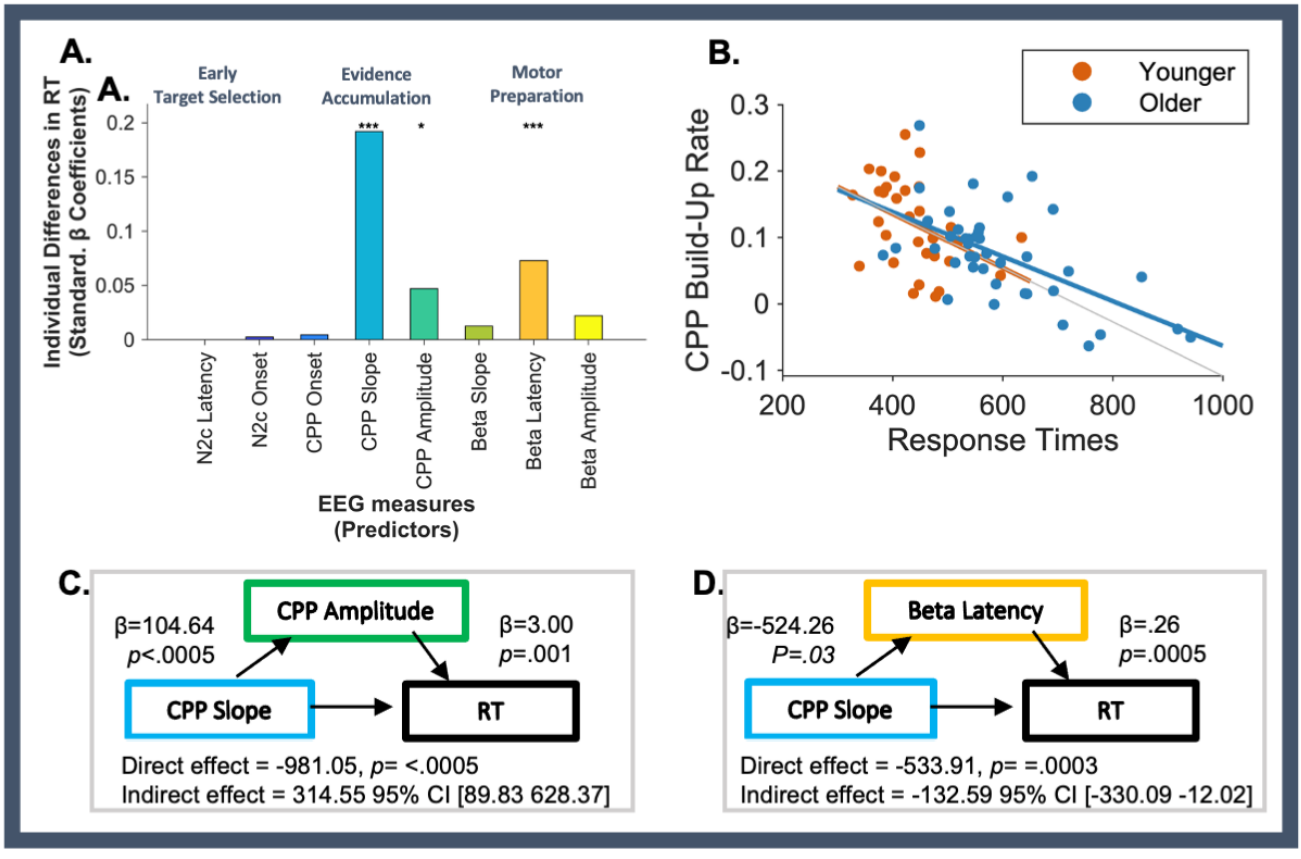


**Figure 4. Individual differences in response speed are captured by sensory evidence accumulation build-up rate. A.** Associations between response speed (RT) and the EEG variables. Results from the final regression model of RT are reported in Table 1. *Note* the absolute value of all standardised beta values were plotted for visualisation purposes and nuisance variables entered from the first step in the model are not visualised here. **B.** The relationship between CPP build-up rate and RT for older and younger adults. CPP build-up rate was directly associated with an individuals' response speed. Moreover, an individuals' capacity to accumulate sensory evidence indirectly impacts response speed by influencing CPP amplitude **(C)** and Beta latency (**D**), both of which mediate the association between CPP build-up rate and response speed.

Finally, to validate these results, we calculated the Bayes Factor Inclusion probabilities ($BF_{10}$) with a Bayesian linear regression using a Jeffrey–Zellner–Siow prior (JZS[78]; *r* scale covariates = 0.354), which can be interpreted such that $BF_{inclusion}$, or $BF_{10}$ values above 1 indicate strength of evidence in favour of the alternative and values below 1 indicate the strength of evidence in favour of the null. In keeping with the frequentist analyses, the Bayesian regression model for RT indicated strong support for the alternative hypothesis for age*RT ($BF_{10}$= 129.39), age ($BF_{10}$=13404.20), CPP slope ($BF_{10}$=1307.327), CPP amplitude ($BF_{10}$=12.64), and LHB latency ($BF_{10}$=50.81). There was no statistical evidence to suggest that N2c amplitude ($BF_{10}$=.74), N2c latency ($BF_{10}$=.55), CPP onset ($BF_{10}$=.75), LHB slope ($BF_{10}$=.74) or LHB amplitude ($BF_{10}$=.95) influenced RT (see Supplementary Table 3 for further details of this Bayesian linear model).

The findings above indicate that CPP build-up rate, CPP amplitude, and LHB latency exerted direct and partially independent influences over RT. On the basis of previous work, we assume that the impact of both CPP amplitude and LHB latency on RT, is, at least in part, determined by accumulated sensory evidence, reflected in temporally preceding CPP build-up rate[51,52,60,62]. We tested this by assessing whether the influence of CPP amplitude and LHB latency on RT was mediated by CPP build-up rate. In both cases, bootstrapped mediation analyses (5000 samples) indicated that this was the case (CPP build-up rate→CPP amplitude→RT indirect effect 281.98, bootstrapped SE 168.06, CI [18.00 669.46]; CPP build-up rate→ LHB latency→RT indirect effect -220.68, bootstrapped SE 102.48, CI [-459.99 -58.22; Fig. 4C, D]). This demonstrates that variability in age-related deficits in RT captured by CPP amplitude and LHB latency are dependent, at least partly, on individual differences in the rate at which sensory evidence can be accumulated. These results suggest that the CPP build-up rate constitutes a critical contributor to interindividual differences in response speed.

## Evidence accumulation build-up rate moderates the relationship between environmental enrichment and response speed

The results thus far demonstrate that individual differences in behaviour (response speed) are meaningfully captured both by an individual's level of environmental enrichment and by three task-related neural metrics (the build-up rate of evidence accumulation, amplitude of evidence accumulation, and timing of motor preparatory activity). This raises the possibility that the relationship between EE and response speed might differ according to individual differences in evidence accumulation build-up rate. To address this, we tested whether each of the three neural markers significantly moderated the relationship between CRI leisure and RT using three separate moderation models, *Bonferroni*-corrected for multiple comparisons (alpha .05/3 moderation models => alpha-corrected threshold = .016). These results revealed a specific moderating influence of CPP build-up rate on the association between EE (CRI Leisure) and RT, as evidenced by a CRI Leisure by CPP build up rate interaction (coefficient = 32.00, se=10.55, $t$=3.03, $p$=.005, CI [10.61 53.39]), which remained significant when covarying for (age; coefficient = 31.45, se=10.81, $t$=2.91, $p$=.006, CI [9.51 53.38], Fig. 5). In contrast, no moderating influence was observed for CPP amplitude (coefficient = .14, se=09.71, $t$=1.39, $p$=.17, CI [-.06 .33]) or LHB latency (coefficient = -.02, se=.006, $t$=-2.48, $p$=.02, CI [-.03 0]). Follow-up analyses exploring the

conditional effects of the predictor at values of the moderator revealed that the relationship between EE and RT was strongest in the older adults with shallower evidence accumulation build-up rates (Fig 5; CPP slope .0034, Coeff = -4.23, *SE*=1.17, *t*=-3.61, *p*=.0009, 95% CI = [-6.60, -1.86]; CPP slope .0724, Coeff=-2.02, *SE*=.80, *t*=-2.52, *p*=.02, 95% CI = [-3.65 -.39], CPP slope .1405, Coeff =.16, *SE*=.98, *t*=.16, *p*=.87, 95% CI [-1.84, 2.16]). These findings accord with previous findings from the neurocognitive reserve literature, whereby the discrepancy between markers of brain health and behaviour are accounted for by EE. As such, these findings further suggest that the CPP build-up rate captures meaningful information relating to the neurophysiological health of the ageing brain.

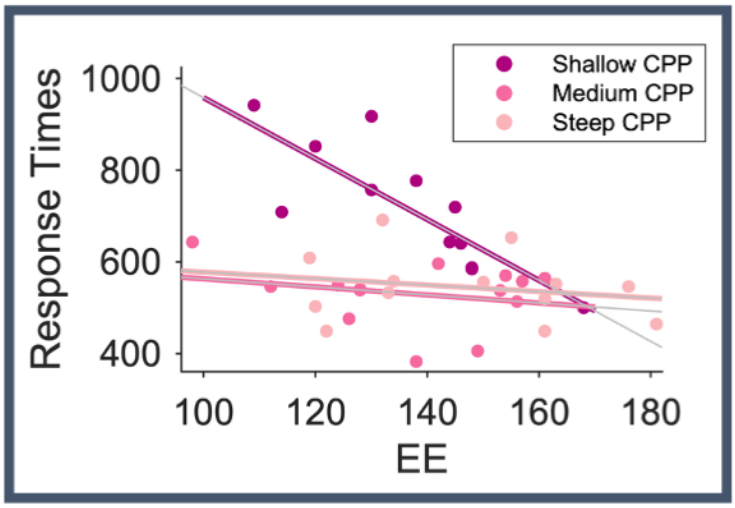



**Figure 5. Moderation model demonstrating the relationship between EE and RT as moderated by CPP build-up rate.** *Note* all analyses were conducted using continuous variables but are visualised here with three bins of equal size for CPP build-up rate.

**Table 2. Results from a Regression Analysis Examining the Moderation of the Relationship between RT and Exposure to Environmental Enrichment in Older Adults by Neural Metrics of Evidence Accumulation Rate**

| | | Coeff | *SE* | *t* | *p* | *95% CI* |
|---|---|---|---|---|---|---|
| Intercept | $i_1$ | 1259.45 | 163.17 | 7..72 | <.0005 | [928.53 1590.38] |
| CPP build-up rate (X) | $b_1$ | -5420.91 | 1493.02 | -3.63 | .0009 | [-8448.95 -2392.88] |
| EE (M) | $b_2$ | -4.34 | 1.20 | -3.62 | .0009 | [-6.76 -1.91] |
| CPP build-up rate ×EE (XM) | $b_3$ | 32.00 | 10.55 | 3.03 | .0045 | [10.61 53.39] |

$R^2$=.53, $MSE$=8102.33, $F_{3,36}$=13.55, $p$<.0005

## Feasibility of EEG markers of evidence accumulation build-up rate as a scalable proxy for neurocognitive health

Our findings provide evidence that the CPP build-up rate is mechanistically linked to an extensively validated marker of neurocognitive health – response speed – in older adults. This invites the possibility that this neural marker may be used by large-scale studies as an objective, cost-effective neurophysiological marker of ageing brain health. Both our results presented here, and a large body of previous research e.g.[41,60,62,68], has measured the CPP using a single electrode (most typically 'electrode *Pz*'). This affords clear benefits for reliably assessing this signal using low-density electrode arrays with either in-lab or portable EEG systems. Determining the minimum number of trials that permits a reliable measurement of CPP parameters, such as the CPP build-up rate, is therefore crucial for facilitating eventual clinical translation.

