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Sabrina Lenzoni

The neural correlates of metacognitive awareness

Thesis presented to the Programa de Pós-graduação em Psicologia of PUC-Rio in partial fulfillment of the requirements for the degree of Doutor em Psicologia.

Advisor: Prof. Daniel Correa Mograbi

Rio de Janeiro,
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Dedication

For Sandra and Roberto

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Abstract

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Loss of insight in own's cognitive abilities can be a feature of a wide range of neurological disorders and can be relevant for clinical outcomes and rehabilitation effectiveness. Furthermore, recent research has shown that changes in metacognition can also characterize healthy aging and interfere with everyday life activity, increasing the incidence of a set of behaviours affecting health and decision making. Considering the subjective nature of self-awareness and the lack of consensus on assessment instruments to measure metacognitive abilities, it is important to elucidate the neuroarchitecture of metacognitive awareness and identify biomarkers of metacognitive functions. The current thesis explores this topic through four articles. According to the Cognitive Awareness Model (CAM), different type of self-awareness impairments depends on different profiles of neurocognitive dysfunctions, such as *mnemonic* and *executive anosognosia*. The former is discussed in Article 1, which focuses on mechanisms underlying impaired self-awareness in Alzheimer's disease. Specifically, the evidence suggests that Alzheimer's patients rely on outdated information about the self and are unable to consolidate new information as consequence of anterograde and retrograde amnesia. Moreover, neuroimaging findings show that fronto-cingulate and temporal degeneration are implicated in self-awareness impairments. Article 2 focused instead on neural mechanisms underlying *executive anosognosia*. A systematic review of event-related potential studies investigating self-monitoring in neurological disorders was conducted to understand the contribution of different brain structures to error monitoring. Specifically, the study focused on the error-related negativity (ERN) and the error positivity (Pe), which index error detection and error awareness, respectively. The findings suggest the presence of domain-general processing of error detection relying on cingulo-opercular areas and basal ganglia, but it was also hypothesized that lesions outside the fronto-basal monitoring network may lead to domain-specific deficits. To test the domain-

specificity hypothesis, an event-related potential study was conducted (Article 3). A group of young and older adults completed a perceptual and a memory flanker task and the findings demonstrated that it is possible to differentiate self-monitoring processes across cognitive domains. Moreover, Pe findings demonstrated a global decline of error awareness in aging. Interestingly, in older adults only, within-task increase in Pe was specific to the memory domain, suggesting the presence of learning effects for memory but not for perceptual decisions. It was hypothesized that error awareness impairments may be associated with sensory decline in aging. Thus, Article 4 investigated the association between Pe and stimulus-locked potentials in young and older adults during memory flanker task performance, in order to understand the contribution of sensory or memory processes to age-related changes in error awareness. The findings showed that efficient stimulus recollection was associated with higher error awareness in both young and older adults and that reduced error awareness in older adults was associated with impairments in perceptual processing of stimuli. Overall, this work contributes to our understanding of neurocognitive processes underlying metacognitive awareness and neural correlates of different types of anosognosia and supports the multidimensional conceptualization of metacognitive awareness delineated by the CAM. The study's results offer novel insights into neural markers of metacognitive processes that can serve clinical assessment and the development of cognitive training and rehabilitation.

Keywords

Self-awareness; metacognition; ERPs; ERN; Pe; neurological disorders; aging

Resumo

Lenzoni, Sabrina; Mograbi, Daniel Correa. Os correlatos neurais da consciência metacognitiva. Rio de Janeiro, 2022. 220p. Tese de Doutorado - Departamento de Psicologia, Pontifícia Universidade Católica do Rio de Janeiro.

A perda de insight nas próprias habilidades cognitivas pode ser uma característica de uma ampla gama de distúrbios neurológicos e pode ser relevante para os resultados clínicos e a eficácia da reabilitação. Além disso, pesquisas recentes têm mostrado que alterações na metacognição também podem caracterizar o envelhecimento saudável e interferir nas atividades da vida cotidiana, aumentando a incidência de um conjunto de comportamentos que afetam a saúde e a tomada de decisões. Considerando a natureza subjetiva da autoconsciência e a falta de consenso sobre instrumentos de avaliação para medir habilidades metacognitivas, é importante elucidar a neuroarquitetura da consciência metacognitiva e identificar biomarcadores de funções metacognitivas. A presente tese explora este tema através de quatro artigos. De acordo com o Modelo de Consciência Cognitiva (CAM), diferentes tipos de comprometimento da autoconsciência dependem de diferentes perfis de disfunções neurocognitivas, como anosognosia mnemônica e executiva. O primeiro é discutido no Artigo 1, que se concentra nos mecanismos subjacentes à autoconsciência prejudicada na doença de Alzheimer. Especificamente, as evidências sugerem que os pacientes com Alzheimer dependem de informações desatualizadas sobre si mesmos e são incapazes de consolidar novas informações como consequência da amnésia anterógrada e retrógrada. Além disso, achados de neuroimagem mostram que a degeneração fronto-cingulada e temporal estão implicadas em deficiências de autoconsciência. O Artigo 2, por sua vez, concentrou-se nos mecanismos neurais subjacentes à anosognosia executiva. Foi realizada uma revisão sistemática de estudos potenciais relacionados a eventos que investigam o automonitoramento em distúrbios neurológicos foi realizada para entender a contribuição de diferentes estruturas cerebrais para o monitoramento de erros. Especificamente, o estudo concentrou-se na negatividade relacionada ao erro (ERN) e na positividade do erro (Pe), que indexam a detecção de erros e a consciência do erro, respectivamente. Os

achados sugerem a presença de processamento de domínio geral de detecção de erros com base em áreas cingulo-operculares e gânglios da base, mas também foi levantada a hipótese de que lesões fora da rede de monitoramento fronto-basal podem levar a déficits específicos de domínio. Para testar a hipótese de especificidade de domínio, foi realizado um estudo de potencial relacionado a eventos (Artigo 3). Um grupo de adultos jovens e idosos completou uma tarefa de flanker perceptual e de memória, e os resultados demonstraram que é possível diferenciar processos de automonitoramento em domínios cognitivos. Além disso, os achados de Pe demonstraram um declínio global da consciência de erro no envelhecimento. Curiosamente, apenas em adultos mais velhos, o aumento de Pe dentro da tarefa foi específico para o domínio da memória, sugerindo a presença de efeitos de aprendizagem para a memória, mas não para as decisões perceptivas. Foi levantada a hipótese de que as deficiências na percepção do erro podem estar associadas ao declínio sensorial no envelhecimento. Assim, o Artigo 4 investigou a associação entre Pe e potenciais bloqueados por estímulo em adultos jovens e idosos durante o desempenho da tarefa flanker de memória, a fim de entender a contribuição dos processos sensoriais ou de memória para mudanças relacionadas à idade na consciência de erro. Os resultados mostraram que a lembrança eficiente de estímulos foi associada a uma maior consciência de erro em adultos jovens e mais velhos e que a redução da consciência de erro em adultos mais velhos foi associada a deficiências no processamento perceptivo de estímulos. No geral, este trabalho contribui para nossa compreensão dos processos neurocognitivos subjacentes à consciência metacognitiva e correlatos neurais de diferentes tipos de anosognosia e apóia a conceituação multidimensional da consciência metacognitiva delineada pelo CAM. Os resultados do estudo oferecem novos insights sobre marcadores neurais de processos metacognitivos que podem servir para avaliação clínica e desenvolvimento de treinamento e reabilitação cognitiva.

Palavras-chave

Autoconsciência; metacognição; ERPs; ERN; Pe; doenças neurológicas; envelhecimento

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List of abbreviations

ABI	Acquired brain injury
ABM	Autobiographical memory
ACC	Anterior cingulate cortex
AD	Alzheimer's disease
ALS	Amyotrophic lateral sclerosis
CA	Cerebellar ataxia
CAM	Cognitive awareness model
CD	Cerebellar degeneration
CRN	Correct-related negativity
DMN	Default mode network
Ea	Error awareness
Ec	Error correction
EEG	Electroencephalography
ERN	Error-related negativity
ERP	Event-related potential
fMRI	Functional magnetic resonance
HC	Healthy controls
HD	Huntington's disease
IMTC	Inferior medial temporal cortex
LPC	Late positive complex
MCI	Mild cognitive impairment
mPFC	Medial prefrontal cortex
MS	Multiple sclerosis
MTT	Multiple trace theory
MVPA	Multivariate pattern analysis
OCD	Obsessive compulsive disorder
OFC	Orbitofrontal cortex
OHC	Old healthy controls
PCC	Posterior cingulate cortex
PDB	Personal data base
PDB	Parkinson's disease

Pe	Error positivity
PEA	Post-error accuracy
PES	Post-error slowing
PET	Positron-emission tomography
PFC	Prefrontal cortex
PRO	Predicted response-outcome
PTA	Post-traumatic amnesia
rCBF	Regional cerebral blood flow
RT	Reaction time
SPECT	Single-photon emission computed tomography
TBI	Traumatic brain injury
TS	Tourette syndrome
VMPFC	Ventromedial prefrontal cortex
YHC	Young healthy controls

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I. Theoretical background

1. Metacognitive awareness: theory and neuroanatomy

Self-awareness can be defined as the capacity of becoming the object one's own attention (Morin, 2011). The first-person perspective ("I" or "me") defines the subjective perception and interpretation of the surrounding world, making us the protagonist of our life experiences and allowing us to reflect on ourselves. Being self-aware also means wondering about future or hypothetical scenarios, predicting "what I would do" and "how I could feel" but also trying to empathize and imagining how others could feel ("If I were you, I would feel/I would think"). The concept of self-knowledge has been referenced since the ancient Greece and has been among the fundamentals of philosophical and religious beliefs throughout history. However, the study of self-awareness has become part of the neuroscientific research in the last decades (Lou et al., 2017).

Self-awareness is a multidimensional construct that encompasses subjective experiences that give rise to the sense of self (Huntley et al., 2021; Mograbi et al., 2021). The concept of self-awareness comprises several dissociable but interconnected processes which can individually dominate specific experiences of awareness and vary in complexity, i.e., the degree of involvement of higher order processes (Mograbi et al., 2021; Morin, 2006). Some of the facets of self-awareness are the sense of bodily physiological states and the subjective experience of somatic feelings (interoception; Craig, 2002), the sense of body posture and movement in space (proprioception; Tuthill & Azim, 2018), and the sense of controlling voluntary actions and, through their execution, their outcomes and consequences on the external world (agency; Haggard, 2017). Moreover, autobiographical memory is a core component of self-awareness, through both episodic memories (life events and experiences) and personal semantics (general knowledge about the self). Self-based memories define the perception of ourselves, our abilities and shape our expectations (Mograbi et al., 2021).

The ability to monitor and regulate our emotion contribute to self-aware experiences by evaluating emotional feelings, their valence and strength, and their

association with specific events, by implementing cognitive and behavioural strategies to suppress them, and by planning future actions to avoid or to anticipate unwanted emotions (Gross, 2013). A similar but commonly described as dissociate process concerns cognitive abilities. Metacognition has been described as "thinking about thinking" and refers to the knowledge about our own cognitive abilities and to the capacity to monitor and regulate our cognitive functions (Dunlosky & Metcalfe, 2009; Flavell, 1979; S. M. Fleming et al., 2012). Poor metacognition commonly refers to inaccurate assessments (i.e., overestimating or underestimating) of one's learning, behavioural performance, or cognitive abilities.

The Cognitive Awareness Model (CAM) provides an excellent framework to explain mechanisms neurocognitive underlying metacognition. The CAM (Figure 1) has been developed to investigate self-awareness in Alzheimer's disease (AD), and later revised to account for other clinical conditions (Agnew & Morris, 1998; Hannesdottir & Morris, 2007; Morris & Hannesdottir, 2004; Morris & Mograbi, 2013). This model (Figure 1). A core component of the CAM is the *Cognitive Comparator Mechanism*, which enables the formation of accurate metacognitive judgments by comparing current abilities and information stored in the memory systems. Specifically, the *Personal Database System* stores semantic information about the self, including the information about personal abilities, that develops from socio-cultural experiences (i.e., our abilities in relation to others) and from memories about life events, that are stored in the *Autobiographical Conceptual Memory System*. In turn, autobiographical memories formation relies on episodic and working memory, thus highlighting the relevance of mnemonic processes for the emergence of self-awareness.

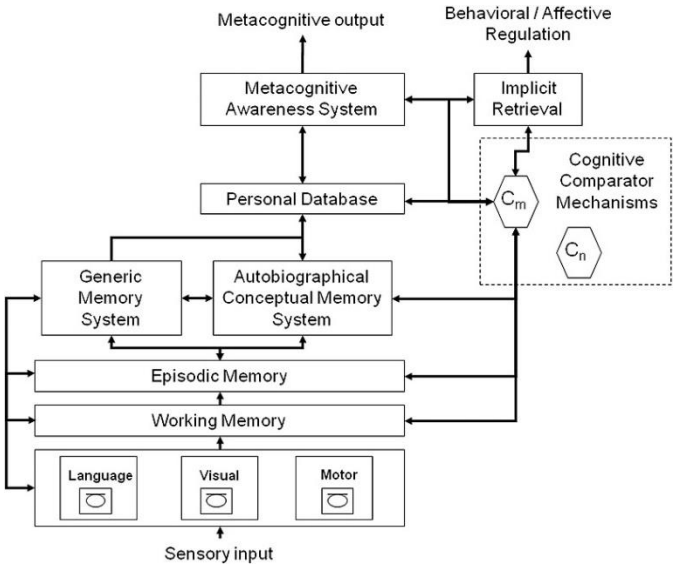

Figure 1. Schematic representation of the Cognitive Awareness Model (Morris & Mograbi, 2013).

The term *anosognosia*, from the Ancient Greek ἀ- (a-, "not, without") and νόσος (nósos, "disease") and γνῶσις (gnôsis, "knowledge"), is commonly used in the context of neurological disorders and refers to lack of awareness of disorder or specific symptoms/deficits (Mograbi & Morris, 2018). The term anosognosia has been often used to refer to metacognition deficits in the context of neurological conditions, such as in the case anosognosia for memory deficits in AD (Souchay, 2007). Anosognosia is a heterogenous condition, as suggested by evidence showing that underlying neural dysfunction and associated symptoms can vary (Agnew & Morris, 1998; Gainotti, 2018; Marcel et al., 2004). It has been found that anosognosia worsens as function of disease severity (Avondino & Antoine, 2016), that level of awareness can vary across symptoms (Antoine et al., 2013), and that anosognosia for memory deficits can be dissociated from awareness of other domains (Antoine et al., 2013)

Different types of anosognosia have been identified, as a result of different impairments in the system (Morris & Mograbi, 2013). Primary anosognosia refers to the compromise of the Metacognitive Awareness System, which can be described

as an emergent process that conveys signals from memory and monitoring systems. In this case, information about self-evaluation is not available to explicit awareness, even though errors can be detected through monitoring mechanisms and correctly integrated with mnemonic processes. However, implicit awareness is still intact (Mograbi & Morris, 2013), as indicated by behavioural signs, such as motor adjustments (Cocchini, Beschin, et al., 2010) or emotional reactions, such as facial expressions (Mograbi et al., 2012) following performance failures, which can be recorded even when individuals do not explicitly acknowledge error commission and do not benefit from feedback. The neural underpinnings of this condition have been linked to a breakdown in interregional connectivity rather than modular deficits, including top-down modulation and bottom-up integration processes (Morris & Mograbi, 2013).

Mnemonic anosognosia is caused by memory deficits, such as in the case of AD. AD is a progressive neurodegenerative disease and the most common type of dementia (Alzheimer's Association, 2018). AD pathogenesis includes accumulation of β-amyloid plaques and tau-containing neurofibrillary tangles in the hippocampus (Braak et al., 1993; Montine et al., 2012), leading to neuronal death and subsequent cortical atrophy (Archer et al., 2006; Mormino et al., 2009) and altered functional connectivity (Allen et al., 2007; Sheline & Raichle, 2013). Among the cognitive deficits associated with disease progression, memory decline is a key feature of AD (Jahn, 2013; Morris & Kopelman, 1986) and plays a major role in self-awareness impairments and loss of sense of self (Morris & Mograbi, 2013) Specifically, anterograde amnesia prevents the update of information about the self and gradual retrograde amnesia leads to deterioration of recent memory and preservation of remote and outdated material, resulting in a 'Petrified Self' (Mograbi et al., 2009). Interestingly, neuroimaging research has highlighted the role of frontal lobe dysfunction in lack of awareness in AD and three explanations have been suggested by Mograbi et al. (2009). A first possibility that frontal lobe dysfunction is implicated in impaired retrieval of recent memories. Another explanation is that "petrified self" phenomenon is necessary but not sufficient for the occurrence of anosognosia in AD and that is coupled to deficits of a belief evaluation system, subserved by prefrontal functions. A third hypothesis is that in

some cases anosognosia in AD may arise from global or local dysfunction of the comparator system.

Executive anosognosia in fact refers to the Cognitive Comparator Mechanisms dysfunction. Type I comparators are feed-forward mechanisms operating at local levels (e.g., sensory, motor) subserved by frontal cortico-subcortical loops. Therefore, frontal lobe dysfunction may account for a central dysfunction in monitoring processes, while domain-specific deficits can result in loss of awareness of certain functions, such in the cases of hemiplegia (Bottini et al., 2018), aphasia (Cocchini, Gregg, et al., 2010), and apraxia (Canzano et al., 2014; Scandola et al., 2021). Type 2 comparators are higher-order mechanisms that incorporate signals from type I comparators with information from multiple processes (e.g., mnemonic, attentional) and give rise to awareness of deficit (Mograbi & Morris, 2013). A growing body of evidence has confirmed the central role of the medial prefrontal cortex (mPFC), including the anterior cingulate cortex (ACC) in metacognitive processes (S. M. Fleming & Dolan, 2012; Metcalfe & Schwartz, 2016; Vaccaro & Fleming, 2018). In fact, this region has been shown to play a crucial role in performance monitoring, including error detection, response correction, and feedback evaluation during task performance (Holroyd & Yeung, 2012). Recent evidence suggesting the co-existence of domain-general and domain-specific neural patterns underlying metacognitive decisions in the medial prefrontal cortex supports the idea of local mechanisms of monitoring processes (Morales et al., 2018). Furthermore, studies focusing on behavioural performance showed dissociations of metacognitive abilities across cognitive domains (Bellon et al., 2020; Chapman et al., 2018; Dentakos et al., 2019; Rouault et al., 2018).

2. Clinical relevance of metacognitive awareness

Assessing awareness of cognitive impairment or decline has several implications for clinical assessment and rehabilitation purposes. In AD, the presence of anosognosia is associated with worse treatment outcomes (Cosentino et al., 2011; Koltai et al., 2001), higher incidence of risky behaviours (Starkstein, 2014), increased caregiver burden (Seltzer et al., 1997; Starkstein, 2014; Turró-Garriga et al., 2013), increased likelihood of institutionalization and care costs (Turró-Garriga

et al., 2016). Moreover, anosognosia has been shown to be a predictor of conversion from mild cognitive impairment to AD (Gerretsen et al., 2017; Spalletta et al., 2014; Tabert et al., 2002; Therriault et al., 2018). Importantly, self-awareness has been shown to play a major role in functional decline in healthy aging (Arora et al., 2021). It has been observed that healthy older adults are less aware of their mistakes (Harty et al., 2013; Sim et al., 2020), and that reduced metacognitive abilities may affect daily activities such as engagement in occupational therapy (Mihaljcic et al., 2017), financial decision making (Yu et al., 2022), and medication management (Cooper et al., 2005).

Self- awareness impairments have also been reported in acquired brain injury (ABI) by several studies (Bach & David, 2006; Dromer et al., 2021; Prigatano & Sherer, 2020; Robertson & Schmitter-Edgecombe, 2015; Sherer, Bergloff, Levin, et al., 1998; Sherer et al., 2003). Reduced awareness in ABI has been linked to limited understanding of the impact of post-injury deficits (J. Fleming & Strong, 1995), resistance to treatment (Katz et al., 2002), diminished ability to develop compensatory strategies (Ownsworth & Fleming, 2005). Conversely, higher metacognitive awareness following ABI has been associated with better functional outcomes (Yeo et al., 2021) and interventions aimed at improving self-awareness showed beneficial effects on independence in activity of daily living (Villalobos et al., 2019). Specifically, error monitoring has been identified as unique predictor of functional outcomes such as community reintegration (Robertson & Schmitter-Edgecombe, 2015). Recent findings have shown the benefits of error-based learning over errorless learning (Ownsworth et al., 2017), highlighting the relevance of self-monitoring and self-regulation and the use of metacognitive skills training for rehabilitation programs (J. Fleming et al., 2017).

Recent research has explored metacognitive deficits and emphasized their relationship with neurocognitive impairments and disease comorbidities in other clinical populations, such as multiple sclerosis (Mazancieux et al., 2019), Parkinson's disease (Maier & Prigatano, 2017), chronic fatigue syndrome (Jacobsen et al., 2016), epilepsy (Fisher & Noble, 2017), functional cognitive disorder (Bhome et al., 2019), and amyotrophic lateral sclerosis (La Foresta et al., 2015). It is therefore imperative to identify reliable indexes of metacognitive abilities and to include them in neuropsychological assessment to better

comprehend patient needs and develop appropriate interventions. Measures of awareness of cognitive deficits that have been previously used include discrepancy scores between patient's rating and caregiver's or clinician's ratings (Dourado et al., 2014; Migliorelli et al., 1995; Prigatano, 1996; Reed et al., 1993; Sherer, Bergloff, Boake, et al., 1998), structured interviews (J. M. Fleming et al., 1996; Reed et al., 1993), and semi-structured interviews (Ownsworth et al., 2000). Many of these instruments include sub-components to allow investigation of deficits in specific cognitive and functional domains (de Ruijter et al., 2020; Dromer et al., 2021).

Other methodologies have focused on online performance indexes, comparing performance scores and self-evaluation (Clare et al., 2002; Fischer et al., 2004), correlations between performance and confidence judgments (Nelson, 1984), error detection (Hester et al., 2005), prospective ratings (Dunlosky & Metcalfe, 2009), and signal detection theory measures (S. M. Fleming & Lau, 2014; Maniscalco & Lau, 2012). Hundreds of methods have been designed to capture different facets of cognitive awareness in healthy and clinical populations. However, considering the subjective nature of self-awareness, it is very difficult to find unbiased measures of metacognitive functions (Dunlosky & Metcalfe, 2009; S. M. Fleming & Lau, 2014; Vuorre & Metcalfe, 2022) and currently there is no consensus on tools to implement in clinical settings (de Ruijter et al., 2020; Dromer et al., 2021).

3. Electrophysiological signatures of metacognitive processes

Research on neurodegeneration and aging has highlighted the importance of identifying objective measures of biological processes that characterize medical states and clinical outcomes (Belleville & Bherer, 2012; di Tella et al., 2021; Horvath et al., 2018; Meghdadi et al., 2021; Nuzzo et al., 2014; Parnetti et al., 2019; Poil et al., 2013; Solís-Vivanco et al., 2015). Biomarkers (Strimbu & Tavel, 2010) of neurocognitive dysfunctions are commonly obtained using neuroimaging techniques, such as functional magnetic resonance (fMRI) and electroencephalography (EEG). EEG is a non-invasive technique that records in vivo electrical activity from the scalp. The EEG signal reflects the summation of postsynaptic potentials synchronously generated in the pyramidal cells (Kirschstein

& Köhling, 2009). These oscillations include different frequency bands, ranging from delta (1-3 Hz), theta (4-7 Hz), alpha (8–12 Hz), beta (13–30 Hz), and gamma (30–100 Hz). EEG is commonly used in neurological practice to detect epileptic seizures, to assess sleep functions, and for clinical evaluation of disorder of consciousness (Kennett, 2012).

One major drawback of EEG is its low spatial resolution which limits the estimation of deep sources of neural activity (inverse problem). However, throughout the years several methodologies have been developed to localize the source of EEG signals (Jatoi et al., 2014). Nonetheless, the temporal resolution of EEG is excellent and allows imaging of brain activity changes in the range of milliseconds. For this reason, cognitive neuroscience research has extensively used this technique to explore the course mental processes. Event-related potentials (ERPs) refer to EEG changes that are time-locked to an event, such as the presentation of a stimulus or a response during task performance (Luck, 2014). Throughout the years, EEG research has identified ERP reflecting sensory processes such as visual and evoked potentials, attention, and more complex cognitive processes, such as performance monitoring.

The error-related negativity (ERN; Gehring et al., 1993) or error negativity (Falkenstein et al., 1991) is a negative response-locked potential peak between 0 and 150ms at fronto-central electrodes. ERN amplitude is commonly larger following errors than correct responses, which, in the latter case, can also be referred to as correct-related negativity (CRN; Falkenstein et al., 2000). The ERN has been observed throughout the lifespan (Hämmerer et al., 2014), using several experimental paradigms (Riesel et al., 2013), in different sensory modalities (Falkenstein et al., 1991), at varying levels of task difficulty (Endrass et al., 2012). The neural source of the ERN has been localized in the ACC by several studies (Brázdil et al., 2005; Debener, 2005; Dehaene et al., 1994; Reinhart and Woodman, 2014; Van Veen and Carter, 2002). According to one of the proposed models (Reinforcement Learning; Holroyd & Coles, 2002), the ACC conveys error signals from the basal ganglia through phasic dopaminergic activity, as supported by evidence from pharmacological manipulation of dopamine in healthy individuals (Webber et al., 2021). The functional role of the ERN has been debated in the past. The most popular account is the Mismatch Theory, according to which the ERN

reflects a system of error detection which compares intended (correct) and actual response representation (Dehaene, 2018; Falkenstein et al., 1991; Gehring et al., 1993; Scheffers & Coles, 2000). The major consensus is that the ERN represents a preconscious system of performance monitoring (Weinberg et al., 2015).

The error positivity (Pe) is a positive potential that peaks between 200 and 400ms after the ERN at posterior sites (Falkenstein et al., 1991). Similar to the ERN, Pe amplitude is larger for errors as compared to correct responses. Source localization studies have identified various neural origins including ACC (Herrmann et al., 2004), posterior cingulate cortex/ precuneus (O'Connell et al., 2007) and anterior insula (Dhar et al., 2011). It has been shown that ERN and Pe reflect two independent systems (di Gregorio et al., 2018) but the functional significance of the Pe is less understood (Overbeek et al., 2005). A growing body of evidence suggests that Pe reflects conscious error processing (Boldt & Yeung, 2015; Endrass et al., 2007; Murphy et al., 2012; Nieuwenhuis et al., 2001; O'Connell et al., 2007), suggesting that Pe reflects a metacognitive decision variable underlying a process of evidence accumulation (Desender et al., 2021). In line with the *Evidence Accumulation* hypothesis, error awareness emerges from the integration of evidence about error commission, including sensory, proprioceptive, interoceptive, and cognitive systems, which recruit somatosensory areas and the cingulo-opercular network (Steinhauser & Yeung, 2010, 2012; Ullsperger et al., 2010; Wessel et al., 2011).

II. Objectives

In line with theoretical background, the present thesis will be composed in two parts. The first part will focus on metacognition and performance monitoring in neurological disorders and the second part on characterizing neurophysiological mechanisms of error detection and error awareness in healthy young and older adults.

The first parts consist of two literature reviews, aiming at:

- Providing a review of the past ten years of evidence on mechanisms and neural correlates of anosognosia in Alzheimer's disease
- Understanding what neurological disorders present performance monitoring impairments, as indicated by alteration of ERN and Pe, and their relation with clinical factors.

The second part consists of two research articles, with the following objectives:

- To investigate domain-specificity of performance monitoring ERPs
- To elucidate mechanisms underlying decline of error awareness in aging.

III. Article Selection

Article 1

Lenzoni, S., Morris, R. G., & Mograbi, D. C. (2020). The petrified self 10 years after: current evidence for mnemonic anosognosia. *Frontiers in Psychology*, *11*, 465. https://doi.org/10.3389/fpsyg.2020.00465

Abstract

Lack of awareness about disease, its symptoms and consequences, also termed anosognosia, is a common feature of Alzheimer's disease (AD). It has been hypothesized that memory disorder may be a key contributing factor to anosognosia, with people with AD not being able to update their personal information about performance and relying on older consolidated material about ability. This potentially outdated sense of self has been named, as a metaphor, the petrified self. In the current review, evidence from the past 10 years in relation to this concept is critically appraised. In particular, focus is given to empirical evidence produced on anterograde memory deficits about performance, the profile of autobiographical retrograde memory loss and the role of frontal lobes in anosognosia in AD. Finally, wider consequences of this metaphor for the understanding of selfhood in dementia are discussed.

Keywords

anosognosia; awareness; memory; Alzheimer's disease; dementia.

1. Introduction

Whilst anosognosia is more generally defined as lack of awareness about neurological impairment or illness, it also can be applied specifically to Alzheimer's Disease (AD), in which patients are frequently unaware of their cognitive deficits and the consequences of their clinical condition (Mograbi et al., 2009, 2012; Mograbi & Morris, 2018). This has been shown to be associated with earlier institutionalisation (Horning et al., 2014) worse prognosis (Orfei et al., 2007), reduced treatment compliance (Patel & Prince, 2001) and higher exposure to dangerous behaviors (Starkstein et al., 2007). In addition, loss of awareness has been linked to greater burden in relatives or caregivers (Seltzer et al., 1997; Verhülsdonk et al., 2013).

The manner in which the neurocognitive mechanisms supporting awareness are damaged in AD has been elucidated by experimental studies and theoretical formulations. Our formulation has been the Cognitive Awareness Model (CAM; Agnew & Morris, 1998; Morris & Hannesdottir, 2004; Morris & Mograbi, 2013), where lack of explicit awareness is thought to be the result of cognitive impairments at different levels, with anosognosia being characterised by its heterogeneity. This includes different types of anosognosia, including, (1) Primary anosognosia, where there is either a breakdown in connectivity, leading to impairments in bottom-up integration, or top-down modulation; (2) Executive anosognosia, which involves dysfunction of higher-level monitoring abilities that lead to impaired self appraisal and performance evaluation; and (3) Mnemonic anosognosia, in which lack of awareness is caused by specific types of memory impairment.

Under this framework, M. anosognosia is thought to be the main type in AD and is characterised by a failure in updating and integrating personal information to a personal data base (PDB), resulting in an outdated self-concept. Here, the PDB refers to a repository of information about the self, contrasting with more general semantic memories. In a paper in which we developed this notion with reference to the supporting experimental studies, this phenomenon was given a metaphorical term, namely, "the Petrified Self", (Mograbi et al., 2009). Two elements were highlighted in this 'stone' metaphor: (1) Limited updates in self-concept because of anterograde amnesia caused by degeneration of neuronal structures that support

declarative memory acquisition, such as the hippocampus; and (2) a preserved core of identity based on remote autobiographical memory (ABM), in particular of the semantic type, which has long been consolidated.

It is now ten years since the Petrified Self term was used and since then a considerable amount of new evidence has been produced about the relationship between memory, self and awareness in AD. Accordingly, in the current review, we consider this evidence, including data about ABM, as well as new insights into the relationship between anterograde amnesia and anosognosia in AD. In addition, as a comparison, the mounting neuroimaging evidence about the role of the frontal lobes in anosognosia in AD is appraised. We conclude by discussing reactions to the Petrified Self metaphor and potential implications for how we view people with dementia.

2. The remote self in AD

A key notion of the Petrified Self is that personal knowledge is shaped by or even represented in the form of remote memories. In the last decade, several studies have tried to elucidate the temporal pattern of ABM deficits across different lifetime periods in AD. Specifically, retrieval of remote material seems to be better preserved than recent memories. Overall, it has been highlighted that in AD ABM impairments are characterised by a temporal gradient for both episodic and semantic components (De Simone et al., 2016; Kirk & Berntsen, 2018; Leyhe et al., 2009; Thomann et al., 2012).

Only few studies reported no differences in memory retrieval across different lifetime epochs (Irish, Hornberger, et al., 2011), despite a poorer performance in comparison to healthy controls (Irish et al., 2014, 2018). It is possible that this is due to methodological reasons. For instance, it has been previously shown that the use of the Autobiophical Interview (AI; Levine et al., 2002) tends to diminish the temporal gradient due to fewer memories being allocated to a higher number of epochs (Barnabe et al., 2012).

Furthermore, recent evidence supports Multiple Trace Theory (MTT), according to which semantic memory retrieval is independent from the hippocampus and

mediated by the neocortex after a certain consolidation period (Moscovitch, Rosenbaum, et al., 2005), while episodic retrieval is subserved by medial temporal lobe/hippocampus regardless of the lifetime period of acquisition (Moscovitch, Westmacott, et al., 2005). For example, the presence of a temporal gradient is more consistent for episodic memory (Philippi et al., 2012), including vividness and details specificity (Donix et al., 2010; Irish, Lawlor, et al., 2011; Müller et al., 2016; Seidl et al., 2011), while personal semantics can be relatively preserved in AD (Martinelli et al., 2013). Moreover, it has been suggested that the degree of impairment of semantic ABM may depend on the stage of the disease. While episodic autobiographical memory can be affected in early AD, semantic components may be preserved, becoming impaired only in more severe stages of the condition (Kirk & Berntsen, 2018; Seidl et al., 2011). The temporal gradient is also evident from the early stages of dementia for episodic ABM, while semantic ABM seems to be characterised by a flatter distribution across lifetime periods (Seidl et al., 2011). Therefore, a decline in semantic ABM appears to be dissociated from damage to the primary episodic memory support structures, such as the hippocampus, being affected by later neocortex degeneration.

Although AD memory performance is worse than in mild cognitive impairment (MCI), the deterioration pattern has been shown to be similar for both episodic and semantic ABM in both conditions (Leyhe et al., 2009), with preserved semantic ABM and episodic impairments characterised by a temporal gradient (Seidl et al., 2011) and lower detail specificity (Donix et al., 2010; Seidl et al., 2011).

Although longitudinal studies of autobiographical memory in AD are scarce (e.g., Starkstein et al., 2005), cross-sectional comparisons between AD and MCI may illuminate the change in the content of memories over time with the progression of the condition. The general findings indicate that episodic memory is impaired across life epochs in AD in relation to MCI, but that remote semantic memories are preserved at similar levels in comparison to MCI (Hirjak et al., 2017; Leyhe et al., 2009) and even healthy controls (Thomann et al., 2012). This suggests that autobiographical memory deteriorates as a function of dementia severity, but that episodic impairments are seen from the earlier stages of the condition, whereas autobiographical semantic loss is observed only later on in the course of the illness.

Interestingly, it has been reported that remote memories are proportionally more frequently retrieved by people with AD (De Simone et al., 2016; Müller et al., 2016) and that detail specificity is positively associated to retrieval frequency of memories (Müller et al., 2016). Hence, retrieval frequency may modulate vividness and temporal gradient effects, as a result of the semanticisation process of more frequently retrieved memories, which would gradually acquire independence from medial temporal structures.

This notion is also supported by evidence from an fMRI investigation of the neural correlates of ABM in AD (Meulenbroek et al., 2010). The study showed enhanced activation of frontal regions (inferior frontal gyrus, ventromedial prefrontal cortex) which was inversely associated with hippocampal volume, suggesting that following hippocampal degeneration memory retrieval may rely more on frontal structures, mediating the activation of more preserved memories that probably have undergone semanticisation (Meulenbroek et al., 2010). Moreover, it has been shown that episodic, but not semantic, ABM retrieval impairments are associated with changes in hippocampal morphology (Thomann et al., 2012).

Recent research on the extent to which the hippocampus is involved in ABM revealed opposite results. Philippi et al. (2012) reported that the left hippocampus is associated to remote memory retrieval, while the right hippocampus is correlated with retrieval of more recent memories. Additionally, the authors hypothesize the presence of a rostrocaudal gradient depending on retention duration: lesions to left anterior regions are implicated in impairments of remote memories retrieval while more posterior lesions are linked to deficits in encoding, consolidation or retrieval of recent memories. Another study reported that remote episodic memory retrieval correlates with lateral and left posterior hippocampus (including CA1-3 and subiculum), while more recent memories relied on the left hippocampal head (border of CA1, CA2, and subiculum), in a sample composed by healthy older adults, MCI, and AD (Thomann et al., 2012).

Taken together, these findings describe the profile of ABM impairment in AD, which may play a pivotal role in self-knowledge and self-continuity in this condition. In fact, recent evidence supports a bidirectional relation between the self and memory. Martinelli et al. (2013) showed that AD is characterized by the lack

of relation between autobiographical episodes, self-concept and self-defining memories typically seen in healthy individuals. Moreover, AD patients show alterations in strength and complexity of their sense of self and tend to produce fewer memories tied to the self, with self-concept changes being associated with lower memory integration abilities (Ben Malek et al., 2019). Alterations of the self in AD have also been shown to be associated to memories characterised by lower specificity, fewer contextual details (El Haj & Antoine, 2017) and self-defining memory episodicity (Martinelli et al., 2013). Crucially, memory deficits in AD patients appear to interfere with the ability to remember and acknowledge how past events define themselves.

It is important to highlight that these changes are relative, typically defined in comparison to healthy older adults. Research into life stories (El Haj et al., 2019) has shown AD patients with mild dementia can retrieve ABMs to reflect on self-continuity, being able to maintain a life story and, interestingly, to be more concerned about changes in self-continuity as compared as healthy controls. This suggests that self-concept in AD, particularly during the milder stages of the condition, does not remain unchanged, a common misinterpretation of the Petrified self metaphor. These changes, however, are mediated by the profile of memory impairment of AD.

Finally, it is worth noting that research on ABM has also been approached in its relation with future episodic simulation, suggesting a similarity between remembering the past and imaging the future in AD (Addis et al., 2009; el Haj et al., 2015; El Haj et al., 2015). Specifically, (el Haj et al., 2015) showed that AD patients tend to evoke similar themes during past and future thinking, with this pattern also extending to self-defining memories. In the case of AD, this may contribute to a lack of appreciation of future consequences of their condition, projecting self-concepts tied to remote memories into future imaging.

3. Anterograde amnesia and anosognosia in AD

Evidence from the past 10 years strongly supports the notion that, despite showing fairly accurate predictions of performance, people with AD exhibit a failure in transferring information from online performance and actual experience to the

PDB, resulting in a stable but outdated self-evaluation. The metamemory literature shows that AD patients can make accurate predictions about their performance (E. Bertrand et al., 2018; J. M. Bertrand et al., 2019; Gallo et al., 2012) and use appropriately extrinsic and intrinsic factors in these predictions (Thomas et al., 2013). Moreover, it has been shown that metacognitive judgments in AD are similar regardless the presence of feedback (Chapman et al., 2018; Cosentino et al., 2016), thus suggesting a failure of integrating information about ongoing performance to make more accurate predictions. Crucially, it has been shown that even when prediction accuracy is higher in post- than in pre-test conditions, after 1 h delay AD patients estimation return to be as low as in the pre-test condition (Stewart et al., 2010). This suggests that performance monitoring is fairly preserved in AD and that metacognitive impairments in this group may derive from lack of updating of personal information.

Dodson et al. (2011) reported impairments in episodic memory monitoring in AD for item-by-item confidence accuracy, but accurate predictions at task level. They also observe that in a condition of additional exposure to test material, there is improved memory performance in AD, that does not differ from normal controls, but that still is accompanied by metamemory deficits. Similarly, in a study investigating metacognitive abilities through an associative learning paradigm, AD patients showed reduced online monitoring, presenting impairments in feeling of knowing and retrospective judgments in item-by-item judgments, but preserved sensitivity to extrinsic and intrinsic factors and feedback when asked to predict general performance (Rosen et al., 2014). Despite important methodological issues affecting results, such as procedure complexity and task difficulty, findings from Dodson et al. (2011) and Rosen (2014) suggest that although patients may have monitoring impairments, there is still some preserved calibration, with patients being able to revise their initial estimations of ability, particularly when prompted about performance.

Further insight comes from research investigating neuropsychological intervention outcomes for memory and metamemory abilities (Silva et al., 2017). The authors compared judgments of learning about memory performance before and after cognitive training. Pre-test scores showed that AD patients tend to overestimate their memory performance. Post-training scores revealed that the training improve

both memory and metamemory scores, but prediction of performance continued to be an overestimation of actual abilities. People with AD are able to retain online metamemory information, but this is not incorporated into longer term representations.

4. The role of the frontal lobes revisited

One aspect not fully developed in the 2009 article referred to the role of the frontal lobes in anosognosia in relation to AD. In that context, three main hypotheses were suggested for an association between frontal lobe dysfunction and AD anosognosia: difficulties in error monitoring, impairments in memory retrieval and alterations in belief evaluation systems. Emerging new evidence, mainly from neuroimaging studies, allows a critical revision of these notions.

Structural imaging studies investigating the relation between gray matter volume changes and anosognosia in AD mainly reported an association between frontal atrophy and self-awareness. In particular, anterior cingulate cortex integrity has been associated to lack of awareness (Guerrier et al., 2018) and metamemory deficits (E. Bertrand et al., 2018). Another study analysing metamemory abilities reported an association with right insula volume but also strong correlations between anterior and posterior cingulate cortex that may have been significant with a larger sample size (Cosentino et al., 2015). Interestingly, Spalletta et al. (2014) investigated anosognosia-related structural changes in amnestic mild cognitive impairment (MCI) patients, comparing patients who converted (CONV) to AD and those who did not (NON-CONV) after 5 years. Their results show different relations with anosognosia for the two groups: specifically, awareness for the memory domain in the CONV group was associated with anterior cingulate cortex (ACC) and inferior frontal gyrus volume, while temporal structures were associated to different awareness measures in the NON-CONV group.

Further evidence for frontal involvement comes from research on dementia subtypes, including AD, frontotemporal dementia and primary progressive aphasia (Shany-Ur et al., 2014), indicating that the tendency to overestimate overall functioning is associated to changes in cortical and subcortical frontal regions. Only one study reported divergent results, suggesting that anosognosia in AD and MCI

is mediated by temporal degeneration, including the hippocampus (Tondelli et al., 2018). Surprisingly, Senturk et al. (2017) found no correlation between cortical thickness and anosognosia in early AD and amnestic MCI patients, possibly due to sample characteristics, such as dementia severity, methodological issues (e.g., ROIs choice) and the potential contribution of non-cognitive factors to anosognosia.

Task-related functional magnetic resonance imaging (fMRI) studies highlight the role of the frontal lobes, in particular ventromedial prefrontal cortex (VMPFC) and ACC, for self-referential processes in relation to unawareness in AD (Genon et al., 2014; Zamboni et al., 2013). Moreover, Amanzio et al. (2011) found differences in functional activation during a response inhibition task between AD patients with preserved and impaired awareness, indicating reduced recruitment of frontocingulate, parietal and temporal areas for the unaware AD group. Positron-emission tomography (PET) studies consistently reported that frontal lobe dysfunction has been associated with anosognosia, with hypometabolism in dorsal ACC (Guerrier et al., 2018), dorsomedial PFC and superior frontal sulcus (Jedidi et al., 2014), orbitofrontal cortex and posterior regions as posterior cingulate cortex (PCC) and precuneus (Perrotin et al., 2015). Interestingly, evidence from single-photon emission computed tomography (SPECT) research investigating regional cerebral blood flow (rCBF) suggests that anosognosia in AD is not only related to frontal dysfunction but also to compensational mechanisms reflected by higher rCBF in parieto-occipital regions (Tagai et al., 2018). However, further research is needed to confirm this hypothesis.

Lastly, resting-state functional connectivity maps studies provide crucial evidence elucidating the neural correlates of anosognosia in AD, suggesting that unawareness may emerge from decreased interregional connectivity between and within medial prefrontal cortex and medial temporal regions. In particular, it has been shown a relation between memory self-appraisal and decreased MPFC connectivity with dorsolateral PFC, anterior cingulate cortex and hippocampus (Ries et al., 2012) and an association between anosognosia and disrupted connectivity between PCC and orbitofrontal cortex (OFC) and between OFC and hippocampus (Perrotin et al., 2015). A study conducted by Berlingeri et al. (2015) found significant functional connectivity reduction in unaware AD patients within the inferior medial temporal cortex (IMTC) and VMPFC networks, but only disconnection between IMTC and

hippocampus and insular cortex correlated with anosognosia severity. Finally, a more recent study reported that memory awareness correlated with the degree of disconnection between hippocampus and retrosplenial cortex extending to the ventral PCC and right posterior inferior parietal lobe; in addition, anosognosia was associated with decreased connectivity between hippocampus and VMPFC (Antoine et al., 2019).

Taken together, the evidence suggests that default mode networks (DMN) alterations may mediate self-appraisal and self-knowledge about cognitive functioning in dementia. This may reflect the pivotal role of frontal dysfunction in impaired self-awareness, with frontal lobe alterations leading to executive anosognosia (Morris & Mograbi, 2013; Mograbi & Morris, 2014). Moreover, the fMRI evidence reviewed is in line with the current notion that executive functions are mediated by anterior-posterior connectivity, thus indicating that anosognosia may be linked to disconnection within a monitoring network. In addition, the association between self-awareness deficits with medial temporal atrophy and intraregional connectivity suggests that anosognosia in AD may also depend on mnemonic dysfunction. The involvement of memory impairment in anosognosia in AD is further reinforced by evidence of decreased connectivity between frontal and temporal regions, indicating that a disconnection process can lead to alterations of self-appraisal and self-awareness. This may reflect, for instance, limited access to incident memory when engaging and self-evaluation processes. Conversely, lack of input from monitoring processes may contribute to failures in updating knowledge about the self. In any case, the possibility of unawareness emerging as a disconnection syndrome should be further explored in studies using structural and functional neuroimaging approaches.

The role of the frontal lobes in beliefs evaluation systems remains little explored in relation to anosognosia in AD. This has been suggested as an important factor in cases of unawareness for other clinical conditions, such as in stroke (Vuilleumier, 2004), with some empirical evidence supporting the notion (e.g., Venneri & Shanks, 2004; Vocat et al., 2013). It has been suggested that global measures of awareness in AD may be more vulnerable to beliefs (Chapman et al., 2018), but future studies are needed to investigate this issue. Figure 1 summarises the reviewed evidence in relation to the Petrified self concept.

PLEASE INSERT FIGURE 1 HERE

5. What is in a metaphor and concluding thoughts

The notion of a "petrified self" was first suggested as a metaphor for a self-concept that was stable, but outdated in time. Metaphors are literally false. The idea was highlighting a core identity in people with AD, based on autobiographical memory material that had been long consolidated, while, at the same time, acknowledging the difficulties caused by anterograde amnesia in the updating of self-concept. These difficulties have been suggested not only in the case of AD (e.g. De Simone et al., 2016; Kirk & Berntsen, 2018; Leyhe et al., 2009; Thomann et al., 2012), but also in other cases of hippocampal amnesia (e.g., Rosenbaum et al., 2005, but see also Elward & Vargha-Khadem, 2018).

What was never implied in the metaphor was that people with AD are dead inside, ossified or immune to change. There is, in fact, important evidence that contradicts these notions. For example, all rehabilitation and cognitive stimulation efforts (e.g., E. Bertrand et al., 2019) attest to the potential of improvement of people with dementia and other patients with amnesia (Wilson, 2009). Specifically related to awareness of difficulties, the notion of implicit awareness (Mograbi & Morris, 2013) suggests that behavioural and affective change may happen without explicit knowledge by patients. Similarly, emergent awareness (Moro, 2013) indicates that engaging in activities linked to deficits may promote increased awareness.

Additionally, it is important to highlight that the metaphor applies to narrative aspects of the self, i.e., those that are more directly linked to verbal questioning of self-concept. Selfhood is a complex phenomenon, with many different "selves" or self processes, as indicated in the original formulation about the petrified self (Mograbi et al., 2009). Additionally, it is important to highlight that the metaphor applies to narrative aspects of the self, i.e., those that are more directly linked to verbal questioning of self-concept. Selfhood is a complex phenomenon, with many different "selves" or self processes, as indicated in the original formulation about the petrified self (Baird & Thompson, 2018).

The field of dementia studies has demonstrated the importance of terminology and its potential impact on malignant social psychology. While care should be taken in how we use words to describe the condition, it is also important not to deny the profile of cognitive impairments that may affect selfhood in dementia. When allied with a careful approach, awareness can be a powerful tool for understanding or improvement.

References

Addis, D. R., Sacchetti, D. C., Ally, B. A., Budson, A. E., & Schacter, D. L. (2009). Episodic simulation of future events is impaired in mild Alzheimer's disease. Neuropsychologia, 47(12), 2660–2671. https://doi.org/10.1016/j.neuropsychologia.2009.05.018

Agnew, S. K., & Morris, R. G. (1998). The heterogeneity of anosognosia for memory impairment in Alzheimer's disease: A review of the literature and a proposed model. Aging & Mental Health, 2(1), 7–19. https://doi.org/10.1080/13607869856876

Amanzio, M., Torta, D. M. E., 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, 134(4), 1061–1076. https://doi.org/10.1093/brain/awr020

Antoine, N., Bahri, M. A., Bastin, C., Collette, F., Phillips, C., Balteau, E., Genon, S., & Salmon, E. (2019). Anosognosia and default mode subnetwork dysfunction in Alzheimer's disease. Human Brain Mapping, 40(18), 5330–5340. https://doi.org/10.1002/hbm.24775

Baird, A., & Thompson, W. F. (2018). The Impact of Music on the Self in Dementia. Journal of Alzheimer's Disease, 61(3), 827–841. https://doi.org/10.3233/JAD-170737

Barnabe, A., Whitehead, V., Pilon, R., Arsenault-Lapierre, G., & Chertkow, H. (2012). Autobiographical memory in mild cognitive impairment and Alzheimer's disease: A comparison between the Levine and Kopelman interview methodologies. Hippocampus, 22(9), 1809–1825. https://doi.org/10.1002/hipo.22015

Ben Malek, H., Philippi, N., Botzung, A., Cretin, B., Berna, F., Manning, L., & Blanc, F. (2019). Memories defining the self in Alzheimer's disease. Memory, 27(5), 698–704. https://doi.org/10.1080/09658211.2018.1554080

Berlingeri, M., Ravasio, A., Cranna, S., Basilico, S., Sberna, M., Bottini, G., & Paulesu, E. (2015). Unrealistic representations of "the self": A cognitive

neuroscience assessment of anosognosia for memory deficit. Consciousness and Cognition, 37(6), 160–177. https://doi.org/10.1016/j.concog.2015.08.010

Bertrand, E., Azar, M., Rizvi, B., Brickman, A. M., Huey, E. D., Habeck, C., Landeira-Fernandez, J., Mograbi, D. C., & Cosentino, S. (2018). Cortical thickness and metacognition in cognitively diverse older adults. Neuropsychology, 32(6), 700–710. https://doi.org/10.1037/neu0000458

Bertrand, E., Naylor, R., Laks, J., Marinho, V., Spector, A., & Mograbi, D. C. (2019). Cognitive stimulation therapy for brazilian people with dementia: examination of implementation' issues and cultural adaptation. Aging and Mental Health. https://doi.org/10.1080/13607863.2018.1488944

Bertrand, J. M., Mazancieux, A., Moulin, C. J. A., Béjot, Y., Rouaud, O., & Souchay, C. (2019). In the here and now: Short term memory predictions are preserved in Alzheimer's disease. Cortex, 119, 158–164. https://doi.org/10.1016/j.cortex.2019.03.027

Chapman, S., Colvin, L. E., Vuorre, M., Cocchini, G., Metcalfe, J., Huey, E. D., & Cosentino, S. (2018). Cross domain self-monitoring in anosognosia for memory loss in Alzheimer's disease. Cortex, 101, 221–233. https://doi.org/10.1016/j.cortex.2018.01.019

Cosentino, S., Brickman, A. M., Griffith, E., Habeck, C., Cines, S., Farrell, M., Shaked, D., Huey, E. D., Briner, T., & Stern, Y. (2015). The right insula contributes to memory awareness in cognitively diverse older adults. Neuropsychologia, 75, 163–169. https://doi.org/10.1016/j.neuropsychologia.2015.05.032

Cosentino, S., Zhu, C., Bertrand, E., Metcalfe, J., Janicki, S., & Cines, S. (2016). Examination of the metacognitive errors that contribute to anosognosia in Alzheimer's disease. Cortex, 84, 101–110. https://doi.org/10.1016/j.cortex.2016.08.003

De Simone, M. S., Fadda, L., Perri, R., Aloisi, M., Caltagirone, C., & Carlesimo, G. A. (2016). Does retrieval frequency account for the pattern of autobiographical memory loss in early Alzheimer's disease patients? Neuropsychologia, 80, 194–200. https://doi.org/10.1016/j.neuropsychologia.2015.11.024

Dodson, C. S., Spaniol, M., O'Connor, M. K., Deason, R. G., Ally, B. A., & Budson, A. E. (2011). Alzheimer's disease and memory-monitoring impairment: Alzheimer's patients show a monitoring deficit that is greater than their accuracy deficit. Neuropsychologia, 49(9), 2609–2618. https://doi.org/10.1016/j.neuropsychologia.2011.05.008

Donix, M., Brons, C., Jurjanz, L., Poettrich, K., Winiecki, P., & Holthoff, V. A. (2010). Overgenerality of autobiographical memory in people with amnestic mild cognitive impairment and early Alzheimer's disease. Archives of Clinical Neuropsychology, 25(1), 22–27. https://doi.org/10.1093/arclin/acp098

El Haj, M., & Antoine, P. (2017). Discrepancy between subjective autobiographical reliving and objective recall: The past as seen by Alzheimer's disease patients. Consciousness and Cognition, 49, 110–116. https://doi.org/10.1016/j.concog.2017.01.009

El Haj, M., Antoine, P., & Kapogiannis, D. (2015). Flexibility decline contributes to similarity of past and future thinking in Alzheimer's disease. Hippocampus. https://doi.org/10.1002/hipo.22465

el Haj, M., Antoine, P., & Kapogiannis, D. (2015). Similarity between remembering the past and imagining the future in Alzheimer's disease: Implication of episodic memory. Neuropsychologia, 66, 119–125. https://doi.org/10.1016/j.neuropsychologia.2014.11.015

El Haj, M., Boudoukha, A., Antoine, P., Moustafa, A. A., Gallouj, K., & Allain, P. (2019). Memories Supporting Myself: Autobiographical Memory Supports Self-Continuity in Alzheimer's Disease. Journal of Alzheimer's Disease, 70(4), 1217–1224. https://doi.org/10.3233/JAD-190440

Elward, R. L., & Vargha-Khadem, F. (2018). Semantic memory in developmental amnesia. In Neuroscience Letters. https://doi.org/10.1016/j.neulet.2018.04.040

Gallo, D. A., Cramer, S. J., Wong, J. T., & Bennett, D. A. (2012). Alzheimer's disease can spare local metacognition despite global anosognosia: Revisiting the confidence-accuracy relationship in episodic memory. Neuropsychologia. https://doi.org/10.1016/j.neuropsychologia.2012.06.005

Genon, S., Bahri, M. A., Collette, F., Angel, L., D'Argembeau, A., Clarys, D., Kalenzaga, S., Salmon, E., & Bastin, C. (2014). Cognitive and neuroimaging evidence of impaired interaction between self and memory in Alzheimer's disease. Cortex, 51(1), 11–24. https://doi.org/10.1016/j.cortex.2013.06.009

Guerrier, L., Le Men, J., Gane, A., Planton, M., Salabert, A. S., Payoux, P., Dumas, H., Bonneville, F., Péran, P., & Pariente, J. (2018). Involvement of the Cingulate Cortex in Anosognosia: A Multimodal Neuroimaging Study in Alzheimer's Disease Patients. Journal of Alzheimer's Disease : JAD, 65(2), 443–453. https://doi.org/10.3233/JAD-180324

Hirjak, D., Wolf, R. C., Remmele, B., Seidl, U., Thomann, A. K., Kubera, K. M., Schröder, J., Maier-Hein, K. H., & Thomann, P. A. (2017). Hippocampal formation alterations differently contribute to autobiographic memory deficits in mild cognitive impairment and Alzheimer's disease. Hippocampus, 27(6), 702–715. https://doi.org/10.1002/hipo.22726

Horning, S. M., Melrose, R., & Sultzer, D. (2014). Insight in Alzheimer's disease and its relation to psychiatric and behavioral disturbances. International Journal of Geriatric Psychiatry, 29(1), 77–84. https://doi.org/10.1002/gps.3972

Irish, M., Hornberger, M., el Wahsh, S., Lam, B. Y. K., Lah, S., Miller, L., Hsieh, S., Hodges, J. R., & Piguet, O. (2014). Grey and White Matter Correlates of Recent and Remote Autobiographical Memory Retrieval – Insights from the Dementias. PLoS ONE, 9(11), e113081. https://doi.org/10.1371/journal.pone.0113081

Irish, M., Hornberger, M., Lah, S., Miller, L., Pengas, G., Nestor, P. J., Hodges, J. R., & Piguet, O. (2011). Profiles of recent autobiographical memory retrieval in semantic dementia, behavioural-variant frontotemporal dementia, and Alzheimer's disease. Neuropsychologia, 49(9), 2694–2702. https://doi.org/10.1016/j.neuropsychologia.2011.05.017

Irish, M., Landin-Romero, R., Mothakunnel, A., Ramanan, S., Hsieh, S., Hodges, J. R., & Piguet, O. (2018). Evolution of autobiographical memory impairments in Alzheimer's disease and frontotemporal dementia – A longitudinal neuroimaging study. Neuropsychologia, 110(March 2017), 14–25. https://doi.org/10.1016/j.neuropsychologia.2017.03.014

Irish, M., Lawlor, B. A., O'Mara, S. M., & Coen, R. F. (2011). Impaired capacity for autonoetic reliving during autobiographical event recall in mild Alzheimer's disease. Cortex, 47(2), 236–249. https://doi.org/10.1016/j.cortex.2010.01.002

Jedidi, H., Feyers, D., Collette, F., Bahri, M. A., Jaspar, M., D'Argembeau, A., Salmon, E., & Bastin, C. (2014). Dorsomedial prefrontal metabolism and unawareness of current characteristics of personality traits in Alzheimer's disease. Social Cognitive and Affective Neuroscience, 9(10), 1458–1463. https://doi.org/10.1093/scan/nst132

Kirk, M., & Berntsen, D. (2018). The life span distribution of autobiographical memory in Alzheimer's disease. Neuropsychology. https://doi.org/10.1037/neu0000486

Levine, B., Svoboda, E., Hay, J. F., Winocur, G., & Moscovitch, M. (2002). Aging and autobiographical memory: Dissociating episodic from semantic retrieval. Psychology and Aging. https://doi.org/10.1037/0882-7974.17.4.677

Leyhe, T., Müller, S., Milian, M., Eschweiler, G. W., & Saur, R. (2009). Impairment of episodic and semantic autobiographical memory in patients with mild cognitive impairment and early Alzheimer's disease. Neuropsychologia, 47(12), 2464–2469. https://doi.org/10.1016/j.neuropsychologia.2009.04.018

Martinelli, P., Anssens, A., Sperduti, M., & Piolino, P. (2013). The influence of normal aging and Alzheimer's disease in autobiographical memory highly related to the self. Neuropsychology, 27(1), 69–78. https://doi.org/10.1037/a0030453

Meulenbroek, O., Rijpkema, M., Kessels, R. P. C., Rikkert, M. G. M. O., & Fernández, G. (2010). Autobiographical memory retrieval in patients with Alzheimer's disease. NeuroImage, 53(1), 331–340. https://doi.org/10.1016/j.neuroimage.2010.05.082

Mograbi, D. C., Brown, R. G., & Morris, R. G. (2009). Anosognosia in Alzheimer's disease – The petrified self. Consciousness and Cognition, 18(4), 989–1003. https://doi.org/10.1016/j.concog.2009.07.005

Mograbi, D. C., Ferri, C. P., Sosa, A. L., Stewart, R., Laks, J., Brown, R., & Morris, R. G. (2012). Unawareness of memory impairment in dementia: a population-based

study. International Psychogeriatrics, 24(6), 931–939. https://doi.org/10.1017/S1041610211002730

Mograbi, D. C., & Morris, R. G. (2014). On the Relation among Mood, Apathy, and Anosognosia in Alzheimer's Disease. Journal of the International Neuropsychological Society, 20(1), 2–7. https://doi.org/10.1017/S1355617713001276

Mograbi, D. C., & Morris, R. G. (2018). Anosognosia. Cortex, 103, 385–386. https://doi.org/10.1016/j.cortex.2018.04.001

Moro, V. (2013). The interaction between implicit and explicit awareness in anosognosia: Emergent awareness. In Cognitive Neuroscience. https://doi.org/10.1080/17588928.2013.853656

Morris, R. G., & Hannesdottir, K. (2004). Loss of "awareness" in Alzheimer's Disease. In The Cognitive Neuropsychology of Alzheimer's Disease (pp. 275–296).

Morris, R. G., & Mograbi, D. C. (2013). Anosognosia, autobiographical memory and self knowledge in Alzheimer's disease. Cortex, 49(6), 1553–1565. https://doi.org/10.1016/j.cortex.2012.09.006

Moscovitch, M., Rosenbaum, R. S., Gilboa, A., Addis, D. R., Westmacott, R., Grady, C., McAndrews, M. P., Levine, B., Black, S., Winocur, G., & Nadel, L. (2005). Functional neuroanatomy of remote episodic, semantic and spatial memory: a unified account based on multiple trace theory. Journal of Anatomy, 207(1), 35–66. https://doi.org/10.1111/j.1469-7580.2005.00421.x

Moscovitch, M., Westmacott, R., Gilboa, A., Addis, D. R., Rosenbaum, R. S., Viskontas, I., Priselac, S., Svoboda, E., Ziegler, M., Black, S., Gao, F., Grady, C., Freedman, M., Köhler, S., Leach, L., Levine, B., McAndrews, M. P., Nadel, L., Proulx, G., … Winocur, G. (2005). Hippocampal Complex Contribution to Retention and Retrieval of Recent and Remote Episodic and Semantic Memories: Evidence from Behavioral and Neuroimaging Studies of Healthy and Brain-Damaged People. In Dynamic Cognitive Processes (pp. 333–380). Springer-Verlag. https://doi.org/10.1007/4-431-27431-6_14

Müller, S., Mychajliw, C., Reichert, C., Melcher, T., & Leyhe, T. (2016). Autobiographical Memory Performance in Alzheimer's Disease Depends on

Retrieval Frequency. Journal of Alzheimer's Disease, 52(4), 1215–1225. https://doi.org/10.3233/JAD-151071

Orfei, M. D., Robinson, R. G., Prigatano, G. P., Starkstein, S., Rüsch, N., Bria, P., Caltagirone, C., & Spalletta, G. (2007). Anosognosia for hemiplegia after stroke is a multifaceted phenomenon: A systematic review of the literature. Brain, 130(12), 3075–3090. https://doi.org/10.1093/brain/awm106

Patel, V., & Prince, M. (2001). Ageing and mental health in a developing country: who cares? Qualitative studies from Goa, India. Psychological Medicine, 31(1), 29–38. https://doi.org/10.1017/S0033291799003098

Perrotin, A., Desgranges, B., Landeau, B., Mézenge, F., La Joie, R., Egret, S., Pélerin, A., de la Sayette, V., Eustache, F., & Chételat, G. (2015). Anosognosia in Alzheimer disease: Disconnection between memory and self-related brain networks. Annals of Neurology, 78(3), 477–486. https://doi.org/10.1002/ana.24462

Philippi, N., Noblet, V., Botzung, A., Després, O., Renard, F., Sfikas, G., Cretin, B., Kremer, S., Manning, L., & Blanc, F. (2012). MRI-Based Volumetry Correlates of Autobiographical Memory in Alzheimer's Disease. PLoS ONE, 7(10), e46200. https://doi.org/10.1371/journal.pone.0046200

Ries, M. L., McLaren, D. G., Bendlin, B. B., GuofanXu, Rowley, H. A., Birn, R., Kastman, E. K., Sager, M. A., Asthana, S., & Johnson, S. C. (2012). Medial prefrontal functional connectivity—Relation to memory self-appraisal accuracy in older adults with and without memory disorders. Neuropsychologia, 50(5), 603–611. https://doi.org/10.1016/j.neuropsychologia.2011.12.014

Rosen, H. J., Alcantar, O., Zakrzewski, J., Shimamura, A. P., Neuhaus, J., & Miller, B. L. (2014). Metacognition in the behavioral variant of frontotemporal dementia and Alzheimer's disease. Neuropsychology, 28(3), 436–447. https://doi.org/10.1037/neu0000012

Rosenbaum, R. S., Köhler, S., Schacter, D. L., Moscovitch, M., Westmacott, R., Black, S. E., Gao, F., & Tulving, E. (2005). The case of K.C.: contributions of a memory-impaired person to memory theory. Neuropsychologia, 43(7), 989–1021. https://doi.org/10.1016/j.neuropsychologia.2004.10.007

Seidl, U., Lueken, U., Thomann, P. A., Geider, J., & Schröder, J. (2011). Autobiographical Memory Deficits in Alzheimer's Disease. Journal of Alzheimer's Disease, 27(3), 567–574. https://doi.org/10.3233/JAD-2011-110014

Seltzer, B., Vasterling, J. J., Yoder, J., & Thompson, K. A. (1997). Awareness of Deficit in Alzheimer's Disease: Relation to Caregiver Burden. The Gerontologist, 37(1), 20–24. https://doi.org/10.1093/geront/37.1.20

Senturk, G., Bilgic, B., Arslan, A. B., Bayram, A., Hanagasi, H., Gurvit, H., & Emre, M. (2017). Cognitive and anatomical correlates of anosognosia in amnestic mild cognitive impairment and early-stage Alzheimer's disease. International Psychogeriatrics, 29(2), 293–302. https://doi.org/10.1017/S1041610216001812

Shany-Ur, T., Lin, N., Rosen, H. J., Sollberger, M., Miller, B. L., & Rankin, K. P. (2014). Self-awareness in neurodegenerative disease relies on neural structures mediating reward-driven attention. Brain, 137(8), 2368–2381. https://doi.org/10.1093/brain/awu161

Silva, A. R., Pinho, M. S., Macedo, L., Souchay, C., & Moulin, C. (2017). Mnemonic anosognosia in Alzheimer's disease is caused by a failure to transfer online evaluations of performance: Evidence from memory training programs. Journal of Clinical and Experimental Neuropsychology, 39(5), 419–433. https://doi.org/10.1080/13803395.2016.1231799

Spalletta, G., Piras, F., Piras, F., Sancesario, G., Iorio, M., Fratangeli, C., Cacciari, C., Caltagirone, C., & Orfei, M. D. (2014). Neuroanatomical correlates of awareness of illness in patients with amnestic mild cognitive impairment who will or will not convert to Alzheimer's disease. Cortex, 61, 183–195. https://doi.org/10.1016/j.cortex.2014.10.010

Starkstein, S. E., Boller, F., & Garau, L. (2005). A Two-Year Follow-Up Study of Remote Memory in Alzheimer's Disease. The Journal of Neuropsychiatry and Clinical Neurosciences, 17(3), 336–341. https://doi.org/10.1176/jnp.17.3.336

Starkstein, S. E., Jorge, R., Mizrahi, R., Adrian, J., & Robinson, R. G. (2007). Insight and danger in Alzheimer's disease. European Journal of Neurology, 14(4), 455–460. https://doi.org/10.1111/j.1468-1331.2007.01745.x

Stewart, G., McGeown, W. J., Shanks, M. F., & Venneri, A. (2010). Anosognosia for memory impairment in Alzheimer's disease. Acta Neuropsychiatrica, 22(4), 180–187. https://doi.org/10.1111/j.1601-5215.2010.00463.x

Tagai, K., Shinagawa, S., Kada, H., Inamura, K., Nagata, T., & Nakayama, K. (2018). Anosognosia in mild Alzheimer's disease is correlated with not only neural dysfunction but also compensation. Psychogeriatrics, 18(2), 81–88. https://doi.org/10.1111/psyg.12287

Thomann, P. A., Seidl, U., Brinkmann, J., Hirjak, D., Traeger, T., Wolf, R. C., Essig, M., & Schroder, J. (2012). Hippocampal morphology and autobiographic memory in mild cognitive impairment and Alzheimer's disease. Current Alzheimer Research, 9(4), 507–515. https://doi.org/10.2174/156720512800492558

Thomas, A. K., Lee, M., & Balota, D. A. (2013). Metacognitive monitoring and dementia: How intrinsic and extrinsic cues influence judgments of learning in people with early-stage Alzheimer's disease. Neuropsychology, 27(4), 452–463. https://doi.org/10.1037/a0033050

Tondelli, M., Barbarulo, A. M., Vinceti, G., Vincenzi, C., Chiari, A., Nichelli, P. F., & Zamboni, G. (2018). Neural Correlates of Anosognosia in Alzheimer's Disease and Mild Cognitive Impairment: A Multi-Method Assessment. Frontiers in Behavioral Neuroscience, 12(May), 1–11. https://doi.org/10.3389/fnbeh.2018.00100

Venneri, A., & Shanks, M. F. (2004). Belief and awareness: reflections on a case of persistent anosognosia. Neuropsychologia, 42(2), 230–238. https://doi.org/10.1016/S0028-3932(03)00171-4

Verhülsdonk, S., Quack, R., Höft, B., Lange-Asschenfeldt, C., & Supprian, T. (2013). Anosognosia and depression in patients with Alzheimer's dementia. Archives of Gerontology and Geriatrics, 57(3), 282–287. https://doi.org/10.1016/j.archger.2013.03.012

Vocat, R., Saj, A., & Vuilleumier, P. (2013). The riddle of anosognosia: Does unawareness of hemiplegia involve a failure to update beliefs? Cortex. https://doi.org/10.1016/j.cortex.2012.10.009

Vuilleumier, P. (2004). Anosognosia: The Neurology of Beliefs and Uncertainties. Cortex, 40(1), 9–17. https://doi.org/10.1016/S0010-9452(08)70918-3

Wilson, B. A. (2009). Memory rehabilitation: Integrating theory and practice. Guildford Press.

Zamboni, G., Drazich, E., McCulloch, E., Filippini, N., Mackay, C. E., Jenkinson, M., Tracey, I., & Wilcock, G. K. (2013). Neuroanatomy of impaired self-awareness in Alzheimer's disease and mild cognitive impairment. Cortex, 49(3), 668–678. https://doi.org/10.1016/j.cortex.2012.04.011

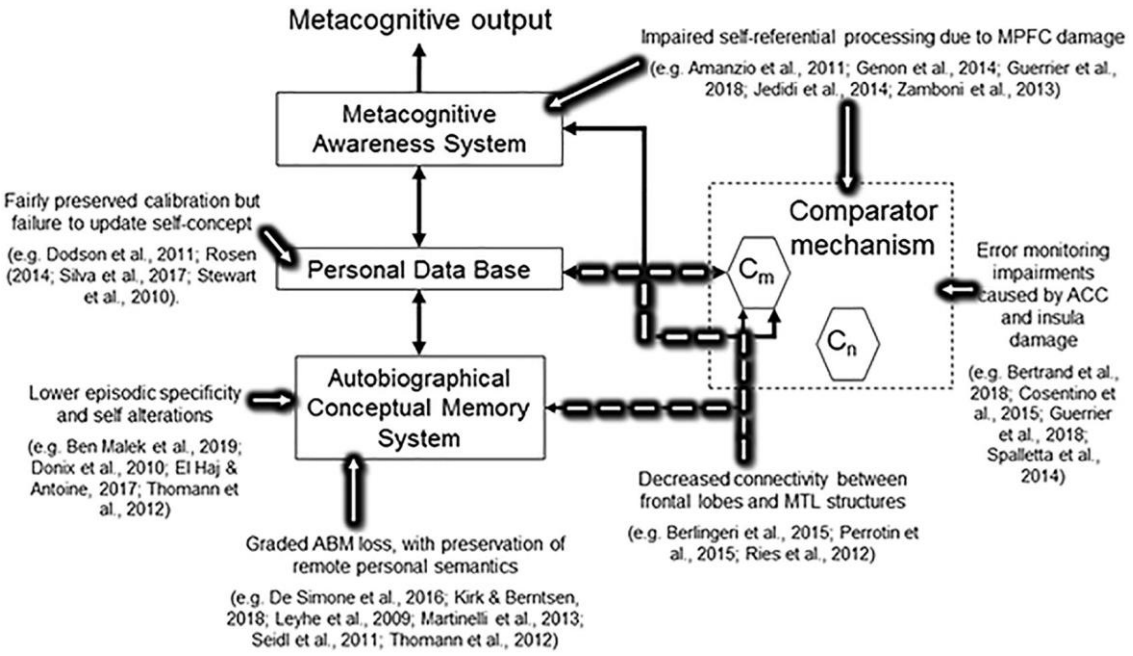

Figure 1. Simplified version of the Cognitive Awareness Model showing current evidence in relation to the main concepts of the Petrified Self.

Article 2

Lenzoni, S., Baker, J., Sumich, A. L., & Mograbi, D. C. (2022). New insights into neural networks of error monitoring and clinical implications: a systematic review of ERP studies in neurological diseases. Reviews in the Neurosciences, 33(2), 161-179. https://doi.org/10.1515/revneuro-2021-0054

Abstract

Error monitoring allows for the efficient performance of goal-directed behaviours and successful learning. Furthermore, error monitoring as a metacognitive ability may play a crucial role for neuropsychological interventions, such as rehabilitation. In the past decades, research has suggested two electrophysiological markers for error monitoring: the error-related negativity (ERN) and the error positivity (Pe), thought to reflect, respectively, error detection and error awareness. Studies on several neurological diseases have investigated the alteration of the ERN and the Pe, but these findings have not been summarized. Accordingly, a systematic review was conducted to understand what neurological conditions present alterations of error monitoring event-related potentials and their relation with clinical measures. Overall, ERN tended to be reduced in most neurological conditions while results related to Pe integrity are less clear. ERN and Pe were found to be associated with several measures of clinical severity. Additionally, we explored the contribution of different brain structures to neural networks underlying error monitoring, further elaborating on the domain-specificity of error processing and clinical implications of findings. In conclusion, electrophysiological signatures of error monitoring could be reliable measures of neurological dysfunction and a robust tool in neuropsychological rehabilitation.

Keywords

ERPs; error positivity; error-related negativity; neurology; self-monitoring

1. Introduction

Error monitoring is crucial to successfully perform goal-directed behaviours (Ullsperger et al., 2014) and for adaptive control in daily life (Krönke et al., 2018; Overmeyer et al., 2021). Over the last decades, error processing has captured the attention of clinical research, showing that deficient error monitoring characterizes various mental disorders (for example Clayson et al., 2020; Meyer, 2016; Riesel et al., 2019). Moreover, error monitoring, as a metacognitive ability involved in online cognitive control, is thought to contribute to the emergence of self-awareness (R. G. Morris & Mograbi, 2013).

According to the Cognitive Awareness Model (Agnew & Morris, 1998; Mograbi & Morris, 2014), performance monitoring plays a key role in in the integrity of metacognitive awareness. Error monitoring impairments, underlying frontal cortico-subcortical loops can occur at multiple levels and result in anosognosia, that is defined as the lack of awareness of symptoms or deficits in clinical conditions, such as neurological disorders (Mograbi & Morris, 2018). A central dysfunction of monitoring mechanisms would result in executive anosognosia while domain-specific impairments would lead to local, domain-specific unawareness, such as anosognosia for hemiplegia.

Self-awareness has been extensively investigated in neurodegenerative diseases and acquired brain injury (Amanzio et al., 2020; Chavoix & Insausti, 2017; Leung & Liu, 2011; Mazancieux et al., 2019; Prigatano & Sherer, 2020). Crucially, impaired self-awareness in neurological conditions can hinder rehabilitation (Medley & Powell, 2010; Ownsworth & Clare, 2006; Trahan et al., 2006) and community reintegration (Kelley et al., 2014). Self-awareness, and specifically error awareness, is believed to be essential for successful rehabilitation of cognitive functions (Dockree et al., 2015; Leung & Liu, 2011). Common approaches in rehabilitation are errorless and error-based learning. In the first case, the training consists of observing and practicing only correct actions, through the support of the therapist who prevents the patient from performing errors (Haslam & Kessels, 2018). This approach has been shown to be effective for memory impairments (Clare & Jones, 2008; Dunn & Clare, 2007; Ehlhardt et al., 2008). Instead, error-based learning training focuses on a trial-and-error process, including prompt and feedback provided by the psychotherapist, allowing self-correction and facilitating strategy

use (Toglia, 2011). Recently, it has been shown that error-based learning can be more effective than errorless learning approaches in rehabilitation for brain injury patients, improving patient's self-awareness and allowing skill transfer (Ownsworth et al., 2017), thus highlighting the relevance of error detection and correction for clinical conditions. Critically, Overmeyer et al. (2021) showed that error-related negativity (ERN) can predict self-control in real life behavior. Assessing error-related integrity in brain injury patients may be indicative of self-monitoring abilities within specific cognitive domains and thus guide the choice of rehabilitation techniques. Therefore, functional brain biomarkers of error monitoring could potentially become a robust tool for clinical assessment and rehabilitation.

Electroencephalography (EEG) research has identified two event-related potentials (ERPs) underpinning error processing: the error-related negativity (ERN) and the error positivity (Pe). The ERN is a negative deflection occurring around 50ms over fronto-central sites following error commission (Gehring et al., 1993). According to the Mismatch Theory, the ERN reflects the mismatch between action efferent copies and top-down representations of intended (correct) and actual response (Dehaene, 2018; Falkenstein et al., 1991; Gehring et al., 1993; Scheffers & Coles, 2000). Alternatively, it has been proposed that the ERN represents the degree of conflict between competing representations (Conflict Monitoring theory; Botvinick et al., 2001; Yeung et al., 2004).

The neural generator of the ERN has been localized in the anterior cingulate cortex (ACC; Brázdil et al., 2005; Debener, 2005; Dehaene et al., 1994; Reinhart & Woodman, 2014; van Veen & Carter, 2002). According to Reinforcement Learning theory, the ERN is mediated by changes in levels of phasic dopaminergic activity in the basal ganglia resulting in inhibitory error signaling from the basal ganglia to the ACC (Holroyd & Coles, 2002). According to the Predicted Response-Outcome (PRO) model (Alexander & Brown, 2011), learning processes relying on medial prefrontal cortex function follow standard rules of probability. The authors also proposed that error effects may reflect the comparison between actual and intended outcomes, while conflict derives from the prediction of multiple responses and their outcomes. Evidence from behavioural studies, focusing on post-error slowing, suggested that unexpected events (either correct responses or errors) elicit a

maladaptive shift of the attention away from the ongoing task (Notebaert et al., 2009). A more recent account of error processing is the adaptive orienting theory (Wessel, 2018), which posits that errors, as unexpected events, trigger a series of adaptive automatic processes, including rapid motor and cognitive suppression, and subsequent attentional reorienting. This is supported by further research on the association between the ERN and attentional post-error adjustments, showing temporal proximity between ERN and subsequent attentional reallocation, and that the strength of post-error adjustments varies with ERN amplitude (Steinhauser & Andersen, 2019). The consensus among different theoretical accounts is that the ERN indexes a performance monitoring system, that enables learning and behavioral adjustments (Weinberg et al., 2015).

The Pe is a later positive component, peaking at centro-parietal sites between 200-500ms after error commission (Falkenstein et al., 1991; Overbeek et al., 2005). It has been shown that ERN and Pe represent two independent systems of error monitoring (di Gregorio et al., 2018; Overbeek et al., 2005). However, the functional role of Pe is still debated. It has been proposed that Pe is a P3b-like component, associated with motivational significance of the response (Overbeek et al., 2005; Ridderinkhof et al., 2009), that may reflect working memory updating (Donchin & Coles, 1988; Polich, 2007). In this context, post-error processing has been associated with locus coeruleus-norepinephrine system activity (Nieuwenhuis et al., 2005; Overbeek et al., 2005; Ridderinkhof et al., 2009), as one possible input into the salience network (Wessel, 2018). Error awareness and processing of salience have been shown to rely on overlapping neural networks, involving the anterior insula, dorsal ACC, thalamus, supplementary motor area, and parietal regions (Harsay et al., 2012). Previous studies have demonstrated a relationship between Pe and conscious perception of errors (Endrass et al., 2007; Murphy et al., 2012; Nieuwenhuis et al., 2001). In line with the Accumulation Account, the Pe reflects error awareness, which emerges from a process of evidence accumulation about the erroneous response (Steinhauser & Yeung, 2010, 2012; Ullsperger et al., 2010; Wessel et al., 2011). Error awareness is believed to emerge from the integration of different input signals, such as cognitive, sensory, proprioceptive and interoceptive inputs (Ullsperger et al., 2010; Wessel et al., 2011). Ullsperger et al.

(2010) also suggested that the neural network underlying error awareness and the Pe involved structures such as ACC, anterior insula and, somatosensory areas.

The aim of this review is to understand whether ERN and Pe alterations are specific to certain neurological conditions and examine their relation with clinical factors. Furthermore, this evaluation will provide insights to elucidate the role of different brain areas in neural networks underlying error monitoring.

2. Methods

2.1 Search strategy

Article selection was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Moher et al., 2009) guidelines. A flowchart of this selection process is displayed in Figure. 1. PubMed, Scopus and Web of Science databases were systematically searched for eligible studies from inception to January 31st, 2021. The search terms used were: ERP OR "event related" OR "event-related" OR "evoked potential" OR "evoked-potential" AND "error related negativity" OR "error-related negativity" OR ERN OR Ne OR "error positivity" OR Pe. Reference lists from detected studies were also checked for additional unidentified studies.

2.2 Study selection

Only English-language studies were included. Eligible studies fulfilled the following criteria: 1) the study design was cross-sectional; 2) the study included a clinical group with a neurological condition and a control group, as determined by neurological diagnosis; 3) all participants were adults; 4) each group was composed of at least 5 participants; 5) the amplitude and/or the latency of the ERN and/or the Pe were measured by ERP technique. Studies without group-level statistics were excluded. Studies including neurodevelopmental diseases were excluded. Reviews and conferences papers were also excluded.

2.3 Quality assessment

A quality assessment form was devised which focused on sampling, measurement of outcomes and analysis (Table 1). In accordance with the Cochrane Collaboration recommendations (Higgins et al., 2020), an overall score was not generated, with a risk of bias judgment of "yes", "no" or "unclear" being given instead for individual domains. Study quality was assessed by two independent reviewers If a study received more than two "no" or three "unclear" judgments, the study was considered as having poor quality and was excluded from the review.

2.4 Data extraction

The following data were extracted by two independent reviewers: authors, publication year, diagnosis, sample size, task (experimental task and stimuli description), results comparing patient and control behavioural performance and ERPs measures, and correlations between ERPs measures and other measures.

PLEASE INSERT TABLE 1 HERE

PLEASE INSERT FIGURE 1 HERE

3. Results

A total of 41 studies met inclusion criteria and were selected for review. Of these, 39 measured the ERN and 23 the Pe, with 21 measuring both ERPs. ERN and Pe were typically measured at midline electrodes. The most common recording sites of interest were Fz, FCz, Cz, and Pz.

The most common task (n studies=23) to investigate error monitoring was the Flanker Task, which relies on the conflict between task-relevant and task-irrelevant stimuli (Eriksen & Eriksen, 1974). Other tasks based on interference suppression were the Stroop Task (n=3), Letter Discrimination Task (n=1), Simon-type Task (n=1) and the Error Awareness Task (n=1). The Error Awareness Task is a paradigm developed by Hester et al. (2005) and is an adapted version of the Stroop Task, incorporating a Go/NoGo component and a button press response to signal

error awareness. Paradigms involving response inhibition included the Go/NoGo (n=3), the Oddball Task (n=1), and the Stop Signal Task (n=3). The remaining paradigms relied on a variety of cognitive tasks. In the Anti-saccade Task (n=3), participants are asked to quickly perform a saccade to the opposite direction of a cue stimulus presentation (Hallett, 1978). In the Lexical Decision paradigm (n=2), participants are asked to decide whether a string of letters is a word or not (Rubenstein et al., 1971). The Picture-Name Verification task (n=1) consists of the presentation of a word followed by a picture. Participants are then asked to decide whether they semantically matched or not (Wingfield, 1968). Finally, a visual search (n=1) and a visual short-term memory task (n=1) were also employed as experimental paradigms.

Neurological conditions included Alzheimer's disease (n=2), Tourette syndrome (n=3), multiple sclerosis (n=1), amyotrophic lateral sclerosis (n=1), Parkinson's disease (n=11), Huntington's disease (n=4), cerebellar ataxia (n=1), cerebellar degeneration (n=1), focal lesions (n=9+ 1 cerebellar lesion) and traumatic brain injury (n=8). ERPs findings are reviewed by disorder. Cerebellar ataxia, cerebellar degeneration and one study involving cerebellar lesion were grouped together as "Cerebellar Dysfunction". Results of single studies are displayed in Table 2.

3.1 Alzheimer's disease (AD)

Both studies showed lower ERN amplitude in AD patients compared to healthy controls (Ito & Kitagawa, 2005; Mathalon et al., 2002), with the former reporting longer ERN latency in AD. One study reported no difference between AD and controls for Pe amplitude (Mathalon et al., 2002), while Ito and Kitagawa (2005) showed decreased Pe amplitude and prolonged latency in AD. Mathalon et al., (2002) used a Picture-Name Task Verification, while Ito and Kitagawa (2005) a Lexical Decision Paradigm.

3.2 Tourette Syndrome (TS)

All studies reported higher ERN amplitude in TS as compared to controls (Johannes et al., 2002; Schüller et al., 2018; Warren et al., 2020). No differences in ERN latency were reported by Johannes et al., (2002). Pe amplitude was measured in only one study (Schüller et al., 2018), which indicated lower amplitude in TS than healthy controls. No significant correlation was reported between ERPs amplitude and clinical parameters (Schüller et al.,2018; Warren et al., 2020) or neuroepileptic medication (Schüller et al., 2018). Tasks used were the Oddball task (Johannes et al., 2002), Stop Signal Task (Schuller et al., 2018) and the Flanker Task (Warren et al., 2020).

3.3 Multiple Sclerosis (MS)

Only one study investigated error monitoring in MS (López-Góngora et al., 2015) using a Flanker Task with an inhibition of response variant ("Stop task"). They reported higher ERN amplitude in MS as compared to healthy controls. Additionally, correlational analyses showed a negative association between ERN amplitude and time since last relapse and a positive association between ERN amplitude and Multiple Sclerosis Severity Score and Expanded Disability Status Scale.

3.4 Amyotrophic Lateral Sclerosis (ALS)

Only one study using the Flanker task to measure error related ERPs in ALS was found (Seer, Joop, et al., 2017). Results showed no differences in ERN amplitude between ALS and controls. Further analyses on subgroups showed that ALS patients with low executive performance had lower ERN amplitude than ALS patients with high executive performance and controls with low executive performance, while ERN did not differ between controls with low executive performance and controls with high executive performance. Correlational analyses revealed that executive performance negatively correlated with ERN amplitude in the ALS group.

3.5 Cerebellar Dysfunction

A study on patients affected by cerebellar ataxia (CA; Tunc et al., 2019) reported no differences in ERN amplitude between CA and controls, while two studies conducted by the Peterburs group revealed lower ERN in amplitude in patients with cerebellar lesion (CL; Peterburs et al., 2012) and cerebellar degeneration (CD; Peterburs et al., 2015). No alteration of ERN latency was reported in CL (Peterburs et al., 2012). Peterburs et al., 2015 reported that ERN amplitude negatively correlated with grey matter volume in the cerebellum right lobule V and left lobule VIIb/VIIIa. Moreover, Pe amplitude was found to be lower in CA patients (Tunc et al.,2019) and higher in CL patients (Peterburs et al., 2012) as compared to healthy controls. No differences in Pe amplitude between CD and controls were reported (Peterburs et al., 2015). Shorter Pe latency in CL was reported (Peterburs et al., 2012). Peterburs et al. (2015) reported that Pe was positively correlated with grey matter volume in the right posterolateral cerebellum. The task used in the study on CA by Tunc et al. (2019) was a Flanker task while the Anti-saccade Task was the experimental task for CL and CD studies (Peterburs et al., 2012, 2015)

3.6 Parkinson's Disease (PD)

Eight studies reported reduced ERN amplitude in PD (Beste et al., 2009; Falkenstein et al., 2001; Ito & Kitagawa, 2006; Rustamov et al., 2014; Seer, Lange, et al., 2017; Stemmer et al., 2007; Willemssen et al., 2008, 2009). Two studies reported no ERN amplitude differences between PD and controls (Holroyd et al., 2002; Verleger et al., 2013). ERN latency was shown to be unaltered in PD in five studies while Falkenstein et al. (2001) reported no differences for Flanker task performance and shorter latencies for Go/NoGo and Simon-type tasks.

Further analyses have been conducted on medication state in PD. Stemmer et al. (2007) and Beste et al. (2009) found no differences in ERN amplitude between drug-naïve and medicated PD. Another study on treated PD patients (Willemssen et al., 2008) revealed no difference between on- and off-medication groups. Seer, Lange, et al., (2017) reported reduced ERN amplitude in on-medication PD as compared to off-medication PD. Although the on-medication group presented reduced ERN amplitude as compared to healthy controls, no differences were found between off-medication PD and control groups.

Correlational analyses showed that BDI scores were positively associated with ERN amplitude in PD (Willemssen et al., 2009). Seer, Lange, et al. (2017) reported that in the PD off-medication group, ERN amplitude was inversely associated with higher scores in apathy, depression, psychiatric status, and schizotypal scales and positively associated with health status. In the on-medication group reduced ERN amplitude was associated with higher apathy scores.

Pe was measured in only two studies. Ito and Kitagawa (2006) showed that Pe amplitude is reduced in PD while Pe latency did not differ between PD and controls. The other study reported no differences in Flanker task and Simon-type task performance, while Pe amplitude was found to be lower in Go/NoGo performance (Falkenstein et al., 2005). Ito and Kitagawa (2006) used a Lexical Decision Paradigm; Falkenstein et al. (2001, 2005) applied three paradigms: Flanker task, Simon type task, Go/NoGo task. The other ten studies employed the flanker task.

3.7 Huntington's Disease (HD)

ERN amplitude was shown to be lower in HD as compared to healthy controls (Beste et al., 2009) and pre-clinical HD (pHD; Beste et al., 2008, 2007, 2006). No differences in ERN amplitude between pHD and healthy controls were found (Beste et al., 2007). Beste et al. (2009) reported that ERN amplitude did not differ between pHD and young controls, and that was higher in pHD as compared to old controls. Correlation analyses revealed that CAG-index was inversely associated with ERN amplitude (Beste et al., 2006). Additionally, medial frontal gyrus grey matter volume was found to be correlated with ERN amplitude (Beste et al., 2008) No group difference for ERN latency was reported when comparing HD and healthy controls (Beste et al, 2006, Beste et al.,2009), HD and pHC, or pHC and controls (Beste et al., 2009). Beste et al. (2008) did not find Pe amplitude differences between HD and pHD. All studies used a Flanker Task as the experimental paradigm (Beste et al., 2006, 2007, 2008, 2009).

3.8 Focal Lesions

Studies investigating error related ERPs included lesions of the ACC (n=1), lateral prefrontal cortex (n=4), orbitofrontal cortex (n=1), frontopolar cortex (n=1),

temporal cortex (n=1), basal ganglia (n=1), thalamus (n=2), and left hemisphere regions (n=1). Patients with ACC lesions showed reduced ERN amplitude as compared to healthy controls and a participant group (brain damage control) with brain lesions not involving the ACC (Maier et al., 2015). No between group differences in Pe amplitude and latency were found.

One study involving patients presenting basal ganglia lesions showed reduced ERN and Pe amplitude, while no differences in latency were found as compared to healthy controls (Ullsperger & von Cramon, 2006). Three studies on lateral prefrontal cortex (PFC) lesions showed lower ERN amplitude as compared to controls (Gehring & Knight, 2000; Ullsperger et al., 2002; Wessel et al., 2014) Ullsperger and Von Cramon, 2006). In contrast, Gehring and Knight (2000) reported no differences in ERN amplitude between lateral PFC lesions and control groups. No differences in ERN latency were found (Ullsperger et al., 2002; Ullsperger & von Cramon, 2006). Pe amplitude was found to be reduced in lateral PFC patients (Ullsperger et al., 2002; Ullsperger & von Cramon, 2006). Solbakk et al. (2014) reported reduced ERN amplitude and higher Pe amplitude in orbitofrontal lesion patients as compared to healthy controls. The study by Ullsperger et al. (2002) involving a frontopolar and temporal lesion group reported no differences in ERN amplitude and latency and Pe amplitude between lesion groups and controls. Studies on thalamic lesions revealed reduced ERN amplitude (Peterburs et al., 2011; Seifert et al., 2011) and Pe amplitude (Seifert et al., 2011).

Finally, Niessen et al. (2020) reported no differences either in ERN or Pe amplitude and latency between patients with a left hemisphere lesion and healthy controls. Moreover, they found a correlation between ERN latency and lesion size. Peterburs et al (2011) used an anti-saccade task as experimental task, Gehring and Knight used a letter discrimination task, Solbakk et al. (2014) used a stop signal task and Niessen et al. (2020) a Go/NoGo Task. All the other studies used a Flanker task.

3.9 Traumatic Brain Injury (TBI)

Five studies reported reduced ERN amplitude in TBI patients (De Beaumont et al., 2013; Larson et al., 2007, 2009; Pontifex et al., 2009). Two studies showed no differences in ERN amplitude between TBI patients and healthy controls (Larson

et al., 2012; Shen et al., 2020). Olson et al. (2018) reported higher ERN amplitude in TBI as compared to controls. No differences in ERN latency between TBI and controls were found (Larson et al., 2007, 2009; Shen et al., 2020).

Seven studies reported no difference in Pe amplitude between TBI and healthy controls (De Beaumont et al., 2013; Larson et al., 2009, 2012; Logan et al., 2015; Olson et al., 2018; Pontifex et al., 2009; Shen et al., 2020). One study showed reduced Pe amplitude in TBI (Larson et al., 2007). While Larson et al. (2007) reported no differences in Pe latency between TBI and controls, Shen et al. (2020) found longer Pe latency in TBI.

Correlational analyses showed that the ERN was negatively associated with number of prior incidents (Pontifex et al., 2009) and concussions (De Beaumont et al., 2013). Moreover, negative affect was inversely correlated with ERN amplitude (Larson et al., 2009). ERN latency was found to be inversely associated with prior TBIs (Larson et al.,2012)

Pe amplitude was associated with length of post-traumatic amnesia, and negatively associated with time since injury (Larson et al., 2012). Shen et al., (2020) reported that probability of inhibition (likelihood of response inhibition for Stop trials) negatively correlated with Pe latency and positively correlated with Pe amplitude.

Two studies used a Flanker task (Olson et al., 2018; Pontifex et al., 2009), The Stroop task was used in three studies (Larson et al., 2007,2009,2012). The Error Awareness Task was used by Logan et al (2015). Shen et al. (2020) employed a Stop signal task while De Beaumont et al. (2013) employed a visual search task variant and a visual short-term memory task.

PLEASE INSERT TABLE 2 HERE

4. Discussion

This systematic review included 41 articles assessing the ERN and the Pe in neurological disorders including Alzheimer's disease, Tourette's syndrome, multiple sclerosis, amyotrophic lateral sclerosis, Parkinson's disease, Huntington's disease, cerebellar ataxia, cerebellar degeneration, focal lesions and traumatic brain

injury, in comparison with controls. Overall, ERN amplitude tended to be reduced in clinical conditions, with the exception of Tourette syndrome and multiple sclerosis, which seemed to be characterized by enhanced ERN amplitude. Pe amplitude was investigated in fewer studies and did not present a consistent pattern of alteration across different neurological disorders. ERN and Pe latency were generally unaltered with the exception of a few individual studies across different clinical groups. The Flanker Task was the most commonly employed experimental task across neurological conditions, but the use of other paradigms relying on different cognitive processes needs to be considered to discuss contradictory results.

One of the aims of this review was to understand whether alterations of error monitoring are specific to certain neurological conditions, examining the contribution of different brain structures to error monitoring. ERN alterations can be consistently observed in neurological disorders affecting core structures involved in error monitoring. In line with the PRO model (Alexander & Brown, 2011), the medial prefrontal cortex plays a crucial role in error processing. Patients with lesion of the ACC, which is thought to be the ERN neural generator (Brázdil et al., 2005; Debener, 2005; Dehaene et al., 1994; Reinhart & Woodman, 2014; van Veen & Carter, 2002), were found to present reduced ERN (Maier et al., 2015). Similar findings were found in studies in AD (Ito & Kitagawa, 2005; Mathalon et al., 2002), in which ACC dysfunction is well documented (Rosenberg et al., 2015). Interestingly, previous research associated ACC alterations with self-awareness in AD (for a review, see Lenzoni et al., 2020), thus confirming its crucial role in self-monitoring alterations in these patients. According to the Reinforcement Learning theory (Holroyd and Coles, 2002), error signalling relies on mesencephalic dopaminergic activity from the basal ganglia to the ACC. Neurodegenerative disorders affecting basal ganglia and dopamine regulation consistently presented alteration of the ERN. The ERN was shown to be reduced in PD (Beste, et al., 2009; Falkenstein et al., 2001; Ito and Kitagawa, 2006; Rustamov et al., 2014; Seer, Lange, et al., 2017; Stemmer et al., 2007; R. Willemssen et al., 2008; Willemssen et al., 2009) and in HD (Beste et al., 2006; (Beste et al., 2009), while higher ERN amplitude was found in TS (Johannes et al., 2002; Schüller et al., 2018; Warren et al., 2020). Moreover, the ERN was found to be reduced in patients with a focal basal ganglia lesion (Ullsperger and Von Cramon, 2006). These findings show that

changes in dopamine levels mediate performance monitoring processes, as previously suggested by research reporting the impact of dopamine antagonists (Forster et al., 2017; Zirnheld et al., 2004) and dopamine receptors genotypes (Biehl et al., 2011; Krämer et al., 2007) on the ERN. Focal thalamic lesions were also associated with reduced ERN (Peterburs et al., 2011; Seifert et al., 2011). Crucially, the thalamus plays a key role in the generation and updating of mental representation (Wolff & Vann, 2019), and is considered a relay of efferent copies (or corollary discharge) of motor commands (Sommer, 2003). Therefore, thalamic alterations may have a disruptive impact on the cognitive conflict between competing representations and their "translation" into the appropriate motor commands to be selected during task performance.

Interestingly, even when the "core" neural network underlying error monitoring is not directly affected, the ERN may be altered. When analysing the studies involving these neurological conditions, contradictory results can be found and no clear pattern can be defined. However, considering the interaction between the experimental task and the lesion localization, it can be hypothesized that the ERN does not merely rely on the integrity of the structures involved in conflict processing and error detection, but also on the alteration of those cognitive processes mediating the generation of the competing representations. Two studies showed reduced ERN in participants affected by cerebellar dysfunctions (Peterburs et al., 2012, 2015). Peterburs et al. (2012) included patients vascular focal damage to the cerebellum. By comparison, Peterburs et al (2015) patient group, that we labelled CD, included pathologies that primarily affect the cerebellar cortex, such as spinocerebellar ataxia, sporadic adult-onset ataxia, and autosomal dominant ataxia. In both studies the paradigm used was the Antisaccade Task. The third study reported spared ERN amplitude in cerebellar ataxia (Tunc et al, 2019), which includes different types of spinocerebellar ataxia. The task used was the Flanker Task. Therefore, online performance monitoring in patients affected by cerebellar dysfunction results to be impaired for cognitive abilities that rely on cerebellar integrity, such as saccadic eye movement generation, as shown by the Peterburs group. In contrast, error monitoring appears to be spared for functions that are not prominently mediated by cerebellar activation, such as for the Flanker Task. Similarly, the ERN was shown to be reduced in patients with a lateral PFC lesion during the Flanker Task

performance (Ullsperger et al., 2002; Ullsperger & von Cramon, 2006; Wessel et al., 2014), while it was unaltered as compared to controls when performing a letter discrimination task (Gehring & Knight, 2000). Furthermore, the ERN was found to be larger in patients with orbitofrontal lesions during a Stop Signal Task (Solbakk et al., 2014) but was shown to be unaltered during the Flanker Task performance (Ullsperger et al., 2002). This suggests the presence of domain-specific mechanisms underlying error monitoring, that may selectively affect task performance. Beyond a domain-general "core" network, domain-specific neural signals contribute to the generation of competing representations, and therefore, mediate error processing. This notion would imply the existence of domain-specific alterations of representations (and their correctness), supporting theoretical accounts such as the Mismatch Theory (Dehaene, 2018; Falkenstein et al., 1991; Gehring et al., 1993; Scheffers & Coles, 2000) and PRO model (Alexander & Brown, 2011), that emphasize the pivotal role of multiple competing representations and their response outcome during performance monitoring, rather than a general mechanisms of response conflict, as proposed by the Conflict Monitoring hypothesis (Botvinick et al., 2001; Yeung et al., 2004). Although, domain-specificity of metacognitive processes, such as self-monitoring, have been previously discussed (Mograbi & Morris, 2014) and supported by behavioral (Bellon et al., 2020; Chapman et al., 2018; Dentakos et al., 2019) and neuroimaging (Morales et al., 2018) studies, it is yet to be investigated in ERP research. Nonetheless, inconsistent findings within neurological conditions may be also mediated by heterogeneity in methodology, such as task instructions (S. E. Morris et al., 2006), number of error trials (Fischer et al., 2017), or task difficulty (Riesel et al., 2015), and individual differences, such as motivation (Boksem et al., 2006), affective state (Wiswede et al., 2009), or stress (Hu et al., 2019) that may modulate the ERN differently across clinical and healthy populations.

A smaller proportion of the studies included analyses of the Pe. Overall, the results are in line with a functional distinction between ERN and Pe (di Gregorio et al., 2018; Overbeek et al., 2005) as demonstrated by the lack of unidirectional changes across many different neurological conditions. Pe was shown to be unaltered in the presence of ERN reduction (Beste et al., 2008; De Beaumont et al., 2013; Larson et al., 2009; Maier et al., 2015; Olson et al., 2018; Peterburs et al., 2015; Pontifex et

al., 2009), thus supporting the idea that ERN and Pe represent independent systems of error monitoring. Critically, in most of the neurological conditions considered in this review, we have evidence on the Pe from one study only. Therefore, it is more difficult to draw major conclusions about Pe integrity within individual conditions.

However, it is important to observe that the ACC may not have a prominent role in Pe generation, as shown by unaltered Pe in patients with ACC lesion (Maier et al., 2015) and AD (Ito et al., 2005). Among basal ganglia disorders, Pe was reduced in focal lesion (Ullsperger & Von Cramon, 2006) and TS (Tunc et al., 2019) patients. It should be noted that the Ullsperger group reported reduced Pe in lateral PFC lesion patients, and in 5 out of 7 patients, the lesion extended to the insula (Ullsperget et al., 2002; Ullsperger & Von Cramon, 2006). Contradictory results were found in PD patients. Ito and Kitagawa (2006) reported a reduction in Pe, while another study involving three experiments found no differences in Pe amplitude between PD and controls for Flanker Task and Simon-type task performances, and reduced Pe during a Go/NoGo task (Falkenstein et al., 2005). Findings from cerebellar dysfunction are also contradictory, but an association between Pe amplitude and dystonia severity was found in cerebellar ataxia patients (Tunc et al., 2019), suggesting a relation between motor dysfunction and decrease in Pe.

According to the Evidence Accumulation Account (Steinhauser & Yeung, 2010; Ullsperger et al., 2010), the Pe emerges when sufficient evidence about error commission has been accumulated. This would involve the integration of conflict/response information, proprioception, interoception, and sensory inputs (about action performance). It has been hypothesized that brain structures involved in the emergence of error awareness includes cingulate structures, somatosensory areas and anterior insula (Hester et al., 2005; Klein et al., 2013; Ullsperger et al., 2010). Importantly, peripheral and visceral signals could contribute to the generation of the Pe and such factors must be considered in central and peripheral nervous system pathologies that could affect sensorimotor processing. It could be hypothesized that motor diseases, including those affecting the peripheral nervous system, may suffer from changes in sensorimotor information processing that could contribute to accumulation processing underlying error awareness. The anterior insula, integrating signals ascending from peripheral pathways, plays a key role in

interoceptive awareness (Chen et al., 2021), and the ACC are key nodes of the salience network (Uddin, 2015). In line with Adaptive Orienting theory, the salience network, as well as the frontobasal ganglia network, are involved in post-error processing (Wessel, 2018). Recent evidence on cross-network interactions involved in cognitive control suggests that that salience network may play a crucial role in real-life self-control by initiating switching between default mode and executive networks (Krönke et al., 2020), thus underlining the critical involvement of anterior insula and ACC in self-monitoring and self-regulation.

Nevertheless, error awareness and its relation with the Pe has not been explored in neurological conditions, except for Logan et al. (2015). In their study, they used the Error Awareness Task which allowed to investigate ERPs differences for aware and unaware errors in TBI patients. Although they found no differences between patients and controls, a significant effect of awareness on Pe amplitude in both groups was observed. Such experimental manipulations can be critical in the analyses of the Pe, by potentially revealing differences otherwise undetectable, and exploring the differential association between aware and unaware errors with other measures.

Moreover, we explored whether lesion lateralization was associated with error monitoring system dysfunction; one recent study's sample included only patients with left hemisphere lesion (Niessen et al., 2020), reporting no group differences in either ERN or Pe. In the rest of the studies, the patient group included either both hemispheres lesions or bilateral lesions, and in the first case, no subgroup analyses exploring lateralization effect was conducted. Therefore, considering the heterogeneity of lesion localization and size in the study by Niessen et al. (2020), it is difficult to discuss any potential hemispheric asymmetry of the performance monitoring system.

Importantly, some methodological issues need to be acknowledged when considering the presence of inconsistent findings across and within neurological conditions. The studies reviewed present relevant differences in sample size (ranging from 6 to 36 for the clinical group), experimental manipulations (Fischer et al., 2017; Mathewson et al., 2005; S. E. Morris et al., 2006), and quantification of ERP-related metrics (Overbeek et al., 2005). Moreover, a large part of the studies employed the Flanker task (n=23/41), and the number of studies focusing on

specific neurological disorders is unbalanced, with, for example, wider research on Parkinson's disease (n=11) and TBI (n=8) and limited investigation of multiple sclerosis (n=1), amyotrophic lateral sclerosis (n=1).

The second aim of this review was to investigate the associations between error-related ERPs and clinical factors. Overall, across different neurodegenerative disorders, we can observe that ERN and Pe are associated with disease severity measures (n=14/17 including correlational analysis). Pe amplitude was shown to be associated with dystonia severity in CA patients (Tunc et al., 2019) and both ERN and Pe with cerebellar grey matter volume in CD (Peterburs et al., 2015). In MS patients, ERN correlated with time since last relapse and disease severity measures (Lopez-Gongora et al., 2015). In HD, the ERN correlated with size of CAG repetitions (Beste et al, 2009), which is typically used as a severity index (Duyao et al., 1993; Rosenblatt et al., 2006), and with medial frontal grey matter volume (Beste et al., 2008). This suggests that error monitoring ERPs may represent a reliable measure of neurodegeneration processes.

Given the heterogeneity of neurological profiles, clinical outcomes and recovery trajectories in TBI patients (Azouvi et al., 2017; Bigler, 2001; Chastain et al., 2009; Green et al., 2008; Perlbarg et al., 2009; Rabinowitz et al., 2018), it is cautious to say that we cannot establish whether ERPs alterations are specific for this neurological condition. However, this line of research provided relevant knowledge about the association between clinical factors and error monitoring. Several measures of trauma severity were found to be associated with the ERN across many studies. ERN amplitude and latencies were shown to be associated with higher number of TBIs (De Beaumont et al., 2013; Larson et al., 2007; Pontifex et al., 2009) and Pe amplitude was found to be correlated with post-traumatic amnesia length and time since the injury (Larson et al., 2012). Among others, post-traumatic amnesia is considered a strong predictor of clinical outcomes (Ponsford et al., 2016), thus suggesting that error-related ERPs may not only index injury severity but also predict outcomes after TBI. Importantly, self-awareness impairments are very common in TBI (Prigatano, 2005; Sherer et al., 1998, 2003) and multi-dimensional measures of self-awareness, including error monitoring, play a critical role in TBI interventions (Robertson & Schmitter-Edgecombe, 2015; Simmond & Fleming, 2003). In TBI patients, performance monitoring deficits were

found to be associated with activity of the dorsal ACC and anterior insula (Ham et al., 2014), supporting the relevance of these areas for error processing.

Moreover, the relations between the ERN and depression (Willemssen et al., 2008; Seer, Lange et al., 2017), negative affect (Larson et al., 2009) and psychiatric symptoms (Seer, Lange et al., 2017) point out the critical relevance of error monitoring in clinical profiles of neurological disorders. Extensive research on psychiatric conditions identified impairment of error processing, as reflected by ERN alterations. For example, the ERN has been proposed as endophenotype of internalizing disorders (Olvet & Hajcak, 2008; Weinberg et al., 2015), specifically of obsessive-compulsive disorder (Riesel, 2019), anxiety (Riesel et al., 2019), and as candidate biomarker of depression (Clayson et al., 2020). Reduction of reduction of error-related ERPs have also been reported in psychopathy (Vallet et al., 2021), schizophrenia (Bates et al., 2002; Foti et al., 2012; Simmonite et al., 2012), and bipolar disorder (Minzenberg et al., 2014; Morsel et al., 2014). Further research is needed to extend the knowledge about overlapping neural networks underlying performance monitoring in neurological and psychiatric disorders. For instance, TS and obsessive compulsive disorder (OCD) have been suggested to share pathophysiological mechanisms, possibly reflecting similarities between tics and repetitive behaviours associated with cortico-striato-thalamo-cortical circuitry dysfunction (Hartmann & Millet, 2018). Moreover, OCD is often present as a comorbidity of TS (Sheppard, 1999). Although performance monitoring in these two clinical populations has never been directly compared, previous research showed that the ERN is typically enhanced, suggesting hyperactive error signals in both conditions (Warren et al., 2020). Warren et al. (2020) observed that increased ERN in TS may reflect compensatory mechanisms that allow successful behavioural performance.

As shown by the current review, all the studies (n=3) including TS showed higher ERN amplitude but comparable behavioural performance as compared to healthy controls. A similar phenomenon has been highlighted by Lopez-Góngora et al. (2015) concerning error monitoring in MS. However, this hypothesis is not supported by findings from other neurological disorders, in which no systematic pattern linking ERN to task performance, especially for accuracy and reaction times, can be identified. A limited number of studies within and across neurological

disorders included measures of post-error adjustments, such as post-error slowing (n=16) and post-error accuracy (n=4). Nonetheless, no consistent association between post-error measures and ERPs was found.

In conclusion, these findings highlight the link between error monitoring networks, self-awareness, and neurocognitive rehabilitation outcomes. Error-related ERPs may be employed in assessment protocols to evaluate patient ability to monitor their own functioning and understand the severity of their conditions. Error monitoring ERPs can also be important measures for rehabilitation effectiveness, because error detection and correction can be critical for (re)learning mechanisms (Ownsworth et al., 2017).

Future research should investigate domain-specificity of error monitoring and the role of functional disconnection within performance monitoring networks. This would extend our knowledge on brain processes underlying error monitoring and provide useful information on specific cognitive deficits for neuropsychological assessment and rehabilitation. Furthermore, future studies investigating error monitoring in neurological disorders would benefit from including: 1) both ERN and Pe amplitude and latency analyses; 2) experimental manipulations to distinguish aware and unaware errors in order to explore the relation between Pe, error awareness, and other variables; 3) clinical and neurocognitive measures, and assessment of psychiatric comorbidities; 4) subgroup analyses exploring differences between left and right hemisphere lesion; 5) behavioural measures of post-error adjustments. Finally, combining EEG with non-invasive brain stimulation techniques could offer new perspectives to elucidate the relative contribution of different brain structures in error monitoring and potential tools for error processing and self-awareness rehabilitation.

References

Agnew, S. K., & Morris, R. G. (1998). The heterogeneity of anosognosia for memory impairment in Alzheimer's disease: A review of the literature and a proposed model. Aging & Mental Health, 2(1), 7–19. https://doi.org/10.1080/13607869856876

Alexander, W. H., & Brown, J. W. (2011). Medial prefrontal cortex as an action-outcome predictor. Nature Neuroscience, 14(10), 1338–1344. https://doi.org/10.1038/nn.2921

Amanzio, M., Bartoli, M., Cipriani, G. E., & Palermo, S. (2020). Executive Dysfunction and Reduced Self-Awareness in Patients With Neurological Disorders. A Mini-Review. Frontiers in Psychology, 11. https://doi.org/10.3389/fpsyg.2020.01697

Azouvi, P., Arnould, A., Dromer, E., & Vallat-Azouvi, C. (2017). Neuropsychology of traumatic brain injury: An expert overview. In Revue Neurologique. https://doi.org/10.1016/j.neurol.2017.07.006

Bates, A. T., Kiehl, K. A., Laurens, K. R., & Liddle, P. F. (2002). Error-related negativity and correct response negativity in schizophrenia. Clinical Neurophysiology. https://doi.org/10.1016/S1388-2457(02)00154-2

Bellon, E., Fias, W., & de Smedt, B. (2020). Metacognition across domains: Is the association between arithmetic and metacognitive monitoring domain-specific? PLOS ONE, 15(3), e0229932. https://doi.org/10.1371/journal.pone.0229932

Beste, C., Saft, C., Andrich, J., Gold, R., & Falkenstein, M. (2006). Error processing in Huntington's disease. PLoS ONE, 1(1). https://doi.org/10.1371/journal.pone.0000086

Beste, C., Saft, C., Konrad, C., Andrich, J., Habbel, A., Schepers, I., Jansen, A., Pfleiderer, B., & Falkenstein, M. (2008). Levels of error processing in Huntington's disease: A combined study using event-related potentials and voxel-based morphometry. Human Brain Mapping, 29(2), 121–130. https://doi.org/10.1002/hbm.20374

Beste, C., Saft, C., Yordanova, J., Andrich, J., Gold, R., Falkenstein, M., & Kolev, V. (2007). Functional compensation or pathology in cortico-subcortical interactions

in preclinical Huntington's disease? Neuropsychologia, 45(13), 2922–2930. https://doi.org/10.1016/j.neuropsychologia.2007.06.004

Beste, C., Willemssen, R., Saft, C., & Falkenstein, M. (2009). Error processing in normal aging and in basal ganglia disorders. Neuroscience, 159(1), 143–149. https://doi.org/10.1016/j.neuroscience.2008.12.030

Biehl, S. C., Dresler, T., Reif, A., Scheuerpflug, P., Deckert, J., & Herrmann, M. J. (2011). Dopamine transporter (dat1) and dopamine receptor d4 (drd4) genotypes differentially impact on electrophysiological correlates of error processing. PLoS ONE. https://doi.org/10.1371/journal.pone.0028396

Bigler, E. D. (2001). The lesion(s) in traumatic brain injury: Implications for clinical neuropsychology. Archives of Clinical Neuropsychology. https://doi.org/10.1016/S0887-6177(00)00095-0

Boksem, M. A. S., Meijman, T. F., & Lorist, M. M. (2006). Mental fatigue, motivation and action monitoring. Biological Psychology. https://doi.org/10.1016/j.biopsycho.2005.08.007

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. Psychological Review, 108(3), 624–652. https://doi.org/10.1037/0033-295X.108.3.624

Brázdil, M., Roman, R., Daniel, P., & Rektor, I. (2005). Intracerebral Error-Related Negativity in a Simple Go/NoGo Task. Journal of Psychophysiology, 19(4), 244–255. https://doi.org/10.1027/0269-8803.19.4.244

Chapman, S., Colvin, L. E., Vuorre, M., Cocchini, G., Metcalfe, J., Huey, E. D., & Cosentino, S. (2018). Cross domain self-monitoring in anosognosia for memory loss in Alzheimer's disease. Cortex, 101, 221–233. https://doi.org/10.1016/j.cortex.2018.01.019

Chastain, C. A., Oyoyo, U. E., Zipperman, M., Joo, E., Ashwal, S., Shutter, L. A., & Tong, K. A. (2009). Predicting outcomes of traumatic brain injury by imaging modality and injury distribution. Journal of Neurotrauma. https://doi.org/10.1089/neu.2008.0650

Chavoix, C., & Insausti, R. (2017). Self-awareness and the medial temporal lobe in neurodegenerative diseases. Neuroscience & Biobehavioral Reviews, 78, 1–12. https://doi.org/10.1016/j.neubiorev.2017.04.015

Chen, W. G., Schloesser, D., Arensdorf, A. M., Simmons, J. M., Cui, C., Valentino, R., Gnadt, J. W., Nielsen, L., Hillaire-Clarke, C. S., Spruance, V., Horowitz, T. S., Vallejo, Y. F., & Langevin, H. M. (2021). The Emerging Science of Interoception: Sensing, Integrating, Interpreting, and Regulating Signals within the Self. Trends in Neurosciences, 44(1), 3–16. https://doi.org/10.1016/j.tins.2020.10.007

Clare, L., & Jones, R. S. P. (2008). Errorless Learning in the Rehabilitation of Memory Impairment: A Critical Review. Neuropsychology Review, 18(1), 1–23. https://doi.org/10.1007/s11065-008-9051-4

Clayson, P. E., Carbine, K. A., & Larson, M. J. (2020). A registered report of error-related negativity and reward positivity as biomarkers of depression: P-Curving the evidence. International Journal of Psychophysiology, 150, 50–72. https://doi.org/10.1016/j.ijpsycho.2020.01.005

De Beaumont, L., Beauchemin, M., Beaulieu, C., & Jolicoeur, P. (2013). Long-term attenuated electrophysiological response to errors following multiple sports concussions. Journal of Clinical and Experimental Neuropsychology, 35(6), 596–607. https://doi.org/10.1080/13803395.2013.800023

Debener, S. (2005). Trial-by-Trial Coupling of Concurrent Electroencephalogram and Functional Magnetic Resonance Imaging Identifies the Dynamics of Performance Monitoring. Journal of Neuroscience, 25(50), 11730–11737. https://doi.org/10.1523/JNEUROSCI.3286-05.2005

Dehaene, S. (2018). The Error-Related Negativity, Self-Monitoring, and Consciousness. Perspectives on Psychological Science, 13(2), 161–165. https://doi.org/10.1177/1745691618754502

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a Neural System for Error Detection and Compensation. Psychological Science, 5(5), 303–305. https://doi.org/10.1111/j.1467-9280.1994.tb00630.x

Dentakos, S., Saoud, W., Ackerman, R., & Toplak, M. E. (2019). Does domain matter? Monitoring accuracy across domains. Metacognition and Learning, 14(3), 413–436. https://doi.org/10.1007/s11409-019-09198-4

di Gregorio, F., Maier, M. E., & Steinhauser, M. (2018). Errors can elicit an error positivity in the absence of an error negativity: Evidence for independent systems of human error monitoring. NeuroImage, 172(September 2017), 427–436. https://doi.org/10.1016/j.neuroimage.2018.01.081

Dockree, P. M., Tarleton, Y. M., Carton, S., & FitzGerald, M. C. C. (2015). Connecting Self-Awareness and Error-Awareness in Patients with Traumatic Brain Injury. Journal of the International Neuropsychological Society, 21(7), 473–482. https://doi.org/10.1017/S1355617715000594

Donchin, E., & Coles, M. G. H. (1988). Is the P300 component a manifestation of context updating? Behavioral and Brain Sciences, 11(03), 357. https://doi.org/10.1017/S0140525X00058027

Dunn, J., & Clare, L. (2007). Learning face–name associations in early-stage dementia: Comparing the effects of errorless learning and effortful processing. Neuropsychological Rehabilitation, 17(6), 735–754. https://doi.org/10.1080/09602010701218317

Duyao, M., Ambrose, C., Myers, R., Novelletto, A., Persichetti, F., Frontali, M., Folstein, S., Ross, C., Franz, M., Abbott, M., Gray, J., Conneally, P., Young, A., Penney, J., Hollingsworth, Z., Shoulson, I., Lazzarini, A., Falek, A., Koroshetz, W., … Macdonald, M. (1993). Trinucleotide repeat length instability and age of onset in Huntington's disease. Nature Genetics. https://doi.org/10.1038/ng0893-387

Ehlhardt, L. A., Sohlberg, M. M., Kennedy, M., Coelho, C., Ylvisaker, M., Turkstra, L., & Yorkston, K. (2008). Evidence-based practice guidelines for instructing individuals with neurogenic memory impairments: What have we learned in the past 20 years? Neuropsychological Rehabilitation, 18(3), 300–342. https://doi.org/10.1080/09602010701733190

Endrass, T., Reuter, B., & Kathmann, N. (2007). ERP correlates of conscious error recognition: Aware and unaware errors in an antisaccade task. European Journal of Neuroscience, 26(6), 1714–1720. https://doi.org/10.1111/j.1460-9568.2007.05785.x

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. Perception & Psychophysics, 16(1), 143–149. https://doi.org/10.3758/BF03203267

Falkenstein, M., Hielscher, H., Dziobek, I., Schwarzenau, P., Hoormann, J., Sundermann, B., & Hohnsbein, J. (2001). Action monitoring, error detection, and the basal ganglia: An ERP study. NeuroReport, 12(1), 157–161. https://doi.org/10.1097/00001756-200101220-00039

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. Electroencephalography and Clinical Neurophysiology, 78(6), 447–455. https://doi.org/10.1016/0013-4694(91)90062-9

Falkenstein, M., Willemssen, R., Hohnsbein, J., & Hielscher, H. (2005). Error processing in Parkinson's disease: The error positivity (Pe). Journal of Psychophysiology. https://doi.org/10.1027/0269-8803.19.4.305

Fischer, A. G., Klein, T. A., & Ullsperger, M. (2017). Comparing the error-related negativity across groups: The impact of error- and trial-number differences. Psychophysiology, 54(7), 998–1009. https://doi.org/10.1111/psyp.12863

Forster, S. E., Zirnheld, P., Shekhar, A., Steinhauer, S. R., O'Donnell, B. F., & Hetrick, W. P. (2017). Event-related potentials reflect impaired temporal interval learning following haloperidol administration. Psychopharmacology, 234(17), 2545–2562. https://doi.org/10.1007/s00213-017-4645-2

Foti, D., Kotov, R., Bromet, E., & Hajcak, G. (2012). Beyond the Broken Error-Related Negativity: Functional and Diagnostic Correlates of Error Processing in Psychosis. Biological Psychiatry, 71(10), 864–872. https://doi.org/10.1016/j.biopsych.2012.01.007

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A Neural System for Error Detection and Compensation. Psychological Science, 4(6), 385–390. https://doi.org/10.1111/j.1467-9280.1993.tb00586.x

Gehring, W. J., & Knight, R. T. (2000). Prefrontal-cingulate interactions in action monitoring. Nature Neuroscience. https://doi.org/10.1038/74899

Green, R. E., Colella, B., Christensen, B., Johns, K., Frasca, D., Bayley, M., & Monette, G. (2008). Examining Moderators of Cognitive Recovery Trajectories After Moderate to Severe Traumatic Brain Injury. Archives of Physical Medicine and Rehabilitation. https://doi.org/10.1016/j.apmr.2008.09.551

Hallett, P. E. (1978). Primary and secondary saccades to goals defined by instructions. Vision Research, 18(10), 1279–1296. https://doi.org/10.1016/0042-6989(78)90218-3

Ham, T. E., Bonnelle, V., Hellyer, P., Jilka, S., Robertson, I. H., Leech, R., & Sharp, D. J. (2014). The neural basis of impaired self-awareness after traumatic brain injury. Brain. https://doi.org/10.1093/brain/awt350

Harsay, H. A., Spaan, M., Wijnen, J. G., & Ridderinkhof, K. R. (2012). Error Awareness and Salience Processing in the Oddball Task: Shared Neural Mechanisms. Frontiers in Human Neuroscience, 6. https://doi.org/10.3389/fnhum.2012.00246

Hartmann, A., & Millet, B. (2018). Repetitive movements and behaviors in neurological and psychiatric practice: Distinctions and similarities between Tourette disorder and obsessive–compulsive disorder. Revue Neurologique, 174(4), 199–202. https://doi.org/10.1016/j.neurol.2018.01.364

Haslam, C., & Kessels, R. P. C. (2018). Errorless Learning in Neuropsychological Rehabilitation. In C. Haslam & R. P. C. Kessels (Eds.), Errorless Learning in Neuropsychological Rehabilitation: Mechanisms, Efficacy and Application. Routledge. https://doi.org/10.4324/9781315660738

Hester, R., Foxe, J. J., Molholm, S., Shpaner, M., & Garavan, H. (2005). Neural mechanisms involved in error processing: A comparison of errors made with and without awareness. NeuroImage, 27(3), 602–608. https://doi.org/10.1016/j.neuroimage.2005.04.035

Higgins, J., Thomas, J., Chandler, J., Cumpston, M., Li, T., Page, M., & Welch, V. (2020). Cochrane Handbook for systematic reviews of interventions version 6.1 (updated September 2020) (Cochrane, Ed.).

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity.

Psychological Review, 109(4), 679–709. https://doi.org/10.1037/0033-295X.109.4.679

Holroyd, C. B., Praamstra, P., Plat, E., & Coles, M. G. H. (2002). Spared error-related potentials in mild to moderate Parkinson's disease. Neuropsychologia, 40(12), 2116–2124. https://doi.org/10.1016/S0028-3932(02)00052-0

Hu, N., Hu, X., Xu, Z., Li, Q., Long, Q., Gu, Y., & Chen, A. (2019). Temporal dynamic modulation of acute stress on error processing in healthy males. Psychophysiology. https://doi.org/10.1111/psyp.13398

Ito, J., & Kitagawa, J. (2005). Error processing in patients with Alzheimer's disease. Pathophysiology, 12(2), 97–101. https://doi.org/10.1016/j.pathophys.2005.02.003

Ito, J., & Kitagawa, J. (2006). Performance Monitoring and Error Processing During a Lexical Decision Task in Patients With Parkinson's Disease. Journal of Geriatric Psychiatry and Neurology, 19(1), 46–54. https://doi.org/10.1177/0891988705284716

Johannes, S., Wieringa, B. M., Nager, W., Müller-Vahl, K. R., Dengler, R., & Münte, T. F. (2002). Excessive action monitoring in Tourette syndrome. Journal of Neurology, 249(8), 961–966. https://doi.org/10.1007/s00415-002-0657-9

Kelley, E., Sullivan, C., Loughlin, J. K., Hutson, L., Dahdah, M. N., Long, M. K., Schwab, K. A., & Poole, J. H. (2014). Self-Awareness and Neurobehavioral Outcomes, 5 Years or More After Moderate to Severe Brain Injury. Journal of Head Trauma Rehabilitation, 29(2), 147–152. https://doi.org/10.1097/HTR.0b013e31826db6b9

Klein, T. A., Ullsperger, M., & Danielmeier, C. (2013). Error awareness and the insula: links to neurological and psychiatric diseases. Frontiers in Human Neuroscience, 7. https://doi.org/10.3389/fnhum.2013.00014

Krämer, U. M., Cunillera, T., Càmara, E., Marco-Pallarés, J., Cucurell, D., Nager, W., Bauer, P., Schüle, R., Schöls, L., Rodriguez-Fornells, A., & Münte, T. F. (2007). The impact of catechol-O-methyltransferase and dopamine D4 receptor genotypes on neurophysiological markers of performance monitoring. Journal of Neuroscience. https://doi.org/10.1523/JNEUROSCI.4229-07.2007

Krönke, K.-M., Wolff, M., Mohr, H., Kräplin, A., Smolka, M. N., Bühringer, G., & Goschke, T. (2018). Monitor yourself! Deficient error-related brain activity predicts real-life self-control failures. Cognitive, Affective, & Behavioral Neuroscience, 18(4), 622–637. https://doi.org/10.3758/s13415-018-0593-5

Krönke, K.-M., Wolff, M., Shi, Y., Kräplin, A., Smolka, M. N., Bühringer, G., & Goschke, T. (2020). Functional connectivity in a triple-network saliency model is associated with real-life self-control. Neuropsychologia, 149, 107667. https://doi.org/10.1016/j.neuropsychologia.2020.107667

Larson, M. J., Clayson, P. E., & Farrer, T. J. (2012). Performance monitoring and cognitive control in individuals with mild traumatic brain injury. Journal of the International Neuropsychological Society, 18(2), 323–333. https://doi.org/10.1017/S1355617711001779

Larson, M. J., Kaufman, D. A. S., Kellison, I. L., Schmalfuss, I. M., & Perlstein, W. M. (2009). Double Jeopardy! The Additive Consequences of Negative Affect on Performance-Monitoring Decrements Following Traumatic Brain Injury. Neuropsychology, 23(4), 433–444. https://doi.org/10.1037/a0015723

Larson, M. J., Kaufman, D. A. S., Schmalfuss, I. M., & Perlstein, W. M. (2007). Performance monitoring, error processing, and evaluative control following severe TBI. Journal of the International Neuropsychological Society, 13(6), 961–971. https://doi.org/10.1017/S1355617707071305

Lenzoni, S., Morris, R. G., & Mograbi, D. C. (2020). The Petrified Self 10 Years After: Current Evidence for Mnemonic anosognosia. Frontiers in Psychology, 11. https://doi.org/10.3389/fpsyg.2020.00465

Leung, D. P. K., & Liu, K. P. Y. (2011). Review of self-awareness and its clinical application in stroke rehabilitation. International Journal of Rehabilitation Research, 34(3), 187–195. https://doi.org/10.1097/MRR.0b013e3283487f31

Logan, D. M., Hill, K. R., & Larson, M. J. (2015). Cognitive control of conscious error awareness: error awareness and error positivity (Pe) amplitude in moderate-to-severe traumatic brain injury (TBI). Frontiers in Human Neuroscience, 9. https://doi.org/10.3389/fnhum.2015.00397

López-Góngora, M., Escartín, A., Martínez-Horta, S., Fernández-Bobadilla, R., Querol, L., Romero, S., Mañanas, M. À., & Riba, J. (2015). Neurophysiological Evidence of Compensatory Brain Mechanisms in Early-Stage Multiple Sclerosis. PLOS ONE, 10(8), e0136786. https://doi.org/10.1371/journal.pone.0136786

Maier, M. E., Di Gregorio, F., Muricchio, T., & Di Pellegrino, G. (2015). Impaired rapid error monitoring but intact error signaling following rostral anterior cingulate cortex lesions in humans. Frontiers in Human Neuroscience, 9(June). https://doi.org/10.3389/fnhum.2015.00339

Mathalon, D. H., Fedor, M., Faustman, W. O., Gray, M., Askari, N., & Ford, J. M. (2002). Response-monitoring dysfunction in schizophrenia: An event-related brain potential study. Journal of Abnormal Psychology, 111(1), 22–41. https://doi.org/10.1037/0021-843X.111.1.22

Mathewson, K. J., Dywan, J., & Segalowitz, S. J. (2005). Brain bases of error-related ERPs as influenced by age and task. Biological Psychology, 70(2), 88–104. https://doi.org/10.1016/j.biopsycho.2004.12.005

Mazancieux, A., Souchay, C., Casez, O., & Moulin, C. J. A. (2019). Metacognition and self-awareness in Multiple Sclerosis. Cortex, 111, 238–255. https://doi.org/10.1016/j.cortex.2018.11.012

Medley, A. R., & Powell, T. (2010). Motivational Interviewing to promote self-awareness and engagement in rehabilitation following acquired brain injury: A conceptual review. Neuropsychological Rehabilitation, 20(4), 481–508. https://doi.org/10.1080/09602010903529610

Meyer, A. (2016). Developing Psychiatric Biomarkers: a Review Focusing on the Error-Related Negativity as a Biomarker for Anxiety. Current Treatment Options in Psychiatry, 3(4), 356–364. https://doi.org/10.1007/s40501-016-0094-5

Minzenberg, M. J., Gomes, G. C., Yoon, J. H., Swaab, T. Y., & Carter, C. S. (2014). Disrupted action monitoring in recent-onset psychosis patients with schizophrenia and bipolar disorder. Psychiatry Research: Neuroimaging, 221(1), 114–121. https://doi.org/10.1016/j.pscychresns.2013.11.003

Mograbi, D. C., & Morris, R. G. (2014). The developing concept of implicit awareness: A rejoinder and reply to commentaries on Mograbi and Morris (2013).

Cognitive Neuroscience, 5(3–4), 138–142. https://doi.org/10.1080/17588928.2014.905522

Mograbi, D. C., & Morris, R. G. (2018). Anosognosia. Cortex, 103, 385–386. https://doi.org/10.1016/j.cortex.2018.04.001

Moher, D., Liberati, A., Tetzlaff, J., & Altman, D. G. (2009). Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. BMJ, 339(jul21 1), b2535–b2535. https://doi.org/10.1136/bmj.b2535

Morales, J., Lau, H., & Fleming, S. M. (2018). Domain-general and domain-specific patterns of activity supporting metacognition in human prefrontal cortex. Journal of Neuroscience, 38(14), 3534–3546. https://doi.org/10.1523/JNEUROSCI.2360-17.2018

Morris, R. G., & Mograbi, D. C. (2013). Anosognosia, autobiographical memory and self knowledge in Alzheimer's disease. Cortex, 49(6), 1553–1565. https://doi.org/10.1016/j.cortex.2012.09.006

Morris, S. E., Yee, C. M., & Nuechterlein, K. H. (2006). Electrophysiological analysis of error monitoring in schizophrenia. Journal of Abnormal Psychology, 115(2), 239–250. https://doi.org/10.1037/0021-843X.115.2.239

Morsel, A. M., Morrens, M., Temmerman, A., Sabbe, B., & de Bruijn, E. R. (2014). Electrophysiological (EEG) evidence for reduced performance monitoring in euthymic bipolar disorder. Bipolar Disorders, 16(8), 820–829. https://doi.org/10.1111/bdi.12256

Murphy, P. R., Robertson, I. H., Allen, D., Hester, R., & O'Connell, R. G. (2012). An electrophysiological signal that precisely tracks the emergence of error awareness. Frontiers in Human Neuroscience, 6(MARCH 2012), 1–16. https://doi.org/10.3389/fnhum.2012.00065

Niessen, E., Ant, J. M., Bode, S., Saliger, J., Karbe, H., Fink, G. R., Stahl, J., & Weiss, P. H. (2020). Preserved performance monitoring and error detection in left hemisphere stroke. NeuroImage: Clinical. https://doi.org/10.1016/j.nicl.2020.102307

Nieuwenhuis, S., Aston-Jones, G., & Cohen, J. D. (2005). Decision making, the P3, and the locus coeruleus--norepinephrine system. Psychological Bulletin, 131(4), 510–532. https://doi.org/10.1037/0033-2909.131.4.510

Nieuwenhuis, S., Richard Ridderinkhof, K., Blom, J., Band, G. P. H., & Kok, A. (2001). Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. Psychophysiology, 38(5), 752–760. https://doi.org/10.1017/S0048577201001111

Notebaert, W., Houtman, F., Opstal, F. Van, Gevers, W., Fias, W., & Verguts, T. (2009). Post-error slowing: An orienting account. Cognition, 111(2), 275–279. https://doi.org/10.1016/j.cognition.2009.02.002

Olson, R. L., Brush, C. J., Ehmann, P. J., Buckman, J. F., & Alderman, B. L. (2018). A history of sport-related concussion is associated with sustained deficits in conflict and error monitoring. International Journal of Psychophysiology, 132, 145–154. https://doi.org/10.1016/j.ijpsycho.2018.01.006

Olvet, D. M., & Hajcak, G. (2008). The error-related negativity (ERN) and psychopathology: Toward an endophenotype. In Clinical Psychology Review (Vol. 28, Issue 8, pp. 1343–1354). https://doi.org/10.1016/j.cpr.2008.07.003

Overbeek, T. J. M., Nieuwenhuis, S., & Ridderinkhof, K. R. (2005). Dissociable components of error processing: On the functional significance of the Pe vis-à-vis the ERN/Ne. In Journal of Psychophysiology (Vol. 19, Issue 4, pp. 319–329). https://doi.org/10.1027/0269-8803.19.4.319

Overmeyer, R., Berghäuser, J., Dieterich, R., Wolff, M., Goschke, T., & Endrass, T. (2021). The Error-Related Negativity Predicts Self-Control Failures in Daily Life. Frontiers in Human Neuroscience, 14. https://doi.org/10.3389/fnhum.2020.614979

Ownsworth, T., & Clare, L. (2006). The association between awareness deficits and rehabilitation outcome following acquired brain injury. Clinical Psychology Review, 26(6), 783–795. https://doi.org/10.1016/j.cpr.2006.05.003

Ownsworth, T., Fleming, J., Tate, R., Beadle, E., Griffin, J., Kendall, M., Schmidt, J., Lane-Brown, A., Chevignard, M., & Shum, D. H. K. (2017). Do People With Severe Traumatic Brain Injury Benefit From Making Errors? A Randomized

Controlled Trial of Error-Based and Errorless Learning. Neurorehabilitation and Neural Repair, 31(12), 1072–1082. https://doi.org/10.1177/1545968317740635

Perlbarg, V., Puybasset, L., Tollard, E., Lehéricy, S., Benali, H., & Galanaud, D. (2009). Relation between brain lesion location and clinical outcome in patients with severe traumatic brain injury: A diffusion tensor imaging study using voxel-based approaches. Human Brain Mapping. https://doi.org/10.1002/hbm.20817

Peterburs, J., Gajda, K., Koch, B., Schwarz, M., Hoffmann, K. P., Daum, I., & Bellebaum, C. (2012). Cerebellar lesions alter performance monitoring on the antisaccade task-An event-related potentials study. Neuropsychologia, 50(3), 379–389. https://doi.org/10.1016/j.neuropsychologia.2011.12.009

Peterburs, J., Pergola, G., Koch, B., Schwarz, M., Hoffmann, K. P., Daum, I., & Bellebaum, C. (2011). Altered error processing following vascular thalamic damage: Evidence from an antisaccade task. PLoS ONE, 6(6). https://doi.org/10.1371/journal.pone.0021517

Peterburs, J., Thürling, M., Rustemeier, M., Göricke, S., Suchan, B., Timmann, D., & Bellebaum, C. (2015). A cerebellar role in performance monitoring – Evidence from EEG and voxel-based morphometry in patients with cerebellar degenerative disease. Neuropsychologia, 68, 139–147. https://doi.org/10.1016/j.neuropsychologia.2015.01.017

Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. Clinical Neurophysiology, 118(10), 2128–2148. https://doi.org/10.1016/j.clinph.2007.04.019

Ponsford, J. L., Spitz, G., & Mckenzie, D. (2016). Using post-traumatic amnesia to predict outcome after traumatic brain injury. Journal of Neurotrauma. https://doi.org/10.1089/neu.2015.4025

Pontifex, M. B., O'Connor, P. M., Broglio, S. P., & Hillman, C. H. (2009). The association between mild traumatic brain injury history and cognitive control. Neuropsychologia, 47(14), 3210–3216. https://doi.org/10.1016/j.neuropsychologia.2009.07.021

Prigatano, G. P. (2005). Impaired self-awareness after moderately severe to severe traumatic brain injury. In Acta Neurochirurgica, Supplementum. https://doi.org/10.1007/3-211-27577-0_5

Prigatano, G. P., & Sherer, M. (2020). Impaired Self-Awareness and Denial During the Postacute Phases After Moderate to Severe Traumatic Brain Injury. Frontiers in Psychology, 11. https://doi.org/10.3389/fpsyg.2020.01569

Rabinowitz, A. R., Hart, T., Whyte, J., & Kim, J. (2018). Neuropsychological Recovery Trajectories in Moderate to Severe Traumatic Brain Injury: Influence of Patient Characteristics and Diffuse Axonal Injury. Journal of the International Neuropsychological Society. https://doi.org/10.1017/S1355617717000996

Reinhart, R. M. G., & Woodman, G. F. (2014). Causal Control of Medial-Frontal Cortex Governs Electrophysiological and Behavioral Indices of Performance Monitoring and Learning. Journal of Neuroscience, 34(12), 4214–4227. https://doi.org/10.1523/JNEUROSCI.5421-13.2014

Ridderinkhof, K. R., Ramautar, J. R., & Wijnen, J. G. (2009). To P E or not to P E : A P3-like ERP component reflecting the processing of response errors. Psychophysiology, 46(3), 531–538. https://doi.org/10.1111/j.1469-8986.2009.00790.x

Riesel, A. (2019). The erring brain: Error-related negativity as an endophenotype for OCD-A review and meta-analysis. Psychophysiology, 56(4), e13348. https://doi.org/10.1111/psyp.13348

Riesel, A., Klawohn, J., Grützmann, R., Kaufmann, C., Heinzel, S., Bey, K., Lennertz, L., Wagner, M., & Kathmann, N. (2019). Error-related brain activity as a transdiagnostic endophenotype for obsessive-compulsive disorder, anxiety and substance use disorder. Psychological Medicine, 49(7), 1207–1217. https://doi.org/10.1017/S0033291719000199

Riesel, A., Richter, A., Kaufmann, C., Kathmann, N., & Endrass, T. (2015). Performance monitoring in obsessive-compulsive undergraduates: Effects of task difficulty. Brain and Cognition, 98, 35–42. https://doi.org/10.1016/j.bandc.2015.05.002

Robertson, K., & Schmitter-Edgecombe, M. (2015). Self-awareness and traumatic brain injury outcome. Brain Injury, 29(7–8), 848–858. https://doi.org/10.3109/02699052.2015.1005135

Rosenberg, P. B., Nowrangi, M. A., & Lyketsos, C. G. (2015). Neuropsychiatric symptoms in Alzheimer's disease: What might be associated brain circuits? Molecular Aspects of Medicine, 43–44, 25–37. https://doi.org/10.1016/j.mam.2015.05.005

Rosenblatt, A., Liang, K. Y., Zhou, H., Abbott, M. H., Gourley, L. M., Margolis, R. L., Brandt, J., & Ross, C. A. (2006). The association of CAG repeat length with clinical progression in Huntington disease. Neurology. https://doi.org/10.1212/01.wnl.0000204230.16619.d9

Rubenstein, H., Lewis, S. S., & Rubenstein, M. A. (1971). Homographic entries in the internal lexicon: Effects of systematicity and relative frequency of meanings. Journal of Verbal Learning and Verbal Behavior, 10(1), 57–62. https://doi.org/10.1016/S0022-5371(71)80094-4

Rustamov, N., Rodriguez-Raecke, R., Timm, L., Agrawal, D., Dressler, D., Schrader, C., Tacik, P., Wegner, F., Dengler, R., Wittfoth, M., & Kopp, B. (2014). Attention shifting in Parkinson's disease: An analysis of behavioral and cortical responses. Neuropsychology, 28(6), 929–944. https://doi.org/10.1037/neu0000099

Scheffers, M. K., & Coles, M. G. H. (2000). Scheffers MK , Coles MG . Performance monitoring in a confusing world : error related brain activity , judgements of response ... Performance Monitoring in a Confusing ' World : and Types of Errors. Journal of Experimental Psychology: Human Perception and Performance, 26(1), 141–151. https://doi.org/10.1037/0096-1523.26.1.141

Schüller, T., Gruendler, T. O. J., Huster, R., Baldermann, J. C., Huys, D., Ullsperger, M., & Kuhn, J. (2018). Altered electrophysiological correlates of motor inhibition and performance monitoring in Tourette's syndrome. Clinical Neurophysiology, 129(9), 1866–1872. https://doi.org/10.1016/j.clinph.2018.06.002

Seer, C., Joop, M., Lange, F., Lange, C., Dengler, R., Petri, S., & Kopp, B. (2017). Attenuated error-related potentials in amyotrophic lateral sclerosis with executive

dysfunctions. Clinical Neurophysiology, 128(8), 1496–1503. https://doi.org/10.1016/j.clinph.2017.05.007

Seer, C., Lange, F., Loens, S., Wegner, F., Schrader, C., Dressler, D., Dengler, R., & Kopp, B. (2017). Dopaminergic modulation of performance monitoring in Parkinson's disease: An event-related potential study. Scientific Reports, 7. https://doi.org/10.1038/srep41222

Seifert, S., Von Cramon, D. Y., Imperati, D., Tittgemeyer, M., & Ullsperger, M. (2011). Thalamocingulate interactions in performance monitoring. Journal of Neuroscience, 31(9), 3375–3383. https://doi.org/10.1523/JNEUROSCI.6242-10.2011

Shen, I. H., Lin, Y. J., Chen, C. L., & Liao, C. C. (2020). Neural correlates of response inhibition and error processing in individuals with mild traumatic brain injury: An event-related potential study. Journal of Neurotrauma, 37(1), 115–124. https://doi.org/10.1089/neu.2018.6122

Sheppard, D. (1999). Tourette's and comorbid syndromes Obsessive compulsive and attention deficit hyperactivity disorder. a common etiology? Clinical Psychology Review, 19(5), 531–552. https://doi.org/10.1016/S0272-7358(98)00059-2

Sherer, M., Bergloff, P., Levin, E., High, W. M., Oden, K. E., & Nick, T. G. (1998). