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Executive control from healthy ageing to cognitive impairment: A systematic review of stroop and simon effects using psychophysiological and imaging techniques

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ABSTRACT

Background: The prevalence of cognitive impairment and dementia in the ageing population emphasises the need for strategies to mitigate cognitive decline. While research on Alzheimer's Disease (AD) has focused on early risk factors, less attention has been paid to protective factors such as cognitive reserve (CR) and cognitive control (CC). Methods: This review examines age-related changes in the prefrontal cortex (PFC) in healthy ageing and cognitive impairment. We analysed studies using Stroop and Simon tasks in conjunction with EEG, EEG/ERP, fNIRS and fMRI. Results: Of the 1411 articles reviewed, 49 studies met our criteria. The results suggest that the Stroop and Simon effects are essential for distinguishing between healthy ageing and cognitive impairment. Increased activity of the PFC supports task performance, especially in cognitive ageing. However, when compensatory mechanisms fail, deficits in Stroop and Simon effects may indicate cognitive impairment and reduced activation of the PFC. Conclusion: This review emphasises the critical role of CR in attenuating age-related cognitive decline and highlights the importance of the PFC in maintaining CC.

1. Introduction

Population aging presents significant opportunities and challenges and has profound implications for global health and economic systems. In line with the United Nations Decade of Healthy Ageing (2021–2030), it is imperative to improve our understanding and strategies to promote healthy aging while mitigating cognitive impairments such as dementia (World Health Organization, 2020). Traditionally, research has identified risk factors and early biomarkers for Alzheimer's disease (AD), focusing on cardiovascular and metabolic diseases as well as lifestyle influences (Livingston et al., 2020; Niotis et al., 2024).

However, less attention has been paid to protective factors that could support healthy aging and delay cognitive decline (Pappalettera et al., 2024; Turrini et al., 2023). In particular, cognitive reserve (CR) can be considered a crucial mediating factor that moderates the relationship between structural and cognitive decline. Although some researchers have distinguished between CR and brain reserve in terms of differences in cognitive processing strategies that can help compensate for neuronal damage and individual differences in the neurobiological state of the brain, respectively (Stern, 2009; Stern et al., 2023), other researchers have argued that the distinction between these two concepts is somewhat artificial, as cognition is inherently brain-dependent (Cabeza et al., 2018).

This reserve reflects the brain's ability to effectively utilize neural resources to compensate for age-related cognitive decline, potentially delaying the onset of cognitive impairment (Cabeza et al., 2018). It is crucial for maintaining cognitive function and emotional regulation and serves as a buffer against dementia (Amanzio et al., 2023a, 2023b;

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Corbo et al., 2023; Opdebeeck et al., 2016). Promoting CR through activities such as cognitive training can improve executive functions (EFs), which are particularly vulnerable to age-related decline (Basak et al., 2008).

EFs, also referred to as cognitive control (CC), are responsible for coordinating top-down mental processes and actions in accordance with current goals and future plans and are primarily mediated by the prefrontal cortex (PFC) (Egner, 2017; Miller and Cohen, 2001). It is widely agreed that there are three core EFs: Inhibition (including self-control, and interference control), working memory (WM), and cognitive flexibility (set shifting) (Lehto et al., 2003; Miyake et al., 2000).

Interestingly, it is a major challenge to understand how the PFC is organized to mediate the broad range of cognitive processes referred to as CC/EFs (Friedman and Robbins, 2022). These include inhibiting automatic or dominant responses, controlling interference, switching between tasks, coordinating multiple tasks, updating WM, monitoring, and higher-order EFs processes such as reasoning, problem solving, and planning (Diamond, 2013; Friedman and Miyake, 2017; Jurado and Rosselli, 2007). Neuropsychological tests such as the Stroop (MacLeod, 1991; Stroop, 1935) and Simon tasks (Simon, 1990) are valuable for studying CC/EFs, including inhibition, shifting, and updating (Miyake et al., 2000), and for investigating the neural mechanisms associated with these processes using advanced psychophysiological and imaging techniques. These tasks provide important insights into how the CC/EFs rely on the PFC and related regions, which are among the first cognitive functions to decline with age (Hasher et al., 2007).

The dorsolateral prefrontal cortex (DLPFC) modulates task-relevant information processing through top-down control and plays a crucial role in the preparation and maintenance of task rules as well as in the monitoring and regulation of cognitive performance. The DLPFC facilitates important actions by exerting CC/EFs over motor behavior (Hoshi, 2006; Miller and Cohen, 2001), with the rostral midlateral prefrontal regions (Brodmann areas, BA, 10 and 46) playing a special role (Moayedi et al., 2015; Petrides, 2005a).

Despite the well-documented role of the DLPFC in various CC/EFs, the existence of functional subdivisions within the DLPFC remains unclear (Jung et al., 2022). Numerous fMRI studies report activation of the DLPFC, but the exact location and extent of activation sites vary depending on the executive task (Bahlmann et al., 2015; Kohn et al., 2014; Nee et al., 2007; Rottschy et al., 2012; Zheng et al., 2008). Cieslik et al. (2013) investigated the subdivision of the DLPFC using a meta-analysis of task-dependent and task-independent connectivity. In this study, the DLPFC was divided into two regions with different connectivity profiles: an anterior subregion, which is co-activated with the anterior cingulate cortex (ACC), and a posterior subregion, which is co-activated with the parietal cortex. Functional characterization by forward and backward inference revealed that the anterior network is involved in attentional processes such as monitoring motor responses, action inhibition, and behavioral adjustments (e.g., in the Stroop task), whereas the posterior cluster is more involved in action control processes that depend on stimulus processing and WM (e.g., in the n-back task) (Cieslik et al., 2013). These findings indicate functional variation along a rostral-caudal axis in the DLPFC (Petrides, 2005b) and, together with its anatomical diversity, suggest that the DLPFC consists of functionally distinct subregions. The DLPFC is also functionally and structurally connected to the insula and the ACC (Catani et al., 2012; Cieslik et al., 2013; Jung et al., 2022). As core components of the Multiple Demand Network, these regions play a role in cognitively demanding tasks such as CC/EFs and in responding to the uncertainty of emotional arousal (Camilleri et al., 2018; Menon and Uddin, 2010; Seeley et al., 2007). In addition, a neural circuit involved in goal-directed behaviors connects subregions of the DLPFC directly to the basal ganglia and thalamus via corticostriatal projections (Alexander et al., 1986; Jarbo and Verstynen, 2015). In particular, the anatomical connections between the basal ganglia and the DLPFC form a neural circuit that supports action control (Cieslik et al., 2013; Petrides, 2005a).

Maintaining the function of the PFC is crucial for effective CC/EFs (Miller and Cohen, 2001). On the other hand, declining PFC function impairs the ability to manage conflicting responses and ignore distractions, resulting in diminished executive control (Braver and Barch, 2002; Raz, 2000; Stuss and Craik, 2019). Studies have shown that the reduction in gray matter volume in the PFC is more pronounced with age, while other areas, such as the sensory and entorhinal cortex, shrink less (Raz, 2000). Ageing may also impair frontostriatal circuitry, leading to decreased EFs and secondary memory problems (Buckner, 2004). In particular, the medial PFC undergoes significant changes in healthy older adults at high risk for dementia, and AD patients, which are exacerbated by hypertension and metabolic syndrome (Fjell et al., 2009; Raz et al., 2005).

Neuropsychological tests such as the Stroop task (MacLeod, 1991; Stroop, 1935) and the Simon task (Simon, 1990) are valuable for the assessment of CC/EFs in healthy individuals and for the assessment of executive control in older adults with subjective cognitive decline (SCD) or objective cognitive impairment.

Although the Stroop and Simon tasks address different types of conflict, both draw on similar top-down CC mechanisms involving the PFC, albeit via different neural pathways (Liu et al., 2004). These tasks provide a unique opportunity to investigate age-related differences in brain activity, with older adults showing either increased or decreased activity compared to younger individuals. The underlying mechanisms, including compensatory or inefficient neuronal recruitment such as dedifferentiation (e.g., Park et al., 2012), remain poorly understood, particularly with regard to the distinction between physiological and pathological aging and between cognitive and executive control processes.

Our systematic review uniquely examines neuropsychological paradigms of CC/EFs, such as the Stroop and Simon tasks, and the associated neural mechanisms using advanced cognitive neuroscience techniques, including electroencephalography (EEG), functional near-infrared spectroscopy (fNIRS), and functional magnetic resonance imaging (fMRI). For the purposes of this systematic review, the term "cognitive reserve" is used to better capture the adaptive aspects of the cognitive processes and brain networks involved in tasks such as Simon and Stroop, rather than structural differences in the brain.

By examining the specific neural basis of the Stroop and Simon effects and considering the differences between these techniques, this review aims to answer several key questions: 1) How does CR contribute to the maintenance of cognitive performance? 2) How do age-related cortical changes and compensatory mechanisms occur? 3) What role do CR mechanisms play in the detection of SCD? 4) How can these mechanisms falter in cognitive disorders characterized by dysexecutive control, such as mild cognitive impairment (MCI)?

Early detection of deficits in cognitive and executive control associated with progression from SCD to MCI and dementia is critical for timely and effective intervention. Identifying these neural markers of executive control dysfunction could provide strategies for intervention and prevention and ultimately contribute to improved quality of life for older adults at risk of cognitive decline.

2. Material and methods

The study selection and the data collection adhered to the updated Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Page et al., 2021).

2.1. Systematic search and study selection

A systematic literature search was conducted in five electronic databases: PubMed, Scopus, Embase, Web of Science (WoS) and PsycInfo, up to 11 June 2024. No filters or limits were used and no reference time period was specified. Furthermore, only original articles in English were considered and no restrictions regarding gender and ethnicity were

made.

The search string was as follows: (aging OR ageing OR older adults OR older people OR older population OR elder*) AND (simon OR simon task OR stroop OR stroop task) AND (event-related potential OR erp OR electroencephalography OR eeg OR functional near-infrared spectroscopy OR fnirs OR functional magnetic resonance imaging OR fmri) AND (healthy aging OR healthy ageing OR healthy older adults OR healthy older people OR healthy older population OR healthy elder* OR subjective cognitive decline OR scd OR subjective cognitive complaint OR subjective cognitive impairment OR mild cognitive impairment OR mci).

We also hand-searched for reports in scientific journals. This ad hoc method enables the identification of studies that could neither be found nor retrieved in the major electronic databases (Cochrane Training, 2024).

2.2. Inclusion and exclusion criteria

We only included studies that met the following criteria: (a) with human subjects; (b) with subjects aged $\geq$ 60 years; (c) with the Simon task and/or the Stroop task; (d) with healthy older adults, individuals with SCD, and/or MCI; and (e) with EEG or EEG/ERP, fNIRS and/or fMRI technology.

We excluded studies with the following characteristics: (a) with non-human species or animal models; (b) with subjects < 60 years of age; (c) without Simon task and/or Stroop task; and (d) without EEG or EEG/ERP, fNIRS and/or fMRI signals.

To further evaluate the mechanisms of cognitive control, we also examined the following groups from the included studies: (a) younger healthy adults and (b) patients 60 years and older with different chronic conditions and/or neurocognitive disorders (e.g., psychiatric, vascular, and neurodegenerative diseases).

2.3. Data screening and selection

Three authors (S.M., G.E.C., and R.M.) independently selected studies based on title, abstract, and full text. Any discrepancies were discussed and clarified under the supervision of another author (M.A.). After the eligibility criteria for each study were discussed, two authors (S.M. and G.E.C.) independently analysed the population characteristics, sample size, procedures, study design and results. In case of disagreement, a consensus was reached between all authors or the judgement of a third author (M.A.) was obtained.

Cohen's K was used to assess the level of agreement regarding the inclusion and exclusion of studies: % agreement 100; Cohen's K: 1.

2.4. Data extraction

This review was conducted to find useful information on the activity of the PFC in relation to cognitive and executive control processes in older adults. It also considers individuals with subjective cognitive decline and MCI and focuses on studies using Simon or Stroop tasks with EEG or EEG/ERP, fNIRS and fMRI.

After removing duplicates from the search terms, the titles and abstracts were screened, followed by a full-text review of the articles to determine whether they were eligible to be reviewed.

Three reviewers (G.E.C., S.M. and R.M.) independently extracted data on study characteristics and measures.

Finally, M.A. validated the extracted results against the records and discussed any discrepancies with the other authors (G.E.C. and S.M.).

2.5. Methodological quality assessment

The Mixed Methods Appraisal Tool (MMAT, Hong et al., 2018) was used to assess the quality of empirical studies, including primary research based on experiments, observations, or simulations (Abbott, 1998; Porta et al., 2014). It is not applicable to non-empirical studies such as reviews and theoretical papers. The MMAT is capable of evaluating the most common study methodologies and designs, but it does not cover specific designs like economic and diagnostic accuracy studies, for which other appraisal tools might be more appropriate.

The MMAT is specifically designed for qualitative, quantitative, and mixed methods studies. It assesses methodological quality in five different classes: qualitative research, randomised controlled trials (RCTs), non-randomized studies, quantitative descriptive studies, and mixed methods studies. Each included study was assessed for quality using the MMAT (Hong et al., 2018), providing appropriate criteria tailored to each research design. Qualitative, RCTs, and non-randomized studies were each evaluated on five criteria, while mixed-methods studies were assessed on 15 criteria, covering the qualitative and quantitative component, and the integration of findings. Each criterion was rated as sufficiently met or not, resulting in scores out of 5 for qualitative and quantitative studies, and out of 15 for mixed-methods studies. Two reviewers (R.M. and G.E.C.) independently conducted the appraisals, and final ratings were established through discussion and consultation with a third reviewer (M.A.). No overall score was calculated for the studies, in line with recommendations from Hong et al. (2018) and Glenny (2005), as these scores can "obscure" specific methodological strengths and weaknesses (Hong et al., 2019; Viswanathan et al., 2012). Instead, the MMAT ratings for each criterion were detailed to better inform readers about the quality of the included studies (see Supplementary Table S1 for MMAT evaluations).

3. Results

3.1. Study selection

A total of 1411 studies were found: 656 in PubMed, 47 in Scopus, 216 in Embase, 282 in WoS and 210 in PsycInfo.

After the removal of 268 duplicates and an initial screening of 654 titles and abstracts, 489 titles were subjected to full-text screening, which resulted in 45 studies that met the inclusion criteria. Specifically, 1098 articles were excluded based on the title, abstract, and full-text screening. The reasons for exclusion were as follows:

- 21 dealt with irrelevant topics (inadequate topic);
- 381 did not refer to older adults (wrong age group);
- 110 used a different task for executive functions (wrong cognitive task);
- 20 were not written in English (wrong language);
- 61 used other procedures (wrong procedure);
- 1 did not study humans (wrong species);
- 347 did not use EEG or EEG/ERP, fNIRS or fMRI signals (wrong technique);
- 157 were not original articles (40 books, 31 conference papers, 32 reviews, 22 study protocols, 5 meta-analyses, 14 single cases, 6 pilot studies, 5 dissertations).

In addition, we identified 4 studies by hand searching.

After the identification and screening phase, 49 studies met our eligibility criteria and were included:

- 39 studies analysed cortical activation during the Stroop task using EEG (n = 4), EEG/ERP (n = 8), fNIRS (n = 12), and fMRI (n = 15);
- 8 studies analysed cortical activation during the Simon task using EEG/ERP;
- 2 studies analysed cortical activation during a combined Simon and Stroop task (1 using EEG/ERP and 1 using fMRI).

The flowchart for the selection of studies is shown in Fig. 1.

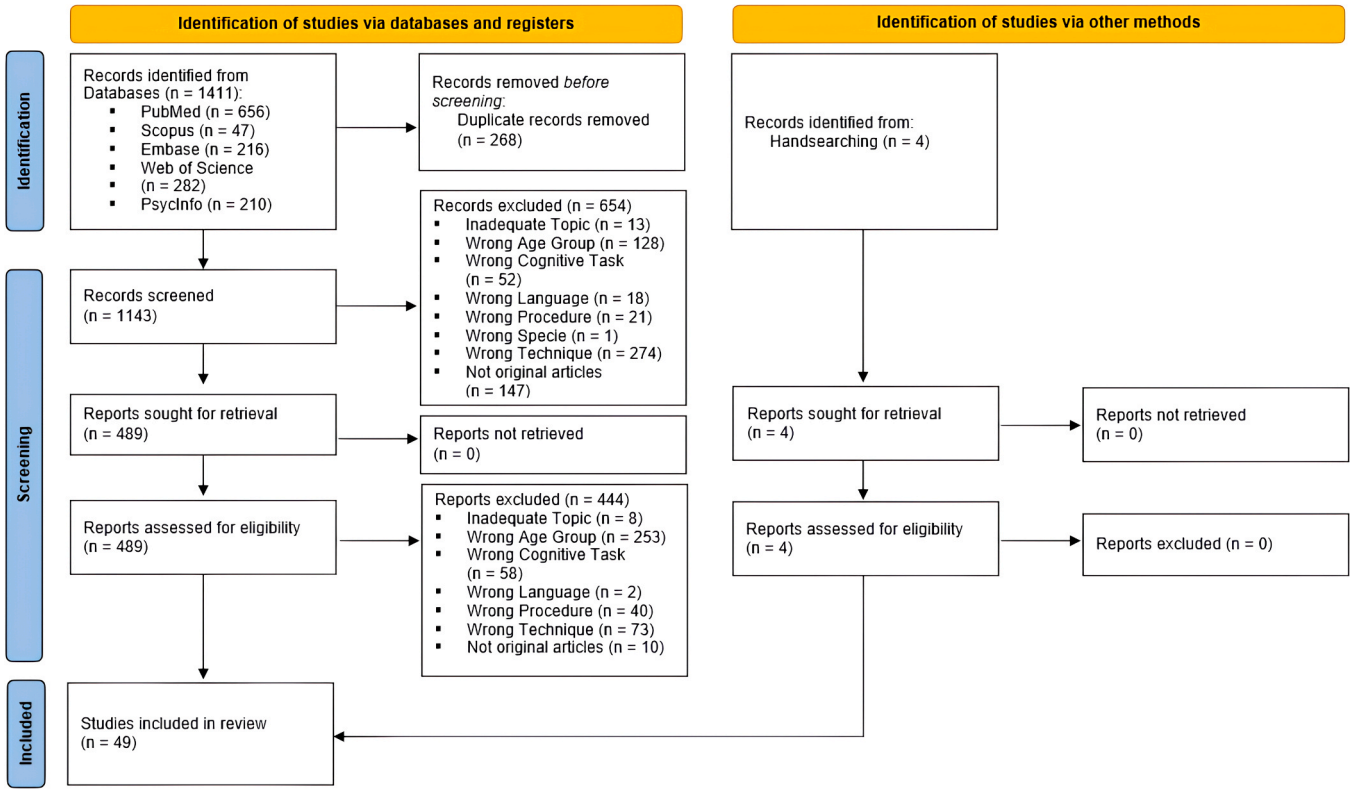

Fig. 1. PRISMA 2020 flow diagram of study selection that included searches of databases, registers and other sources (Page et al#, 2021).

3.2. Description of the selected studies

3.2.1. General characteristics of the different samples in the included studies

The general characteristics of the samples from the included studies (e.g., demographic data) are listed in Table 1 (see also Supplementary Table S2 for additional "all participants" data). Subjects were divided into six reference groups: young adults (YA), middle-aged adults (MA), healthy older adults (HA), SCD participants, MCI patients and subjects with other clinical conditions such as AD, Parkinson's disease (PD), transient ischemic attack (TIA), vascular cognitive impairment (VCI), depression (D) and human immunodeficiency virus (HIV). In addition, specific subgroups within these clinical pathologies include amnestic MCI (aMCI), single domain amnestic MCI (sdaMCI), multidomain amnestic MCI (mdaMCI), multidomain non-amnestic MCI (mdnaMCI) and MCI due to PD (PDMCI). It should be noted that SCD is an umbrella term that is described with different labels, such as subjective memory impairment or subjective memory complaints (SMC), as indicated by Abdulrab and Heun (2008) and Jessen et al. (2014). Therefore, outcomes labelled as SMC in studies such as Cespón et al. (2018) were classified as SCD in this review.

Other measures used in the studies are neuropsychological assessments such as the Mini Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA). Various techniques were used to examine brain activity, including EEG, fNIRS and fMRI during specific interference-task conditions (e.g. neutral, congruent, incongruent) and cognitive reserve classifications (low cognitive reserve, LCR; high cognitive reserve, HCR). The studies also took into account differences in genotype (e.g., Val/Val, Val/Met, Met/Met) and diseases such as cerebral microangiopathy (CMA) and stroke (SP).

The included studies spanned 27 years, from 1997 to 2024.

3.2.2. Behavioural results for Simon and Stroop tasks taking into account the different groups

Tables 2 and 3 show the reaction times (RTs) and error rates (ERs) for congruent and incongruent conditions in the Simon and Stroop tasks as mean and standard deviation (SD). To maintain homogeneity between studies, ERs were calculated from accuracy data (percentage of correct responses) where appropriate. For missing or incomplete data, the authors of the included studies were contacted to obtain behavioural measures of Stroop and Simon effects.

For each study that provided these values, the effect size of the interference effect for both RTs and ERs was computed using the Cohen's *d* index (Lakens, 2013). Namely, Cohen's *d* was computed by dividing the mean difference (M_{diff}) between incongruent and congruent conditions by the average standard deviation: $M_{diff}/(SD_{congr}+SD_{incongr})/2$ (Lakens, 2013). The difference in Cohen's *d* was considered small for values between 0.2 and 0.5, moderate for values between 0.5 and 0.8, and large for values greater than 0.8 (Cohen, 1988).

As indicated in the tables, RTs and ERs were analysed for different groups, including young adults, middle-aged adults and healthy-aged participants, as well as subjects with SCD, and patients with MCI, AD, PD, Stroke Patients and other clinical conditions such as Vascular Cognitive Impairment, Transient Ischemic Attack and Human Immunodeficiency Virus. Within MCI, subgroups such as Single Domain Amnestic MCI (sdaMCI), Multiple Domains Amnestic MCI (mdaMCI), Multiple Domains Non-Amnestic MCI (mdnaMCI) and MCI due to Parkinson's Disease (PDMCI) were also analysed. Cognitive reserve was considered with Low Cognitive Reserve (LCR) and High Cognitive Reserve (HCR) categories, and other factors such as abnormal (CH-PAT) and normal (CH-NAT) Aβ/tau ratios were reported.

Some studies reported additional genotype data (e.g., Val/Val, Val/Met, Met/Met), which were analysed for their potential impact on executive control. In cases where error rates or reaction times were not available (N.A.) or were not reported (N.R.), these limitations were detailed. Where data were obtained directly from the authors or where

Table 1
General characteristics of the different samples in the included studies.

Study	Study Design	Participants			Gender	Age	Education in years	MMSE	MoCA	Task	Task version	Task condition	Technique
		group	subgroups	N	W/M	M ± SD (range)	M ± SD (range)	M ± SD (range)	M ± SD (range)				
Arakaki et al. (2022)	cross sectional	HA	CH-NAT	20	15/5	75.1 ± 7.5	15.7 ± 2.3	29.3 ± 1.3	N.R.	Stroop	standard or partially modified	congruent, incongruent	EEG
		HA	CH-PAT	21	16/5	76.2 ± 8.4	16.3 ± 1.9	29.2 ± 0.98					
Burin and Kawashima, (2021)	RCT	HA	1PP	21	11/10	70.57 ± 6.51	14.25 ± 2.46			Stroop	standard or partially modified	congruent, incongruent, neutral	fNIRS
		HA	3PP	21	17/4	72.86 ± 4.66	13.45 ± 1.95						
Byun et al. (2024)	clinical trial	HA	control	41	10/31	67.7 (65.9–69.6)	13.9 (13.3–14.5)	29.0 (28.6–29.4)		Stroop	standard or partially modified	congruent, incongruent, neutral	fNIRS
		HA	mild exercise	40	30/10	68.7 (67.0–70.3)	13.4 (12.7–14.1)	29.1 (28.7–29.4)					
Cespón et al. (2013)	cross sectional	HA		25	11/14	65.0 ± 8.1	10.8 ± 5.6	28.4 ± 1.3		Simon	modified	CDCP, IDCP, CDIP, IDIP	EEG/ERP
		MCI	sda	17	7/10	67.0 ± 9.1	8.9 ± 4.3	27.2 ± 1.9					
		MCI	mda	13	7/6	71.0 ± 9.2	9.2 ± 4.8	23.5 ± 1.7					
Cespón et al. (2015a)	cross sectional	HA		18	11/7	68.3 ± 1.68	11.1 ± 1.24	28.5 ± 0.38		Simon	modified	CDCP, IDCP, CDIP, IDIP	EEG/ERP
		MCI	sda	13	5/8	69.1 ± 1.98	10.1 ± 1.46	26.9 ± 0.45					
		MCI	mda	12	7/5	71.2 ± 2.06	9.2 ± 1.52	23.7 ± 0.47					
Cespón et al. (2015b)	cross sectional	HA		15		66.0 ± 7.5	10.2 ± 5.4	28.3 ± 1.2		Simon	modified	CDCP, IDCP, CDIP, IDIP	EEG/ERP
		MCI	mdna	11		66.5 ± 9.7	6.9 ± 2.9	24.4 ± 2.4					
		MCI	sda	16		67.1 ± 9.3	9.0 ± 4.3	27.2 ± 1.9					
		MCI	mda	11		69.6 ± 9.0	9.8 ± 5.0	23.7 ± 1.6					
Cespón et al. (2018)	cross sectional	SCD	low	18	13/5	65.1 ± 9.1	9.1 ± 5.4	28.2 ± 1.2		Simon	modified	CDCP, IDCP, CDIP, IDIP	EEG/ERP
		SCD	high	16	13/3	64.6 ± 7.1	8.4 ± 2.8	27.6 ± 1.6					
Cespón et al. (2022)	cross sectional	YA		21	16/5	22.6 ± 2.59				Simon	standard or partially modified	congruent, incongruent	EEG/ERP
		HA		20	10/10	67.0 ± 3.11							
Cespón et al. (2023)	cross sectional	HA	LCR	34		72.2 ± 4.3				Simon & Stroop	Simon, spatial Stroop	c-C, i-C, i-I,c-I	EEG/ERP
			HCR	35		69.6 ± 4.9							
Faulkner et al. (2017)	cross sectional	HA		15	2/13	61.5 ± 7.1				Stroop	standard or partially modified	congruent, incongruent	fNIRS
		other	TIA	11	2/9	65.1 ± 10.1							
Gajewski et al. (2012)	cross sectional	HA	Val/Val	79	58/21	70.8 ± 4.7		28.3 ± 1.9		Stroop	standard or partially modified	congruent, incongruent	EEG/ERP
			Val/Met - Met/Met	52	23/28	70.2 ± 4.3		28.8 ± 1.4					
Gajewski and Falkenstein, (2015)	cross sectional	HA	Low active	20	0/20	73.6 ± 4.9	3.6 ± 1.2	28.7 ± 0.9		Stroop	standard or partially modified	congruent, incongruent	EEG/ERP
			Active	20	0/20	72.7 ± 4.3	4.0 ± 0.6	29.0 ± 0.8					
Gajewski et al. (2020)	cross sectional	YA		36	19/17	25.1 ± 2.7 (19−33)		N.A.		Stroop	standard or partially modified	congruent, incongruent	EEG/ERP
		MA		58	0/58	46.5 ± 4.5 (40−53)							
		HA	old high performers	50		69.8 ± 3.8	3.8 ± 1.1	28.8 ± 1.6					

(*continued on next page*)

Table 1 (*continued*)

Study	Study Design	Participants			Gender	Age	Education in years	MMSE	MoCA	Task	Task version	Task condition	Technique
		HA	old mid performers	51		70.2 ± 4.8	3.4 ± 1.2	28.5 ± 1.2					
		HA	old low performers	51		70.9 ± 3.7	3.2 ± 1.1	28.5 ± 1.4					
Gauthier et al. (2013)	cross sectional	YA		31	10/21	23.74 ± 0.52[a]	17.03 ± 0.38[a]	N.A.		Stroop	standard or partially modified	congruent, incongruent, switching	fMRI
		HA		31	14/17	63.53 ± 0.87[a]	16.01 ± 0.57[a]	28.81 ± 0.20[a]					
Goenarjo et al. (2021)	cross sectional	MA	higher-fit	8	0/8	57.9 ± 1.6				Stroop	standard or partially modified	congruent, incongruent, switching	fNIRS
		MA	lower-fit	6	0/6	57.3 ± 2.0							
		HA	higher-fit	5	0/5	64.8 ± 3.6							
		HA	lower-fit	5	0/5	65.7 ± 2.9							
Hamasaki et al. (2018a)	cross sectional	YA		22	7/15	27 ± 1 (20–39)				Stroop	standard or partially modified	congruent, incongruent	fNIRS
		MA		22	12/10	55 ± 1 (50–59)							
		HA	60 s	40	31/9	64 ± 0 (60–69)							
		HA	70 s	14	8/6	74 ± 1 (70–79)							
Hamasaki et al. (2018b)	cross sectional	HA	low stiffness	31	15/16	61.4 ± 6.5				Stroop	standard or partially modified	congruent, incongruent	fNIRS
		HA	high stiffness	31	25/6	64.4 ± 6.2							
Heiberg et al. (2023)	cross sectional	HA		22	12/10	64.5[b] (58.5–69)			> 25	Stroop	standard or partially modified	congruent, incongruent, neutral	fNIRS
		other	SP	21	11/10	64[b] (57–71)			26[b] (25–27)				
Huang et al. (2022)	cross sectional	YA		20	12/8	23.7 ± 3.9 (18–35)				Stroop	standard or partially modified	congruent, incongruent	fNIRS
		HA		13	8/6	63.9 ± 4.0 (60–72)							
Ji et al. (2019)	cross sectional	HA		20	9/11	65.6 ± 1.32	11.25 ± 1.48	27.90 ± 0.62		Stroop	standard or partially modified	congruent, incongruent, switching	fNIRS
Katayama et al. (2023)	cross sectional	HA		402	229/173	73 (70–78)[b]	12 (12–16)[b]	29 (27–30)[b]		Simon	modified	congruent, incongruent, no response	EEG/ERP
		MCI		47	20/27	73 (69–78)[b]	12 (12–14)[b]	28 (26–29)[b]					
Kaufmann et al. (2008)	cross sectional	HA		9		68.3 ± 7.5		29.0 ± 1.2		Stroop	modified (numerical)	congruent, incongruent, neutral	fMRI
		MCI		6		69.8 ± 5.3		24.8 ± 1.2					
Kricheldorff et al. (2023)	cross sectional	HA		30	12/18	59.4 ± 6.8	16 ± 3.2	> 25		Stroop	modified (numerical)	congruent (mostly), incongruent (mostly)	EEG
		other	PD	30	8/22	64 ± 9.6	16.2 ± 3	> 25					
Laguë-Beauvais et al. (2013)	cross sectional	YA		21	13/8	24.38 ± 4.47 (19–36)	16.00 ± 2.37	N.A.		Stroop	modified	incongruent, neutral, switching	fNIRS
		HA		19	16/3	64.00 ± 3.40 (59–69)	14.37 ± 3.17	29.00 ± 1.15					
Langenecker et al. (2004)	cross sectional	YA		13	7/6	26.3 ± 5.5	17.2 ± 3.3			Stroop	standard or partially modified	congruent, incongruent, neutral	fMRI
		HA		13	8/5	71.1 ± 5.4	17.8 ± 2.8	28.4 ± 1.56					

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Table 1 (*continued*)

Study	Study Design	Participants			Gender	Age	Education in years	MMSE	MoCA	Task	Task version	Task condition	Technique
Lau et al. (2020)	RCT	MCI	placebo	18	13/5	65.94 ± 3.61	N.A.	N.R.		Stroop	standard or partially modified	congruent, incongruent	fMRI
		MCI	p. minor	18	15/3	66.89 ± 4.00	N.A.	N.R.		Stroop	standard or partially modified	congruent, incongruent	fMRI
Li et al. (2009)	cross sectional	HA		9	5/4	65.2 ± 7.2 (57–68)	7.1 ± 4.6	28.8 ± 0.9		Stroop	standard or partially modified	incongruent	fMRI
		MCI		9	4/5	63.4 ± 4.6 (52–67)	7.2 ± 3.1	26.4 ± 4.2					
		other	AD	10	5/5	65.8 ± 6.1 (54–69)	6.8 ± 2.7	16.7 ± 2.6					
Li et al. (2012)	cross sectional	HA		11	6/5	64.9 ± 4.2	6.8 ± 4.2	28.0 ± 2.2		Stroop	standard or partially modified	incongruent	fMRI
		other	SIVD	12	6/6	65.5 ± 5.7	7.6 ± 3.2	16.5 ± 4.7	14.6 ± 3.8				
		other	SIVCIND	12	6/6	66.2 ± 4.9	6.9 ± 3.6	26.6 ± 1.7	22.3 ± 1.5				
Manard et al. (2017)	cross sectional	YA		20	8/12	23.5 ± 3.22 (21–30)	14.80 ± 1.82			Stroop	modified	congruent (mostly), incongruent (mostly), neutral	fMRI
		HA		20	12/8	65.1 ± 3.8 (61–74)	15.45 ± 3.3						
Mason et al. (2022)	cross sectional	HA		33	21/12	73.7 ± 0.7		29.4 ± 1.0		Stroop	modified (verbal)	congruent, incongruent, nominal	fNIRS
		other	CAS	33	10/23	73.6 ± 0.5		29.5 ± 1.0					
Mathis et al. (2009)	cross sectional	YA		12	5/7	26.8 ± 3.4 (22–30)				Stroop	standard or partially modified	congruent, incongruent, neutral	fMRI
		MA		12	8/4	51.7 ± 3.1 (46–55)							
		HA		12	3/8	62.8 ± 3.0 (60–68)							
Milham et al. (2002)	cross sectional	YA		12	5/7	23 (21–27)				Stroop	standard or partially modified	congruent, incongruent, neutral	fMRI
		HA		10	3/7	68 (60–75)							
Müller-Oehring et al. (2022)	cross sectional	HA		28	16/12	61 ± 9 (45–76)	17 ± 2 (12–21)			Stroop	modified (match-to-sample)	congruent, incongruent	fMRI
		other	HIV	31	13/18	60 ± 7 (47–78)	14 ± 2 (10–19)						
		other	PD	35	14/21	65 ± 8 (49–79)	17 ± 2 (12–21)						
Nombela et al. (2014)	cross sectional	YA	20 s	17		(20–29)				Stroop	modified (animal)	congruent, incongruent	EEG
		YA	30 s	20		(30–39)							
		MA	40 s	17		(40–49)							
		MA	50 s	18		(50–59)							
		HA	60 s	18		(60–69)							
Onur et al. (2011)	RCT	YA		15	6/9	24.23 ± 3.09	N.R.			Simon & Stroop	visual–spatial version	congruent, incongruent	fMRI
		HA		13	5/8	63.81 ± 6.00	N.R.						
Pišljar et al. (2013)	cross sectional	HA		24	14/10	72.8 ± 6	8.9 ± 2.7	29.4 ± 0.6		Stroop	standard or partially modified	congruent, incongruent, neutral, verbal, reading	EEG/ERP
		other	D	34	20/14	72.8 ± 4.2	8.2 ± 1.9	28.7 ± 1.0					
Puente et al. (2014)	cross sectional	HA		26	16/10	74 ± 5.5	17 ± 2.3	28.0 ± 2 (23–30)		Stroop	standard or partially modified	congruent, incongruent, neutral	fMRI

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Table 1 (*continued*)

Study	Study Design	Participants			Gender	Age	Education in years	MMSE	MoCA	Task	Task version	Task condition	Technique
		MCI		17	10/7	75 ± 6.3	14.4 ± 3.4	25.9 ± 2.4 (19–30)					
Ramos-Goicoa et al. (2016)	cross sectional	HA*		45	28/17	65.4 ± 9.2	10.1 ± 5.3	28.5 ± 1.3		Stroop	standard or partially modified	congruent, incongruent, neutral	EEG/ERP
		MCI*	amnestic	39	21/18	70.7 ± 9.1	9.9 ± 5.2	25.5 ± 2.5					
Sánchez-Moguel et al. (2018)	cross sectional	HA	normal-EEG	22	13/9	65.54 ± 5.21	15.81 ± 5.13			Stroop	modified (counting)	congruent, incongruent	EEG/ERP
			theta-EEG	22	13/9	67.59 ± 5.81	15.31 ± 3.84						
Sánchez-Moguel et al. (2022)	cross sectional	HA	normal-EEG	23	13/10	> 60	> 9	> 27		Stroop	modified (counting)	congruent, incongruent	EEG/ERP
			theta-EEG	23	13/10	> 60	> 9	> 27					
Schroeter et al. (2007)	cross sectional	HA		12	7/5	64.5 ± 2 (62–67)				Stroop	standard or partially modified	congruent, incongruent, neutral	fNIRS
		other	CMA	12	2/10	61.2 ± 4.9 (54–68)							
Schulte et al. (2011)	cross sectional	YA		19	10/9	23.6 ± 3 (19–30)	15.8 ± 1.1			Stroop	modified (match-to-sample)	congruent, incongruent	fMRI
		HA		14	6/8	71 ± 8.7 (58–85)	17.3 ± 2.7	N.A.					
Singh et al. (2018)	cross sectional	HA		28	11/17	69.2 ± 9.2	16.6 ± 3.1	28.8 ± 1.0		Simon	modified	congruent, incongruent	EEG/ERP
		other	PD	28	11/17	69.8 ± 8.59	17.3 ± 3.24	28.6 ± 1.06					
Singh et al. (2023)	cross sectional	HA		49	23/26	70.9 ± 1.1	N.R.		26.7 ± 0.3	Simon	standard or partially modified	congruent, incongruent	EEG/ERP
		MCI	PDMCI	34	9/25	71.2 ± 1.4	N.R.		23.7 ± 0.2				
		other	PD	47	20/27	66.0 ± 1.1	N.R.		27.4 ± 0.2				
		other	PDD	19	3/16	70.2 ± 1.9	N.R.		17.8 ± 0.8				
Tam et al. (2015)	cross sectional	YA		15	9/6	23.5 ± 1.6	17.2 ± 1.0		N.R.	Stroop	standard or partially modified	congruent, incongruent, neutral	fMRI
		HA		28	17/11	64.8 ± 8.7	15.9 ± 3.4		N.R.				
Vidoni et al. (2013)	cross sectional	HA		18	9/9	72.2 ± 7.2	N.R.	29.2 (27–30)		Stroop	modified (word/arrow)	congruent, incongruent	fMRI
		other	AD	16	7/9[c]	74.9 ± 7.4	N.R.	26.8 (24–30)					
West and Bell, (1997)	cross sectional	YA		20	10/10	20.30 (18–28)	13.9	28.28		Stroop	modified (spatially integrated/separated)	congruent, incongruent	EEG
		HA		19	10/9	70.47 (62–78)	14.8	28.0					
You et al. (2021)	RCT	MCI	placebo	10	7/3	63.40 ± 2.41 (60–75)	10.40 ± 3.69	26.58 ± 1.35		Stroop	standard or partially modified	congruent, incongruent, neutral	fMRI
		MCI	CC supplement	10	7/3	64.00 ± 4.00 (60–75)	10.90 ± 3.54	27.09 ± 1.38					
Zurrón et al. (2014)	cross sectional	YA		11	3/8	20.5 ± 1.4	N.R.			Stroop	standard or partially modified	congruent, incongruent	EEG/ERP
		HA		11	3/8	67.6 ± 6.4	N.R.						

Note. MMSE: Mini Mental State Examination. MoCA: Montreal Cognitive Assessment. N: Number. W/M: Women/Men. M: Mean. SD: Standard Deviation. HA: Healthy Ageing. N.R. Assessed but not Reported. EEG: Electroencephalography. CH-NAT: normal Aβ/tau ratios. CH-PAT: abnormal Aβ/tau ratios. RCT: Randomized Controlled Trial. fNIRS: functional Near Infrared Spectroscopy. CDCP: Compatible Direction and Compatible Position. IDCP: Incompatible Direction and Compatible Position. CDIP: Compatible Direction and Incompatible Position. IDIP: Incompatible Direction and Incompatible Position. ERP: Event-Related Potential. sdaMCI: single domain amnestic MCI. mdaMCI: multiple domains amnestic MCI. mdnaMCI: multiple domains non amnestic MCI. SMC: Subjective Memory Complaints. LCR: Low Cognitive Reserve. c-C: congruent-congruent. i-C: incongruent-congruent. i-I: incongruent-incongruent. c-I: congruent-Incongruent. HCR: High Cognitive Reserve. TIA: Transient Ischemic Attack. Val/Val: Val/Val genotype carriers. Val/Met: Val/Met genotype carriers. Met/Met: Met/Met genotype carriers. MA: Middle-aged Adults. fMRI: functional Magnetic Resonance Imaging. SP: Stroke Patients. MCI: Mild Cognitive Impairment. PD: Parkinson's Disease. AD: Alzheimer's Disease.

SIVD: Subcortical Ischemic Vascular Dementia. SIVCIND: Subcortical Ischemic Vascular Cognitive Impairment No Dementia. CAS: Carotid Atherosclerosis. HIV: Human Immune-deficiency Virus. D: Depression. aMCI: amnestic MCI. CMA: Cerebral Micro Angiopathy. PDMCI: MCI due to Parkinson's Disease. PDD: Dementia in PD. CC Supplement: Cosmos caudatus Supplement.

median values were reported, this has also been reported. Overall, these results provide a comprehensive overview of executive control task performance across a wide range of groups and conditions.

As shown in Figs. 2 and 3 and in supplementary Table S3, S4, S5 the proportion of studies for each reference group (YA, MA, HA, SCD, MCI, and other clinical pathologies) was analysed using the calculated effect size of the interference effects for RTs and ERs. Effect sizes were categorised as non-computable, small, moderate or large. Due to the small number of eligible studies for SCD and other clinical pathologies (<5 studies), these groups were excluded from the figures.

The largest number of studies involved healthy older adults and MCI patients. In the healthy ageing group, a large effect size for RTs was observed in 65 % (21/33) of studies and in 47 % (14/30) for ERs. In MCI patients, only 9 % (1/11) of studies reported a large interference effect for RTs, while 46 % (6/13) reported a large effect for ERs.

Fig. 2 illustrates the proportion of studies in the different groups (YA, MA, HA, MCI) that reported effect sizes for RTs in the Simon and Stroop tasks. Fig. 3 similarly shows the proportion of studies reporting effect sizes for ERs. Both figures illustrate that large effect sizes predominate in the healthy ageing and MCI groups, while smaller or no effect sizes are reported in the other groups.

3.2.3. Cortical activation during the Simon and Stroop tasks, taking into account the different groups

Table 4 summarises the results of EEG, EEG/ERP, fNIRS and fMRI studies, focusing on the regions of interest (ROIs) and the main patterns of cortical activation and deactivation when comparing different groups and conditions (congruent vs. incongruent). The table includes results from studies with YA, MA, HA, SCD, MCI, and other clinical populations such as AD, PD and stroke patients.

Key regions activated or deactivated in these studies during the Stroop and Simon tasks included the DLPFC, ventrolateral prefrontal cortex (VLPFC) and ACC, areas commonly associated with cognitive control and conflict monitoring. Activation differences were also detected in BA based on BOLD fMRI signals (dependent on blood oxygen levels), and fNIRS data showed changes in oxygenated (oxy-Hb) and deoxygenated (deoxy-Hb) haemoglobin levels.

In some studies, ROIs were influenced by certain clinical characteristics, such as abnormal vs. normal Aβ/tau ratios (CH-PAT vs. CH-NAT), genetic factors such as brain-derived neurotrophic factor (BDNF) polymorphisms (Val/Val, Val/Met, Met/Met genotypes), or high and low cognitive reserve. For example, participants with higher cognitive reserve showed different activation patterns in prefrontal areas than participants with low cognitive reserve.

Table 4 and Table S3 also include data from interventions such as physical exercise, cognitive exercise or reading control, showing how these activities can influence cortical activation during task performance. In addition, studies with specific subgroups, such as sdaMCI, mdaMCI, mdnaMCI and individuals with SMC, also reported different activation patterns in the ROIs.

The results indicate that both the task condition (congruent vs. incongruent) and individual differences (e.g. clinical pathology, genetic predisposition, CR) significantly influence the cortical activation patterns during the Stroop and Simon tasks.

Supplementary Table S6 shows additional pre-post data for clinical trials, including behavioural results and brain activation findings.

Finally, the main cerebral findings of the Stroop and Simon effects were summarized in Table 5.

4. Factors that influence executive control

In this section, we examine the factors influencing the Stroop and Simon effects and their neural activations in the included studies. These effects refer to cognitive interference between automatic and controlled mental processes that lead to slower reaction times and a higher error rate in the incongruent condition compared to the congruent condition

Table 2
Main reactions times findings.

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main RT findings between groups
Stroop studies								
Arakaki et al. (2022)								Comparisons not statistically significant
RT incongruent			973.37 ± 164.9[a]				All participants	
RT congruent			763.79 ± 112.03[a]					
Stroop effect			209.58					
Cohen's *d*			1.51					
RT incongruent			994.57 ± 186.64				CH-NAT	
RT congruent			784.92 ± 125.16					
Stroop effect			209.65					
Cohen's *d*			1.34					
RT incongruent			952.18 ± 143.23				CH-PAT	
RT congruent			742.66 ± 98.90					
Stroop effect			209.52					
Cohen's *d*			1.73					
Burin and Kawashima, (2021)								Comparisons not statistically significant
RT incongruent			1933.42 ± 330.45				All participants	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
RT incongruent			N.A.				1PP	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
RT incongruent			N.A.				3PP	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
Byun et al. (2024)								
RT incongruent			1623.80				control	No significant difference at baseline Stroop Interference (incongruent - neutral trials) between the control group and the mild exercise group.
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
RT incongruent			1577.60				mild exercise	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
Faulkner et al. (2017)								• HA < TIA
RT incongruent			N.A.			N.A.	TIA	
RT congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
Gajewski et al. (2012)								• Val/Val > Val/Met-Met/Met
RT incongruent			1105.00 ± 180.83[a]				All participants	
RT congruent			955.00 ± 172.82[a]					
Stroop effect			150.00					
Cohen's *d*			0.85					
RT incongruent			N.A.				Val/Val	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
RT incongruent			N.A.				Val/Met-Met/Met	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
Gajewski and Falkenstein, (2015)								Comparisons not statistically significant
RT incongruent			1106.00 ± 178.98[a]				All participants	

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Table 2 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main RT findings between groups
RT congruent			976.00 ± 178.98[a]					
Stroop effect			130.00					
Cohen's *d*			0.73					
RT incongruent			N.A.				Low active	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
RT incongruent			N.A.				Active	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
Gajewski et al. (2020)								• HA > YA
RT incongruent	885.85 ± 125.47[a,c]	1001.45 ± 176.25[a,c]	1430.41 ± 195.30[a,c]				All participants	• HA > MA
RT congruent	762.54 ± 122.16[a,c]	879.59 ± 159.73[a,c]	1149.07 ± 187.13[a,c]					
Stroop effect	123.31	121.86	281.34					
Cohen's *d*	1.00	0.73	1.47					
RT incongruent			1149.52 ± 178.40[a,c]				old high performers	
RT congruent			919.34 ± 147.74[a,c]					
Stroop effect			230.18					
Cohen's *d*			1.41					
RT incongruent			1407.01 ± 178.24[a,c]				old mid performers	
RT congruent			1130.63 ± 172.52[a,c]					
Stroop effect			276.38					
Cohen's *d*			1.58					
RT incongruent			1734.72 ± 229.27[a,c]				old low performers	
RT congruent			1397.25 ± 241.15[a,c]					
Stroop effect			337.48					
Cohen's *d*			1.43					
Gauthier et al. (2013)								Both control and inhibition - switching condition:
RT incongruent	898.20 ± 212.02[a]		1239.91 ± 524.81[a]					• HA > YA
RT congruent	575.52 ± 139.97[a]		696.78 ± 129.29[a]					
Stroop effect	322.68		543.13					
Cohen's *d*	1.83		1.71					
Goenarjo et al. (2021)								• HA > MA
RT incongruent			1009.90 ± 206.50				All participants	• HA lower-fit > HA higher-fit
RT congruent			861.30 ± 132.30					
Stroop effect			148.60					
Cohen's *d*			0.88					
RT incongruent		930.70 ± 106.60	1085.90 ± 132.90				Higher-fit	
RT congruent		841.70 ± 89.20	853.70 ± 13.60					
Stroop effect		89.00	232.20					
Cohen's *d*		0.91	3.17					
RT incongruent		840.00 ± 160.90	1264.10 ± 177.40				Lower-fit	
RT congruent		744.30 ± 99.60	1040.80 ± 111.40					
Stroop effect		99.70	223.30					
Cohen's *d*		0.73	1.55					
Hamasaki et al. (2018a)								Stroop interference time (difference between EASY and HARD conditions)
RT incongruent	N.A.	N.A.	N.A.					• HA > YA
RT congruent	N.A.	N.A.	N.A.					YA (0.32), 50 s (0,32), 60 s (0,41), 70 s (0,58)
Stroop effect	N.A.	N.A.	N.A.					Stroop interference:
Cohen's *d*	N.A.	N.A.	N.A.					• high stiffness > low stiffness
Hamasaki et al. (2018b)								

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Table 2 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main RT findings between groups
RT incongruent			N.A.				All participants	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
RT incongruent			N.A.				Low stiffness	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
RT incongruent			N.A.				High stiffness	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
Heiberg et al. (2023)								Congruent and incongruent:
RT incongruent			1520.00 ± 380.00			1850.00 ± 380.00	SP	• SP > HA
RT congruent			1370.00 ± 280. 00			1580.00 ± 350.00		
Stroop effect			150.00			270.00		
Cohen's *d*			0.45			0.74		
Huang et al. (2022)								Congruent and incongruent:
RT incongruent	725.00		1053.00					• HA > YA
RT congruent	211.00		529.00					
Stroop effect	514.00		524.00					
Cohen's *d*	N.A.		N.A.					
Ji et al. (2019)								The Stroop effect and Cohen's d cannot be calculated
RT incongruent			N.A.					because the executive task
RT congruent			N.A.					contains both incongruent
Stroop effect			N.A.					and congruent trials
Cohen's *d*			N.A.					
Kaufmann et al. (2008)								Overall response latencies were
RT incongruent $_{physical}$			704.50 ± 101.50[a,d]		685.50 ± 74.50[a,d]			comparable between groups.
RT incongruent $_{numerical}$			713.50 ± 103.50[a,d]		688.00 ± 118.00[a,d]			
Stroop effect			N.A.		N.A.			
Cohen's *d*			N.A.		N.A.			
Kricheldorff et al. (2023)								• PD > HA
RT incongruent			N.A.			N.A.	PD	
RT congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
Laguë-Beauvais et al. (2013)								Interference:
RT incongruent	807.89 ± 131.27		1033.58 ± 99.43					• HA > YA
RT congruent	N.A.		N.A.					
Stroop effect	N.A.		N.A.					
Cohen's *d*	N.A.		N.A.					
Langenecker et al. (2004)								Comparisons not statistically significant
RT incongruent	799.00 ± 134.00		851.00 ± 115.00					
RT congruent	693.00 ± 111.00		733.00 ± 133.00					
Stroop effect	106.00		118.00					
Cohen's *d*	0.87		0.95					
Lau et al. (2020)								N.R.
RT incongruent					N.A.			
RT congruent					N.A.			
Stroop effect					N.A.			
Cohen's *d*					N.A.			
RT incongruent					N.A.			
RT congruent					N.A.			
Stroop effect					N.A.			
Cohen's *d*					N.A.			
Li et al. (2009)								• AD > MCI > HA
RT incongruent			528.41 ± 97.55		595.03 ± 157.21	678.32 ± 155.39	AD	
RT congruent			N.A.		N.A.	N.A.		
Stroop effect			N.A.		N.A.	N.A.		

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Table 2 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main RT findings between groups
Cohen's *d*			N.A.		N.A.	N.A.		
Li et al. (2012)								• SIVD > SIVCIND > HA
RT incongruent			531.46 ± 98.76			714.63 ± 145.61	SIVD	
RT congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
RT incongruent						627.18 ± 139.55	SIVCIND	
RT congruent						N.A.		
Stroop effect						N.A.		
Cohen's *d*						N.A.		
Manard et al. (2017)								• HA > YA
RT incongruent	841.00 ± 142.30[a]		1019.00 ± 192.30[a]					
RT congruent	733.00 ± 143.10[a]		852.00 ± 143.10[a]					
Stroop effect	108.00		167.00					
Cohen's *d*	0.76		1.00					
Mason et al. (2022)								Comparisons not statistically significant
RT incongruent			1040.00[b]			1090.00[b]	CAS	
RT congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
Mathis et al. (2009)								All conditions:
RT incongruent	1126.00 ± 98.00	1258.00 ± 65.00	1278.00 ± 61.00					• YA < HA
RT congruent	1070.00 ± 85.00	1200.00 ± 95.00	1190.00 ± 67.00					• YA < MA
Stroop effect	56.00	58.00	88.00					
Cohen's *d*	0.61	0.73	1.38					
Milham et al. (2002)								Interference effect:
RT incongruent	N.A.		N.A.					• HA > YA
RT congruent	N.A.		N.A.					
Stroop effect	N.A.		N.A.					
Cohen's *d*	N.A.		N.A.					
Müller-Oehring et al. (2022)								Comparisons not statistically significant
RT incongruent			857.50[a]			897.50[a]	HIV	
RT congruent			799.00[a]			825.00[a]		
Stroop effect			58.50			72.50		
Cohen's *d*			N.A.			N.A.		
RT incongruent						890.00[a]	PD	
RT congruent						830.00[a]		
Stroop effect						60.00		
Cohen's *d*						N.A.		
Nombela et al. (2014)								Significant differences of all groups with
RT incongruent	820.00 (20 s)[a]	920.00 (40 s)[a]	960.00 (60 s)[a]					respect to HA.
RT congruent	700.00 (20 s)[a]	780.00 (40 s)[a]	810.00 (60 s)[a]					
Stroop effect	120.00 (20 s)	140.00 (40 s)	150.00					
Cohen's *d*	N.A.	N.A.	N.A.					
RT incongruent	900.00 (30 s)[a]	975.00 (50 s)[a]						
RT congruent	680.00 (30 s)[a]	810.00 (50 s)[a]						
Stroop effect	220.00 (30 s)	165.00 (50 s)						
Cohen's *d*	N.A.	N.A.						
Pisljar et al. (2013)								• D > HA
RT incongruent			1124.43 ± 137.64[d]			1426.68 ± 226.08[d]	D	
RT congruent			868.95 ± 117.44[d]			1104.90 ± 199.09[d]		
Stroop effect			255.48			321.79		
Cohen's *d*			2.00			1.51		
Puente et al. (2014)								Comparisons not statistically significant
RT incongruent			1084.60 ± 182.00		1173.50 ± 337.70			

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Table 2 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main RT findings between groups
RT congruent			837.50 ± 131.10		864.80 ± 167.90			
Stroop effect			247.10		308.70			
Cohen's *d*			1.58		1.22			
Ramos-Goicoa et al. (2016)								
RT incongruent		549.80 ± 108.30	601.70 ± 152.00		553.80 ± 76.80		MA aMCI	• HA > MA
RT congruent		529.70 ± 85.10	563.80 ± 132.50		517.20 ± 61.00			
Stroop effect		20.10	37.90		36.60			
Cohen's *d*		0.21	0.27		0.53			
RT incongruent					626.20 ± 102.90		HA aMCI	
RT congruent					582.30 ± 88.80			
Stroop effect					43.90			
Cohen's *d*					0.46			
Sánchez-Moguel et al. (2018)								Stroop interference:
RT incongruent			707.46 ± 63.21[a]				All participants	• normal EEG > Theta EEG
RT congruent			651.25 ± 58.93[a]					
Stroop effect			56.21					
Cohen's *d*			0.92					
RT incongruent			721.80 ± 53.83				Normal-EEG	
RT congruent			652.88 ± 52.61					
Stroop effect			68.92					
Cohen's *d*			1.30					
RT incongruent			702.12 ± 72.60				Theta-EEG	
RT congruent			649.62 ± 65.25					
Stroop effect			52.50					
Cohen's *d*			0.76					
Sánchez-Moguel et al. (2022)								The Theta-EEG group showed fewer differences
RT incongruent			713.09 ± 66.01[a]				All participants	Between stimulus types than
RT congruent			657.53 ± 65.03[a]					the Normal-EEG group.
Stroop effect			54.56					
Cohen's *d*			0.83					
RT incongruent			727.47 ± 59.21				Normal-EEG	
RT congruent			658.97 ± 59.13					
Stroop effect			68.50					
Cohen's *d*			1.16					
RT incongruent			698.71 ± 72.81				Theta-EEG	
RT congruent			656.10 ± 70.93					
Stroop effect			42.61					
Cohen's *d*			0.59					
Schroeter et al. (2007)								• CMA > HA
RT incongruent			1543.00 ± 298.00[a]			2571.00 ± 890.00[a]	CMA	
RT congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
Schulte et al. (2011)								Stroop effects:
RT incongruent	863.30 ± 223.00[a]		773.10 ± 203.99[a]					• HA < YA
RT congruent	800.70 ± 212.15[a]		747.30 ± 190.48[a]					
Stroop effect	62.60		25.80					
Cohen's *d*	0.29		0.13					
Tam et al. (2015)								Comparisons not statistically significant

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Table 2 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main RT findings between groups
RT incongruent	684.60 ± 144.60		747.70 ± 116.70					
RT congruent	567.50 ± 123.20		607.80 ± 98.00					
Stroop effect	117.10		139.90					
Cohen's *d*	0.87		1.30					
Vidoni et al. (2013)								N.R.
RT incongruent			1075.00 ± 204.00[b]			1103.00 ± 195.00[b]	AD	
RT congruent			940.00 ± 213.00[b]			991.00 ± 171.00[b]		
Stroop effect			135.00			112.00		
Cohen's *d*			0.65			0.61		
West and Bell, (1997)								Interference:
RT incongruent	750.00[a,b]		1100.00[a,b]					Comparisons not statistically significant
RT congruent	612.50[a,b]		822.00[a,b]					
Stroop effect	137.50		278.00					
Cohen's *d*	N.A.		N.A.					
You et al. (2021)								N.R.
RT incongruent					1574.09 ± 96.21[d]			
RT congruent					1174.51 ± 105.61[d]			
Stroop effect					399.58			
Cohen's *d*					N.A.			
RT incongruent					1557.39 ± 83.21[d]			
RT congruent					1157.39 ± 196.04[d]			
Stroop effect					400.00			
Cohen's *d*					N.A.			
Zurrón et al. (2014)								• HA > YA
RT incongruent	647.00 ± 79.00		951.00 ± 170.00					
RT congruent	587.00 ± 88.00		827.00 ± 134.00					
Stroop effect	60.00		124.00					
Cohen's *d*	0.72		0.82					
Simon studies								
Cespón et al. (2013)								Comparisons not statistically significant
RT incongruent			595.00 ± 85.00		631.00 ± 123.00		sdaMCI	
RT congruent			544.00 ± 86.00		580.00 ± 109.00			
Simon effect			51.00		51.00			
Cohen's *d*			0.44	.	0.44			
RT incongruent					664.00 ± 121.00		mdaMCI	
RT congruent					595.00 ± 97.00			
Simon effect					69.00			
Cohen's *d*					0.63			
Cespón et al. (2015a)								N.R.
RT incongruent			624.80 ± 109.46[a]		636.20 ± 109.60[a]		sdaMCI	
RT congruent			567.50 ± 98.00[a]		583.30 ± 98.70[a]			
Simon effect			52.90		52.90			
Cohen's *d*			0.51		0.51			
RT incongruent					667.30 ± 109.46[a]		mdaMCI	
RT congruent					611.20 ± 97.68[a]			
Simon effect					56.10			
Cohen's *d*					0.54			
Cespón et al. (2015b)								Comparisons not statistically significant
RT incongruent			604.00 ± 89.00		602.00 ± 48.00		mdnaMCI	
RT congruent			551.00 ± 92.00		565.00 ± 55.00			
Simon effect			37.00		37.00			
Cohen's *d*			0.72		0.72			

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Table 2 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main RT findings between groups
RT incongruent					636.00 ± 125.00		sdaMCI	
RT congruent					584.00 ± 112.00			
Simon effect					52.00			
Cohen's *d*					0.44			
RT incongruent					667.00 ± 128.00		mdaMCI	
RT congruent					599.100.00			
Simon effect					68.00			
Cohen's *d*					0.60			
Cespón et al. (2018)								Comparisons not statistically significant
RT incongruent				593.00 ± 80.00			Low SMC	
RT congruent				555.00 ± 88.00				
Simon effect				62.00				
Cohen's *d*				0.94				
RT incongruent				614.00 ± 71.00			High SMC	
RT congruent				552.00 ± 61.00				
Simon effect				62.00				
Cohen's *d*				0.94				
Cespón et al. (2022)								Both congruent and incongruent:
RT incongruent	413.00		553.00					• HA > YA
RT congruent	384.00		502.00					
Simon effect	51.00		51.00					
Cohen's *d*	N.A.		N.A.					
Katayama et al. (2023)								Both congruent and incongruent:
RT incongruent			610.00 ± 90.00[c]		670.00 ± 100.00[c]			• MCI > HA
RT congruent			580.00 ± 90.00[c]		630.00 ± 100.00[c]			
Simon effect			40.00		40.00			
Cohen's *d*			0.40		0.40			
Singh et al. (2018)								Comparisons not statistically significant
RT incongruent			590.00[a]			600.00[a]	PD	
RT congruent			550.00[a]			560.00[a]		
Simon effect			40.00			40.00		
Cohen's *d*			N.A.			N.A.		
Singh et al. (2023)								For the Simon task, there was a main effect
RT incongruent			558.00[b]		562.00[b]	522.00[b]	PDMCI	of Group but no interaction. These findings
RT congruent			537.00[b]		520.00[b]	499.00[b]	PD	indicate that PD-related cognitive
Simon effect			N.A.		N.A.	N.A.		dysfunction does not reliably affect
Cohen's *d*			N.A.		N.A.	N.A.		behavioural indicators of control
RT incongruent						540.00[b]	PDD	adaptation during incongruent Simon trials.
RT congruent						N.A.		
Simon effect						N.A.		
Cohen's *d*						N.A.		
Simon and Stroop studies								
Cespón et al. (2023)								N.R.
RT incongruent (Simon)			593.95 ± 72.75[a]				All participants	
RT congruent (Simon)			542.40 ± 66.50[a]					
Simon effect			51.55					
Cohen's *d* (Simon)			0.74					
RT incongruent (Stroop)			653.30 ± 97.10[a]					
RT congruent (Stroop)			568.95 ± 78.35[a]					
Stroop effect			84.35					
Cohen's *d* (Stroop)			0.96					
RT incongruent (Simon)			605.50 ± 87.90				LCR	
RT congruent (Simon)			550.30 ± 72.20					
Simon effect			55.20					
Cohen's *d* (Simon)			0.69					
RT incongruent (Stroop)			677.90 ± 114.50					

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Table 2 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main RT findings between groups
RT congruent (Stroop)			583.60 ± 73.80					
Stroop effect			94.30					
Cohen's *d* (Stroop)			1.00					
RT incongruent (Simon)			582.40 ± 57.60				HCR	
RT congruent (Simon)			534.50 ± 60.80					
Simon effect			47.90					
Cohen's *d* (Simon)			0.81					
RT incongruent (Stroop)			628 70 ± 79.70					
RT congruent (Stroop)			554.30 ± 82.90					
Stroop effect			74.40					
Cohen's *d* (Stroop)			0.92					
Onur et al. (2011)								Interference:
RT incongruent	N.A.		N.A.					• HA > YA
RT congruent	N.A.		N.A.					
Simon effect	N.A.		N.A.					
Stroop effect	N.A.		N.A.					
Cohen's *d*	N.A.		N.A.					

Note. Reaction Time (RT) values are expressed in milliseconds as mean ± standard deviation. YA: Young Adults. MA: Middle-aged Adults. HA: Healthy Ageing. SCD: Subjective Cognitive Decline. MCI: Mild Cognitive Impairment. CH-NAT: normal Aβ/tau ratios. CH-PAT: abnormal Aβ/tau ratios. N.A. Not available or not available for individual subgroups, as the studies only included one group. TIA: Transient Ischemic Attack. Val/Val: Val/Val genotype carriers. Val/Met: Val/Met genotype carriers. Met/Met: Met/Met genotype carriers. SP: Stroke Patients. PD: Parkinson's Disease. AD: Alzheimer's Disease. SIVD: Subcortical Ischemic Vascular Dementia. SIVCIND: Subcortical Ischemic Vascular Cognitive Impairment No Dementia. CAS: Carotid Atherosclerosis. HIV: Human Immune-deficiency Virus. D: Depression. aMCI: amnestic MCI. CMA: Cerebral Micro Angiopathy. N.C: Not Computable. N.R.: Not Reported. sdaMCI: single domain amnestic MCI. mdaMCI: multiple domains amnestic MCI. mdnaMCI: multiple domains non amnestic MCI. SMC: Subjective Memory Complaints. PDMCI: MCI due to Parkinson's Disease. PDD: Dementia in PD. LCR: Low Cognitive Reserve. HCR: High Cognitive Reserve.

[a] The values were obtained by calculations based on the data available in the study (e.g., subgroups, standard errors, figures).

[b] Median values.

[c] Data received by the authors.

[d] Data only for correct responses.

(Liu et al., 2004). We highlight how age-related differences, positive lifestyle interventions (e.g. cognitive and/or physical training, nutritional supplements), biological influences (e.g. genetics, AD biomarkers), subjective or objective cognitive impairments (SCD, MCI and dementia), health conditions (healthy ageing) and chronic diseases (neurological, e.g. AD, PD, psychiatric, e.g. depression, and cerebral vascular diseases) influence executive control.

4.1. EEG results in the Stroop task

4.1.1. Age-related differences

Nombela et al. (2014) and West and Bell (1997) found that older adults show slower RTs and lower accuracy in the Stroop task compared to younger adults. Specifically, Nombela et al. (2014) found that the right posterior parietal cortex, frontobasal areas and left ACC are more involved in conflict resolution in older adults, which is associated with opposing changes in alpha and theta activity. West and Bell (1997) noted that older adults rely more on the posterior attentional system due to a decline in anterior attention systems.

4.1.2. Biological influences (AD biomarkers)

CR plays a key role in maintaining cognitive performance despite the presence of AD biomarkers. Arakaki et al. (2022) showed that healthy individuals with abnormal amyloid/tau ratios exhibited compensatory occipital event-related alpha desynchronisation and increased spectral alpha entropy. Despite these neurobiological changes, which indicate compensatory hyperactivity, no significant differences in performance were found in the Stroop task compared to the control group.

4.1.3. Neurological disease (Parkinson's Disease)

Kricheldorff et al. (2023) showed that patients with PD had slower RTs and higher error rates compared to healthy older adults. PD patients also showed impaired proactive cognitive control, as evidenced by reduced modulation of theta power in the mid-frontal area during the Stroop task, indicating impaired executive control strategies.

4.2. EEG/ERP results in the Stroop task

4.2.1. Age-related differences

Zurrón et al. (2014) found that older adults showed longer RTs for incongruent stimuli and delayed N2 and P3b latencies compared to younger adults. In addition, older participants showed increased frontal P150 and reduced parietal P3b amplitudes. This suggests a reorganisation of neural processing in old age to maintain cognitive performance despite longer processing time for incongruent stimuli.

4.2.2. Healthy ageing and positive lifestyle interventions

Sánchez-Moguel et al. (2018) observed in the study with healthy elderly people that the RTs in the group with normal EEG were significantly longer than in the group with theta EEG in relation to the Stroop effect interference. In addition, Sánchez-Moguel et al. (2022) observed that excessive theta activity in older adults led to compensatory neurobiological adaptations despite similar accuracy performances.

Physical activity has been shown to improve cognitive control. Gajewski and Falkenstein (2015) reported that physically active older adults performed better on the Stroop task, made fewer errors and showed enhanced frontal cortex activity, as indicated by shorter P2 latency and more negative N2 and N450 amplitudes. In addition, Gajewski et al. (2020) found that CR strengthened by education and physical activity helps to maintain cognitive control performance despite ageing.

Table 3
Main error rates findings.

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main ER findings between groups
Stroop studies								
Arakaki et al. (2022)								Comparisons not statistically significant
ER incongruent			10.00 ± 4.50[a,c]				All participants	
ER congruent			6.00 ± 2.50[a,c]					
Stroop effect			4.00					
Cohen's *d*			1.14					
ER incongruent			10.00 ± 4.00 * *				CH-NAT	
ER congruent			6.00 ± 3.00 * *					
Stroop effect			4.00					
Cohen's *d*			1.14					
ER incongruent			10.00 ± 5.00 * *				CH-PAT	
ER congruent			6.00 ± 2.00 * *					
Stroop effect			4.00					
Cohen's *d*			1.14					
Burin and Kawashima, (2021)								Comparisons not statistically significant
ER incongruent			15.79 ± 1.41				All participants	
ER congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
RT incongruent			N.A.				1PP	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
RT incongruent			N.A.				3PP	
RT congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
Byun et al. (2024)								N.R.
ER incongruent			19.30				control	
ER congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
ER incongruent			15.80				mild exercise	
ER congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
Faulkner et al. (2017)								N.R.
ER incongruent			N.A.			N.A.	TIA	
ER congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
Gajewski et al. (2012)								Incompatible trials (colour naming):
ER incongruent			5.10 ± 6.80[a]				All participants	• Val/Val > Val/Met-Met/Met
ER congruent			1.60 ± 2.28[a]					
Stroop effect			3.50					
Cohen's *d*			0.77					
ER incongruent			6.30 ± 6.22[a]				Val/Val	
ER congruent			1.60 ± 6.22[a]					
Stroop effect			4.70					
Cohen's *d*			0.76					
ER incongruent			3.80 ± 7.21[a]				Val/Met -	
ER congruent			1.60 ± 6.48[a]				Met/Met	
Stroop effect			2.20					
Cohen's *d*			0.32					
Gajewski and Falkenstein, (2015)								Incompatible trials of the color-naming:

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Table 3 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main ER findings between groups
ER incongruent			2.90 ± 3.79[a]				All participants	• Low active > Active
ER congruent			1.30 ± 1.26[a]					
Stroop effect			1.60					
Cohen's *d*			0.63					
ER incongruent			N.A.				Low active	
ER congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
ER incongruent			N.A.				Active	
ER congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
Gajewski et al. (2020)								• HA > YA
ER incongruent	8.17 ± 6.51[a,c]	12.10 ± 11.71[a,c]	13.70 ± 9.58[a,c]				HA All participants	• HA > MA
ER congruent	2.00 ± 2.52[a,c]	2.04 ± 3.45[a,c]	1.82 ± 3.52[a,c]					
Stroop effect	6.17	10.06	11.89					
Cohen's *d*	1.37	1.33	1.81					
ER incongruent			7.81 ± 5.89[a,c]				old high performers	
ER congruent			1.35 ± 2.45[a,c]					
Stroop effect			6.45					
Cohen's *d*			1.55					
ER incongruent			13.06 ± 10.20[a,c]				old middle performers	
ER congruent			1.67 ± 4.18[a,c]					
Stroop effect			11.39					
Cohen's *d*			1.58					
ER incongruent			20.25 ± 12.67[a,c]				old low performers	
ER congruent			2.42 ± 3.93[a,c]					
Stroop effect			17.82					
Cohen's *d*			2.15					
Gauthier et al. (2013)								Both control and inhibition - switching condition:
ER incongruent	3.81 ± 4.56[a,c]		12.61 ± 13.41[a,c]					
ER congruent	0.48 ± 1.33[a,c]		2.42 ± 8.96[a,c]					• HA > YA
Stroop effect	3.33		10.19					
Cohen's *d*	1.13		0.90					
Goenarjo et al. (2021)								Comparisons not statistically significant
ER incongruent			3.30 ± 4.60[c]				All participants	
ER congruent			2.60 ± 2.70[c]					
Stroop effect			0.70					
Cohen's *d*			0.19					
ER incongruent		1.70 ± 1.50[c]	2.00 ± 3.60[c]				Higher-fit	
ER congruent		3.70 ± 3.70[c]	0.70 ± 0.90[c]					
Stroop effect		−2.00	1.30					
Cohen's *d*		−0.77	0.58					
ER incongruent		5.30 ± 7.20[c]	5.00 ± 4.60[c]				Lower-fit	
ER congruent		2.50 ± 1.80[c]	3.00 ± 2.70[c]					
Stroop effect		2.80	2.00					
Cohen's *d*		0.62	0.55					
Hamasaki et al. (2018a)								N.R.
ER incongruent	N.A.	N.A.	N.A.					
ER congruent	N.A.	N.A.	N.A.					
Stroop effect	N.A.	N.A.	N.A.					
Cohen's *d*	N.A.	N.A.	N.A.					
Hamasaki et al. (2018b)								N.R.

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Table 3 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main ER findings between groups
ER incongruent			N.A.				All participants	
ER congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
ER incongruent			N.A.				Low stiffness	
ER congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
ER incongruent			N.A.				High stiffness	
ER congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
Heiberg et al. (2023)								Congruent and incongruent:
ER incongruent			11.20 ± 5.00[b]			24.90 ± 23.70[b]	SP	• SP > HA
ER congruent			1.70 ± 2.20[b]			5.10 ± 7.90[b]		
Stroop effect			9.50			19.80		
Cohen's *d*			2.64			1.25		
Huang et al. (2022)								Accuracy (incongruent):
ER incongruent	5.72[c]		14.93[c]					• HA < YA
ER congruent	3.51[c]		8.27[c]					
Stroop effect	2.21		6.66					
Cohen's *d*	N.A.		N.A.					
Ji et al. (2019)								The Stroop effect and Cohen's d cannot be calculated because the executive task contains both incongruent and congruent trials.
ER incongruent			N.A.					
ER congruent			N.A.					
Stroop effect			N.A.					
Cohen's *d*			N.A.					
Kaufmann et al. (2008)								Comparisons not statistically significant
ER incongruent $_{physical}$			20.00 ± 28.50[a]		36.50 ± 23.00[a]			
ER incongruent $_{numerical}$			41.50 ± 18.50[a]		41.50 ± 20.50[a]			
Stroop effect			N.A.		N.A.			
Cohen's *d*			N.A.		N.A.			
Kricheldorff et al. (2023)								• PD > HA
ER incongruent			N.A.			N.A.	PD	
ER congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
Laguë-Beauvais et al. (2013)								Interference:
ER incongruent	2.00 ± 2.43[c]		5.56 ± 7.00[c]					• HA > YA
ER congruent	N.A.		N.A.					
Stroop effect	N.A.		N.A.					
Cohen's *d*	N.A.		N.A.					
Langenecker et al. (2004)								• HA > YA
ER incongruent	4.90 ± 5.20		16.60 ± 10.00					
ER congruent	2.30 ± 2.00		12.60 ± 8.60					
Stroop effect	2.60		4.00					
Cohen's *d*	0.72		0.43					
Lau et al. (2020)								N.R.
ER incongruent					N.A.			
ER congruent					N.A.			
Stroop effect					N.A.			
Cohen's *d*					N.A.			
ER incongruent					N.A.			
ER congruent					N.A.			
Stroop effect					N.A.			
Cohen's *d*					N.A.			
Li et al. (2009)								• AD > MCI > HA
ER incongruent			4.31 ± 10.58		5.31 ± 6.21	9.36 ± 14.13	AD	
ER congruent			N.A.		N.A.	N.A.		
Stroop effect			N.A.		N.A.	N.A.		
Cohen's *d*			N.A.		N.A.	N.A.		
Li et al. (2012)								• SIVD > SIVCIND > HA

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Table 3 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main ER findings between groups
ER incongruent			3.37 ± 1.35			5.24 ± 2.17	SIVD	
ER congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
ER incongruent						3.44 ± 1.93	SIVCIND	
ER congruent						N.A.		
Stroop effect						N.A.		
Cohen's *d*						N.A.		
Manard et al. (2017)								Comparisons not statistically significant
ER incongruent	4.58 ± 1.23[a,c]		6.33 ± 1.23[a,c]					
ER congruent	1.42 ± 0.85[a,c]		2.67 ± 0.85[a,c]					
Stroop effect	3.16		3.66					
Cohen's *d*	3.04		3.52					
Mason et al. (2022)								Comparisons not statistically significant
ER incongruent			1.00[b,c]			2.00[b,c]	CSA	
ER congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
Mathis et al. (2009)								Accuracy (incongruent condition):
ER incongruent	7.60 ± 5.60[c]	24.00 ± 18.10[c]	21.30 ± 9.30[c]					• YA > HA
ER congruent	2.50 ± 1.80[c]	10.00 ± 9.90[c]	8.30 ± 6.20[c]					• YA > MA
Stroop effect	5.10	14.00	13.00					
Cohen's *d*	1.38	1.00	1.68					
Milham et al. (2002)								Interference effect:
ER incongruent	N.A.		N.A.					• HA > YA
ER congruent	N.A.		N.A.					
Stroop effect	N.A.		N.A.					
Cohen's *d*	N.A.		N.A.					
Müller-Oehring et al. (2022)								Comparisons not statistically significant
ER incongruent	N.A.					N.A.	HIV	
ER congruent	N.A.					N.A.		
Stroop effect	N.A.					N.A.		
Cohen's *d*	N.A.					N.A.		
ER incongruent						N.A.	PD	
ER congruent						N.A.		
Stroop effect						N.A.		
Cohen's *d*						N.A.		
Nombela et al. (2014)								Significant differences of all groups with respect to HA.
ER incongruent	N.A.	N.A.	N.A.					
ER congruent	N.A.	N.A.	N.A.					
Stroop effect	N.A.	N.A.	N.A.					
Cohen's *d*	N.A.	N.A.	N.A.					
Pišljar et al. (2013)								N.R.
ER incongruent			N.A.			N.A.	D	
ER congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
Puente et al. (2014)								Comparisons not statistically significant
ER incongruent			3.00 ± 5.00[c]		8.00 ± 11.00[c]			
ER congruent			1.00 ± 12.00[c]		2.00 ± 3.00[c]			
Stroop effect			2.00		6.00			
Cohen's *d*			0.24		0.86			
Ramos-Goicoa et al. (2016)								• HA > MA
ER incongruent		2.14 ± 1.71[c]	5.43 ± 4.00[c]		5.43 ± 8.86[c]		MA aMCI	
ER congruent		2.71 ± 2.28[c]	6.43 ± 5.14[c]		3.86 ± 4.28[c]			
Stroop effect		−0.57	−1.00		1.57			
Cohen's *d*		−0.29	−0.22		0.34			
ER incongruent					9.14 ± 12.43[c]		HA aMCI	
ER congruent					7.43 ± 5.14[c]			
Stroop effect					1.71			
Cohen's *d*					0.24			
Sánchez-Moguel et al. (2018)								Comparisons not statistically significant

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Table 3 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main ER findings between groups
ER incongruent			21.62 ± 12.56[a,c]				All participants	
ER congruent			13.95 ± 9.03[a,c]					
Stroop effect			7.67					
Cohen's *d*			0.71					
ER incongruent			20.95 ± 12.05[c]				Normal-EEG	
ER congruent			14.06 ± 8.69[c]					
Stroop effect			6.89					
Cohen's *d*			0.66					
ER incongruent			22.28 ± 13.07[c]				Theta-EEG	
ER congruent			13.83 ± 9.37[c]					
Stroop effect			8.45					
Cohen's *d*			0.75					
Sánchez-Moguel et al. (2022)								Comparisons not statistically significant
ER incongruent			24.12 ± 17.24[a,c]				All participants	
ER congruent			16.65 ± 15.71[a,c]					
Stroop effect			7.47					
Cohen's *d*			0.45					
ER incongruent			22.87 ± 14.93[c]				Normal-EEG	
ER congruent			16.45 ± 14.28[c]					
Stroop effect			6.42					
Cohen's *d*			0.44					
ER incongruent			25.37 ± 19.55[c]				Theta-EEG	
ER congruent			16.85 ± 17.14[c]					
Stroop effect			8.52					
Cohen's *d*			0.46					
Schroeter et al. (2007)								Patients with CMA and controls showed a
ER incongruent			12.00 ± 14.00[a]			11.41 ± 14.00[a]	CMA	clear interference effect
ER congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
Schulte et al. (2011)								Comparisons not statistically significant
ER incongruent	N.A.		N.A.					
ER congruent	N.A.		N.A.					
Stroop effect	N.A.		N.A.					
Cohen's *d*	N.A.		N.A.					
Tam et al. (2015)								Comparisons not statistically significant
ER incongruent	N.A.		N.A.					
ER congruent	N.A.		N.A.					
Stroop effect	N.A.		N.A.					
Cohen's *d*	N.A.		N.A.					
Vidoni et al. (2013)								N.R.
ER incongruent			N.A.			N.A.	AD	
ER congruent			N.A.			N.A.		
Stroop effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
West and Bell, (1997)								Accuracy:
ER incongruent	N.A.		N.A.					• HA < YA
ER congruent	N.A.		N.A.					
Stroop effect	N.A.		N.A.					
Cohen's *d*	N.A.		N.A.					
You et al. (2021)								N.R.
ER incongruent					27.50 ± 5.89[c]			
ER congruent					20.50 ± 6.43[c]			
Stroop effect					7.00			
Cohen's *d*					1.14			
ER incongruent					26.00 ± 6.99[c]			

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Table 3 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main ER findings between groups
ER congruent					20.50 ± 4.97[c]			
Stroop effect					5.50			
Cohen's *d*					0.92			
Zurrón et al. (2014)								Comparisons not statistically significant
ER incongruent	4.42 ± 3.8[c]		3.27 ± 3.8[c]					
ER congruent	5.19 ± 3.8[c]		5.57 ± 3.8[c]					
Stroop effect	−0.77		−2.30					
Cohen's *d*	−0.20		−0.61					
Simon studies								
Cespón et al. (2013)								Percentage of Errors (PE):
ER incongruent			7.40 ± 5.20		6.60 ± 5.80		sdaMCI	• mdaMCI > HA
ER congruent			2.60 ± 3.70		2.40 ± 3.00			• mdaMCI > sdaMCI
Simon effect			4.20		4.20			
Cohen's *d*			0.95		0.95			
ER incongruent					11.20 ± 7.10		mdaMCI	
ER congruent					5.10 ± 5.40			
Simon effect					6.10			
Cohen's *d*					0.98			
Cespón et al. (2015a)								Percentage of Errors (PE):
ER incongruent			8.10 ± 5.81[a]		5.40 ± 5.80[a]		sdaMCI	• mdaMCI > HA
ER congruent			2.60 ± 3.64[a]		1.80 ± 3.60[a]			• mdaMCI > sdaMCI
Simon effect			3.60		3.60			
Cohen's *d*			0.42		0.42			
ER incongruent					12.20 ± 5.81[a]		mdaMCI	
ER congruent					5.10 ± 3.49[a]			
Simon effect					7.10			
Cohen's *d*					1.18			
Cespón et al. (2015b)								Comparisons not statistically significant
ER incongruent			2.50 ± 3.10		2.70 ± 3.60		mdnaMCI	
ER congruent			1.80 ± 2.30		1.40 ± 2.60			
Simon effect			1.30		1.30			
Cohen's *d*			0.42		0.42			
ER incongruent					6.80 ± 5.90		sdaMCI	
ER congruent					7.20 ± 4.70			
Simon effect					−0.40			
Cohen's *d*					−0.08			
ER incongruent					6.90 ± 5.00		mdaMCI	
ER congruent					5.50 ± 3.70			
Simon effect					1.40			
Cohen's *d*					0.32			
Cespón et al. (2018)								Comparisons not statistically significant
ER incongruent				6.40 ± 4.50			Low SMC	
ER congruent				2.80 ± 3.70				
Simon effect				3.00				
Cohen's *d*				0.78				
ER incongruent				5.50 ± 4.60			High SMC	
ER congruent				2.50 ± 3.10				
Simon effect				3.00				
Cohen's *d*				0.78				
Cespón et al. (2022)								Comparisons not statistically significant
ER incongruent	7.75[a,c]		7.08[a,c]					
ER congruent	2.37[a,c]		1.78[a,c]					
Simon effect	N.A.		N.A.					
Cohen's *d*	N.A.		N.A.					

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Table 3 (*continued*)

Study	YA	MA	HA	SCD	MCI	other pathologies	subgroups	main ER findings between groups
Katayama et al. (2023)								Accuracy (incongruent):
ER incongruent			5.38 ± 11.60[c,d]		6.94 ± 13.49[c,d]			• MCI < HA
ER congruent			3.89 ± 11.89[c,d]		4.68 ± 13.15[c,d]			
Simon effect			2.26		2.26			
Cohen's *d*			0.17		0.17			
Singh et al. (2018)								Comparisons not statistically significant
ER incongruent			N.A.			N.A.	PD	
ER congruent			N.A.			N.A.		
Simon effect			N.A.			N.A.		
Cohen's *d*			N.A.			N.A.		
Singh et al. (2023)								N.R.
ER incongruent			N.A.		N.A.	N.A.	PDMCI	
ER congruent			N.A.		N.A.	N.A.	PD	
Simon effect			N.A.		N.A.	N.A.		
Cohen's *d*			N.A.		N.A.	N.A.		
ER incongruent						N.A.	PDD	
ER congruent						N.A.		
Simon effect						N.A.		
Cohen's *d*						N.A.		
Simon and Stroop studies								
Cespón et al. (2023)								Number of errors:
ER incongruent (Simon)			4.45 ± 4.84[a,c]				All participants	• LCR > HCR
ER congruent (Simon)			0.85 ± 1.48[a,c]					
Simon effect			3.6					
Cohen's *d* (Simon)			1.14					
ER incongruent (Stroop)			4.30 ± 4.84[a,c]					
ER congruent (Stroop)			0.85 ± 1.76[a,c]					
Stroop effect			3.45					
Cohen's *d* (Stroop)			1.05					
ER incongruent (Simon)			6.10 ± 6.62[c]				LCR	
ER congruent (Simon)			0.97 ± 1.58[c]					
Simon effect			5.13					
Cohen's *d* (Simon)			1.25					
ER incongruent (Stroop)			5.24 ± 5.63[c]					
ER congruent (Stroop)			1.17 ± 1.90[c]					
Stroop effect			4.07					
Cohen's *d* (Stroop)			1.08					
ER incongruent (Simon)			2.80 ± 3.07[c]				HCR	
ER congruent (Simon)			0.73 ± 1.38[c]					
Simon effect			2.07					
Cohen's *d* (Simon)			0.93					
ER incongruent (Stroop)			3.37 ± 4.0[c]					
ER congruent (Stroop)			0.54 ± 1.62[c]					
Stroop effect			2.83					
Cohen's *d* (Stroop)			1.00					
Onur et al. (2011)								Comparisons not statistically significant
ER incongruent	N.A.		N.A.					
ER congruent	N.A.		N.A.					
Simon effect	N.A.		N.A.					
Stroop effect	N.A.		N.A.					
Cohen's *d*	N.A.		N.A.					

Note. Error rate (ER) values are expressed in percentages as mean ± standard deviation. YA: Young Adults. MA: Middle-aged Adults. HA: Healthy Ageing. SCD: Subjective Cognitive Decline. MCI: Mild Cognitive Impairment. CH-NAT: normal Aβ/tau ratios. CH-PAT: abnormal Aβ/tau ratios. N.A. Not available or not available for individual subgroups, as the studies only included one group. N.R.: Not Reported. TIA: Transient Ischemic Attack. Val/Val: Val/Val genotype carriers.
Val/Met: Val/Met genotype carriers. Met/Met: Met/Met genotype carriers. N.C: Not Computable. SP: Stroke Patients. PD: Parkinson's Disease. AD: Alzheimer's Disease. SIVD: Subcortical Ischemic Vascular Dementia. SIVCIND: Subcortical Ischemic Vascular Cognitive Impairment No Dementia. CAS: Carotid Atherosclerosis. HIV: Human Immune-deficiency Virus. D: Depression. aMCI: amnestic MCI. EEG: Electroencephalography. CMA: Cerebral Micro Angiopathy. sdaMCI: single domain

amnestic MCI. mdaMCI: multiple domains amnestic MCI. mdnaMCI: multiple domains non amnestic MCI. SMC: Subjective Memory Complaints. PDMCI: MCI due to Parkinson's Disease. PDD: Dementia in PD. LCR: Low Cognitive Reserve. HCR: High Cognitive Reserve.

[a] The values were obtained by calculations based on the data available in the study (e.g., subgroups, standard errors, figures).

[b] Median values.

[c] The accuracy values (percentage of correct responses) or number of errors have been converted into percentage error rates where available.

[d] Data received by the authors.

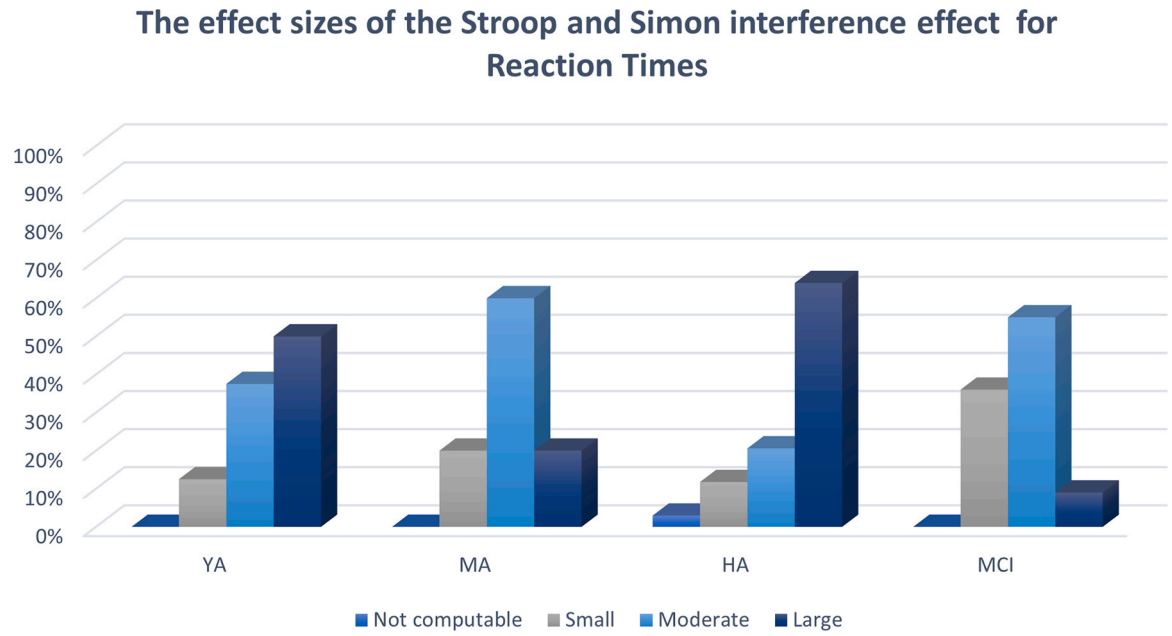

Fig. 2. Proportion of studies for each reference group reporting the effect size (Cohen's *d*) of Stroop and Simon interference effects for RTs. *Note*. YA: Young Adults. MA: Middle-aged Adults. HA: Healthy Ageing. MCI: Mild Cognitive Impairment.

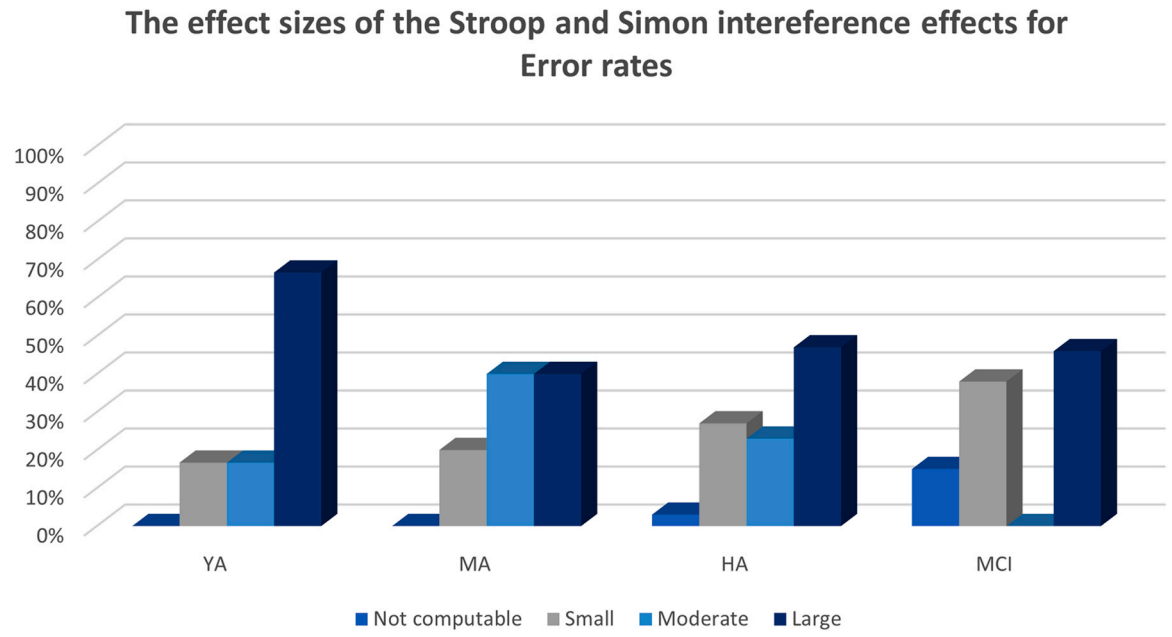

Fig. 3. Proportion of studies for each reference group reporting the effect size (Cohen's *d*) of Stroop and Simon interference effects for ERs. *Note*. YA: Young Adults. MA: Middle-aged Adults. HA: Healthy Ageing. MCI: Mild Cognitive Impairment.

4.2.3. Biological influences (Genetics)

Gajewski et al. (2012) showed that older adults carrying the Met allele of the BDNF Val66Met polymorphism had better interference control and increased N450 components, with shorter RTs and lower error rates compared to Val/Val carriers.

4.2.4. Mild cognitive impairment

Ramos-Goicoa et al. (2016) found that aMCI exacerbates age-related cognitive decline, with slower processing times and deficits in motor response preparation in the Stroop task, demonstrating the impact of cognitive impairment on executive functions.

Table 4
Main cerebral findings.

Study	Technique	Electrodes and/or investigated ROIs	Main findings	Interpretation of the results
Stroop studies				
Arakaki et al. (2022)	EEG	Frontal (Fz, F3, F4), Central (Cz, C3, C4), Parietal (Pz, P3, P4), Left Temporal (F7, T3, T5), Right Temporal (F8, T4, T6), Occipital (O1, O2).	Congruent trials: • Lower late delta power at the occipital region in CH-PATs compared to CH-NATs. Incongruent trials: • Lower late theta power at the occipital region and right temporal region in CH-PATs compared to CH-NATs; • CH-NATs had higher centroparietal theta compared to CH-PATs; • CH-PATs had more negative temporo-occipital alpha and beta in addition to higher parietal theta compared to CH-NATs.	The EEG analysis revealed significant differences in brain activation between cognitively healthy individuals with and without abnormal amyloid/tau levels, indicating that EEG activity could be a useful biomarker for early identification of cognitive changes associated with Alzheimer's disease.
Gajewski et al. (2012)	EEG/ERP	Fronto-central (Fz, FCz, Cz), Centro-parietal (CPz, Pz).	Interference trials: Val/Met - Met/Met group showed enhanced N450 compared to Val/Val.	There was a negative going wave (N450) that overlapped with frontal and parietal P3 and was influenced by compatibility and BDNF genotype. Most importantly, Stroop interference was only significant in the Val/Met-Met/Met group.
Gajewski and Falkenstein, (2015)	EEG/ERP	Fronto-central (Fz, FCz, Cz), Centro-parietal (CPz, Pz).	P2 latency: • delayed for low active group N2 amplitude: • larger in high active group N450 amplitude: • more negative in high active group.	In older people, physical activity correlates with increased brain activity in the prefrontal cortex, which enables better performance in a Stroop interference task.
Gajewski et al. (2020)	EEG/ERP	Central, Frontal, Prefrontal, Occipital, Parietal, Temporal (C3, C4, CP3, CP4, CPz, Cz, F3, F4, F7, F8, FC3, FC4, FCz, Fp1, Fp2, Fpz, Fz, O1, O2, Oz, P3, P4, P7, P8, PO3, PO4, POz, Pz, T7, T8).	P2/N2 complex during reactive task control were larger in the old high performers than low performers and similar to middle-aged or even young participants.	Successful interference control in old age is related to an efficient proactive and reactive mode of cognitive control that enables a level of performance normally observed in younger adults. This suggests that cognitive reserve in old age has a neural substrate in the form of increased brain activity involved in executive control tasks.
Kricheldorff et al. (2023)	EEG	128 active electrodes.	Both groups show a reduction in conflict-related theta activity in the midline front when more conflict is expected. Only the healthy participants showed reduced theta conflict power in items where high conflict was expected (MI).	Parkinson's patients show significant deficits in proactive cognitive control, which is reflected in the midline frontal theta modulation. In contrast to HA participants, PD patients were less able to regulate cognitive control in the reactive control task in tasks with high demands on cognitive control.
Nombela et al. (2014)	EEG	Prefrontal, central, temporal, parietal, occipital (Fp1, Fp2, Fz, C1, C2, C3, C4, Cz, T3, T4, Pz, O1 and O2).	Alpha band (congruent stimuli): • YA (30 s) < HA (60 s) in Fp2 Beta band (congruent and incongruent stimuli): • YA (30 s), MA (40 s) < HA (60 s) in Fp1, Fp2, C3, Cz, C4, T4, O2 Delta band (incongruent stimuli): • YA (30 s) > HA (60 s) in Pz • YA (20 s, 30 s), MA (40 s, 50 s) > HA (60 s) in Fp2 Delta band (congruent stimuli): • YA (30 s) > HA (60 s) in Fp2, T3, T4, O2 • YA (20 s), MA (40 s, 50 s) > HA (60 s) in Fp2 Sub-Alpha band (incongruent stimuli): • MA (40 s) > HA (60 s) in C3, Cz, C4 Sub-Alpha band (congruent stimuli): • YA (20 s) > HA (60 s) in Fp1, Fpz • MA (40 s) > HA (60 s) in Fpz Theta band (incongruent stimuli): • MA (40 s) > HA (60 s) in Fp1, T3, C3, Cz, C4, T4, O2 • MA (40 s) > HA (60 s) in Fp2 Theta band (congruent stimuli): • YA (30 s), MA (40 s) > HA (60 s) in Fp2, C3, C4 • YA (20 s) > HA (60 s) in C3.	The cortical areas involved in conflict resolution are the right posterior parietal area, the fronto-basal area and the left ACC. In addition, Stroop interference is directly influenced by opposing alpha and theta activity and starts to decline until the 60 s as healthy ageing occurs.
Pišljar et al. (2013)	EEG/ERP	Frontal, central, parietal (Fz, Cz, Pz).	Latencies: • No significant difference between groups of the N100, P200, N200 and P300. The P300b wave was absent in patients with depression. Amplitudes:	The specific combination of the pattern of interference effects (i.e., overall RT slowing in addition to abnormal or absent ERPs in all conditions) suggests that information processing and not only motor processing is impaired.

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Table 4 (*continued*)

Study	Technique	Electrodes and/or investigated ROIs	Main findings	Interpretation of the results
			• Patients: abolishment of the late positivity (corresponding to the P300b); • Patients < HA (N200 centro-parietally in all conditions) • Patients < HA (P300a in neutral condition centrally and frontally).	
Ramos-Goicoa et al. (2016)	EEG/ERP	Frontal, central, parietal (Fz, Cz, Pz).	LATENCY: • HA > MA (P150, P3b, N2) • MA aMCI > MA (P3b).	The electrophysiological results suggest that ageing also causes a slowdown in the neural processing of stimuli and a greater investment of resources (even though inefficient) for cognitive control. The results also showed that cognitive status influences Stroop-related neuronal function. The results support the use of ERP parameters as indicators of aMCI.
Sánchez-Moguel et al. (2018)	EEG/ERP	Coronal (frontal, fronto–central, central, centro-parietal, parietal) and Sagittal (left, left medial, medial, right medial, right).	Amplitude (300–500 ms): • Theta-EEG > Normal-EEG Amplitude (500–700 ms): • Normal-EEG > Theta-EEG (negative wave) N500 effect (amplitude of the incongruent condition > amplitude of the congruent condition): • Normal-EEG > Theta-EEG • Normal-EEG > Theta-EEG (interference effect).	People with excessive theta activity show a peculiar ERP pattern that can be observed at multiple levels of the neurocognitive system before clinical signs of cognitive impairment appear. This study is the first to clearly demonstrate a preclinical cognitive deficit in healthy older adults with excessive theta activity in their EEG.
Sánchez-Moguel et al. (2022)	EEG/ERP	Fronto-central, central, centro-parietal, parietal (Fcz, Cz, Cpz, Pz).	Total energy (delta, theta, alpha bands): • Theta-EEG > Normal-EEG Relative energy (delta): • Theta-EEG > Normal-EEG Relative energy (beta, gamma): • Normal-EEG > Theta-EEG Time window 258–516 ms: • Theta-EEG > Normal-EEG (theta, alpha bands) Time windows 258–516, 516–774, and 774–1032 ms: • Theta-EEG > Normal-EEG (delta band).	The signal energy was higher in the Theta- EEG group compared to Normal- EEG. Energy analysis of the ERP using wavelets allows accurate quantification of EEG energy during the Stroop task. These results suggest that the excessive energy in the Theta- EEG group may indicate that more neurons are recruited to solve the task with the same efficiency as in the Normal- EEG group.
West and Bell, (1997)	EEG/ERP	Frontal medial (F3-F4), frontal lateral (F7-F8), parietal (P3-P4), and occipital (O1-O2).	Color-word integrated: • HA ≠ YA (medial and lateral frontal regions, parietal region).	The age-related increase in the Stroop interference effect could be due to a decrease in the functional combinity of the PFC, resulting in an inability to suppress the influence of incongruent word information. In fact, the anterior attention system is more sensitive to the effects of increasing age than the posterior attention system.
Zurrón et al. (2014)	EEG/ERP	Frontal, central, parietal (Fz, Cz, Pz).	Latency: • HA > YA (N2, P3) Amplitude: • HA > YA (P150).	The psychophysiological data suggest that the age-related slowdown is not generalised, as it affects the neural networks underlying the cognitive processes of evaluation and categorisation, but not the perceptual processes. In addition, the ERP data confirm changes in the neural functioning of the older participants during the processing of a Stroop task.
Burin and Kawashima, (2021)	fNIRS	Left DLPFC (CHs 23, 25, and 26).	There was no difference in activation comparing the baselines of the two groups (1pp and 3pp).	At baseline, no significant neural differences were found between the groups. In contrast, increased activation was observed in the left DLPFC after the entire intervention.
Byun et al. (2024)	fNIRS	Left dorsolateral PFC (CHs 13, 14, 16, and 17), left ventrolateral PFC (CHs 2, 6, and 9), left frontopolar area (CHs 3, 7, and 10), right dorsolateral PFC (CHs 35, 36, 39, and 40), right ventrolateral PFC (CHs 26, 29, and 33), right frontopolar area (CHs 25, 28, and 32).	There were no significant differences between pre-conditions for the control and the mild exercise group.	Only a 3-month intervention with mild exercise has been shown to improve executive functions by modulating the neural efficiency of the prefrontal cortex in middle-aged adults, but especially in older subjects.
Faulkner et al. (2017)	fNIRS	PFC.	No between-group differences for prefrontal cortex perfusion were found.	Moderate-intensity exercise increased perfusion and executive function in both TIA and healthy older adults. However, these improvements were not associated with prefrontal cortex perfusion.
Goenarjo et al. (2021)	fNIRS	Dorsolateral and anterior PFC (left and right BAs 9 and 10).	Δ [Deoxy-Hb] in the right PFC: • HA > MA • higher fit > lower fit.	High cardiorespiratory fitness could allow healthy older men to achieve higher PFC oxygenation compared to middle-aged adults.
Hamasaki et al. (2018a)	fNIRS	Left and right PFC.	Δ [Oxy-Hb] in the left PFC: • HA (60 s, 70 s) < YA.	Significant laterality between the left and right prefrontal cortex was observed in young people, but not in middle-aged or older adults. This suggests that cerebral oxygenation in the

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Table 4 (*continued*)

Study	Technique	Electrodes and/or investigated ROIs	Main findings	Interpretation of the results
				left prefrontal cortex may be involved in age-related cognitive decline.
Hamasaki et al. (2018b)	fNIRS	Left and right PFC.	Δ [Oxy-Hb] in the left PFC: • Low stiffness > high stiffness Δ [Oxy-Hb] in low stiffness subjects: • left PFC > right PFC.	In middle-aged and older adults, a significant negative correlation was observed between central arterial stiffness and cerebral oxygenation in the left prefrontal cortex during executive function tasks. Both groups with low arterial stiffness showed higher cerebral oxygenation in the left prefrontal cortex compared to adults with high arterial stiffness. These results suggest that central arterial stiffness may be related to hemodynamics and function in the ageing brain.
Heiberg et al. (2023)	fNIRS	Superolateral, inferolateral, superomedial, inferomedial PFC.	Δ [Deoxy-Hb]: • HA > SP (superomedial, superolateral and inferomedial PFC).	Both groups showed an inverted hemodynamic response in the superolateral and superomedial prefrontal cortex, which was lower in stroke patients compared to healthy older adults.
Huang et al. (2022)	fNIRS	DLPFC, middle temporal gyrus, superior temporal gyrus, temporopolar area, pars triangularis Broca's area, and frontopolar area.	Comparisons not statistically significant (despite older people had higher activation than younger adults).	Both sides of the DLPFC and the right Broca's area were most strongly cortically activated in the Stroop test.
Ji et al. (2019)	fNIRS	Left VLPFC (CHs 1–4), left DLPFC (CHs 5, 6, 7 and 11), right DLPFC (CHs 13–16), posterior right VLPFC (CHs 17–20).	Left DLPFC activation: • PE > RC • CE + PE > RC Right DLPFC activation: • PE > RC • PE > CE • CE + PE > RC • CE + PE > CE.	Combined exercise has been shown to improve cognitive function better than single exercise, which was supported by task-efficient cerebral oxygenation and improved oxygen utilisation during cortical activation in older adults.
Laguë-Beauvais et al. (2013)	fNIRS	The anterior DLPFC (BAs 9, 10 and 46), the posterior DLPFC (BAs 6 and 4), the anterior VLPFC (BAs 10, 45 and 46) and the posterior VLPFC (BAs 4, 6 and 44) for both hemispheres.	Inhibition condition: • YA: No difference in HbO concentration among the prefrontal subregion • HA: small left anterior DLPFC and bilateral posterior DLPFC and VLPFC involvements Switching condition: • YA: Bilateral activation in the anterior PFC with left side prevalence • HA: wider bilateral brain areas • HA: HbR concentration lower than denomination condition.	These results indicate that inhibition and task switching differ in younger adults due to a lack of change in concentration during inhibition and in older adults due to greater utilisation of frontal resources during task switching. The age-related differences in the activation patterns of the prefrontal brain during inhibition and switching suggest that these two mechanisms are differentially altered in the course of normal aging.
Mason et al. (2022)	fNIRS	Bilateral PFC, bilateral sensorimotor cortex.	• Plaque group < HA (left hemisphere of the brain).	A consistent decrease in brain activity was observed in plaque group compared with healthy controls during the incongruous Stroop task.
Schroeter et al. (2007)	fNIRS	Left and right DLPFC (F3/F4-FC3/FC4).	Stroop effect: no significant difference was detected between groups.	The patients showed an early deoxygenation of the blood directly after the start of stimulation as well as a delay and reduction of the vascular response. The latter correlated highly with behavioural performance and neuropsychological deficits. The results indicate that neurovascular coupling and frontal lobe function are impaired in cerebral microangiopathy.
Gauthier et al. (2013)	fMRI	Left and right frontal and parietal lobe.	No significant difference was detected between young adults and healthy ageing.	Although BOLD signal changes elicited by a cognitive task were similar between groups, a lower maximal BOLD response were found in older adults. This suggests that a given BOLD response represents a greater change in neuronal activity in healthy older people than in young adults.
Kaufmann et al. (2008)	fMRI	Frontal, temporal, parietal, occipital and cerebellar regions bilaterally.	BOLD responses: • MCI > HA (frontal, temporal, parietal, occipital and cerebellar regions bilaterally). Congruity effect: • MCI > HA (bilateral occipital regions (including fusiform gyrus bilaterally), left inferior frontal cortex, left inferior parietal cortex and superior temporal gyrus, right cerebellum, right postcentral gyrus as well as in subcortical regions).	MCI patients showed not only stronger but also additional activations in cortical and cerebellar regions, most likely reflecting neurofunctional compensatory processes for the deficient control of executive abilities (i.e., interference processing).
Langenecker et al. (2004)	fMRI	Bilateral frontal, temporal, parietal, occipital, cerebellum, limbic, sub-lobar areas.	Incongruent - congruent: • HA > YA (frontal regions, right frontal gyrus, left inferior frontal gyrus).	The results showed comparable activation regions in general in young and older adults in a Stroop task, with a variety of predominantly prefrontal regions showing higher activation

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Study	Technique	Electrodes and/or investigated ROIs	Main findings	Interpretation of the results
				levels in the left inferior frontal gyrus in older adults during interference. The results are consistent with the hypothesis that older adults recruit additional, particularly prefrontal, areas during task performance.
Lau et al. (2020)	fMRI	Right and left dorsolateral PFC (BAs 9–46).	No significant difference was detected between groups for the Stroop task.	No significant intervention effects were observed in Stroop task-induced DLPFC activation.
Li et al. (2009)	fMRI	Parietal cortex, dorsal anterior cingulate, PFC, insula, basal ganglia.	Incongruent: • MCI > HA (dorsal anterior cingulate, bilateral middle and inferior frontal gyri, bilateral inferior parietal lobule, bilateral insular); • AD < MCI, HA (dorsal anterior cingulate, bilateral middle and inferior frontal gyri, bilateral inferior parietal lobule, bilateral insular).	The activation of the PFC during the Stroop task was different in healthy older adults, AD and MCI patients. Dysfunction of the PFC was observed in AD patients, whereas an efficient compensatory mechanism was observed in MCI subjects. These results indicate differences in the neurophysiological mechanisms of the PFC in the different stages of cognitive impairment.
Li et al. (2012)	fMRI	Dorsal anterior cingulate, bilateral middle and inferior frontal gyri, bilateral inferior parietal lobule, insular and basal ganglia.	Incongruent: • HA, SIVCIND: dorsal anterior cingulate, bilateral middle and inferior frontal gyri, bilateral inferior parietal lobule, insular and basal ganglia. • SIVD: bilateral middle, inferior frontal gyri, insular and inferior parietal lobule. • SIVCIND > HA (dorsal anterior cingulate, bilateral middle and inferior frontal gyri, bilateral inferior parietal lobule, insular ganglia). • SIVD < HA (bilateral middle, inferior frontal gyri, insular and inferior parietal lobule).	Different activation characteristics of the prefrontal cortex were observed in normal aging and different conditions of participants with SIVCI during the performance of the Stroop task. There is dysfunction in SIVD and compensation in SIVCIND.
Manard et al. (2017)	fMRI	Posterior superior temporal, inferior frontal triangularis, inferior frontal operculum inferior temporal, anterior striatum, lateral orbitofrontal.	Interference: • YA: increased activity in left superior temporal gyrus • HA: increased activity in left inferior frontal and temporal areas Reactive control: • HA: increased activity in left inferior frontal areas Proactive control: • HA: increased activity in right middle frontal gyrus and decreased activity in the ACC and right lateral orbitofrontal gyrus.	In the reactive control condition, the increased brain activity was interpreted as a successful compensatory process in an area associated with inhibition. In proactive control situations, changes were observed between areas involved in recognising conflict and maintaining task goals or contextual information to cope with the high level of interference.
Mathis et al. (2009)	fMRI	Right and left DLPFC, right and left VLPFC, bilateral parietal lobule, premotor cortex.	• HA > YA (left VLPFC, left parietal cortex, left DLPFC) • MA > YA (left VLPFC, left parietal cortex) • HA > MA (left DLPFC).	The early effect of age-related impairment of the inhibitory process would affect the left side of the DLPFC, the VLPFC and the parietal lobe before the cortical network is affected bilaterally around the age of 60.
Milham et al. (2002)	fMRI	Anterior cingulate cortex/pre supplementary motor area, middle frontal gyrus, inferior frontal gyrus, superior and inferior parietal lobules, and extrastriate cortex.	• HA < YA (DLPFC, parietal cortices) • HA > YA (temporal cortex, anterior inferior PFC) • HA > YA (ACC sensitivity to presence of conflict).	In older participants, a stronger activation in the temporal cortex was found, which indicates a deeper processing of the word. In addition, older adults were found to have greater activity in the ventral prefrontal cortex, indicating an increased ability of irrelevant representations to access working memory.
Müller-Oehring et al. (2022)	fMRI	DLPFC, superior parietal lobe, inferior parietal lobe, gyrus, middle cingulate cortex.	No significant difference was detected between groups for the Stroop task.	The Stroop effect activates a frontoparietal network that includes bilateral frontal (DLPFC, ACC and medial prefrontal cortex) and bilateral parietal (inferior and superior parietal lobes, angular gyrus) cortex as well as left temporal gyri and right cerebellar regions.
Puente et al. (2014)	fMRI	Orbitofrontal cortex, DLPFC, ACC, posterior parietal cortex.	• MCI > HA in DLPFC, orbitofrontal, and posterior parietal cortex (uncorrected threshold $p < .01$).	Although there are minimal functional brain differences exist, fMRI of inhibition does not currently provide clinicians with a reliable tool for identifying MCI as opposed to traditional neuropsychological techniques.
Schulte et al. (2011)	fMRI	Right and left precuneus, right medial superior frontal gyrus, right and left anterior and middle cingulate cortex, right and left insula, left calcarine sulcus, left superior parietal lobe, left paracentral gyrus, right middle frontal gyrus (BAs 23, 9, 24, 48, 17/18, 5, 4, 46).	Stroop-mix contrast activation: • HA > YA (frontoparietal regions - right BA 9, bilateral BA 23, 24, right BA 48). Stroop-same contrast activation: • HA: occipito-parietal visuomotor (left BA 17/18, left BA 5, left BA 4) • YA > HA (frontal areas – bilateral BA 48, right BA 46)	Different brain networks were active in the older group compared to younger people, suggesting that older adults can adapt efficiently to task demands by using different strategies in conflict resolution.

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Study	Technique	Electrodes and/or investigated ROIs	Main findings	Interpretation of the results
Tam et al. (2015)	fMRI	Frontoparietal attentional cortices (anterior cingulate, inferior frontal gyrus, and superior frontal gyrus).	Incongruent: • HA > YA (posterior cingulate, precentral gyrus, inferior frontal gyrus, temporal lobe); • YA > HA (isthmus of the cingulate retrosplenial cortex). Congruent: • HA > YA (insula and temporal pole) • YA > HA (extrastriate visual cortex).	Older adults have higher reaction time modulated activity in more frontal regions than younger adults, possibly due to age-related compensatory or dedifferentiation mechanisms. These results suggest that although the functional correlates of intraindividual reaction time variability change with age, the basic task-related mechanisms within the frontoparietal attention and default mode network regions remain stable during healthy ageing.
Vidoni et al. (2013)	fMRI	Left and right middle frontal gyri, superior parietal lobule, anterior cingulate gyri and sulci, precentral gyri.	No differences were detected between groups, although there were trends for greater activity for the HA group in the left precentral gyrus and right precuneus.	In people without dementia, higher cardiorespiratory fitness is associated with increased mid-frontal brain activity and decreased anterior cingulate brain activity in tasks requiring executive functions such as attention and conflict resolution.
You et al. (2021)	fMRI	DLPFC, frontal gyrus (inferior, superior, anterior, posterior, middle, precentral).		A significant increase in DLPFC activation was observed only at week 12 of CC supplementation, which may have the ability to improve working memory in older adults with MCI.
Simon studies				
Cespón et al. (2013)	EEG/ERP	Anterior, frontal, central, temporal, parietal, occipital (AFz, AF7, AF8, Fz, F3, F4, F5, F6, F7, F8, FCz, FC1, FC2, FC3, FC4, FT7, FT8, FT9, FT10, Cz, C1, C2, C3, C4, C5, C6, T7, T8, CPz, CP3, CP4, TP7, TP8, TP9, TP10, Pz, P3, P4, P7, P8, P9, P10, PO7, PO8, Oz, O1 and O2).	Amplitude: • mdaMCI < HA (N2pc).	The N2pc amplitude was smaller in participants with mdaMCI than in healthy participants, indicating a decrease in the correlates of the allocation of attentional resources to the target stimulus. In addition, N2pc amplitude proved to be a moderately good biomarker for the subtype of mdaMCI.
Cespón et al. (2015a)	EEG/ERP	Prefrontal, frontal, central, parietal, occipital, temporal.	Latency: • mdaMCI > HA (N2cc) • mdaMCI > sdaMCI (N2cc) Amplitude: • HA > mdaMCI (N2pc).	N2cc could be considered a useful correlate of mdaMCI. Indeed, increased interference was associated with delays in the implementation of cognitive control in mdaMCI participants, as indicated by longer N2cc latencies in this group. On the basis of N2cc latency, mdaMCI participants could be clearly distinguished from healthy elderly and sdaMCI participants.
Cespón et al. (2015b)	EEG/ERP	Prefrontal, frontal, central, parietal, occipital, temporal.	N2 Latency: • mdaMCI > HA • mdaMCI > mdnaMCI • mdaMCI > sdaMCI Amplitude: • mdaMCI < HA (N2pc).	MCI subtypes exhibited differences in ERP correlates of cognitive processes compared to healthy older participants and revealed several potential ERP biomarkers, such as delayed N2 latency and attenuated amplitudes of N2pc. The present results suggest that ERP is a suitable method to obtain early biomarkers for MCI, as it reveals incipient abnormalities that are not yet evident at the behavioural level.
Cespón et al. (2018)	EEG/ERP	Prefrontal, frontal, central, parietal, occipital, temporal.	MFN amplitude (stimulus position): • spatially incompatible > spatially compatible (larger only in HSMC) MFN amplitude (stimulus direction): • spatially incompatible > spatially compatible (larger only in HSMC).	MFN amplitude, related to ACC activity involved in conflict monitoring, was greater only in the HSMC group when the position of the stimulus and/or the direction in which the arrow pointed were incompatible with the required response. Therefore, the HSMC group used greater conflict monitoring activity to maintain performance, which was achieved for the spatial position conflict but not for the arrow direction conflict. These results suggest incipient deficits in monitoring conflict information in participants with high SMC, compared to low SMC.
Cespón et al. (2022)	EEG/ERP	Prefrontal, frontal, central, parietal, occipital, temporal.	N2cc latency: • HA > YA N2pc latency: • HA > YA	Older adults also exhibited longer reaction times and N2pc and N2cc latencies, suggesting that they took longer to focus their attention on the target stimulus and inhibit the tendency to respond to the attended location.
Katayama et al. (2023)	EEG/ERP	Fz, Cz, and Pz.	N2 amplitude: • MCI < HA (only no-response condition).	Clear differences were found between the HA and MCI groups in N2 amplitude (and in the dorsal attention network during resting state-EEG). Taken together, these results suggest that EEG measurements are suitable as neurophysiological markers for the detection of preclinical stages of Alzheimer's disease in older adults.
Singh et al. (2018)	EEG/ERP	Cz.	ERPs for response were similar between control and PD. PD patients had attenuated mid-frontal theta activity specific to response-conflict.	These results suggest that attenuated mid-frontal theta activity may be a novel biomarker for cognitive dysfunction in Parkinson's disease.

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Table 4 (*continued*)

Study	Technique	Electrodes and/or investigated ROIs	Main findings	Interpretation of the results
Singh et al. (2023)	EEG/ERP	Cz.	• Cz: No main effects of group • For time frequency ROIs: Main effect of group on greater delta power and theta power.	This study suggests that cognition is impaired in Parkinson's patients because they cannot modulate cue-triggered midfrontal delta/theta rhythms to perform cognitive tasks, leading to cognitive dysfunction and ultimately contributing to MCI and dementia. Furthermore, these results suggest that midfrontal delta/theta activity is a potential biomarker for cognitive deficits in Parkinson's disease.
Simon and Stroop studies				
Cespón et al. (2023)	EEG/ERP	Left frontal (F3, F5, FC3, FC5), right frontal (F4, F6, FC4, FC6), left parietal (P3, P5, CP3, CP5), and right parietal (P4, P6, CP4, CP6).	Latency: • Stroop: P300 and N2cc earlier in HCR than LCR group Amplitude: • Spatial Stroop: N2cc larger in HCR than LCR • Parietal P300 larger in left compared to right hemisphere in HCR but not in LCR.	Inhibitory abilities were better in older adults with high CR than with low CR, as shown by group-related differences. In addition, earlier N2cc and P300 latencies and increased N2cc amplitudes were found in the more demanding task in HCR than in LCR, suggesting an important role of task difficulty. Only the LCR group showed brain activity patterns typically associated with an aged brain, namely a shift in activity from posterior to anterior and a loss of interhemispheric asymmetry. Thus, high levels of CR are associated with the maintenance of neuronal activity patterns observed in young adults rather than the use of neuronal compensatory mechanisms.
Onur et al. (2011)	fMRI	Pre-supplementary motor area, ACC, intraparietal cortex, precuneus, precentral gyrus, insula, inferior frontal and parietal gyrus.	Interference condition: • YA > HA (left DLPFC) • HA > YA (left Inferior frontal gyrus).	It was observed that differential activation in the left DLPFC in young subjects and left inferior frontal gyrus activity in older subjects is associated with interference control.

Note. ROIs: Region of Interests. EEG: Electroencephalography. CH-PAT: abnormal Aβ/tau ratios. CH-NAT: normal Aβ/tau ratios. ERP: Event-Related Potential. Val/Met: Val/Met genotype carriers. Met/Met: Met/Met. genotype carriers. Val/Val: Val/Val genotype carriers. BDNF: Brain-Derived Neurotrophic Factor. MI: Mostly Incongruent. HA: Healthy Ageing. PD: Parkinson Disease. YA: Young Adults. MA: Middle-aged Adults. ACC: Anterior Cingulate Cortex. RT: Reaction Time. aMCI: amnestic MCI. Ms: milliseconds. fNIRS: functional Near Infrared Spectroscopy. CHs: Channels. 1pp: first-person perspective. 3pp: third-person perspective. DLPFC: Dorsolateral Prefrontal Cortex. PFC: Pre-Frontal Cortex. TIA: Transient Ischemic Attack. BA: Broadmann Area. Deoxy-Hb: Deoxygenated Hemoglobin. Oxy-Hb: Oxygenated Hemoglobin. SP: Stroke patients. VLPFC: Ventrolateral Prefrontal Cortex. PE: Physical Exercise. RC: Reading Control. CE: Cognitive Exercise. Hb0: Oxy-hemoglobin. HbR: Deoxy-hemoglobin. fMRI: functional Magnetic Resonance Imaging. BOLD: Blood Oxygenation Level Dependent. MCI: Mild Cognitive Impairment. AD: Alzheimer's Disease. SIVCIND: Subcortical Ischemic Vascular Cognitive Impairment No Dementia. SIVD: Subcortical Ischemic Vascular Dementia. SIVCI: Subcortical Ischemic Vascular Cognitive Impairment. CC Supplement: Cosmos caudatus Supplement. mdaMCI: multiple domains amnestic MCI. N2cc: central contralateral negativity. sdaMCI: single domain amnestic MCI. N2pc: posterior contralateral negativity. mdnaMCI: multiple domains non amnestic MCI. MFN: Medial Frontal Negativity. HSMC: High Subjective Memory Complaints. SMC: Subjective Memory Complaints. HCR: High Cognitive Reserve. LCR: Low Cognitive Reserve. CR: Cognitive Reserve.

4.2.5. *Psychiatric disorders (Depression)*

Pišljar et al. (2013) observed that individuals with late-onset depression in remission had prolonged RTs, greater Stroop interference and abnormal P300b compared to controls, suggesting residual executive control dysfunction in the absence of clinical symptoms.

4.3. *fNIRS results for the Stroop task*

4.3.1. *Age-related differences*

Huang et al. (2022) and Laguë-Beauvais et al. (2013) reported that older adults showed slower RTs and produced more errors in the Stroop task than younger adults. fNIRS data showed increased activation in prefrontal regions in older adults, suggesting that they require more distributed brain activity to cope with inhibitory control.

4.3.2. *Positive lifestyle interventions*

Ji et al. (2019) showed that physical and cognitive training reduced RTs in older adults and improved brain oxygenation during the Stroop task. A virtual body illusion and light physical training also improved performance on the Stroop task compared to baseline (Burin and Kawashima, 2021; Byun et al., 2024, respectively). Although Byun et al. (2024) found that neuronal efficiency did not change significantly, Burin and Kawashima (2021) showed that shorter RT in the Stroop task was associated with increased neuronal activation in the left DLPFC compared to baseline data.

4.3.3. *Cerebrovascular diseases*

Cerebrovascular diseases such as arterial stiffness and micro-angiopathy significantly impair executive control. Hamasaki et al. (2018a), (2018b) showed that increased cerebrovascular health and diseases was associated with decreased brain oxygenation in the PFC during the Stroop task. In addition, patients with cerebral micro-angiopathy (Schroeter et al., 2007) and acute ischemic stroke (Heiberg et al., 2023) showed impaired neurovascular coupling and executive function. Goenarjo et al. (2021) found that higher cardiorespiratory fitness was associated with better Stroop performance and better oxygenation in the right PFC.

Mason et al. (2022) investigated the relationship between carotid atherosclerosis and brain activation patterns. They found that cognitively normal individuals with carotid atherosclerosis showed lower brain activation during the Stroop task measured by fNIRS, although there were no significant differences in task performance, mean arterial pressure or heart rate. This suggests that vascular disease can alter brain function without directly affecting cognitive performance.

Faulkner et al. (2017) investigated the acute effects of different postures on executive function in patients with TIA whose RTs were longer than those of healthy older people. They also found that Stroop performance improved after training regardless of posture. However, these improvements were not associated with haemodynamic PFC perfusion.

Table 5
Summary of the main cerebral findings of the Stroop and Simon effects.

	EEG/ERP	fNIRS	fMRI
Stroop	• Areas involved in EEG activity related to conflict resolution include the right posterior parietal cortex, the fronto-basal area and the left ACC. Stroop interference is influenced by opposing alpha and theta activity found in healthy individuals over the age of 60. • The EEG/ERP studies show variations in the latency and amplitude of brain waves (i.e., N450, P3, P300b, N2) that either increase or decrease, reflecting changes in cognitive processing and executive control that are age-dependent on factors such as cognitive status, physical activity and genotype. These changes indicate either deterioration or improvement in cognitive function in different groups of participants.	• Physical exercise at a healthy age, both in moderation and in combination, improves cognitive function and neuronal performance. • A change in the oxygen supply to the PFC may be involved in age-related cognitive decline, particularly in the performance of executive tasks.	• There are some differences in PFC activation between young, old and patients with cognitive impairments such as MCI or AD. • The activation of the PFC during the Stroop task is influenced by age, with older people activating more frontal and temporal resources compared to younger people. These so-called compensatory processes compensate for deficits in executive control. • The Stroop task activates a frontoparietal network that is present in both young and older adults. This includes bilateral frontal regions (DLPFC, ACC, medial prefrontal cortex), parietal regions (inferior and superior parietal lobe, angular gyrus), temporal areas and the right cerebellum.
Simon	• Increased N2pc/N2cc latency and decreased amplitude in older adults and individuals with cognitive impairment (e.g., MCI), suggesting that they took longer to focus their attention on the target stimulus and inhibit the tendency to respond to the attended location. • Midfrontal theta attenuation in PD as a potential biomarker for cognitive deficits.		
Stroop/ Simon	• Inhibitory abilities were better in older adults with high CR than in those with low CR. Thus, high levels of CR are associated with the maintenance of neuronal activity patterns rather than the use of neuronal compensatory mechanisms.		• Interference control in the left DLPFC in young subjects and in the left inferior frontal gyrus in older adults.

Note. EEG: Electroencephalography. ERP: Event-Related Potential. fNIRS: functional Near Infrared Spectroscopy. fMRI: functional Magnetic Resonance Imaging. PFC: Prefrontal Cortex. MCI: Mild Cognitive Impairment. AD: Alzheimer's Disease. DLPFC: Dorsolateral Prefrontal Cortex. ACC: Anterior Cingulate Cortex. N2pc: posterior contralateral negativity. N2cc: central contralateral negativity. PD: Parkinson Disease. CR: cognitive reserve.

4.4. fMRI results in the Stroop task

4.4.1. Age-related differences

Langenecker et al. (2004) found that older adults showed lower accuracy compared to younger subjects, but showed greater activation of frontal brain regions, suggesting compensatory mechanisms for maintaining cognitive control function. Milham et al. (2002) similarly reported slower RTs and more errors in older adults, who also showed increased activation in the temporal cortex, reflecting deeper word processing, and the ventral PFC, suggesting greater susceptibility to irrelevant representations in working memory.

Schulte et al. (2011) discovered that older adults showed less Stroop interference than younger adults, which was associated with greater activation in fronto-parietal regions, suggesting compensatory recruitment of additional brain areas. This activation supports cognitive performance in the face of age-related declines in processing speed and accuracy. In addition, age-related changes in brain function are associated with slower reaction times and altered neuronal activity (Tam et al., 2015), reflecting the brain's adaptive responses to cognitive decline.

Mathis et al. (2009) studied YA, MA and HA and found that RTs increased with age, with HA showing the slowest responses. However, in the incongruent Stroop task, the younger adults made more errors than the MA and HA. Neuroimaging data showed that early age-related impairments in inhibitory processing affected the left DLPFC, VLPFC and parietal lobe before leading to bilateral cortical network impairment in individuals over 60.

Manard et al. (2017) found that HA had longer RTs on cognitive control tasks than YA. Although accuracy differences were not significant, fMRI data showed increased activation of the left inferior frontal areas in HA during reactive control tasks, indicating successful compensatory mechanisms. Under proactive control conditions, observed brain activity indicated changes in areas associated with conflict detection and task goal maintenance.

4.4.2. Positive lifestyle interventions

Positive lifestyle interventions such as exercise and, in some cases, nutritional supplements have been shown to improve brain function and delay cognitive decline in older adults. In particular, higher cardiorespiratory fitness has also been associated with better cognitive function and brain activation. Vidoni et al. (2013) reported that individuals with higher cardiorespiratory fitness had higher activity in the middle frontal brain and lower activity in the ACC, suggesting that physical fitness may protect against the cognitive decline associated with AD by improving executive control function. Although no significant differences in performance on the Stroop task were found between HA and patients at risk for AD, trends indicated higher brain activity in regions involved in attention and motor preparation, such as the left precentral gyrus and right precuneus, in the older, high-fit group.

On the other hand, Lau et al. (2020) conducted an RCT with Persicaria minor in individuals with MCI, but found no significant differences in DLPFC activation during the Stroop task between the supplement and placebo groups. No significant intervention effects were observed in other cognitive tasks either, suggesting that this supplement does not directly influence the activation elicited by the Stroop task. In contrast, You et al. (2021) observed that supplementation with Cosmos caudatus led to a significant increase in activation of the DLPFC in older adults with MCI after 12 weeks. While there was no significant change in accuracy, the faster RTs observed in the supplement group were associated with a lower accuracy rate, particularly in the incongruent Stroop condition. These results suggest that supplementation with Cosmos caudatus may increase brain activity in regions critical for working memory and conflict resolution, although there is a trade-off between speed and accuracy.

4.4.3. MCI, Alzheimer's, Parkinson's and cerebrovascular diseases

MCI patients often show increased cortical activation, which indicates compensatory mechanisms. Kaufmann et al. (2008) found that MCI patients activated additional cortical and cerebellar regions that likely compensated for impaired executive control. However, according

to Puente et al. (2014), fMRI differences between MCI and healthy individuals were small, suggesting that traditional neuropsychological instruments are still more reliable than fMRI for the diagnosis of MCI.

Li et al. (2009) compared healthy older adults, individuals with MCI, and AD patients. RTs and error rates increased progressively in all groups, with AD patients performing worst. Neuroimaging showed different patterns: healthy adults showed normal activation of the PFC, MCI patients showed compensatory activation and AD patients showed marked dysfunction in the PFC. These findings suggest that the brain's activation patterns change across varying stages of cognitive impairment.

Studies comparing patients with PD and HIV show similar deficits in frontostriatal and sensorimotor regions, suggesting common neurodegenerative processes. Müller-Oehring et al. (2022) found no significant differences in RTs or accuracy in the Stroop task between YA, HIV patients and patients with PD. However, neuroimaging showed that all groups activated a frontoparietal network that included bilateral frontal regions such as the DLPFC, the ACC and the medial prefrontal cortex as well as bilateral parietal and temporal regions.

In addition, Li et al. (2012) analysed participants with subcortical ischemic vascular dementia (SIVD) and subcortical ischemic vascular cognitive impairment without dementia (SIVCIND). They observed longer RTs and higher error rates in SIVD patients compared to SIVCIND and healthy adults. The fMRI results showed compensatory activation in the SIVCIND participants, while the SIVD patients showed dysfunction of the prefrontal cortex, suggesting that the severity of cognitive decline affects brain activation in tasks requiring executive functions.

Vascular changes also play a crucial role in the interpretation of fMRI results in ageing populations. Gauthier et al. (2013) reported that older adults showed slower RTs and higher error rates compared to YA under both control and inhibition conditions. Although changes in BOLD signalling were similar between groups, older adults exhibited a lower maximal BOLD response, suggesting that a given BOLD signal represents a greater change in neural activity in older than in younger individuals. This suggests that vascular changes may complicate the interpretation of fMRI data in ageing populations, as the underlying neuronal responses may differ markedly despite similar task performance.

4.5. Results for the Simon task and the combined Stroop and Simon task

4.5.1. Age-related differences

Cespón et al. (2022) found that both congruent and incongruent trials in the Simon task resulted in longer RTs in older adults compared to younger adults, although the differences in accuracy were not significant. Neuroimaging showed longer N2pc and N2cc latencies in healthy ageing subjects, suggesting delayed attentional focussing and response inhibition.

Onur et al. (2011) observed that older adults showed a stronger interference effect with different brain activation in Stroop/Simon like tasks. While YA showed stronger DLPFC activity, older adults activated the left inferior frontal gyrus. In all groups, incongruent trials were associated with increased activation of pre-SMA, ACC and IPC, indicating the role of these regions in resolving interference. Pharmacological treatment with levodopa enhanced interference effects in YA, indicating an age-independent effect of dopaminergic modulation on cognitive control.

Cespón et al. (2023) demonstrated that older adults with higher CR had better inhibitory control, as evidenced by faster N200 and P300 latencies, and that they maintained similar neuronal activity patterns as YA. In contrast, individuals with low CR showed neuronal shifts from posterior to anterior regions and less hemispheric asymmetry, indicating a greater reliance on compensatory mechanisms.

4.5.2. Subjective cognitive decline and cognitive impairment

Cespón et al. (2018) found that participants with high subjective memory complaints (i.e. SCD) showed increased medial frontal negativity in conflict tasks, especially when the spatial location of the stimulus conflicted with the required response. This suggests that these individuals invest more effort in conflict monitoring compared to individuals with lower memory complaints.

In addition, Cespón et al. (2013) showed that mdaMCI patients had smaller N2pc amplitudes than healthy adults, suggesting impaired attentional allocation. This ERP component could serve as a moderately good biomarker for the identification of mdaMCI. In another study, Cespón et al. (2015a) emphasised that mdaMCI patients showed significantly longer N2cc latencies, suggesting delayed cognitive control processes during interference resolution. N2cc latency may distinguish mdaMCI patients from healthy controls and other MCI subtypes.

Furthermore, Cespón et al. (2015b) observed that MCI subtypes showed differences in ERP correlates of cognitive processes compared to healthy older participants and discovered several potential ERP biomarkers, such as delayed N2 latency and attenuated amplitudes of N2pc.

Additionally, Katayama et al. (2023) reported that MCI patients showed longer RTs and more errors in incongruent conditions of the Simon task, with significantly lower N2 amplitudes, indicating deficits in attentional control.

4.5.3. Neurological diseases (Parkinson's Disease)

In PD, mid- frontal theta activity during the Simon task has been identified as an important marker of cognitive dysfunction. Singh et al. (2018), (2023) observed that a reduction in mid- frontal theta activity correlated with cognitive decline and disease progression, making it a potential biomarker of PD cognitive impairment and a target for therapeutic intervention.

More specifically, Singh et al. (2023) found that PD patients, particularly those with MCI (PDMCI) or dementia (PDD), had difficulty modulating theta/delta rhythms triggered by a cue. This impairment could contribute to their cognitive deficits, and theta activity in the middle frontal region could serve as a biomarker for cognitive decline in PD.

5. Discussion

This systematic review uniquely explores neuropsychological paradigms of CC and associated neural mechanisms using advanced cognitive neuroscience techniques. By examining the specific neural basis of the Stroop and Simon effects, taking into account the differences between the different techniques, this review aims to identify early neural markers of CC and understanding compensatory mechanisms, such as CR, that help older adults maintain performance despite declining RTs and accuracy.

The Stroop and Simon tasks simulate the cognitive interference and control demands that occur in daily life. These tasks reflect the need to adapt to changing demands and shifting priorities in everyday activities. As cognitive performance declines with age, older adults often rely on compensatory mechanisms to maintain executive control. This is reflected in slower RTs and lower accuracy compared to younger adults.

Central executive dysfunctions – such as deficits in response inhibition, cognitive flexibility and monitoring - are closely related to difficulties in performing instrumental activities of daily living (iADLs) such as managing finances, medication and household tasks. These challenges are particularly pronounced in individuals with MCI and neurodegenerative diseases (e.g., Amanzio et al., 2010, 2011, 2013, 2014, 2016, 2017a, 2017b, 2018, 2021; Palermo et al., 2017, 2018). Research has shown that deficits in inhibition, cognitive flexibility and monitoring, as measured by tasks such as the Stroop task and the TMT-B test, predict deterioration in basic and instrumental activities of daily living (Verreckt et al., 2022).

Identifying these deficits is crucial, as patients with mdaMCI who also have iADL deficits have a higher risk of developing dementia than patients with MCI without iADL deficits (Jekel et al., 2015). Therefore, tasks such as Stroop and Simon are not only useful for the assessment of

CC, but also crucial for identifying individuals at risk of further cognitive decline and functional impairment.

In addition to the age-related decline in EFs, other factors - such as lifestyle, biological influences, health status and diseases - also play an important role in executive control in older people. A multidimensional model, such as the one presented in our review, which considers neuropsychological behavioural data with EEG, ERP, fNIRS and fMRI results, is essential for understanding the dynamic interplay between executive control and compensatory processes, especially in cognitive ageing.

5.1. Influencing factors in executive control

5.1.1. Age-related changes

Age-related changes in cognitive control have been extensively investigated using EEG, ERP, fNIRS and fMRI studies, with a focus on tasks such as the Stroop and Simon tasks, each of which offer unique insights into how the brain compensates for cognitive decline.

In EEG studies of the Stroop task, older adults rely more on the posterior attentional system, while the anterior attentional system declines, as found by Nombela et al. (2014) and West and Bell (1997). Nombela et al. (2014) found increased theta and decreased alpha and beta rhythms in older adults, particularly in the right posterior regions, while West and Bell (1997) observed a decrease in theta activity in the anterior midline, indicating decreased efficiency of the anterior attentional system. ERP studies such as that of Zurrón et al. (2014) found delayed N2 and P3b latencies in older adults, suggesting slower processing speed, while a compensatory shift from posterior to anterior brain regions was observed. Similarly, fNIRS studies showed age-related decreases in connectivity between the DLPFC, with younger participants showing stronger connectivity, while older adults showed increased prefrontal activation, likely compensating for declining cognitive resources (Huang et al., 2022; Laguë-Beauvais et al., 2013). fMRI studies also suggest a compensatory recruitment of frontal regions in older adults, as found by Langenecker et al. (2004), Milham et al. (2002) and Schulte et al. (2011). Tam et al. (2015) found age-related differences in BOLD activity associated with RTs, suggesting adaptive changes in brain function with age. For the Simon task, EEG and ERP studies also show that cognitive control decreases with age. Cespón et al. (2022) showed that older adults exhibited longer RTs and lower cortical excitability in the Simon task, suggesting impaired cognitive control. In ERP studies focussing on MCI, Katayama et al. (2023) found reduced N2 amplitude and dorsal attention network activity in MCI participants, suggesting deficits in attentional control. Further work by Cespón et al. (2013), (2015a), (2015b) found reduced attentional and motor resources, particularly in mdaMCI, which showed more severe impairments compared to sdaMCI. Delayed N2cc latency in mdaMCI participants indicates slower executive control processes and is thus a potential biomarker for AD progression. Finally, mid-frontal theta rhythms were found to be critical for cognitive control in PD, with potential implications for therapeutic interventions.

5.1.2. Positive lifestyle interventions

Lifestyle interventions, including cognitive and physical training, are increasingly recognised for their role in maintaining or improving cognitive function in older adults. Studies using various methods such as ERP, fNIRS and behavioural assessments have provided strong evidence that physical activity, cognitive engagement and nutritional supplements can positively influence brain function, particularly in tasks requiring cognitive control such as the Stroop and Simon tasks. Research has shown that long-term physical activity can improve cognitive performance and neural efficiency. Gajewski and Falkenstein (2015) found that older adults who were physically active over a longer period of time performed better on the Stroop task, especially under high-interference conditions. These individuals exhibited shorter P2 latencies and more pronounced N2 and N450 amplitudes, indicating increased activity in the frontal cortex. These neural markers suggest that regular physical activity improves the brain's ability to deal with conflicting stimuli. This emphasises the role of physical fitness in building CR, which helps to mitigate age-related cognitive decline. In addition to physical activity, CR has been shown to play a crucial role in maintaining cognitive performance in old age. Gajewski et al. (2020) found that older adults with higher levels of education, higher IQ and a history of cognitive engagement showed similar neural patterns to younger individuals in the Stroop task. Specifically, these high-performing older adults showed greater CNV and P2/N2 amplitudes, reflecting more efficient neural processing. These results suggest that CR may help older adults maintain their cognitive control abilities despite age-related brain changes. The combination of cognitive and physical training appears to offer even greater benefits than either intervention alone. Ji et al. (2019) demonstrated that a combined cognitive-physical training programme improved inhibitory control and cerebral oxygenation in older adults more effectively than training alone. This was measured by improved performance in the Stroop task and increased cerebral oxygenation in the prefrontal cortex using fNIRS. This finding highlights the synergistic effects of engaging both the body and mind interventions, leading to improved brain function. Similarly, Burin and Kawashima (2021) conducted a RCT in which older adults were exposed to a virtual body illusion, resulting in improved performance on the Stroop task and increased activation in the DLPFC. This increase in neuronal activation suggests that such innovative cognitive interventions may stimulate prefrontal regions involved in executive control, further enhancing cognitive flexibility and inhibitory processes in older adults. In addition, Byun et al. (2024) found that mild physical training over three months decreased RTs in the Stroop task, although neuronal efficiency scores and oxy-Hb levels in the PFC did not change significantly, suggesting that even light training may have a positive impact on cognitive control. Consistent with the benefits of physical fitness, Vidoni et al. (2013) investigated the relationship between cardiorespiratory fitness and brain activation during the Stroop task. They found that older adults with higher fitness levels had better brain function and greater activation in key cognitive regions than people with AD. This suggests that maintaining physical fitness may protect against cognitive decline and even promote more efficient brain function in healthy ageing individuals. Dietary supplements have also been investigated for their potential to support cognitive health in old age. Lau et al. (2020) and You et al. (2021) investigated the effect of Persicaria minor and Cosmos caudatus and showed that these supplements improved brain activation in the DLPFC in older adults with MCI. This improvement in PFC activation suggests that dietary interventions may help slow cognitive decline in ageing populations, particularly those at higher risk of dementia. These studies show that certain dietary interventions can modulate brain function and thus represent a complementary approach to cognitive and physical training in older adults.

5.1.3. The role of biological, genetic, and disease-related factors

The role of biological, genetic and disease-related factors in cognitive ageing has been investigated in a number of tasks, in particular the Stroop and Simon tasks, using methods such as ERP, EEG and fNIRS. These studies provide important insights into how different conditions, from genetic variations to chronic diseases, affect cognitive control and executive function in older adults. Genetic factors play an important role in cognitive ageing, as demonstrated by research on the Stroop task. Gajewski et al. (2012) investigated the effects of the BDNF Val66Met polymorphism and found that older adults carrying the Met allele showed improved interference control and increased N450 components during the Stroop task compared to Val/Val carriers. This suggests that the Met allele may provide a protective advantage for inhibitory control in old age by modulating fronto-striatal networks. Furthermore, Sánchez-Moguel et al. (2018), (2022) examined EEG profiles in healthy older adults, focusing on those with excessive theta activity, which can signal early cognitive decline. Despite similar behavioural performance,

those with excessive theta activity showed compensatory mechanisms such as increased P300 amplitude and increased signal energy in the theta and alpha bands, indicating neurobiological adaptations. Looking at AD biomarkers, research has found that CR can maintain cognitive control even in the presence of AD markers. Arakaki et al. (2022) reported that cognitively healthy individuals with abnormal amyloid/tau ratios in the CSF exhibited compensatory hyperactivity, such as more negative occipital event-related alpha desynchronisation (ERD) and higher spectral alpha entropy (SE) during the Stroop task. These results suggest that these individuals may be in a transitional phase to cognitive decline in which hyperactive brain responses serve as early compensatory mechanisms. Chronic diseases, including PD, have a marked impact on cognitive control, particularly with regard to proactive control strategies. Kricheldorff et al. (2023) found that people with PD had difficulty proactively adjusting cognitive control during the Stroop task. This failure in conflict resolution was associated with decreased modulation of midlinear-frontal theta energy, highlighting the challenges that PD patients have in adapting their cognitive strategies.

Recent findings emphasize the importance of considering not only cortical areas, but also subcortical regions - such as the basal ganglia and cerebellum - to understand the neural circuits that support cognitive and executive control (Müller-Oehring et al., 2022). As highlighted in studies by Okayasu et al. (2023), the cerebellum, particularly in the dorsolateral hemispheres (crus I/II, lobules VI/VIIb), is heavily involved in a cortico-cerebellar loop with cortical regions, especially in tasks that require conflict resolution, such as the Stroop test. In these tasks, the PFC amplifies cerebellar activity, while the cerebellum inhibits prefrontal activity to filter out irrelevant stimuli and thereby support goal-directed behavior.

In their fMRI study, Müller-Oehring et al. (2022) showed that CC processes activate a lateral fronto-parietal network, while motor control processes involve a cerebellar, precentral and medial prefrontal network. The authors observed activation deficits in both cognitive and motor control regions in PD patients compared to controls, particularly in the dorsolateral frontal, medial frontal and anterior cingulate cortices for CC and in the limbic, frontal, parietal and cerebellar regions for motor control. These findings contribute to a more comprehensive understanding of the neural mechanisms underlying CC/EFs, particularly in neurodegenerative diseases such as Parkinson's disease, in which compensatory activation of the cerebellum attenuates executive control deficits.

In PD, changes are often observed in subcortical-cortical circuits, particularly in the basal ganglia and thalamus. The basal ganglia, including the subthalamic nucleus, play a crucial role in modulating theta rhythms, which are essential for CC. Research has shown that the basal ganglia regulate the flow of information between cortical regions, which is essential for supporting tasks that require inhibition and cognitive flexibility. The thalamus, which acts as a relay station, also facilitates communication between cortical and subcortical areas and plays a key role in the execution of complex cognitive functions.

Impairments in these regions can lead to significant difficulties in cognitive control, as is the case in Parkinson's disease. In these cases, dysfunction in subcortical networks often triggers compensatory activation in other regions, such as the cerebellum, to maintain cognitive performance. This dynamic interplay between cortical and subcortical regions highlights the complexity of cognitive control networks and suggests that a deeper understanding of these networks could lead to therapeutic strategies for diseases such as PD, in which dysfunction of the basal ganglia and thalamic circuits is compensated for by increased activation of the cerebellum to maintain cognitive performance.

Subjective and objective cognitive impairments, including MCI and dementia, also exacerbate age-related cognitive decline. Ramos-Goicoa et al. (2016) demonstrated that MCI participants showed slower neural processing and increased allocation of resources to irrelevant stimuli during the Stroop task, which further impaired motor response preparation. Similarly, Pišljar et al. (2013) found prolonged reaction times and greater Stroop interference accompanied by abnormal P300b waveforms in individuals with late-life depression in remission, suggesting persistent cognitive impairment despite remission of depressive symptoms. Vascular health has a significant impact on executive control as shown by studies using fNIRS during the Stroop task. Hamasaki et al. (2018a), (2018b) found that cerebrovascular health and diseases were associated with reduced cerebral oxygenation and poorer Stroop performance, suggesting that deterioration in executive control in older adults may be due to an impaired haemodynamic response, particularly in the left prefrontal cortex. The study by Hamasaki et al. (2018a) was the only one to find gender differences in behavioral results and brain activation. In particular, the RT of the Stroop effect was longer in the over 70-year-old females than in the younger group. In addition, the female group over 60 years showed a significantly smaller change in oxide Hb signal only in the left PFC than the younger group. In contrast, the different age groups showed no significant differences in the oxide Hb signal and in the RT of the Stroop effect in the male subjects.

Schroeter et al. (2007) confirmed that cerebral microangiopathy reduces brain activation in the frontal lobes and impairs task performance. Mason et al. (2022) investigated carotid arteriosclerosis and found reduced brain activation during the Stroop task in affected individuals, although task performance was not impaired. This illustrates how vascular disease can alter neuronal responses without an immediate cognitive decline being apparent. In addition, Heiberg et al. (2023) found that patients with AIS had impaired executive control and abnormal haemodynamic responses, highlighting the potential of fNIRS as a biomarker of cognitive impairment. Faulkner et al. (2017) and Goenarjo et al. (2021) extended this line of research and showed that higher cardiorespiratory fitness in older adults improves Stroop performance and increases prefrontal oxygenation. In the context of the Simon task, studies on cognitive impairment have identified important biomarkers for tracking executive cognitive decline. Cespón et al. (2018) found that older adults with subjective memory complaints, particularly those with high-grade memory complaints, exhibited increased cognitive effort to maintain performance during the Simon task, as evidenced by higher medial frontal negativity. This indicates increased conflict monitoring. Further work by Cespón et al. (2013), (2015a) on MCI found that participants with mdaMCI showed reduced amplitudes of N2pc and lateralised readiness potential, indicating deficits in attention and motor processing. Delayed N2cc latencies in mdaMCI participants indicate slower executive control and serve as potential early markers for AD. PD has also been investigated using the Simon task. Singh et al. (2018), (2023) found that reduced theta activity in the middle frontal area during response conflict and processing after an error was associated with cognitive dysfunction and disease progression. These results suggest that theta rhythms in the middle frontal area serve as critical biomarkers of cognitive dysfunction in PD and could be targets for therapeutic intervention.

5.2. *Compensatory mechanisms*

This review highlights the critical role of PFC activation in maintaining cognitive control and emphasises the importance of CR in mitigating age-related cognitive decline. Although there are differences between these two paradigms, the review consistently emphasises how both the Stroop and Simon tasks illustrate compensatory mechanisms and the variability in brain activation between healthy ageing and cognitive impairment. Pathological changes, such as inefficient neuronal recruitment (dedifferentiation), are more evident in people with MCI or in neurodegenerative and vascular disorders. The Stroop and Simon tasks offer important insights into these compensation mechanisms. The Stroop task, which focuses on cognitive inhibition and semantic conflict, is highly sensitive to executive dysfunction and cognitive decline. In contrast, the Simon task, which focuses on spatial and motor conflicts, is particularly useful in distinguishing between MCI subtypes and motor-related diseases such as PD. Both tasks show the

compensatory strategies that older adults use and reflect how the brain copes with healthy ageing and cognitive impairment.

5.3. Models of compensation

Compensatory neural processes, as described in models such as the Compensation-Related Utilisation of Neural Circuits Hypothesis (CRUNCH, Reuter-Lorenz and Cappell, 2008) and the Scaffolding Theory of Ageing and Cognition (STAC, Park and Reuter-Lorenz, 2009), are essential for the maintenance of cognitive performance in old age. These models explain how older adults adapt by shifting neuronal activation from posterior to anterior regions, relying on PFC activity to compensate for the decline in posterior areas (PASA model, Davis et al., 2008; Dennis and Cabeza, 2008). However, when these compensatory mechanisms fail, as in MCI, neuronal recruitment becomes inefficient, leading to dedifferentiation (Park et al., 2012).

5.4. Limitations and future directions

This review emphasizes the importance of considering the strengths of each psychophysiological and imaging technique when examining neural activation patterns related to cognitive and executive control, particularly in the Simon and Stroop tasks. To interpret the results, it is crucial to understand how these techniques complement each other, such as the superior spatial resolution of fMRI and the excellent temporal resolution of EEG. Importantly, the review provides a nuanced and novel discussion of how different neuroimaging methods contribute to our understanding of cortical activation in cognitive decline in the aging population, with a particular focus on potential protective factors that may mitigate this decline. In addition, the review emphasizes the importance of demonstrating that the Stroop and Simon effects serve as reliable markers for distinguishing between healthy aging and cognitive impairment.

However, the study also has limitations that provide clues for future research. One important limitation is the heterogeneity of the study designs, including differences in cognitive tasks, psychophysiological and neuroimaging techniques, and participant characteristics. These differences may limit the generalizability of the results. In addition, some studies relied on small sample sizes, which may affect the robustness and reproducibility of the results. Another important limitation is the fact that the literature reviewed was predominantly cross-sectional, which limits the ability to draw causal conclusions about the progression of cognitive decline and the long-term effects of the compensatory mechanisms identified by the CC tasks.

Furthermore, the limited number of studies that have examined brain activation during the Simon task underscores the need for further research to better understand the neural mechanisms underlying cognitive and executive control in aging populations. Expanding research in this area could help clarify whether the Simon task is a reliable marker of cognitive impairment.

Future research should focus on longitudinal studies and standardization of CR assessments to more accurately capture the multidimensional nature of compensatory mechanisms across the lifespan. In addition, further studies are needed to investigate how lifestyle interventions such as cognitive training and physical activity interact with compensatory processes to potentially delay cognitive decline. Expanding research in these areas will provide deeper insights into the role of CR and CC in healthy aging and their potential to prevent or attenuate cognitive impairment in older adults.

6. Conclusion

This systematic review provides crucial insights into the neural mechanisms underlying cognitive and executive control, in the Stroop and Simon tasks. By examining these tasks using a variety of psychophysiological and imaging techniques, we have addressed key questions regarding the role of CR, the effects of age-related cortical changes, the role of CC in the detection of SCD, and the compensatory mechanisms that support cognitive performance in the face of neuronal decline.

The studies reviewed emphasize the critical role of CR in maintaining cognitive performance, allowing for more efficient neural processing and enabling the brain to adapt to age-related changes. This adaptability, particularly in regions essential for EFs (e.g., the DLPFC), facilitates the resolution of cognitive conflicts (e.g., the Stroop and Simon effects) and helps individuals maintain efficiency despite cognitive aging. Increased activity in CR-related brain regions helps compensate for cognitive demands during these tasks, particularly in older adults.

However, age-related changes, particularly in the PFC, result in reduced oxygen supply and increased arterial stiffness, negatively impacting core EFs. Although compensatory mechanisms in regions such as the left inferior frontal gyrus and DLPFC help mitigate these effects, they are less efficient in older adults than in younger individuals. This inefficiency is evident in slower inhibitory control, which is further exacerbated in conditions like MCI and neurodegenerative diseases.

In individuals with SCD, higher memory complaints are correlated with increased engagement in conflict control, suggesting that the ability to resolve cognitive conflicts is critical for maintaining performance. This pattern indicates potential deficits in conflict resolution and monitoring that could serve as early indicators of more severe cognitive decline.

In MCI and neurodegenerative diseases (such as AD, and PD), CC typically deteriorates due to dysfunction in regions responsible for EFs. This leads to greater difficulty in tasks requiring attention, inhibition, and conflict resolution, such as the Stroop and Simon tasks. While compensatory brain activity may occur, it is less efficient than in healthy individuals. EEG studies show that individuals with neurocognitive disorders experience changes in midfrontal delta/theta rhythms during cognitive conflict. Functional neuroimaging studies using fMRI and fNIRS reveal increased activity in brain regions such as the DLPFC, which suggests compensatory mechanisms that help individuals with MCI or dementia cope with executive control deficits. However, even in these cases, compensatory activation in other brain regions (e.g., the cerebellum and temporal areas) may be insufficient to fully offset the cognitive impairments, especially in diseases like AD and PD.

In summary, the findings from EEG, fNIRS, and fMRI studies underscore the significant role of CR in supporting EFs and maintaining CC in aging and neurodegenerative conditions. They also highlight the importance of compensatory mechanisms in the PFC and other brain regions in mitigating cognitive decline. These findings suggest that early detection of CC deficits and the identification of compensatory mechanisms may serve as a foundation for developing interventions aimed at delaying or preventing cognitive impairment offering a window into early markers of cognitive dysfunction. Ultimately, this could lead to improved quality of life for older adults at risk of cognitive decline.

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Declaration of Competing Interest

None of the authors have competing interests to declare.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2025.106121.

Data availability

Data will be made available on request.

References

Abbott, A., 1998. The causal devolution. Sociol. Methods Res. 27 (2), 148–181.

Abdulrab, K., Heun, R., 2008. Subjective memory impairment. A review of its definitions indicates the need for a comprehensive set of standardised and validated criteria. Eur. Psychiatry.: J. Assoc. Eur. Psychiatr. 23 (5), 321–330. https://doi.org/10.1016/j.eurpsy.2008.02.004.

Alexander, G.E., DeLong, M.R., Strick, P.L., 1986. Parallel organization of functionally segregated circuits linking basal ganglia and cortex. Annu. Rev. Neurosci. 9, 357–381. https://doi.org/10.1146/annurev.ne.09.030186.002041.

Amanzio, M., Cipriani, G.E., Bartoli, M., Canessa, N., Borghesi, F., Chirico, A., Cipresso, P., 2023a. The neuropsychology of healthy aging: the positive context of the University of the Third Age during the COVID-19 pandemic. Sci. Rep. 13 (1), 6355. https://doi.org/10.1038/s41598-023-33513-4.

Amanzio, M., Cipriani, G.E., Canessa, N., Borghesi, F., Chirico, A., Cipresso, P., 2023b. A unique neuropsychophysiological approach to objectify emotion (dys)regulation in healthy older adults during the COVID-19 pandemic. Sci. Rep. 13 (1), 23093. https://doi.org/10.1038/s41598-023-50310-1.

Amanzio, M., D'Agata, F., Palermo, S., Rubino, E., Zucca, M., Galati, A., Pinessi, L., Castellano, G., Rainero, I., 2016. Neural correlates of reduced awareness in instrumental activities of daily living in frontotemporal dementia. Exp. Gerontol. 83, 158–164. https://doi.org/10.1016/j.exger.2016.08.008.

Amanzio, M., D'Agata, F., Palermo, S., Rubino, E., Zucca, M., Galati, A., Pinessi, L., Castellano, G., Rainero, I., 2017a. Corrigendum to "Neural correlates of reduced awareness in instrumental activities of daily living in frontotemporal dementia". Exp. Gerontol. 83 (2016), 158–164. https://doi.org/10.1016/j.exger.2017.05.022.

Amanzio, M., Monteverdi, S., Giordano, A., Soliveri, P., Filippi, P., Geminiani, G.C., 2010. Impaired awareness of movement disorders in Parkinson's disease. Brain Cogn. 72 (3), 337–346. https://doi.org/10.1016/j.bandc.2009.10.011.

Amanzio, M., Palermo, S., Stanziano, M., D'Agata, F., Galati, A., Gentile, S., Castellano, G., Bartoli, M., Cipriani, G.E., Rubino, E., Fonio, P., Rainero, I., 2021. Investigating neuroimaging correlates of early frailty in patients with behavioral variant frontotemporal dementia: a MRI and FDG-PET Study. Front. Aging Neurosci. 13, 637796. https://doi.org/10.3389/fnagi.2021.637796.

Amanzio, M., Palermo, S., Zibetti, M., Leotta, D., Rosato, R., Geminiani, G.C., Lopiano, L., 2014. Self-unawareness of levodopa induced dyskinesias in patients with Parkinson's disease. Brain Cogn. 90, 135–141. https://doi.org/10.1016/j.bandc.2014.06.014.

Amanzio, M., Palermo, S., Zucca, M., Rosato, R., Rubino, E., Leotta, D., Bartoli, M., Rainero, I., 2017b. Neuropsychological correlates of pre-frailty in neurocognitive disorders: a possible role for metacognitive dysfunction and mood changes. Front. Med. 4, 199. https://doi.org/10.3389/fmed.2017.00199.

Amanzio, M., Palermo, S., Zucca, M., Rosato, R., Rubino, E., Leotta, D., Bartoli, M., Rainero, I., 2018. Neuropsychological correlates of instrumental activities of daily living in neurocognitive disorders: a possible role for executive dysfunction and mood changes. Int. Psychogeriatr. 30 (12), 1871–1881. https://doi.org/10.1017/S1041610218000455.

Amanzio, M., Torta, D.M., Sacco, K., Cauda, F., D'Agata, F., Duca, S., Leotta, D., Palermo, S., Geminiani, G.C., 2011. Unawareness of deficits in Alzheimer's disease: role of the cingulate cortex. Brain A J. Neurol. 134 (Pt 4), 1061–1076. https://doi.org/10.1093/brain/awr020.

Amanzio, M., Vase, L., Leotta, D., Miceli, R., Palermo, S., Geminiani, G.C., 2013. Impaired awareness of deficits in Alzheimer's disease: the role of everyday executive dysfunction. J. Int. Neuropsychol. Soc. JINS 19 (1), 63–72. https://doi.org/10.1017/S1355617712000896.

Arakaki, X., Hung, S.M., Rochart, R., Fonteh, A.N., Harrington, M.G., 2022. Alpha desynchronization during Stroop test unmasks cognitively healthy individuals with abnormal CSF Amyloid/Tau. Neurobiol. Aging 112, 87–101. https://doi.org/10.1016/j.neurobiolaging.2021.11.009.

Bahlmann, J., Blumenfeld, R.S., D'Esposito, M., 2015. The Rostro-Caudal axis of frontal cortex is sensitive to the domain of stimulus information. Cereb. cortex 25 (7), 1815–1826. https://doi.org/10.1093/cercor/bht419.

Basak, C., Boot, W.R., Voss, M.W., Kramer, A.F., 2008. Can training in a real-time strategy video game attenuate cognitive decline in older adults? Psychol. Aging 23 (4), 765–777. https://doi.org/10.1037/a0013494.

Braver, T.S., Barch, D.M., 2002. A theory of cognitive control, aging cognition, and neuromodulation. Neurosci. Biobehav. Rev. 26 (7), 809–817. https://doi.org/10.1016/s0149-7634(02)00067-2.

Buckner, R.L., 2004. Memory and executive function in aging and AD: multiple factors that cause decline and reserve factors that compensate. Neuron 44 (1), 195–208. https://doi.org/10.1016/j.neuron.2004.09.006.

Burin, D., Kawashima, R., 2021. Repeated exposure to illusory sense of body ownership and agency over a moving virtual body improves executive functioning and increases prefrontal cortex activity in the elderly. Front. Hum. Neurosci. 15, 674326. https://doi.org/10.3389/fnhum.2021.674326.

Byun, K., Hyodo, K., Suwabe, K., Fukuie, T., Ha, M.S., Damrongthai, C., Kuwamizu, R., Koizumi, H., Yassa, M.A., Soya, H., 2024. Mild exercise improves executive function with increasing neural efficiency in the prefrontal cortex of older adults. GeroScience 46 (1), 309–325. https://doi.org/10.1007/s11357-023-00816-3.

Cabeza, R., Albert, M., Belleville, S., Craik, F.I.M., Duarte, A., Grady, C.L., Lindenberger, U., Nyberg, L., Park, D.C., Reuter-Lorenz, P.A., Rugg, M.D., Steffener, J., Rajah, M.N., 2018. Maintenance, reserve and compensation: the cognitive neuroscience of healthy ageing. Nat. Rev. Neurosci. 19 (11), 701–710. https://doi.org/10.1038/s41583-018-0068-2.

Camilleri, J.A., Müller, V.I., Fox, P., Laird, A.R., Hoffstaedter, F., Kalenscher, T., Eickhoff, S.B., 2018. Definition and characterization of an extended multiple-demand network. NeuroImage 165, 138–147. https://doi.org/10.1016/j.neuroimage.2017.10.020.

Catani, M., Dell'acqua, F., Vergani, F., Malik, F., Hodge, H., Roy, P., Valabregue, R., Thiebaut de Schotten, M., 2012. Short frontal lobe connections of the human brain. Cortex A J. devoted Study Nerv. Syst. Behav. 48 (2), 273–291. https://doi.org/10.1016/j.cortex.2011.12.001.

Cespón, J., Chupina, I., Carreiras, M., 2023. Cognitive reserve counteracts typical neural activity changes related to ageing. Neuropsychologia 188, 108625. https://doi.org/10.1016/j.neuropsychologia.2023.108625.

Cespón, J., Galdo-Álvarez, S., Díaz, F., 2013. Electrophysiological correlates of amnestic mild cognitive impairment in a simon task. PloS One 8 (12), e81506. https://doi.org/10.1371/journal.pone.0081506.

Cespón, J., Galdo-Álvarez, S., Díaz, F., 2015a. Inhibition deficit in the spatial tendency of the response in multiple-domain amnestic mild cognitive impairment. An event-related potential study. Front. Aging Neurosci. 7, 68. https://doi.org/10.3389/fnagi.2015.00068.

Cespón, J., Galdo-Álvarez, S., Díaz, F., 2018. Event-related potentials reveal altered executive control activity in healthy elderly with subjective memory complaints. Front. Hum. Neurosci. 12, 445. https://doi.org/10.3389/fnhum.2018.00445.

Cespón, J., Galdo-Álvarez, S., Pereiro, A.X., Díaz, F., 2015b. Differences between mild cognitive impairment subtypes as indicated by event-related potential correlates of cognitive and motor processes in a Simon task. J. Alzheimer's Dis. JAD 43 (2), 631–647. https://doi.org/10.3233/JAD-132774.

Cespón, J., Pellicciari, M.C., Casula, E.P., Miniussi, C., 2022. Age-related changes in cortical excitability linked to decreased attentional and inhibitory control. Neuroscience 495, 1–14. https://doi.org/10.1016/j.neuroscience.2022.05.021.

Cieslik, E.C., Zilles, K., Caspers, S., Roski, C., Kellermann, T.S., Jakobs, O., Langner, R., Laird, A.R., Fox, P.T., Eickhoff, S.B., 2013. Is there "one" DLPFC in cognitive action control? Evidence for heterogeneity from co-activation-based parcellation. Cereb. cortex 23 (11), 2677–2689. https://doi.org/10.1093/cercor/bhs256.

Cochrane Training.Handsearching. https://training.cochrane.org/resource/tsc-induction-mentoring-training-guide/5-handsearching (Accessed 19 July 2024).

Cohen, J., 1988. Statistical Power Analysis for the Behavioral Sciences, 2nd ed. Lawrence Erlbaum Associates, Publishers, Hillsdale, NJ.

Corbo, I., Marselli, G., Di Ciero, V., Casagrande, M., 2023. The protective role of cognitive reserve in mild cognitive impairment: a systematic review. J. Clin. Med. 12 (5), 1759. https://doi.org/10.3390/jcm12051759.

Davis., S.W., Dennis, N.A., Daselaar, S.M., Fleck, M.S., Cabeza, R., 2008. Que PASA? The posterior-anterior shift in aging. Cereb. Cortex 18 (5), 1201–1209. https://doi.org/10.1093/cercor/bhm155.

Dennis, N.A., Cabeza, R., 2008. Neuroimaging of Healthy Cognitive Aging. In: Salthouse, T.A., Craik, F.E.M. (Eds.), Handbook of Aging and Cognition, 3rd edition. Psychological Press, New York, pp. 1–56.

Diamond, A., 2013. Executive functions. Annu. Rev. Psychol. 64, 135–168. https://doi.org/10.1146/annurev-psych-113011-143750.

Egner, T., 2017. The Wiley Handbook of Cognitive Control. Wiley Blackwell. https://doi.org/10.1002/9781118920497.

Faulkner, J., Stoner, L., Grigg, R., Fryer, S., Stone, K., Lambrick, D., 2017. Acute effects of exercise posture on executive function in transient ischemic attack patients. Psychophysiology 54 (8), 1239–1248. https://doi.org/10.1111/psyp.12868.

Fjell, A.M., Walhovd, K.B., Fennema-Notestine, C., McEvoy, L.K., Hagler, D.J., Holland, D., Brewer, J.B., Dale, A.M., 2009. One-year brain atrophy evident in healthy aging. J. Neurosci.: Off. J. Soc. Neurosci. 29 (48), 15223–15231. https://doi.org/10.1523/JNEUROSCI.3252-09.2009.

Friedman, N.P., Miyake, A., 2017. Unity and diversity of executive functions: Individual differences as a window on cognitive structure. Cortex A J. devoted Study Nerv. Syst. Behav. 86, 186–204. https://doi.org/10.1016/j.cortex.2016.04.023.

Friedman, N.P., Robbins, T.W., 2022. The role of prefrontal cortex in cognitive control and executive function. Neuropsychopharmacol. Off. Publ. Am. Coll. Neuropsychopharmacol. 47 (1), 72–89. https://doi.org/10.1038/s41386-021-01132-0.

Gajewski, P.D., Falkenstein, M., Thönes, S., Wascher, E., 2020. Stroop task performance across the lifespan: high cognitive reserve in older age is associated with enhanced proactive and reactive interference control. NeuroImage 207, 116430. https://doi.org/10.1016/j.neuroimage.2019.116430.

Gajewski, P.D., Falkenstein, M., 2015. Long-term habitual physical activity is associated with lower distractibility in a Stroop interference task in aging: behavioral and ERP evidence. Brain Cogn. 98, 87–101. https://doi.org/10.1016/j.bandc.2015.06.004.

Gajewski, P.D., Hengstler, J.G., Golka, K., Falkenstein, M., Beste, C., 2012. The Met-genotype of the BDNF Val66Met polymorphism is associated with reduced Stroop interference in elderly. Neuropsychologia 50 (14), 3554–3563. https://doi.org/10.1016/j.neuropsychologia.2012.09.042.

Gauthier, C.J., Madjar, C., Desjardins-Crépeau, L., Bellec, P., Bherer, L., Hoge, R.D., 2013. Age dependence of hemodynamic response characteristics in human functional magnetic resonance imaging. Neurobiol. Aging 34 (5), 1469–1485. https://doi.org/10.1016/j.neurobiolaging.2012.11.002.

Glenny, A.-M., 2005. No "gold standard" critical appraisal tool for allied health research. Evid. Based Dent. 6 (4), 100–101.

Goenarjo, R., Dupuy, O., Fraser, S., Berryman, N., Perrochon, A., Bosquet, L., 2021. Cardiorespiratory fitness and prefrontal cortex oxygenation during Stroop task in older males. Physiol. Behav. 242, 113621. https://doi.org/10.1016/j.physbeh.2021.113621.

Hamasaki, A., Akazawa, N., Yoshikawa, T., Myoenzono, K., Tagawa, K., Sawano, Y., Nishimura, M., Maeda, S., 2018b. Central artery stiffness is related to cerebral oxygenation hemodynamics during executive function tasks in healthy middle-aged and older adults. Exp. Gerontol. 114, 93–98. https://doi.org/10.1016/j.exger.2018.10.022.

Hamasaki, A., Akazawa, N., Yoshikawa, T., Myoenzono, K., Tagawa, K., Maeda, S., 2018a. Age-related declines in executive function and cerebral oxygenation hemodynamics. Tohoku J. Exp. Med. 245 (4), 245–250. https://doi.org/10.1620/tjem.245.245.

Hasher, L., Lustig, C., Zacks, R., 2007. Inhibitory mechanisms and the control of attention. In: Conway, A.R.A., Jarrold, C., Kane, M.J. (Eds.), Variation in Working Memory. Oxford University Press, pp. 227–249.

Heiberg, A.V., Simonsen, S.A., Schytz, H.W., Iversen, H.K., 2023. Cortical hemodynamic response during cognitive Stroop test in acute stroke patients assessed by fNIRS. NeuroRehabilitation 52 (2), 199–217. https://doi.org/10.3233/NRE-220171.

Hong, Q.N., Fàbregues, S., Bartlett, G., Boardman, F., Cargo, M., Dagenais, P., Gagnon, M.-P., Griffiths, F., Nicolau, B., O'Cathain, A., Rousseau, M.-C., Vedel, I., Pluye, P., 2018. The Mixed Methods Appraisal Tool (MMAT) version 2018 for information professionals and researchers. Educ. Inf. 34 (4), 285–291. https://doi.org/10.3233/EFI-180221.

Hong, Q.N., Pluye, P., Fàbregues, S., Bartlett, G., Boardman, F., Cargo, M., Dagenais, P., Gagnon, M.P., Griffiths, F., Nicolau, B., O'Cathain, A., Rousseau, M.C., Vedel, I., 2019. Improving the content validity of the mixed methods appraisal tool: a modified e-Delphi study. J. Clin. Epidemiol. 111, 49–59.e1. https://doi.org/10.1016/j.jclinepi.2019.03.008.

Hoshi, E., 2006. Functional specialization within the dorsolateral prefrontal cortex: a review of anatomical and physiological studies of non-human primates. Neurosci. Res. 54 (2), 73–84. https://doi.org/10.1016/j.neures.2005.10.013.

Huang, W., Li, X., Xie, H., Qiao, T., Zheng, Y., Su, L., Tang, Z.M., Dou, Z., 2022. Different cortex activation and functional connectivity in executive function between young and elder people during stroop test: an fNIRS study. Front. Aging Neurosci. 14, 864662. https://doi.org/10.3389/fnagi.2022.864662.

Jarbo, K., Verstynen, T.D., 2015. Converging structural and functional connectivity of orbitofrontal, dorsolateral prefrontal, and posterior parietal cortex in the human striatum. J. Neurosci. Off. J. Soc. Neurosci. 35 (9), 3865–3878. https://doi.org/10.1523/JNEUROSCI.2636-14.2015.

Jekel, K., Damian, M., Wattmo, C., Hausner, L., Bullock, R., Connelly, P.J., Dubois, B., Eriksdotter, M., Ewers, M., Graessel, E., Kramberger, M.G., Law, E., Mecocci, P., Molinuevo, J.L., Nygård, L., Olde-Rikkert, M.G., Orgogozo, J.M., Pasquier, F., Peres, K., Salmon, E., Frölich, L., 2015. Mild cognitive impairment and deficits in instrumental activities of daily living: a systematic review. Alzheimer's Res. Ther. 7 (1), 17. https://doi.org/10.1186/s13195-015-0099-0.

Jessen, F., Amariglio, R.E., van Boxtel, M., Breteler, M., Ceccaldi, M., Chételat, G., Dubois, B., Dufouil, C., Ellis, K.A., van der Flier, W.M., Glodzik, L., van Harten, A.C., de Leon, M.J., McHugh, P., Mielke, M.M., Molinuevo, J.L., Mosconi, L., Osorio, R.S., Perrotin, A., Petersen, R.C., Subjective Cognitive Decline Initiative (SCD-I) Working Group, 2014. A conceptual framework for research on subjective cognitive decline in preclinical Alzheimer's disease. Alzheimer's Dement. J. Alzheimer's Assoc. 10 (6), 844–852. https://doi.org/10.1016/j.jalz.2014.01.001.

Ji, Z., Feng, T., Mei, L., Li, A., Zhang, C., 2019. Influence of acute combined physical and cognitive exercise on cognitive function: an NIRS study. PeerJ 7, e7418. https://doi.org/10.7717/peerj.7418.

Jung, J., Lambon Ralph, M.A., Jackson, R.L., 2022. Subregions of DLPFC display graded yet distinct structural and functional connectivity. J. Neurosci. Off. J. Soc. Neurosci. 42 (15), 3241–3252. https://doi.org/10.1523/JNEUROSCI.1216-21.2022.

Jurado, M.B., Rosselli, M., 2007. The elusive nature of executive functions: a review of our current understanding. Neuropsychol. Rev. 17 (3), 213–233. https://doi.org/10.1007/s11065-007-9040-z.

Katayama, O., Stern, Y., Habeck, C., Lee, S., Harada, K., Makino, K., Tomida, K., Morikawa, M., Yamaguchi, R., Nishijima, C., Misu, Y., Fujii, K., Kodama, T., Shimada, H., 2023. Neurophysiological markers in community-dwelling older adults with mild cognitive impairment: an EEG study. Alzheimer's Res. Ther. 15 (1), 217. https://doi.org/10.1186/s13195-023-01368-6.

Kaufmann, L., Ischebeck, A., Weiss, E., Koppelstaetter, F., Siedentopf, C., Vogel, S.E., Gotwald, T., Marksteiner, J., Wood, G., 2008. An fMRI study of the numerical Stroop task in individuals with and without minimal cognitive impairment. Cortex 44 (9), 1248–1255. https://doi.org/10.1016/j.cortex.2007.11.009.

Kohn, N., Eickhoff, S.B., Scheller, M., Laird, A.R., Fox, P.T., Habel, U., 2014. Neural network of cognitive emotion regulation–an ALE meta-analysis and MACM analysis. NeuroImage 87, 345–355. https://doi.org/10.1016/j.neuroimage.2013.11.001.

Kricheldorff, J., Ficke, J., Debener, S., Witt, K., 2023. Impaired proactive cognitive control in Parkinson's disease. Brain Commun. 5 (6), fcad327. https://doi.org/10.1093/braincomms/fcad327.

Laguë-Beauvais, M., Brunet, J., Gagnon, L., Lesage, F., Bherer, L., 2013. A fNIRS investigation of switching and inhibition during the modified Stroop task in younger and older adults. NeuroImage 64, 485–495. https://doi.org/10.1016/j.neuroimage.2012.09.042.

Lakens, D., 2013. Calculating and reporting effect sizes to facilitate cumulative science: a practical primer for t-tests and ANOVAs. Front. Psychol. 4, 863. https://doi.org/10.3389/fpsyg.2013.00863.

Langenecker, S.A., Nielson, K.A., Rao, S.M., 2004. fMRI of healthy older adults during Stroop interference. NeuroImage 21 (1), 192–200. https://doi.org/10.1016/j.neuroimage.2003.08.027.

Lau, H., Shahar, S., Mohamad, M., Rajab, N.F., Yahya, H.M., Din, N.C., Hamid, H.A., 2020. The effects of six months Persicaria minor extract supplement among older adults with mild cognitive impairment: a double-blinded, randomized, and placebo-controlled trial. BMC Complement. Med. Ther. 20 (1), 315. https://doi.org/10.1186/s12906-020-03092-2.

Lehto, J.E., Juujärvi, P., Kooistra, L., Pulkkinen, L., 2003. Dimensions of executive functioning: evidence from children. Br. J. Dev. Psychol. 21, 59–80.

Li, C., Zheng, J., Wang, J., 2012. An fMRI study of prefrontal cortical function in subcortical ischemic vascular cognitive impairment. Am. J. Alzheimer's Dis. Other Dement. 27 (7), 490–495. https://doi.org/10.1177/1533317512455841.

Li, C., Zheng, J., Wang, J., Gui, L., Li, C., 2009. An fMRI stroop task study of prefrontal cortical function in normal aging, mild cognitive impairment, and Alzheimer's disease. Curr. Alzheimer Res. 6 (6), 525–530. https://doi.org/10.2174/156720509790147142.

Liu, X., Banich, M.T., Jacobson, B.L., Tanabe, J.L., 2004. Common and distinct neural substrates of attentional control in an integrated Simon and spatial Stroop task as assessed by event-related fMRI. NeuroImage 22 (3), 1097–1106. https://doi.org/10.1016/j.neuroimage.2004.02.033.

Livingston, G., Huntley, J., Sommerlad, A., Ames, D., Ballard, C., Banerjee, S., Brayne, C., Burns, A., Cohen-Mansfield, J., Cooper, C., Costafreda, S.G., Dias, A., Fox, N., Gitlin, L.N., Howard, R., Kales, H.C., Kivimäki, M., Larson, E.B., Ogunniyi, A., Orgeta, V., Mukadam, N., 2020. Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. Lancet 396 (10248), 413–446. https://doi.org/10.1016/S0140-6736(20)30367-6.

MacLeod, C.M., 1991. Half a century of research on the Stroop effect: an integrative review. Psychol. Bull. 109 (2), 163–203. https://doi.org/10.1037/0033-2909.109.2.163.

Manard, M., François, S., Phillips, C., Salmon, E., Collette, F., 2017. The neural bases of proactive and reactive control processes in normal aging. Behav. brain Res. 320, 504–516. https://doi.org/10.1016/j.bbr.2016.10.026.

Mason, S.A., Al Saikhan, L., Jones, S., James, S.N., Murray-Smith, H., Rapala, A., Williams, S., Sudre, C., Wong, B., Richards, M., Fox, N.C., Hardy, R., Schott, J.M., Chaturvedi, N., Hughes, A.D., 2022. Association between carotid atherosclerosis and brain activation patterns during the Stroop task in older adults: an fNIRS investigation. NeuroImage 257, 119302. https://doi.org/10.1016/j.neuroimage.2022.119302.

Mathis, A., Schunck, T., Erb, G., Namer, I.J., Luthringer, R., 2009. The effect of aging on the inhibitory function in middle-aged subjects: a functional MRI study coupled with a color-matched Stroop task. Int. J. Geriatr. Psychiatry 24 (10), 1062–1071. https://doi.org/10.1002/gps.2222.

Menon, V., Uddin, L.Q., 2010. Saliency, switching, attention and control: a network model of insula function. Brain Struct. Funct. 214 (5-6), 655–667. https://doi.org/10.1007/s00429-010-0262-0.

Milham, M.P., Erickson, K.I., Banich, M.T., Kramer, A.F., Webb, A., Wszalek, T., Cohen, N.J., 2002. Attentional control in the aging brain: insights from an fMRI study of the stroop task. Brain Cogn. 49 (3), 277–296. https://doi.org/10.1006/brcg.2001.1501.

Miller, E.K., Cohen, J.D., 2001. An integrative theory of prefrontal cortex function. Annu. Rev. Neurosci. 24, 167–202. https://doi.org/10.1146/annurev.neuro.24.1.167.

Miyake, A., Friedman, N.P., Emerson, M.J., Witzki, A.H., Howerter, A., Wager, T.D., 2000. The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: a latent variable analysis. Cogn. Psychol. 41 (1), 49–100. https://doi.org/10.1006/cogp.1999.0734.

Moayedi, M., Salomons, T.V., Dunlop, K.A., Downar, J., Davis, K.D., 2015. Connectivity-based parcellation of the human frontal polar cortex. Brain Struct. Funct. 220 (5), 2603–2616. https://doi.org/10.1007/s00429-014-0809-6.

Müller-Oehring, E.M., Hong, J.Y., Poston, K.L., Brontë-Stewart, H.M., Sullivan, E.V., McGlynn, L., Schulte, T., 2022. Neurofunctional characteristics of executive control in older people with HIV infection: a comparison with Parkinson's disease. Brain Imaging Behav. 16 (4), 1776–1793. https://doi.org/10.1007/s11682-022-00645-6.

Nee, D.E., Wager, T.D., Jonides, J., 2007. Interference resolution: insights from a meta-analysis of neuroimaging tasks. Cogn. Affect. Behav. Neurosci. 7 (1), 1–17. https://doi.org/10.3758/cabn.7.1.1.

Niotis, K., Saperia, C., Saif, N., Carlton, C., Isaacson, R.S., 2024. Alzheimer's disease risk reduction in clinical practice: a priority in the emerging field of preventive neurology. Nat. Ment. Health 2, 25–40. https://doi.org/10.1038/s44220-023-00191-0.

Nombela, C., Nombela, M., Castell, P., García, T., López-Coronado, J., Herrero, M.T., 2014. Alpha-theta effects associated with ageing during the Stroop test. PloS One 9 (5), e95657. https://doi.org/10.1371/journal.pone.0095657.

Okayasu, M., Inukai, T., Tanaka, D., Tsumura, K., Shintaki, R., Takeda, M., Nakahara, K., Jimura, K., 2023. The Stroop effect involves an excitatory-inhibitory fronto-

cerebellar loop, 11 Nat. Commun. 14 (1), 27. https://doi.org/10.1038/s41467-022-35397-w.
Onur, Ö.A., Piefke, M., Lie, C.H., Thiel, C.M., Fink, G.R., 2011. Modulatory effects of levodopa on cognitive control in young but not in older subjects: a pharmacological fMRI study. J. Cogn. Neurosci. 23 (10), 2797–2810. https://doi.org/10.1162/jocn.2011.21603.
Opdebeeck, C., Martyr, A., Clare, L., 2016. Cognitive reserve and cognitive function in healthy older people: a meta-analysis. Neuropsychol. Dev. Cogn. Sect. B Aging Neuropsychol. Cogn. 23 (1), 40–60. https://doi.org/10.1080/13825585.2015.1041450.
Page, M.J., McKenzie, J.E., Bossuyt, P.M., Boutron, I., Hoffmann, T.C., Mulrow, C.D., Shamseer, L., Tetzlaff, J.M., Akl, E.A., Brennan, S.E., Chou, R., Glanville, J., Grimshaw, J.M., Hróbjartsson, A., Lalu, M.M., Li, T., Loder, E.W., Mayo-Wilson, E., McDonald, S., McGuinness, L.A., Moher, D., 2021. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ (Clin. Res. Ed.). 372, n71. https://doi.org/10.1136/bmj.n71.
Palermo, S., Lopiano, L., Morese, R., Zibetti, M., Romagnolo, A., Stanziano, M., Rizzone, M.G., Geminiani, G.C., Valentini, M.C., Amanzio, M., 2018. Role of the cingulate cortex in dyskinesias-reduced-self-awareness: an fMRI study on Parkinson's Disease patients. Front. Psychol. 9, 1765. https://doi.org/10.3389/fpsyg.2018.01765.
Palermo, S., Lopiano, L., Zibetti, M., Rosato, R., Leotta, D., Amanzio, M., 2017. A novel framework for understanding reduced awareness of dyskinesias in Parkinson's Disease. Park. Relat. Disord. 39, 58–63. https://doi.org/10.1016/j.parkreldis.2017.03.009.
Pappalettera, C., Carrarini, C., Cappa, S., Caraglia, N., Cotelli, M., Marra, C., Perani, D., Redolfi, A., Spadin, P., Tagliavini, F., Vanacore, N., Vecchio, F., Rossini, P.M., 2024. Challenges to identifying risk versus protective factors in Alzheimer's disease, 10.1038/s41591-024-03158-5. Advance online publication Nat. Med.. https://doi.org/10.1038/s41591-024-03158-5.
Park, J., Carp, J., Kennedy, K.M., Rodrigue, K.M., Bischof, G.N., Huang, C.M., Rieck, J.R., Polk, T.A., Park, D.C., 2012. Neural broadening or neural attenuation? Investigating age-related dedifferentiation in the face network in a large lifespan sample. J. Neurosci. Off. J. Soc. Neurosci. 32 (6), 2154–2158. https://doi.org/10.1523/JNEUROSCI.4494-11.2012.
Park, D.C., Reuter-Lorenz, P., 2009. The adaptive brain: aging and neurocognitive scaffolding. Annu. Rev. Psychol. 60, 173–196. https://doi.org/10.1146/annurev.psych.59.103006.093656.
Petrides, M., 2005b. The rostral–caudal axis of cognitive control within the lateral frontal cortex. From Monkey Brain to Human Brain. The MIT Press, Cambridge.
Petrides, M., 2005a. Lateral prefrontal cortex: architectonic and functional organization. Philos. Trans. R. Soc. Lond. Ser. B Biol. Sci. 360 (1456), 781–795. https://doi.org/10.1098/rstb.2005.1631.
Pišljar, M., Repovš, G., Pirtošek, Z., 2013. Cognition in late onset depression. Psychiatry Res. 210 (1), 89–94. https://doi.org/10.1016/j.psychres.2013.01.032.
Porta, M.S., Greenland, S., Hernán, M., dos Santos Silva, I., Last, J.M., 2014. A dictionary of epidemiology. Oxford University Press, New York.
Puente, A.N., Faraco, C., Terry, D.P., Brown, C., Miller, L.S., 2014. Minimal functional brain differences between older adults with and without mild cognitive impairment during the stroop. Neuropsychol. Dev. Cogn. Sect. B Aging, Neuropsychol. Cogn. 21 (3), 346–369. https://doi.org/10.1080/13825585.2013.824065.
Ramos-Goicoa, M., Galdo-Álvarez, S., Díaz, F., Zurrón, M., 2016. Effect of normal aging and of mild cognitive impairment on event-related potentials to a stroop color-word task. J. Alzheimer's Dis. 52 (4), 1487–1501. https://doi.org/10.3233/JAD-151031.
Raz, N., 2000. Aging of the brain and its impact on cognitive performance: integration of structural and functional findings. In: Craik, F.I.M., Salthouse, T.A. (Eds.), The Handbook of Aging and Cognition. Lawrence Erlbaum Associate, pp. 1–90.
Raz, N., Lindenberger, U., Rodrigue, K.M., Kennedy, K.M., Head, D., Williamson, A., Dahle, C., Gerstorf, D., Acker, J.D., 2005. Regional brain changes in aging healthy adults: general trends, individual differences and modifiers. Cereb. Cortex 15 (11), 1676–1689. https://doi.org/10.1093/cercor/bhi044.
Reuter-Lorentz, P.A., Cappell, K.A., 2008. Neurocognitive aging and the compensation hypothesis. Curr. Dir. Psychol. Sci. 17 (3), 177–182.
Rottschy, C., Langner, R., Dogan, I., Reetz, K., Laird, A.R., Schulz, J.B., Fox, P.T., Eickhoff, S.B., 2012. Modelling neural correlates of working memory: a coordinate-based meta-analysis. NeuroImage 60 (1), 830–846. https://doi.org/10.1016/j.neuroimage.2011.11.050.
Sánchez-Moguel, S.M., Alatorre-Cruz, G.C., Silva-Pereyra, J., González-Salinas, S., Sanchez-Lopez, J., Otero-Ojeda, G.A., Fernández, T., 2018. Two different populations within the healthy elderly: lack of conflict detection in those at risk of cognitive decline. Front. Hum. Neurosci. 11, 658. https://doi.org/10.3389/fnhum.2017.00658.
Sánchez-Moguel, S.M., Baravalle, R., González-Salinas, S., Rosso, O.A., Fernández, T., Montani, F., 2022. Abnormal EEG signal energy in the elderly: a wavelet analysis of event-related potentials during a stroop task. J. Neurosci. Methods 376, 109608. https://doi.org/10.1016/j.jneumeth.2022.109608.
Schroeter, M.L., Cutini, S., Wahl, M.M., Scheid, R., Yves von Cramon, D., 2007. Neurovascular coupling is impaired in cerebral microangiopathy–an event-related Stroop study. NeuroImage 34 (1), 26–34. https://doi.org/10.1016/j.neuroimage.2006.09.001.
Schulte, T., Müller-Oehring, E.M., Chanraud, S., Rosenbloom, M.J., Pfefferbaum, A., Sullivan, E.V., 2011. Age-related reorganization of functional networks for successful conflict resolution: a combined functional and structural MRI study. Neurobiol. Aging 32 (11), 2075–2090. https://doi.org/10.1016/j.neurobiolaging.2009.12.002.
Seeley, W.W., Menon, V., Schatzberg, A.F., Keller, J., Glover, G.H., Kenna, H., Reiss, A.L., Greicius, M.D., 2007. Dissociable intrinsic connectivity networks for salience processing and executive control. J. Neurosci. Off. J. Soc. Neurosci. 27 (9), 2349–2356. https://doi.org/10.1523/JNEUROSCI.5587-06.2007.
Simon, J.R., 1990. The effects of an irrelevant directional cue on human information processing, in: Proctor, R.W., Reeve, T.G. (Eds.), Stimulus-response compatibility: An integrated perspective. North-Holland, pp. 31–86.
Singh, A., Cole, R.C., Espinoza, A.I., Wessel, J.R., Cavanagh, J.F., Narayanan, N.S., 2023. Evoked mid-frontal activity predicts cognitive dysfunction in Parkinson's disease. J. Neurol., Neurosurg., Psychiatry 94 (11), 945–953. https://doi.org/10.1136/jnnp-2022-330154.
Singh, A., Richardson, S.P., Narayanan, N., Cavanagh, J.F., 2018. Mid-frontal theta activity is diminished during cognitive control in Parkinson's disease. Neuropsychologia 117, 113–122. https://doi.org/10.1016/j.neuropsychologia.2018.05.020.
Stern, Y., 2009. Cognitive reserve. Neuropsychologia 47 (10), 2015–2028. https://doi.org/10.1016/j.neuropsychologia.2009.03.004.
Stern, Y., Albert, M., Barnes, C.A., Cabeza, R., Pascual-Leone, A., Rapp, P.R., 2023. A framework for concepts of reserve and resilience in aging. Neurobiol. Aging 124, 100–103. https://doi.org/10.1016/j.neurobiolaging.2022.10.015.
Stroop, J.R., 1935. Studies of interference in serial verbal reactions. J. Exp. Psychol. 18 (6), 643–662. https://doi.org/10.1037/h0054651.
Stuss, D.T., Craik, F.I.M., 2019. The Handbook of Aging and Cognition, third ed. Psychology Press.
Tam, A., Luedke, A.C., Walsh, J.J., Fernandez-Ruiz, J., Garcia, A., 2015. Effects of reaction time variability and age on brain activity during Stroop task performance. Brain Imaging Behav. 9 (3), 609–618. https://doi.org/10.1007/s11682-014-9323-y.
Turrini, S., Wong, B., Eldaief, M., Press, D.Z., Sinclair, D.A., Koch, G., Avenanti, A., Santarnecchi, E., 2023. The multifactorial nature of healthy brain ageing: brain changes, functional decline and protective factors. Ageing Res. Rev. 88, 101939. https://doi.org/10.1016/j.arr.2023.101939.
Verreckt, E., Grimm, E., Agrigoroaei, S., de Saint Hubert, M., Philippot, P., Cremer, G., Schoevaerdts, D., 2022. Investigating the relationship between specific executive functions and functional decline among community-dwelling older adults: results from a prospective pilot study. BMC Geriatr. 22 (1), 976. https://doi.org/10.1186/s12877-022-03559-6.
Vidoni, E.D., Gayed, M.R., Honea, R.A., Savage, C.R., Hobbs, D., Burns, J.M., 2013. Alzheimer disease alters the relationship of cardiorespiratory fitness with brain activity during the stroop task. Phys. Ther. 93 (7), 993–1002. https://doi.org/10.2522/ptj.20120465.
Viswanathan, M., Ansari, M.T., Berkman, N.D., Chang, S., Hartling, L., McPheeters, M., Treadwell, J.R., 2012. Assessing the risk of bias of individual studies in systematic reviews of health care interventions. In: Methods Guide for Effectiveness and Comparative Effectiveness Reviews. AHRQ Methods for Effective Health Care. Agency for Healthcare Research and Quality.
West, R., Bell, M.A., 1997. Stroop color-word interference and electroencephalogram activation: evidence for age-related decline of the anterior attention system. Neuropsychology 11 (3), 421–427. https://doi.org/10.1037//0894-4105.11.3.421.
World Health Organization, 2020. Decade of Healthy Ageing: Plan of Action 2021-2030. ⟨https://cdn.who.int/media/docs/default-source/decade-of-healthy-ageing/decade-proposal-final-apr2020-en.pdf⟩.
You, Y.X., Shahar, S., Mohamad, M., Rajab, N.F., Haron, H., Che Din, N., Abdul Hamid, H., 2021. Neuroimaging Functional Magnetic Resonance Imaging Task-Based Dorsolateral Prefrontal Cortex Activation Following 12 Weeks of Cosmos caudatus Supplementation Among Older Adults With Mild Cognitive Impairment. J. Magn. Reson. Imaging. 54 (6), 1804–1818. https://doi.org/10.1002/jmri.27762.
Zheng, D., Oka, T., Bokura, H., Yamaguchi, S., 2008. The key locus of common response inhibition network for no-go and stop signals. J. Cogn. Neurosci. 20 (8), 1434–1442. https://doi.org/10.1162/jocn.2008.20100.
Zurrón, M., Lindín, M., Galdo-Alvarez, S., Díaz, F., 2014. Age-related effects on event-related brain potentials in a congruence/incongruence judgment color-word Stroop task. Front. Aging Neurosci. 6, 128. https://doi.org/10.3389/fnagi.2014.00128.