To determine this, we performed an analysis with a subset of participants who completed at least 9 task blocks, all of whom had a minimum of 129 valid response-locked ERP trials (see methods). We first created new estimates of both RT and CPP build-up rate by randomly selecting *N* trials (either 20, 40, 60, 80, 100, or 120) from the total pool of 129 trials. We repeated this random data-sampling using *N* trials, 1000 times for each bin size. Accordingly, for each participant, we derived 1000 estimates of RT and CPP build-up rate for each of the six trial sizes (Fig. 5.A, B). We then tested whether the likelihood that the estimates of RT and CPP build-up rate were more likely to deviate from the *true* mean estimates with reduced trial numbers. We addressed this question using two approaches. First, we calculated the signal to noise ratio (SNR) of the CPP build-up rate and RT (calculated as mean / standard deviation) and ran two repeated measures ANOVAs (again with trial bin as the repeated measure). This analysis revealed a significant main effect of bin size for both RT ($F_{5,4995}$=3328.28, $p$<.0005, partial $\eta^2$ =77) and CPP build-up rate ($F_{5,4995}$=247.84, $p$<.0005, partial $\eta^2$ =.20). In both cases the data were best explained by a linear fit (RT: $F_{1,999}$=9351.03, $p$<.0005, partial $\eta^2$ =.9, CPP build-up rate: $F_{1,999}$=688.82, $p$=<.005, partial $\eta^2$ =.41), indicating that increasing the number of trials significantly improved the signal to noise ratio. To verify this pattern of results, we ran Kolmogorov-Smirnov tests on the mean estimates of CPP build-up rate and RT to assess whether the cumulative distribution function (CDF) increased with each reduction in trial number. These results demonstrated that, in general, when the number of trials was reduced from 120 the width of the distribution (CDF) changed, as can be observed in Fig 5. A, B; KS <.05 for 120 trials vs all other number of trials; see Supplementary Table 7. This pattern of results demonstrates the expected effect that by reducing the number of trials, we increase the likelihood that estimates of both RT and CPP build-up rate deviate from the *true* mean estimates. Our critical question here, however, is at what level of SNR do we obtain reliable and behaviourally meaningful estimates of the relationship between RT and evidence accumulation build-up rate.

In the aforementioned results, we demonstrated a large effect size for the relationship between CPP build-up rate and RT (Pearson's *r*)= -.60. Cohen's (1988) cut-off for a large effect size is .5. As such, we defined the minimum number of trials at which a reliable CPP estimate can be derived as the number at which we can observe a strong effect size (i.e., an effect size greater or equal to .5) for the relationship between RT and CPP build-up rate. To investigate this, we calculated the direct relationship, using Pearson's correlation, between CPP build-up rate and RT for each of the 1000 permutations for each of the 6 bin sizes (20 up until 120 trials; Fig 5C). We then ran a Bayesian one sample *t*-test to test whether the estimates of effect size (*r*) for each bin size were significantly larger than -.5. We found infinite support for the alternative hypothesis that the effect sizes for the relationship between RT and CPP build-up rate with 120, 100, 80, 60, and 40 trials were larger than .5 (all $BF_{01}$=∞; see descriptive statistics Table 3). However, this was not the case for the estimates derived using 20 trials. Here, Bayes factor analyses revealed strong support for the null hypothesis ($BF_{01}$=648.42), i.e., that the estimates of effect size were not greater than -.5 (Fig 5C, Table 3). As such, these results indicate that 40 response-locked trials are the minimum number of trials that will allow for a reliable estimation of the CPP build-up rate / RT relationship. With current paradigm timings, 40 trials could be obtained in less than 5 minutes, highlighting the potential for isolating reliable EEG metrics of evidence accumulation over relatively short time scales. For detailed calculations of these timings (allowing for both variability in behavioural performance and quality of the EEG data) see Supplementary Results 3.

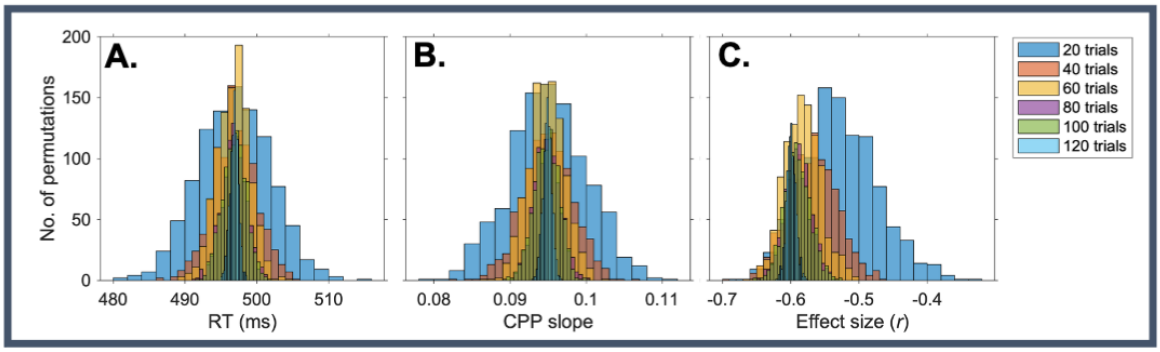



**Figure 5. A-C. Reliable Estimates of the Relationship between RT and Evidence Accumulation Build-Up Rate Can be Obtained With Reduced Trial Numbers**. For each participant we randomly selected 1000 estimates of RT (**A**) and CPP build-up rate (slope; **B**) for each of the six trial bin sizes (see legend). Reducing the number of trials reduced the signal to noise ratio and increased the likelihood that estimates of both RT and CPP build-up rate deviated from the *true* mean estimates (A, B). Critically, strong effect sizes (>.50) for the relationship between CPP build-up rate and RT were observed with as few as 40 trials (**C**) suggesting that this neurophysiological marker of sensory evidence accumulation may be developed as a translatable assessment of brain health for older adults.

## DISCUSSION

Here, we provide direct support for the hypothesis that build-up rates of sensory evidence accumulation are a critical neurophysiological mechanism underpinning the preservation of response speed in older adults. First, sensory evidence accumulation was not only directly related to response speed in older adults but also indirectly impacted performance by modulating subsequent neurophysiological processes, namely the decision criterion and the timing of the motor response. Second, consistent with the concept of neurocognitive reserve, a lifetime of EE offset age-related deficits in response speed. Critically, CPP slope moderated this association, such that the mitigating influence of EE on age-related declines in response times was most pronounced for individuals with relatively less efficient evidence accumulation (shallower build-up rates of CPP). This suggests that evidence accumulation build-up rates may offer rich information about which older individuals may benefit most from engaging with enriched environments.

The results presented here are in keeping with the concept of cognitive reserve as defined by a recent consensus paper[13] (but see[12,15,16]), whereby the proxy of reserve (here EE captured by the CRIq) exerts a moderating influence on the relationship between markers of brain health and cognitive function[13]. Our findings show that when evidence accumulation build-up rates are relatively shallower, individuals with relatively higher EE can nonetheless maintain faster response speeds than those with lower EE. One of the predominant principles of cognitive reserve is that high EE individuals are less reliant on established markers of brain health for facilitating behaviour. As such our findings accord with a large body of work in both healthy and pathological ageing conditions demonstrating that EE facilitates a neuroprotective buffer to cognitive function in spite of objective markers indicative of poor brain health (e.g. grey matter atrophy in healthy individuals[17], amyloid plaques and tangles in Alzheimer's patients[18]).

The mechanisms by which a lifetime of EE facilitates the preservation of cognitive function are unclear[12,13,15,16] and an important question for future work will be to understand the neurobiological substrates. Emerging evidence suggests that connectivity throughout select neural networks (e.g. functional segregation of resting state brain networks[80]) and structural (white matter integrity[81]) may be candidate neurobiological mechanisms. Specifically, a number of studies have shown that connectivity within the fronto-parietal networks (FPN) accounts for substantial inter-individual variability in neurocognitive resilience in older adults (e.g.[82–85]). Recently, we have shown in healthy younger adults that individual differences in connectivity within the dorsal FPN (white matter macrostructural organisation of the superior longitudinal fasciculus (SLF), and resting state functional connectivity within the dorsal FPN) varied according to the CPP build-up rate[62]. In older adults, increasing evidence suggests that the SLF varies according to both levels of EE[85], and risk factors for neurocognitive decline (e.g.[84]). As such, an intriguing question for future work is whether EE might act to alter the white matter structure of the SLF to preserve the efficiency of sensory evidence accumulation rates in later years.

A critical question for public health and neurorehabilitation is precisely what types of engagement are particularly effective for promoting neurocognitive health. Here, we find that engaging in leisure activities, particularly the use of new technology, engaging in social activities, and attending events (including conferences and exhibitions) drove relationships with response speed. The focus of our work was to explore the neural bases of epidemiologically defined markers of cognitive health (i.e., response speed and EE)[17,18]. However, we hope that our results will spur the development of novel interventions based on enriching lifestyle factors to better understand scalable, affordable public health interventions to induce lasting, positive changes in ageing brain function and cultivate resilience to cognitive decline.

Although slowed response times are often seen in healthy neurocognitive ageing (e.g.[25,27]), it is not the case that older adults show response time deficits across all tasks. Both modelling and neurophysiological

work has demonstrated that age-related differences in drift rate, CPP build-up rate, and response times are task specific (e.g.[41], see[50] for a review). A key insight from decision modelling work with older adults has been that slowed RTs may not relate purely to sluggish information processing but might actually reflect a strategic preference for greater caution reflected in higher decision bounds (e.g.[35–37]). We however found no evidence to suggest that older adults adopted a high decision criterion and, in fact, found weak evidence that older adults in our cohort reached a lower bound than their younger counterparts. Thus, using the CPP as a neurophysiological measurement of the evidence accumulation process, we provide additional support for recent observations that increased decision bounds in older adults are not generalisable to all scenarios[41]. Although neural metrics of the decision bound accounted for independent variation in response speeds, this relationship was contingent on the build-up rate of the CPP, such that slower build up rates of sensory evidence corresponded to lower decision bounds. As such, our findings indicate that response speed deficits obtained on an easy detection task in older adults result from a core deficit in the formation of perceptual decisions, as opposed to a more cautious approach to the decision-making process.

Consistent reports of age-related deficits in motor preparation have been reported (see[50] for review). The findings presented here demonstrate that age-related slowing in motor preparatory activity, indexed by later timing of activity in the beta band, are at least partially attributed to slower build-up rates of sensory evidence accumulation. Motor difficulties in neuropathological conditions (e.g. Parkinson's disease) arise from progressive degeneration within the motor system but have been shown to improve with interventions targeting higher order cognitive areas[86,87]. Our findings put forward a neurophysiological mechanism which could facilitate such effects, potentially through strengthening of white matter dorsal fronto-parietal pathways[55].

Finally, identifying the precise stage of information processing driving slowed response speed with aging might hold valuable prognostic information and could provide a sensitive addition to future large-scale epidemiological and translational studies. Here, we provide mechanistic evidence from a targeted and comprehensive EEG analysis to promote such further studies. In our current and recent investigations of the CPP, we have measured this signal from a single EEG electrode located over centro-parietal scalp regions (e.g.[41,62]). We present further evidence here that we can obtain reliable (large effect sizes) and meaningful (strongly predictive of response speed) measurements of the CPP build-up rate with as few as forty trials. Taken together, our work suggests that measuring the CPP via low density and potentially portable EEG, might have significant translational value. This would provide an objective, cost-effective, and clinically translatable phenotype that might be used to assay an older individual's vulnerability to future cognitive decline, monitor the success of treatment protocols (e.g. drug discovery trials), provide a brain-based target for neurorehabilitation interventions (e.g. closed-loop neurofeedback training, and non-invasive brain stimulation), or isolate an objective neurophysiological signature to investigate network dynamics underpinning response speed deficits (e.g.[90]). Future neuroimaging work could test these hypotheses directly by incorporating short EEG assessments of decision making.

Taken together our findings suggest that neural metrics of evidence accumulation build-up rate index an important facet of neurocognitive vulnerability in the ageing brain. Moreover, they suggest that, akin to grey matter atrophy measured with fMRI, CPP build-up rate holds promise as an EEG marker indexing a critical facet of neurophysiological vulnerability of the ageing brain that could be incorporated into large scale epidemiological studies.

# METHODS

## EXPERIMENTAL MODEL AND SUBJECT DETAILS

Seventy-eight healthy volunteers were recruited for this study. Two older adults were excluded due to age ranges more than two standard deviations from the mean (these participants were originally recruited as age-matched controls for a parallel brain injury study). A further four older participants were excluded from analysis for various reasons: one was ambidextrous, one was experiencing a current depressive episode and two had scores of 19 and 21, respectively, on the Montreal Cognitive Assessment (MoCA[79]), suggesting possible cognitive impairment. The final sample included 31 and 41 older participants (see Table 2 for demographic information). All participants reported being right-handed, had normal or corrected to normal vision, had no history of neurological or psychiatric disorder, and had no head injury resulting in loss of consciousness. Ethical approval was obtained from the Monash Health and Monash University Human Research Ethics Committee prior to the commencement of the study. The experimental protocol was approved and carried out in accordance with the approved guidelines. All participants were volunteers naive to the experimental hypothesis being tested and each provided written informed consent.

**Table 2. Demographic Information Reported values are *M* (*SD*)**

| | **Age (*yrs*)** | **Gender** | **Education (yrs)** | ***MoCA*** |
|---|---|---|---|---|
| **Experiment 1** | | | | |
| Younger Adults (*N*=31) | 23.65 (2.87) | 17 female (54.80%) | 15.90 (2.27) | *NA* |
| Older Adults (*N*=41) | 72.41 (5.61) | 26 female (63.40%) | 16.49 (3.48) | 27.46 (1.75) |

## METHOD DETAILS

### Neurophysiological investigation of response speed

Electroencephalography (EEG) was recorded continuously while participants performed a variant of the random-dot motion perceptual decision-making task (Fig. 1[52,68–70]) During this task, participants fixated centrally and monitored two patches of 150 moving dots (each dot = 6x6 pixels), presented peripherally in each hemifield. During random motion, these dots were placed randomly throughout the patch on each frame. During coherent motion, within one hemifield a proportion (90%) of the dots was randomly selected on each frame to be displaced in either a downward or upward direction on the following frame, with a motion speed of 5° per second. Targets were defined by this seamless transition from random motion to coherent motion (Fig. 1; please note, images in figures 1, 2, and 3 are composite images). Participants signalled target detection with a speeded button press using their right index finger (RT). Targets were separated by intervals of random motion of 1.8, 2.8, or 3.8 s (randomized throughout each

block). Targets remained on the screen for 1s, or until the participant pressed the button indicating their detection of coherent motion. The 12 possible trial types (each a combination of one of the 3 periods of random motion, 2 target locations, and 2 coherent motion directions) occurred in a pseudorandom order with the constraint that each different trial type arose twice every 24 trials. All younger adults ($N$=31) performed 8-9 blocks of the task. $N$=22 older adults (who were initially recruited to the study) similarly performed 8-9 blocks, while the remaining older adults (who were later recruited to the study, $N$=19) performed 4-5 blocks of the task at 90 % coherent motion, and a further 4-5 blocks of the task at 25% coherent motion, the latter of which was not analysed for the current study. Critically, a series of $t$ tests revealed there were no significant behavioural differences between the older participants recruited for the longer versus shorter task duration(RT $F_{1,.39}$ =1.72, $p$=.19; Accuracy $F_{1,39}$=.02, $p$=.88) or any of the neurophysiological markers (N2c amplitude $F_{1,39}$ =.08, $p$=.77; N2c latency $F_{1,39}$=-.10, $p$=.76; CPP onset $F_{1,39}$=.82, $p$=.37; CPP slope $F_{1,39}$=.67, $p$=.42; CPP amplitude $F_{1,39}$=.11, $p$=.74; LHB slope $F_{1,39}$=.90, $p$=.35), LHB amplitude $F_{1,39}$=.52, $p$=.48), or LHB latency $F_{1,39}$=.0, $p$=.99). As such the data were combined to examine the impact of environmental enrichment on neural and behavioural signatures of response speed. All participants were given a short break of 30-60 s between each block. An SR Research EyeLink eye tracker (Eye- Link version 2.04, SR Research/SMI) recorded eye movements, to ensure that participants maintained fixation. The centre of each random-dot motion patch was at a visual angle 10° either side and 4° below the fixation square; each patch covered 8° visual angle and consisted of 150 6 x 6 pixel white dots. If a fixation break occurred during a trial (either a blink or a gaze deviation >4° left or right of centre, detected via EyeLink1000, SR Research Ltd), the task halted (stationary dots). Once fixation returned to the central fixation dot, the trial restarted. The fixation dot remained on screen throughout the entire task; however, the two peripheral patches were only present when the trial was initiated by the participant's fixation on the central point. The task was run using MATLAB (MathWorks) and the Psychophysics Toolbox extensions[91–93].

### EEG pre-processing

Continuous EEG was acquired from 64 scalp electrodes using a BrainAmp DC system (Brain Products), digitized at 500 Hz. Data were processed using a combination of custom scripts and EEGLAB[94] routines implemented in MATLAB (MathWorks). A 35 Hz low-pass filter was applied to the data off-line using a fourth-order Butterworth filter, noisy channels were interpolated via spherical spline, and the data were re-referenced to the average reference. Epochs were extracted from the continuous data from -200 to 1500 ms around target onset. For both the ERP and LHB signals, the epochs were baselined with respect to 100 to 0 ms before target onset. A trial was excluded from the analysis if any of the following conditions applied: (1) if RTs were ≤150 ms (pre-emptive responses) or ≥ 1800 ms (responses after coherent motion offset); (2) if the EEG from any channel exceeded 100 μV during the interval from 100 ms before target onset to 100 ms after response; or (3) if central fixation was broken by blinking or eye movement 3° left or right of centre, during the interval between 100 ms before target onset and 100 ms after response. Please note, EyeLink data were not saved for $N$=5 out of the $N$=41 older adults due to a technical error and this final step was therefore not included for this subset of participants. Nonetheless fixation was monitored in real-time during task performance as described in the preceding section so no trials with eye movements >4° from centre were included. To minimise the interaction between overlapping ERP components, the data were subjected to Current Source Density transformation[95].

The N2c component was measured contralateral to the target location, respectively, at electrodes P7 and P8[68,70], and the CPP was measured at electrode Pz[51,52,59,68,70]. The N2c and CPP signals were aggregated to average waveforms as a function of target hemifield for each participant. N2c latency was identified as the time point with the most negative amplitude value in the stimulus-locked waveform between 150-400 ms, whereas N2c amplitude was measured as the mean amplitude inside a 100 ms window centred on the

stimulus-locked grand average peak of the N2c collapsed across hemifield[70]. Onset latency of the CPP was measured by performing running sample point by sample point t tests against zero using a 25ms sliding window across each participant's stimulus-locked CPP waveforms. CPP onset was defined as the first point at which the amplitude reached significance at the 0.05 level for 45 consecutive points[70,96,97]. CPP build-up rate was defined as the slope of a straight line fitted to the response-locked waveform[51,52], with the time window defined individually for each participant from -150 to 50 ms post-response. CPP amplitude was measured as the mean amplitude between -50 and +50ms around the participants' individual response.

### Data processing

Outliers were defined in SPSS using the interquartile range (IQR), separately for the younger and older adults. The interquartile range is the 3rd quartile (75th percentile) minus the 1st quartile (25th percentile). A value was identified as an outlier if either of the following conditions were met: if the value was <25th percentile - 1.5*IQR or if the value was >75th percentile + 1.5*IQR. Using this method, no outliers were identified.

### Assessment of Environmental Enrichment

Participants completed the Cognitive Reserve Index questionnaire (CRIq)[74], a standardised semi-structured interview designed to estimate an individual's level of lifetime cognitive enrichment through a formal computational model. This model encompasses an individual's education, work and leisure activities across the lifespan with consideration given to the participant's age, providing both an overall age-stratified and standardised Cognitive Reserve Index (CRI) and individual standardised subscale scores for each of the three components. One participant did not complete the CRIq due to time constraints.

Participants first reported the number of years in which they had engaged in formal education and additional vocational training. All occupations held since the individual was 18 years old were categorised using the five-point point scale provided by the CRI. These ranged from low skilled manual work (e.g. level 1 includes occupations like call centre operator, and gardener) to highly responsible or intellectual occupation (e.g. level 5 includes managing director of a big company or surgeon). Participants were additionally asked about their involvement in leisure activities that may be repeated with varying frequencies over the lifetime, including but not limited to reading, volunteering, socialising, managing accounts, going on holidays/trips. Activities were grouped into weekly, monthly, annual and fixed frequency activities, and then into whether they were completed never, rarely, often or always, and for how many years of life. Participant engagement in each of these domains is summarised in Supplementary Tables 10-12.

# STATISTICAL ANALYSIS

## The Relationship between Age, Behaviour and EEG

To assess age-related differences in behaviour, two one-way ANOVAs were conducted on Accuracy and RT. Next, to test whether the older and younger adults differed across N2c, CPP, and LHB dynamics, eight one-way ANOVAs were conducted with the EEG variables (N2c latency, and amplitude, CPP onset, build-up rate, and amplitude, LHB build-up rate, LHB amplitude, and LHB latency) as dependent variables, and age as a factor. To assess whether inter-individual differences in RT on the perceptual decision-making paradigm (RT) varied as a function of EEG signals of perceptual decision-making, the EEG parameters which differed in older versus younger adults ($BF_{10}$>1) were each added sequentially into regression models in a hierarchical fashion[68]. Order of entry was determined by the temporal order in the perceptual decision-making process: early target selection (N2c latency); evidence accumulation (CPP onset, build-up rate, and amplitude), and motor preparation (LHB amplitude, and LHB latency). This hierarchical entry method was implemented to assess whether each of the separate neurophysiological signals improved the model fit for RT over and above the signals that temporally preceded them. All neurophysiological signals that improved the model fit for RT were entered into a separate regression model to obtain accurate parameter estimates. The Age*RT interaction term was entered as the first predictor in the model, and Age was entered as the second predictor. Age, and the product term (Age*RT) were both centred (all raw scores for each participant were subtracted from the mean score of the variable) to reduce multicollinearity. Please note all statistical tests were two-sided. Effect sizes of regression models were calculated using Cohen's $F^2$ using the following formula: ($R^2$/(1-$R^2$)). Behavioural data was visualised using *RainCloudPlots* for MATLAB[98,99]. The EEG signals were visualised using GRAMM for MATLAB[100]

## Moderation Models

To elucidate the moderating effects of evidence accumulation rate, amplitude, and beta latency on the relationship between EE and response speed, three moderation analyses were performed using the PROCESS computational toolbox[101,102], *Bonferroni*-corrected for multiple comparisons (alpha .05/3 moderation models).

## Confirmatory Bayesian Analyses

For all Bayesian modelling, results were compared with the null model, and JASP default settings were used (JZS prior, regression analyses: r scale .354, ANOVA analyses: r scale fixed effects .5). BF10 values are reported throughout and can be interpreted such that values above 1 indicate strength of evidence in favour of the alternative and values below 1 strength of evidence in favour of the null hypothesis.

## Minimum Trial Analysis

The minimum trial analyses included all participants (N=53) who completed 8 or more blocks of the task. One individual was identified as an outlier (>2SD from the mean) with regards the number of trials included (N=109 trials) and was excluded, therefore resulting in a total of *N*=52 participants. All of these 52 participants had a minimum of 129 valid response-locked trials, which we used to investigate remaining questions (*M*=183.06; *SD*=19.95; range 129-207).

For the Bayes Factor analyses, default priors in JASP were used (Cauchy prior of .707), and the alternative hypothesis was set at measure 1 ~= measure 2.

**Table 3. Effect sizes for the relationship between RT and CPP build-up rates for 6 different trial sizes.** Only at 20 trials did Bayes factor analyses reveal strong support for the null hypothesis that estimates of effect size were not greater than .5.

| *r* CPP build-up rate-CPP | Mean | SD |
|---|---|---|
| 20 trials | -0.527 | 0.055 |
| 40 trials | -0.568 | 0.035 |
| 60 trials | -0.583 | 0.026 |
| 80 trials | -0.590 | 0.019 |
| 100 trials | -0.591 | 0.019 |
| 120 trials | -0.599 | 0.007 |

# SUPPLEMENTARY RESULTS 1

**Effects of environmental enrichment are independent of level of IQ**

To investigate whether the observed relationship between EE and response speed could reflect individual differences in IQ, we estimated premorbid intelligence in a subset of participants (n=36). There was no direct association between IQ and response speed. Critically, the relationship between EE and response speed remained significant after covarying for IQ, indicating that the relationship between enrichment of cognition was not due to individual differences in intelligence.

More specifically, a subset (*N*=36) of the older adults completed word reading tasks commonly used to estimate premorbid IQ based on the Wechsler Adult Intelligence Scale – Fourth Edition (WAIS-IV)[103]. Of these individuals *n*=17 completed the Test of Premorbid Function (ToPF), while the other *n*=19 completed the National Adult Reading Test (NART)[104], using updated norms. Outliers were defined in SPSS using the IQR, consistent with the main analyses, separately for both cohorts of older adults. One outlier was detected for the ToPF and was subsequently removed and imputed using the mean value from their group. The two cohorts differed on estimates of IQ score derived using the different word lists ($t_{34}$=2.09, $p$=.04), with a subsequent Bayesian independent samples *t* test suggesting moderate evidence for a difference between the two groups, $BF_{10}$ =3.26. This is likely attributable to established differences in the estimations produced by the measure. Nonetheless, we considered it useful to investigate using the data available to us, whether our effects could be attributed to a relationship between response speed and IQ. For this, we ran a hierarchical linear regression of RT, with IQ entered as the first step in the model. IQ did not account for a statistically significant proportion of the variance in RT, indicating no direct influence of premorbid intelligence on response speed ($F_{(1,34)}$ =.07, $p$=.80, $R^2_{adj}$ =-.03). Critically, when the CRI sub-scales were added to the second step in this hierarchical model, the relationship between EE and response speed remained significant ($R^2_{adj}$ =.17, $R^2_{change}$ =.26, $p$=.02; $F_{(4,31)}$ =2.77, $p$=.04), demonstrating that the observed relationship between EE and response speed cannot be attributed to IQ.

# SUPPLEMENTARY RESULTS 2

**Characterisation of older adult participation in leisure activities**

To explore the leisure activities which may drive the apparent effect of EE on response speed, we compared the activities of those with higher versus lower levels of lifetime leisure engagement. To do so, we first devised two groups of older adults based on a median split of their engagement in leisure activities. Those with CRI *Leisure* subscores above the overall median score of 138.00 were considered *High Engagement* (*n*=20), while those with a subscore equal to or lower than 138.00 were deemed *Low Engagement* (*n*=21). We then examined each participant's responses to individual activities on the CRIq. Participants first indicated whether they participated in the activity *Often/Always*, or *Never/Rarely* over the course of their lifetime, and further specified for how many years they engaged *Often/Always*. For participants who engaged in an activity *Often/Always* for at least one year of life, we calculated separate values for their engagement in each activity, representing the percentage of life years spent engaging in each activity since 18 years of age using the following formula: ((Years of activity)/(Age – 18)) * 100. Note that rounding within the CRIq causes some individuals to exceed 100.00% of life spent participating in a given activity. For example, if an individual worked as a nurse for 3 years, this is rounded up to 5 years, as per the standardised questionnaire administration guidelines. Finally, we determined the percentage of individuals in each group who engaged in each activity *Often/Always*, and the mean

percentage of life spent engaging in each activity for each group, the results of which are demonstrated in Supplementary Table 5.

Subsequently, we investigated group differences through a series of *t* tests. Those with *High Engagement*, compared to those with *Low Engagement* in leisure activities, spent a significantly greater proportion of their lives using modern technology ($t_{39}$=4.37, $p$<.001), engaging in social activities ($t_{26.88}$=4.49, $p$<.001), and attending events such as conferences, exhibitions and concerts ($t_{32}$=3.98, $p$<.001). Using Bonferroni correction resulting in an adjusted $a$=.003, there were no significant differences in cinema or theatre attendance ($t_{11.09}$=2.85, $p$=.01), vacationing ($t_{24.83}$=3.11, $p$=.01), driving ($t_{22.96}$=2.11, $p$=.05), reading books ($t_{28.12}$=2.18, $p$=.04), or engaging in hobbies such as sports and games ($t_{37}$=2.31, $p$=.03). No other significant differences were found (all $p$>.05).

## SUPPLEMENTARY RESULTS 3

### Calculation of the time necessary to assess 40 trials of the CPP

Results from the minimum trial analyses indicate that a minimum of 40 trials would be sufficient to derive valid and behaviourally meaningful estimates of CPP build up rate. These trials are derived using the response-locked EEG signals, following data cleaning, for correctly identified target stimuli (coherently moving dots). Below we provide calculations both for the average time we expect necessary to obtain 40 valid trials, and for a 'worst case scenario'.

### Calculations using mean values

Mean accuracy was 96%. In order to get 40 valid response locked trials, participants would need to be administered an extra 4% (2 trials), i.e., 42 trials in total. We calculated the percentage of EEG trials which were rejected by data cleaning i.e., (rejected trials/(rejected trials + valid trials), and on average 15% of trials were excluded following the preprocessing steps. In order to obtain 42 **valid** EEG trials (post data cleaning), an additional 15% of data would need to be collected, so 48 trials in total.
In this case, we would present the participant with

- 48 coherent motion trials at 1 seconds each
    - = 48seconds total
- Each target trial would be preceded by random motion at three variable intervals (1.8s. 2.8s, & 3.8s; 48 targets/3 random motion periods =16 at each time period)
- [(1.8*16) +(2.8*16) + (3.8*16)]
- [28 + 44.8 + 60.8]
    - =133.6seconds
- Total time (48s +133.6s) = 181.6 seconds (3.03 minutes)

### Calculations using the worst-case scenario values (i.e., participants with lowest accuracy and noisiest EEG data)

The participant with the lowest accuracy on the task correctly identified 71% of targets, and as such in order to get 40 valid trials would need an extra 29% (12 trials), i.e., 52 trials in total. While, on average 15% of participant EEG trials were rejected during processing, in the noisiest dataset we rejected 44% of trials. In this scenario, in order to obtain 52 valid trials post cleaning we would need an extra 44% of data (i.e., 75 trials in total).

In this case, we would present the participant with

- 75 coherent motion trials at 1 seconds each
    - = 75 seconds total
- Each target trial would be preceded by random motion at three variable intervals (1.8s. 2.8s, & 3.8s; 75 targets/3 random motion periods =25 at each time period)
- [(1.8*25) +(2.8*25) + (3.8*25)]
- [45 + 70 + 95]
    - =210seconds
- Total time (75s + 210s) = 285 seconds (4.75 minutes)

We note that these times do not account for EEG set-up time. Future work should address the minimum participant preparation time and reliability of these results from low density, portable electrode arrays.

# SUPPLEMENTARY RESULTS 4

### Temporal Dynamics of Evidence Accumulation are Robust Age-Related Indicators

We next examined group-level differences in the eight electrophysiological markers using a series of one-way ANOVAs, *Bonferroni*-corrected for multiple comparisons (alpha .05/8 EEG components => alpha-corrected threshold = .006) and supplemented these with Bayesian analyses to indicate the strength of evidence in support of the null hypothesis. No statistically significant difference was observed between older and younger adults in the latency of early target selection signals (N2*c;* $F_{1,70}$ =2.75, $p$=.10, $BF_{10}$=.79, Supplementary Table 1 for plots and additional analyses see Supplementary Fig. 2), and although there was weak evidence to suggest that the amplitude of the N2c differed between groups ($F_{1,70}$ =6.01, $p$=.02, partial $\eta^2$=.08, $BF_{10}$=3.05, Supplementary Table 1, Supplementary Fig. 1A), this did not survive correction for multiple comparisons.

In line with recent reports[41], the older adults differed from their younger counterparts in metrics of evidence accumulation (the CPP). More specifically, timing delays were observed for several parameters of the CPP in older individuals; they showed a later onset (later CPP onset; $F_{1,70}$=14.8, $p$<.001, partial $\eta^2$=.18, $BF_{10}$=96.52, Supplementary Table 1, Supplementary Fig. 1B) and slower build-up rate (shallower CPP build-up rate (slope); $F_{1,70}$ =8.03, $p$=.006, partial $\eta^2$=.10, $BF_{10}$=6.90, Supplementary Table 1, Supplementary Fig. 1B). No groups differences were observed for the amplitude at response (CPP amplitude; $F_{1,70}$=5.88, $p$=.02, partial $\eta^2$=.08, $BF_{10}$=2.89, Supplementary Table 1). We did not observe differences in motor preparatory activity between the two groups (LHB build-up rate (slope) $F_{1,70}$=.31, $p$=.58, $BF_{10}$=.28; stimulus-aligned peak LHB latency $F_{1,70}$ =4.74, $p$=.03, partial $\eta^2$=.06, $BF_{10}$=1.81, LHB amplitude $F_{1,70}$=3.79, $p$=.06, partial $\eta^2$=.05, $BF_{10}$=1.22, Supplementary Fig. 1C). For additional LHB analyses, see Supplementary Results 5. Together the inferential and Bayesian statistics demonstrate that the CPP onset and build-up rate are robust age-related indicators.

# SUPPLEMENTARY RESULTS 5

### Stimulus-locked beta latency as a valid marker of motor preparatory activity

To verify that motor preparatory activity was accurately captured by our stimulus-locked measure of beta latency, and to exclude the possibility that EE could be impacting RT through an influence over motor preparatory activity, two response-locked beta metrics were derived and explored in relation to RT: response-locked beta slope (build-up rate) and response-locked beta amplitude (threshold; Supplementary Fig. 4 below). Beta slope was defined as the slope of a straight line fitted to the response- locked waveform, with the time window defined individually for each participant between 300 to 50 ms pre-response, and baselined to -450 to -350ms. Beta amplitude was measured as the mean amplitude of a 100ms window centred on a participants' response (i.e., -50 to +50ms around response). A stepwise linear regression model was used to identify which of the three beta measures (peak stimulus-locked latency, along with slope, and amplitude at the time of response) was the best predictor of RT (Criteria: probability of $F$ to enter <=.05, probability of $F$ to remove >=.1). The resulting model of RT included only stimulus-locked beta latency, indicating that this was the most appropriate EEG metric for capturing independent variance in RT (beta latency: standardized $\beta$=.54, $t$=5.25, $p$<.001, 95% CI [.28 .62]; beta slope: $\beta$=.10, $t$=.95, $p$=.34, beta amplitude $\beta$=-.12, $t$=-1.14, $p$=.26 model $F_{1,68}$=27.60, $p$<.001). In line with previous work (e.g.[51,62], this result suggests that beta latency is a valid marker of task-relevant motor preparatory activity accounting for independent variance in response speed.

# SUPPLEMENTARY TABLES

**Supplementary Table 1.** ***Note*** **all values signify mean (M) and standard deviations (SD). EEG signals that differed significantly between the older and younger adults (according to both Bonferroni-corrected frequentist and Bayesian analyses) are highlighted in bold.** ***Note*****, weak (anecdotal) evidence is provided for the group difference in N2c amplitude**

| | Younger Adults (*N*=31) | Older Adults (*N*=41) |
|---|---|---|
| N2c Amplitude (uV/m²) | -10.03 (8.35) | -15.98 (11.36) |
| N2c Latency (ms) | 266.16 (55.61) | 285.78 (44.81) |
| **CPP Onset (ms)** | **250.84 (80.39)** | **345.17 (117.16)** |
| **CPP Build-up Rate** | **.12 (.07)** | **.07 (.07)** |
| CPP Amplitude (uV/m²) | 24.32 (13.75) | 17.20 (11.16) |
| LHB Latency (ms) | 471.58 (98.18) | 549.02 (178.46) |
| LHB Slope | -.0059 (.0057) | -.0068 (.0067) |
| LHB Amplitude | -1.32 (1.26) | -2.04 (1.73) |

**Supplementary Table 2: Hierarchical linear regression model statistics examining how each neurophysiological marker contributed to RT, over and above the contributions made by those processes that temporally preceded.**

**ANOVA** (Dependent Variable: RT)

| Model | | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|---|
| Model 1: Predictors: (Constant), Age*RT | | | | | | |
| 1 | Regression | 98463.640 | 1 | 98463.640 | 6.242 | .015[b] |
| | Residual | 1088413.948 | 69 | 15774.115 | | |
| | Total | 1186877.588 | 70 | | | |
| Model 2: Predictors: (Constant), Age*RT, Age | | | | | | |
| 2 | Regression | 614865.329 | 2 | 307432.665 | 36.547 | .000[c] |
| | Residual | 572012.259 | 68 | 8411.945 | | |
| | Total | 1186877.588 | 70 | | | |
| Model 3: Predictors: (Constant), Age*RT, Age, N2c Amplitude | | | | | | |
| 3 | Regression | 628653.677 | 3 | 209551.226 | 25.151 | .000[d] |
| | Residual | 558223.911 | 67 | 8331.700 | | |
| | Total | 1186877.588 | 70 | | | |
| Model 4. Predictors: (Constant), Age*RT, Age, N2c Amplitude, CPP onset | | | | | | |
| 4 | Regression | 628654.248 | 4 | 157163.562 | 18.582 | .000[e] |

| | | | | | | |
|---|---|---|---|---|---|---|
| | Residual | 558223.340 | 66 | 8457.929 | | |
| | Total | 1186877.588 | 70 | | | |
| Model 5. Predictors: (Constant), Age*RT, Age, N2c Amplitude, CPP onset, CPP Slope | | | | | | |
| 5 | Regression | 768362.958 | 5 | 153672.592 | 23.867 | .000[f] |
| | Residual | 418514.630 | 65 | 6438.687 | | |
| | Total | 1186877.588 | 70 | | | |
| Model 6: Predictors: (Constant), Age*RT, Age, N2c Amplitude, CPP onset, CPP Slope, CPP Amplitude | | | | | | |
| 6 | Regression | 825207.343 | 6 | 137534.557 | 24.338 | .000[g] |
| | Residual | 361670.245 | 64 | 5651.098 | | |
| | Total | 1186877.588 | 70 | | | |
| Model 7: Predictors: (Constant), Age*RT, Age, N2c Amplitude, CPP onset, CPP Slope, CPP Amplitude, LHB Amplitude | | | | | | |
| 7 | Regression | 827509.280 | 7 | 118215.611 | 20.724 | .000[h] |
| | Residual | 359368.308 | 63 | 5704.259 | | |
| | Total | 1186877.588 | 70 | | | |
| Model 8: Predictors: (Constant), Age*RT, Age, N2c Amplitude, CPP onset, CPP Slope, CPP Amplitude, LHB Amplitude, LHB Latency | | | | | | |
| 8 | Regression | 886238.175 | 8 | 110779.772 | 22.846 | .000[i] |
| | Residual | 300639.413 | 62 | 4849.023 | | |
| | Total | 1186877.588 | 70 | | | |

**Supplementary Table 3: Bayesian linear regression model statistics examining how each neurophysiological marker contributed to RT. Note. $BF_{inclusion}$ values above 1 indicate the strength of evidence in favour of the alternative hypothesis and are highlighted in bold.**

**Posterior Summaries of Coefficients**

| | | | | | | 95% Credible Interval | |
|---|---|---|---|---|---|---|---|
| **Coefficient** | **Mean** | **SD** | **P(incl)** | **P(incl \| data)** | **BF inclusion** | **Lower** | **Upper** |
| Intercept | -2.429 | 8.337 | 1.000 | 1.000 | 1.000 | -17.937 | 13.996 |
| **Age*RT** | **0.013** | **0.004** | **0.500** | **0.992** | **129.391** | **0.005** | **0.019** |
| **Age** | **2.273** | **0.451** | **0.500** | **1.000** | **13404.200** | **1.347** | **3.098** |
| **CPP Build-up Rate** | **-759.195** | **179.370** | **0.500** | **0.999** | **1307.327** | **-1118.297** | **-439.427** |
| **CPP Amplitude** | **2.168** | **1.041** | **0.500** | **0.927** | **12.640** | **0.000** | **3.752** |
| **Beta Latency** | **0.221** | **0.074** | **0.500** | **0.981** | **50.810** | **0.074** | **0.391** |
| N2c Amplitude | -0.316 | 0.688 | 0.500 | 0.426 | 0.742 | -2.303 | 0.492 |
| N2c Latency | 0.011 | 0.108 | 0.500 | 0.354 | 0.549 | -0.188 | 0.309 |
| CPP Onset | 0.037 | 0.076 | 0.500 | 0.428 | 0.749 | -0.043 | 0.237 |
| LHB Slope | 791.174 | 1752.233 | 0.500 | 0.427 | 0.744 | -1543.816 | 5360.611 |
| LHB Amplitude | -4.823 | 7.777 | 0.500 | 0.488 | 0.954 | -22.822 | 3.141 |

**Supplementary Table 4: Bayesian Linear Regression Model the relationship between RT and Cognitive Reserve.**

**Model Comparison**

| Models | P(M) | P(M \| data) | $BF_M$ | $BF_{10}$ | $R^2$ |
|---|---|---|---|---|---|
| Null model | 0.250 | 0.077 | 0.251 | 1.000 | 0.000 |
| CRI Leisure + CRI Work + CRI Education | 0.250 | 0.380 | 1.837 | 4.917 | 0.266 |
| CRI Leisure + CRI Work | 0.083 | 0.317 | 5.104 | 12.311 | 0.263 |
| CRI Leisure | 0.083 | 0.110 | 1.358 | 4.269 | 0.154 |
| CRI Leisure + CRI Education | 0.083 | 0.074 | 0.874 | 2.860 | 0.190 |
| CRI Work | 0.083 | 0.022 | 0.253 | 0.873 | 0.065 |
| CRI Education | 0.083 | 0.011 | 0.121 | 0.421 | 0.020 |
| CRI Work + CRI Education | 0.083 | 0.009 | 0.102 | 0.358 | 0.067 |

**Supplementary Table 5** Frequency and duration of leisure activity engagement for those with high and low CRI Leisure (devised by median split).

| | Overall (n = 41) | | High CR-Leisure (n = 20) | | Low CR-Leisure (n = 21) | | Significant difference for proportion of time engaged |
|---|---|---|---|---|---|---|---|
| | Percentage of People Engaged | Proportion of Life Engaged | Percentage of People Engaged | Proportion of Life Engaged | Percentage of People Engaged | Proportion of Life Engaged | |
| **Activities with weekly frequency** | | | | | | | |
| Reading newspapers and magazines | 90.2% (*n*=37) | 82.89% (32.25) | 100.0% (*n*=20) | 89.28% (26.81) | 81.0% (*n*=17) | 75.38% (37.08) | ns. |
| Housework (cooking, ironing, washing, etc) | 97.6% (*n*=40) | 85.61% (32.85) | 100.0% (*n*=20) | 89.71% (33.06) | 95.2% (*n*=20) | 81.50% (32.97) | ns. |
| Driving (not biking) | 95.1% (*n*=39) | 97.34% (14.39) | 95.0% (*n*=19) | 102.02% (5.84) | 95.2% (*n*=20) | 92.89% (18.41) | * |
| Leisure activities (sports, hunting, dancing, cards, bowling, etc) | 95.1% (*n*=39) | 53.58% (31.65) | 95.0% (*n*=19) | 64.97% (19.84) | 95.2% (*n*=20) | 42.76% (30.11) | * |
| **Using new technologies (digital camera, computer, internet, etc)** | **100.0% (*n*=41)** | **72.46% (31.12)** | **100.0% (*n*=20)** | **90.53% (24.51)** | **100.0% (*n*=21)** | **55.26% (27.00)** | *** |
| **Activities with monthly frequency** | | | | | | | |
| **Social activities (parties/going out with friends, local community events, etc)** | **87.8% (*n*=36)** | **51.90% (38.67)** | **90.0% (*n*=18)** | **75.17% (38.25)** | **85.7% (*n*=18)** | **28.64% (21.65)** | *** |
| Cinema or theatre | 41.5% (*n*=17) | 48.25% (34.20) | 45.0% (*n*=9) | 66.79% (36.17) | 38.1% (*n*=8) | 27.38% (15.45) | ** |
| Gardening, handcraft, knitting, etc | 85.4% (*n*=35) | 79.81% (31.07) | 90.0% (*n*=18) | 78.58% (34.77) | 81.0% (*n*=17) | 81.11% (27.64) | ns. |
| Taking care of children or elderly | 63.4% (*n*=26) | 24.39% (13.43) | 60.0% (*n*=12) | 24.87% (13.84) | 66.7% (*n*=14) | 23.99% (13.57) | ns. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Volunteering | 73.2% | 25.39% | 80.0% | 26.11% | 66.7% | 24.57% | ns. |
| | (*n*=30) | (18.38) | (*n*=16) | (23.82) | (*n*=14) | (9.88) | |
| Artistic activities (playing an | 53.7% | 52.26% | 60.0% | 66.41% | 47.6% | 39.68% | ns. |
| instrument, painting, writing, etc) | (*n*=22) | (43.53) | (*n*=12) | (41.23) | (*n*=10) | (43.71) | |
| **Activities with annual frequency** | | | | | | | |
| **Exhibitions, concerts, conferences** | **82.9%** | **59.35%** | **100.0%** | **76.36%** | **66.7%** | **35.06%** | ******* |
| | **(*n*=34)** | **(35.84)** | **(*n*=20)** | **(30.60)** | **(*n*=14)** | **(28.51)** | |
| Holidays | 73.2% | 49.82% | 90.0% | 60.91% | 57.1% | 33.20% | ** |
| | (*n*=30) | (30.42) | (*n*=18) | (33.45) | (*n*=12) | (14.43) | |
| Reading books | 100.0% | 87.69% | 100.0% | 99.13% | 100.0% | 76.81% | * |
| | (*n*=41) | (34.83) | (*n*=20) | (19.26) | (*n*=21) | (42.65) | |
| **Activities with fixed frequency** | | | | | | | |
| Pet care | 73.2% | 53.14% | 85.0% | 58.91% | 61.9% | 45.60% | ns. |
| | (*n*=30) | (36.48) | (*n*=17) | (38.43) | (*n*=13) | (33.75) | |
| Managing one's bank account(s) | 100.0% | 94.20% | 100.0% | 99.65% | 100.0% | 89.00% | ns. |
| | (*n*=41) | (21.20) | (*n*=20) | (13.98) | (*n*=21) | (25.59) | |
| Having children | 82.9% | 2.82 | 70.0% | 2.64 | 95.2% | 2.95 | ns. |
| | (*n*=34) | (.94) | (*n*=14) | (.93) | (*n*=20) | (.94) | |

ns. $p>.05$; * $p\leq.05$; ** $p\leq.01$; *** $p\leq.001$, only these values pass the Bonferroni threshold for significance.

*Note.* All values represent *M*(SD). Values reported for having children are the percentage of people who reported having children, and the mean number of children, respectively.

**Supplementary Table 6.**

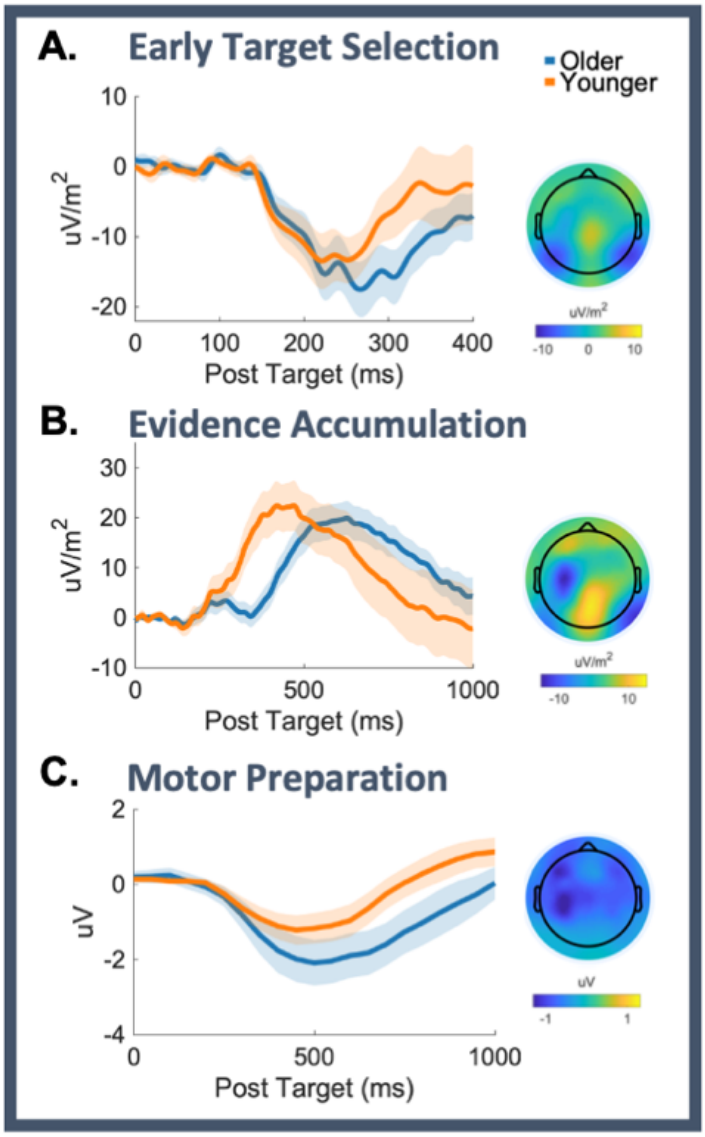
| | | $BF_{01}$ | error % |
|---|---|---|---|
| RT 120 trials | RT 100 trials | 26.185 | 0.004 |
| | RT 80 trials | 22.506 | 0.003 |
| | RT 60 trials | 7.360 | 0.001 |
| | RT 40 trials | 26.381 | 0.004 |
| | RT 20 trials | 28.076 | 0.004 |
| CPP 120 trials | CPP 100 trials | 6.434 | 9.348e -4 |
| | CPP 80 trials | 24.552 | 0.004 |
| | CPP 60 trials | 10.909 | 0.002 |
| | CPP 40 trials | 25.467 | 0.004 |
| | CPP 20 trials | 15.542 | 0.002 |

**Bayesian paired-sample t-tests comparing 120 trials with reduced bin sizes for both RT and CPP slope (build-up rate).**

Supplementary Table 7.

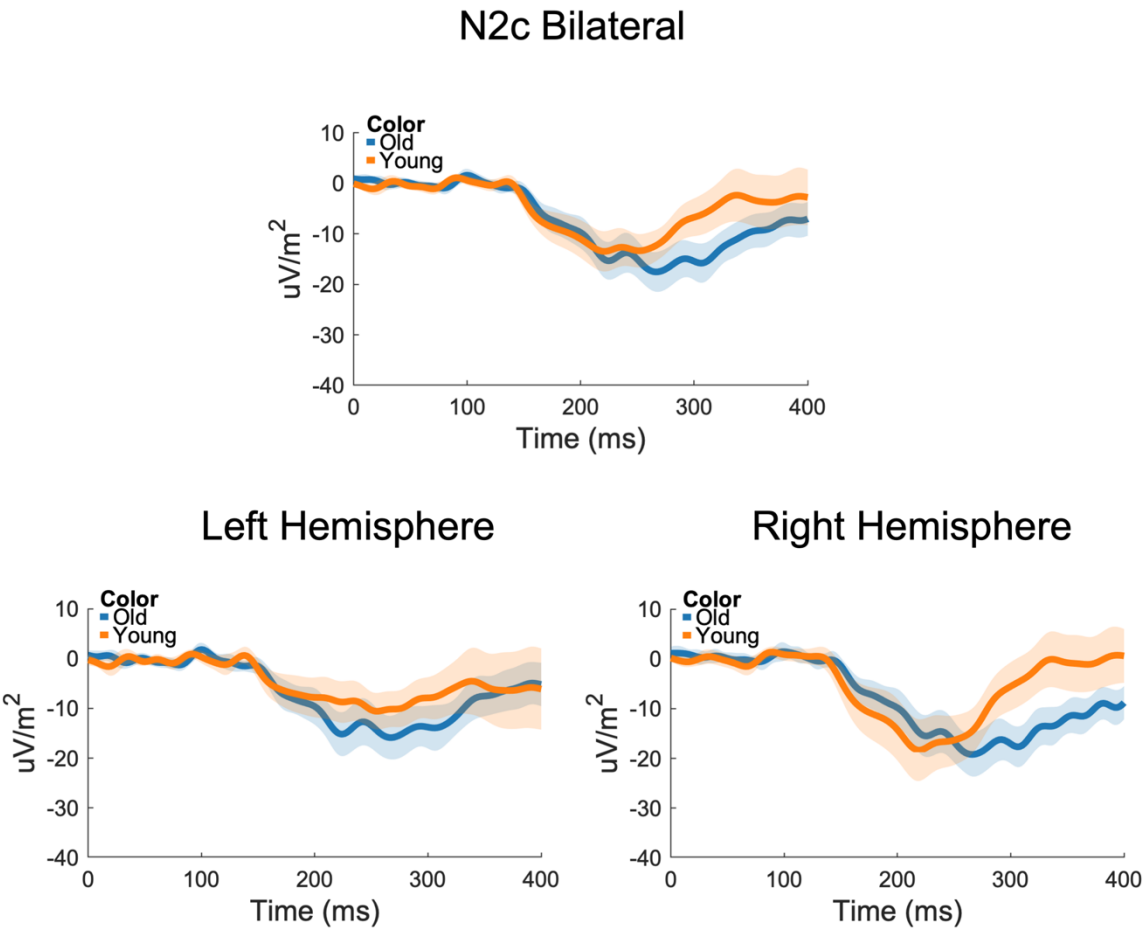
| | 120 vs 100 | 100 vs 80 | 80 vs 60 | 60 vs 40 | 40 vs 20 |
|---|---|---|---|---|---|
| Cumulative Distribution Factor (ks) | | | | | |
| RT (*k*, *p*) | ***k*=.22*** ** | *k* =.03 ns | ***k*=.09*** ** | *k*=.05 ns | ***k*=.14*** ** |
| CPP slope | *k*=.25*** | *k*=.01 ns | *k*=.09*** | *k*=.07** | *k*=.1*** |

*Note.* ks denotes Kolmogorov-Smirnov test, ***, ***p*<.001, **, *p*<.01**



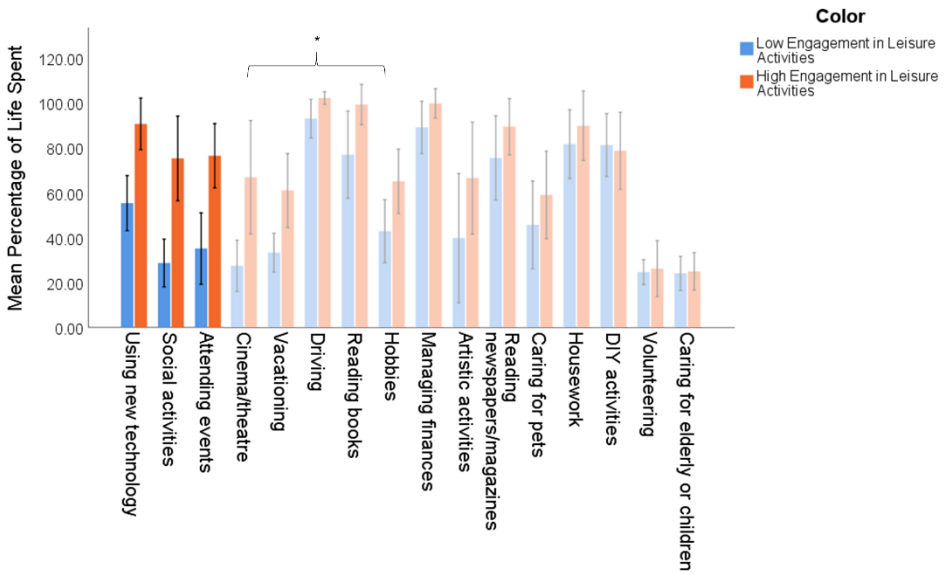

## Supplementary Figure 1. Temporal Dynamics of Evidence Accumulation Are Robust Age-Related Indicators

**A**. The stimulus-aligned N2c waveform (electrodes P7/P8) for older and younger adults. **B.** Stimulus-aligned CPP waveform (electrode Pz) for the two groups. **C.** Stimulus-aligned beta waveform (electrode C3) for the two groups. *Note.* The topoplots depict the spatial distribution of the EEG signal for both groups combined at 150-400ms post-target for the N2c (**A**) -150ms to 50ms aligned to response for the CPP **(B)** and 400-700ms post-target for LHB **(C).**



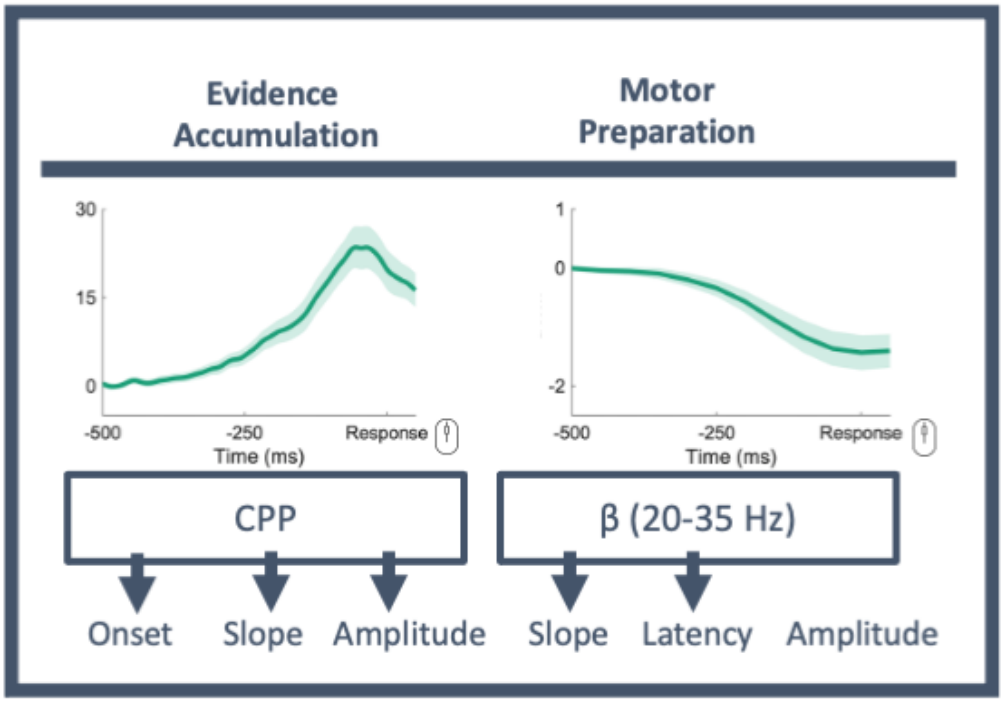

**Supplementary Figure 2. The N2c component, stimulus aligned at electrode P7 (Left Hemisphere), and P8 (Right hemisphere).**

Given the relevance of hemisphere lateralisation for theories of cognitive ageing, we investigated any age-related hemisphere differences in the N2c using 2 (old versus young) X 2 (right hemisphere x left hemisphere) ANOVAs, separately for latency and amplitude . There was no main effect of hemisphere on N2c latency ($F_{1,69}$=3.46, $p$=.07) but there was a significant hemisphere x group interaction term ($F_{1,69}$=11.56, $p$=.001, partial $\eta^2$ =.14). In line with a large body of work highlighting a right hemisphere dominance for early sensory processing, follow up analyses revealed that the younger adults showed a significantly faster right hemisphere N2c latency ($M$=257.07ms, $SD$=51.67) as compared with the left hemisphere (298.77ms, 69.95; $F_{1,29}$=14.99, $p$=.001, partial $\eta^2$ =.34). In contrast, for the older adults there was no hemispheric differences in N2c latency ($F_{1,40}$=1.23, $p$=.28; right hemisphere: $M$=303.59, $SD$=57.56; left hemisphere: $M$=291.37ms, $SD$=54.86), possibly indicative of a reduction in hemispheric asymmetries in the older adults. As compared with the younger adults, the older adults showed slower N2c latencies in the right ($F_{1,69}$=12.32, $p$=.001, partial $\eta^2$ =.15) but not left ($F_{1,69}$=.25, $p$=.62) hemispheres. There was no effect of group on N2c amplitude ($F_{1,69}$=3.33, $p$=.07), nor was there any group x hemisphere interaction term ($F_{1,69}$=.02, $p$=.88).




**Supplementary Figure 3. Differences between the activities of those with relatively higher versus lower levels of lifetime leisure engagement**

Proportion of life spent engaging in particular activities varies between those with an overall higher or lower level of engagement in leisure activities. Significant group differences are presented in bold. * denotes comparisons where $p<.05$, but did not satisfy a Bonferroni adjusted $\alpha=.003$.




**Supplementary Figure 4. CPP and Beta components from Figure 1 visualised here aligned to participants' response.**

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### References

1. Prince, M. *et al.* World Alzheimer Report 2015: The Global Impact of Dementia - An analysis of prevalence, incidence, cost and trends. *Alzheimer's Dis. Int.* 84 (2015) doi:10.1111/j.0963-7214.2004.00293.x.
2. Barker-Collo, S. & Feigin, V. The impact of neuropsychological deficits on functional stroke outcomes. *Neuropsychol. Rev.* **16**, 53–64 (2006).
3. O'Halloran, A. M., Finucane, C., Savva, G. M., Robertson, I. H. & Kenny, R. A. Sustained attention and frailty in the older adult population. *Journals Gerontol. Ser. B Psychol. Sci. Soc. Sci.* (2013).
4. Ball, K., Edwards, J. D., Ross, L. A. & McGwin, G. Cognitive training decreases motor vehicle collision involvement of older drivers. *J. Am. Geriatr. Soc.* **58**, 2107–2113 (2010).
5. Weaver, B., Bédard, M., McAuliffe, J. & Parkkari, M. Using the Attention Network Test to predict driving test scores. *Accid. Anal. Prev.* **41**, 76–83 (2009).
6. Rapp, P. R. & Amaral, D. G. Individual differences in the cognitive and neurobiological consequences of normal aging. *Trends in Neurosciences* vol. 15 340–345 (1992).
7. Norton, S., Matthews, F. E., Barnes, D. E., Yaffe, K. & Brayne, C. Potential for primary prevention of Alzheimer's disease: an analysis of population-based data. *Lancet Neurol.* **13**, 788–794 (2014).
8. Habib, R., Nyberg, L. & Nilsson, L. G. Cognitive and non-cognitive factors contributing to the longitudinal identification of successful older adults in the Betula study. *Aging, Neuropsychol. Cogn.* **14**, 257–273 (2007).
9. Stern, Y., Alexander, G. E., Prohovnik, I. & Mayeux, R. Inverse relationship between education and parietotemporal perfusion deficit in Alzheimer's disease. *Ann. Neurol.* **32**, 371–375 (1992).
10. Valenzuela, M. & Sachdev, P. Brain reserve and dementia: a systematic review. *Psychol. Med.* **36**, 441–454 (2006).
11. Opdebeeck, C., Martyr, A. & Clare, L. Cognitive reserve and cognitive function in healthy older people: A meta-analysis. *Aging, Neuropsychology, and Cognition* vol. 23 40–60 (2016).
12. Cabeza, R. *et al.* Maintenance, reserve and compensation: the cognitive neuroscience of healthy ageing. *Nat. Rev. Neurosci.* **19**, 701–710 (2018).
13. Stern, Y. *et al.* Whitepaper: Defining and investigating cognitive reserve, brain reserve, and brain maintenance. *Alzheimer's Dement.* **16**, 1305–1311 (2020).
14. Stern, Y. *et al.* Mechanisms underlying resilience in ageing. *Nat. Rev. Neurosci. 2019 204* **20**, 246–246 (2019).
15. Cabeza, R. *et al.* Reply to 'Mechanisms underlying resilience in ageing'. *Nat. Rev. Neurosci. 2019 204* **20**, 247–247 (2019).
16. Stern, Y. *et al.* Mechanisms underlying resilience in ageing. *Nature Reviews Neuroscience* vol. 20 246

(2019).
17. Chan, D. *et al.* Lifestyle activities in mid-life contribute to cognitive reserve in late-life, independent of education, occupation, and late-life activities. *Neurobiol. Aging* **70**, 180–183 (2018).
18. Xu, H. *et al.* Association of Lifespan Cognitive Reserve Indicator With Dementia Risk in the Presence of Brain Pathologies. *JAMA Neurol.* **76**, 1184–1191 (2019).
19. Stern, Y. Cognitive reserve in ageing and Alzheimer's disease. *Lancet Neurol.* **11**, 1006–1012 (2012).
20. Ojala-Oksala, J. *et al.* Educational history is an independent predictor of cognitive deficits and long-term survival in postacute patients with mild to moderate ischemic stroke. *Stroke* **43**, 2931–2935 (2012).
21. Sumowski, J. F., Chiaravalloti, N. & Deluca, J. Cognitive reserve protects against cognitive dysfunction in multiple sclerosis. *J. Clin. Exp. Neuropsychol.* **31**, 913–926 (2009).
22. Bonner-Jackson, A. *et al.* Cognitive reserve and brain reserve in prodromal Huntington's disease. *J. Int. Neuropsychol. Soc.* **19**, 739–750 (2013).
23. Gregory, T., Nettelbeck, T., Howard, S. & Wilson, C. Inspection Time: A biomarker for cognitive decline. *Intelligence* **36**, 664–671 (2008).
24. Deary, I. J., Johnson, W. & Starr, J. M. Are processing speed tasks biomarkers of cognitive aging? *Psychol. Aging* **25**, 219–228 (2010).
25. Ritchie, S. J., Tucker-Drob, E. M. & Deary, I. J. A strong link between speed of visual discrimination and cognitive ageing. *Curr. Biol.* (2014) doi:10.1016/j.cub.2014.06.012.
26. Bublak, P. *et al.* Staged decline of visual processing capacity in mild cognitive impairment and Alzheimer's disease. *Neurobiol. Aging* **32**, 1219–1230 (2011).
27. Kochan, N. A. *et al.* Reaction Time Measures Predict Incident Dementia in Community-Living Older Adults: The Sydney Memory and Ageing Study. *Am. J. Geriatr. Psychiatry* **24**, 221–231 (2016).
28. Salthouse, T. A. The processing-speed theory of adult age differences in cognition. *Psychol. Rev.* **103**, 403–428 (1996).
29. Edwards, J. D. *et al.* Speed of processing training results in lower risk of dementia. *Alzheimer's Dement. Transl. Res. Clin. Interv.* **3**, 603–611 (2017).
30. Ball, K. *et al.* Effects of cognitive training interventions with older adults: A randomized controlled trial. *J. Am. Med. Assoc.* **288**, 2271–2281 (2002).
31. Rebok, G. W. *et al.* Ten-year effects of the advanced cognitive training for independent and vital elderly cognitive training trial on cognition and everyday functioning in older adults. *J. Am. Geriatr. Soc.* **62**, 16–24 (2014).
32. Wolinsky, F. D. *et al.* The effect of speed-of-processing training on depressive symptoms in ACTIVE. *Journals Gerontol. - Ser. A Biol. Sci. Med. Sci.* **64**, 468–472 (2009).
33. Wolinsky, F. D. *et al.* The effects of the ACTIVE cognitive training trial on clinically relevant declines in health-related quality of life. *Journals Gerontol. - Ser. B Psychol. Sci. Soc. Sci.* **61**, S281–S287 (2006).
34. Lee, T. *et al.* Leisure activity, health, and medical correlates of neurocognitive performance among monozygotic twins: The older Australian twins study. *Journals Gerontol. - Ser. B Psychol. Sci. Soc. Sci.* **69**, 514–522 (2014).
35. Ratcliff, R., Thapar, A., Gomez, P. & McKoon, G. A Diffusion Model Analysis of the Effects of Aging in the Lexical-Decision Task. *Psychol. Aging* **19**, 278 (2004).
36. Ratcliff, R., Thapar, A. & McKoon, G. Aging and individual differences in rapid two-choice decisions. *Psychon. Bull. Rev.* **13**, 626–635 (2006).
37. Ratcliff, R., Thapar, A. & McKoon, G. Aging, practice, and perceptual tasks: A diffusion model analysis. *Psychol. Aging* **21**, 353–371 (2006).
38. Ratcliff, R. & McKoon, G. Aging effects in item and associative recognition memory for pictures and words. *Psychol. Aging* **30**, 669–674 (2015).
39. McKoon, G. & Ratcliff, R. Aging and IQ effects on associative recognition and priming in item recognition. *J. Mem. Lang.* **66**, 416–437 (2012).
40. Sanctis, P. De *et al.* Enhanced and bilateralized visual sensory processing in the ventral stream may be a feature of normal aging. *Neurobiol. Aging* **29**, 1576–1586 (2008).
41. McGovern, D. P., Hayes, A., Kelly, S. P. & O'Connell, R. G. Reconciling age-related changes in behavioural and neural indices of human perceptual decision-making. *Nat. Hum. Behav.* **2**, 955–966 (2018).
42. Falkenstein, M., Yordanova, J. & Kolev, V. Effects of aging on slowing of motor-response

generation. in *International Journal of Psychophysiology* vol. 59 22–29 (2006).
43. Yordanova, J., Kolev, V., Hohnsbein, J. & Falkenstein, M. Sensorimotor slowing with ageing is mediated by a functional dysregulation of motor-generation processes: Evidence from high-resolution event-related potentials. *Brain* **127**, 351–362 (2004).
44. Gazzaley, A. *et al.* Age-related top-down suppression deficit in the early stages of cortical visual memory processing. *Proc. Natl. Acad. Sci. U. S. A.* **105**, 13122–13126 (2008).
45. Zanto, T. P., Toy, B. & Gazzaley, A. Delays in neural processing during working memory encoding in normal aging. *Neuropsychologia* **48**, 13–25 (2010).
46. Wiegand, I. *et al.* Neural correlates of age-related decline and compensation in visual attention capacity. *Neurobiol. Aging* **35**, 2161–2173 (2014).
47. Thapar, A., Ratcliff, R. & McKoon, G. A diffusion model analysis of the effects of aging on letter discrimination. *Psychol. Aging* **18**, 415–429 (2003).
48. Ratcliff, R., Thapar, A. & McKoon, G. The effects of aging on reaction time in a signal detection task. *Psychol. Aging* **16**, 323–341 (2001).
49. Forstmann, B. U. *et al.* The speed-accuracy tradeoff in the elderly brain: A structural model-based approach. *J. Neurosci.* **31**, 17242–17249 (2011).
50. Dully, J., McGovern, D. P. & O'Connell, R. G. The impact of natural aging on computational and neural indices of perceptual decision making: A review. *Behav. Brain Res.* **355**, 48–55 (2018).
51. O'Connell, R. G., Dockree, P. M. & Kelly, S. P. A supramodal accumulation-to-bound signal that determines perceptual decisions in humans. *Nat. Neurosci.* **15**, 1729–1735 (2012).
52. Kelly, S. P. & O'Connell, R. G. Internal and external influences on the rate of sensory evidence accumulation in the human brain. *J. Neurosci.* **33**, 19434–19441 (2013).
53. Philiastides, M. G., Heekeren, H. R. & Sajda, P. Human Scalp Potentials Reflect a Mixture of Decision-Related Signals during Perceptual Choices. *J. Neurosci.* **34**, 16877–16889 (2014).
54. Rungratsameetaweemana, N., Itthipuripat, S., Salazar, A. & Serences, J. T. Expectations do not alter early sensory processing during perceptual decision-making. *J. Neurosci.* **38**, 5632–5648 (2018).
55. Spitzer, B., Waschke, L. & Summerfield, C. Selective overweighting of larger magnitudes during noisy numerical comparison. *Nat. Hum. Behav.* **1**, 1–8 (2017).
56. von Lautz, A., Herding, J. & Blankenburg, F. Neuronal signatures of a random-dot motion comparison task. *Neuroimage* **193**, 57–66 (2019).
57. van Vugt, M. K., Beulen, M. A. & Taatgen, N. A. Relation between centro-parietal positivity and diffusion model parameters in both perceptual and memory-based decision making. *Brain Res.* **1715**, 1–12 (2019).
58. Loughnane, G. M. *et al.* Catecholamine Modulation of Evidence Accumulation during Perceptual Decision Formation: A Randomized Trial. *J. Cogn. Neurosci.* **31**, 1044–1053.
59. Twomey, D. M., Murphy, P. R., Kelly, S. P. & O'Connell, R. G. The classic P300 encodes a build-to-threshold decision variable. *Eur. J. Neurosci.* **42**, 1636–1643 (2015).
60. Steinemann, N. A., O'Connell, R. G. & Kelly, S. P. Decisions are expedited through multiple neural adjustments spanning the sensorimotor hierarchy. *Nat. Commun.* **9**, 535 (2018).
61. Murphy, P. R., Robertson, I. H., Harty, S. & O'Connell, R. G. Neural evidence accumulation persists after choice to inform metacognitive judgments. *Elife* **4**, 1–23 (2015).
62. Brosnan, M. B. *et al.* Evidence accumulation during perceptual decisions in humans varies as a function of dorsal frontoparietal organization. *Nat. Hum. Behav.* **4**, 844–855 (2020).
63. Veldsman, M. *et al.* Cerebrovascular risk factors impact frontoparietal network integrity and executive function in healthy ageing. *Nat. Commun. 2020 111* **11**, 1–10 (2020).
64. Gilbert, J. R. & Moran, R. J. Inputs to prefrontal cortex support visual recognition in the aging brain. *Sci. Rep.* **6**, 1–9 (2016).
65. Brosnan, M. B. *et al.* Plasticity of the Right-Lateralized Cognitive Reserve Network in Ageing. *Cereb. Cortex* **28**, 1749–1759 (2017).
66. Brosnan, M. B. *et al.* Prefrontal modulation of visual processing and sustained attention in aging, a tDCS–EEG coregistration approach. *J. Cogn. Neurosci.* **30**, 1630–1645 (2018).
67. Berry, A. S., Zanto, T. P., Clapp, W. C., Hardy, J. L. & Delahunt, P. B. The influence of perceptual training on working memory in older adults. *PLoS One* **5**, e11537 (2010).
68. Newman, D. P., Loughnane, G. M., Kelly, S. P., O'Connell, R. G. & Bellgrove, M. A. Visuospatial asymmetries arise from differences in the onset time of perceptual evidence accumulation. *J.*

*Neurosci.* **37**, 3378–3385 (2017).
69. Newsome, W. T., Britten, K. H. & Movshon, J. A. Neuronal correlates of a perceptual decision. *Nature* **341**, 52–54 (1989).
70. Loughnane, G. M. *et al.* Target selection signals influence perceptual decisions by modulating the onset and rate of evidence accumulation. *Curr. Biol.* **26**, 496–502 (2016).
71. Zhou, S.-H., Loughnane, G., O'Connell, R., Bellgrove, M. A. & Chong, T. T.-J. Distractors Selectively Modulate Electrophysiological Markers of Perceptual Decisions. *J. Cogn. Neurosci.* **33**, 1020–1031 (2021).
72. Nunez, M. D., Gosai, A., Vandekerckhove, J. & Neuroimage, R. S. The latency of a visual evoked potential tracks the onset of decision making. *Neurobiol. Aging* (2019) doi:10.2307/j.ctt1x76fxb?refreqid=search-gateway:8fbe92577502515959c53d3aa626b667.
73. Nunez, M. D., Vandekerckhove, J. & Srinivasan, R. How attention influences perceptual decision making: Single-trial EEG correlates of drift-diffusion model parameters. *J. Math. Psychol.* **76**, 117–130 (2017).
74. Nucci, M., Mapelli, D. & Mondini, S. Cognitive Reserve Index questionnaire (CRIq): a new instrument for measuring cognitive reserve. *Aging Clin. Exp. Res.* (2012) doi:10.3275/7800.
75. Park, D. C. *et al.* The Impact of Sustained Engagement on Cognitive Function in Older Adults: The Synapse Project. *Psychol. Sci.* **25**, 103–112 (2014).
76. Sachdev, P. S. *et al.* The contribution of twins to the study of cognitive ageing and dementia: The Older Australian Twins Study. *Int. Rev. Psychiatry* **25**, 738–747 (2013).
77. Robertson, I. H. A right hemisphere role in cognitive reserve. *Neurobiol. Aging* **35**, 1375–1385 (2014).
78. Rouder, J. N. & Morey, R. D. The Nature of Psychological Thresholds. *Psychol. Rev.* **116**, 655–660 (2009).
79. Nasreddine, Z. S. *et al.* The Montreal Cognitive Assessment, MoCA: A brief screening tool for mild cognitive impairment. *J. Am. Geriatr. Soc.* **53**, 695–699 (2005).
80. Ewers, M. *et al.* Segregation of functional networks is associated with cognitive resilience in Alzheimer's disease. *Brain* **144**, 2176–2185 (2021).
81. O'Sullivan, M. *et al.* Evidence for cortical 'disconnection' as a mechanism of age-related cognitive decline. *Neurology* **57**, 632–638 (2001).
82. Franzmeier, N., Duering, M., Weiner, M., Dichgans, M. & Ewers, M. Left frontal cortex connectivity underlies cognitive reserve in prodromal Alzheimer disease. *Neurology* **88**, 1054–1061 (2017).
83. Franzmeier, N. *et al.* Resting-state connectivity of the left frontal cortex to the default mode and dorsal attention network supports reserve in mild cognitive impairment. *Front. Aging Neurosci.* **9**, 270 (2017).
84. Veldsman, M. *et al.* Cerebrovascular risk factors impact frontoparietal network integrity and executive function in healthy ageing. *Nat. Commun.* **11**, 1–10 (2020).
85. Brosnan, M. B., Shalev, N., Ramduny, J., Sotiropoulos, S. N. & Chechlacz, M. Right fronto-parietal networks mediate the neurocognitive benefits of enriched environments. *bioRxiv* 2021.09.01.458571 (2021) doi:10.1101/2021.09.01.458571.
86. Subramanian, L. *et al.* Real-time functional magnetic resonance imaging neurofeedback for treatment of Parkinson's disease. *J. Neurosci.* **31**, 16309–16317 (2011).
87. Subramanian, L. *et al.* Functional magnetic resonance imaging neurofeedback-guided motor imagery training and motor training for parkinson's disease: Randomized trial. *Front. Behav. Neurosci.* **10**, 1–13 (2016).
88. Weigard, A. & Sripada, C. Task-General Efficiency of Evidence Accumulation as a Computationally Defined Neurocognitive Trait: Implications for Clinical Neuroscience. *Biol. Psychiatry Glob. Open Sci.* **1**, 5–15 (2021).
89. Sripada, C. & Weigard, A. Impaired Evidence Accumulation as a Transdiagnostic Vulnerability Factor in Psychopathology. *Front. Psychiatry* **12**, 627179 (2021).
90. Vidaurre, D., Smith, S. M. & Woolrich, M. W. Brain network dynamics are hierarchically organized in time. *Proc. Natl. Acad. Sci. U. S. A.* **114**, 12827–12832 (2017).
91. Pelli, D. G. The VideoToolbox software for visual psychophysics: transforming numbers into movies. *Spat. Vis.* **10**, 437–442 (1997).
92. Brainard, D. H. The Psychophysics Toolbox. *Spat. Vis.* **10**, 433–436 (1997).

93. Cornelissen, F. W., Peters, E. M. & Palmer, J. The Eyelink Toolbox: Eye tracking with MATLAB and the Psychophysics Toolbox. *Behav. Res. Methods, Instruments, Comput.* **34**, 613–617 (2002).
94. Delorme, A. & Makeig, S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J. Neurosci. Methods* **134**, 9–21 (2004).
95. Kayser, J. & Tenke, C. E. Principal components analysis of Laplacian waveforms as a generic method for identifying ERP generator patterns: I. Evaluation with auditory oddball tasks. *Clin. Neurophysiol.* **117**, 348–368 (2006).
96. Foxe, J. J. & Simpson, G. V. Flow of activation from V1 to frontal cortex in humans: A framework for defining 'early' visual processing. *Exp. Brain Res.* **142**, 139–150 (2002).
97. Kelly, S. P., Gomez-Ramirez, M. & Foxe, J. J. Spatial Attention Modulates Initial Afferent Activity in Human Primary Visual Cortex. *Cereb. Cortex* **18**, 2629–2636.
98. Allen, M., Poggiali, D., Whitaker, K., Marshall, T. R. & Kievit, R. A. Raincloud plots: A multi-platform tool for robust data visualization [version 1; peer review: 2 approved]. *Wellcome Open Res.* **4**, (2019).
99. Allen, M., Poggiali, D., Whitaker, K., Marshall, T. R. & Kievit, R. RainCloudPlots tutorials and codebase. (2018) doi:10.5281/ZENODO.1402959.
100. Morel, P. Gramm: grammar of graphics plotting in Matlab. *J. Open Source Softw.* **3**, 568 (2018).
101. Hayes, A. F. PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling. (2012).
102. Hayes, A. F. Introduction to Mediation, Moderation, and Conditional Process Analysis: A Regression-Based Approach. New York, NY: The Guilford Press. *J. Educ. Meas.* **51**, 335–337 (2014).
103. Wechsler, D. *Wechsler Adult Intelligence Scale–Revised. San Antonio, TX: NCS Pearson* vol. 22 (1981).
104. Nelson, H. E. The National Adult Reading Test (NART): Test Manual. *Wind. UK NFER-Nelson* **124**, 0–25 (1982).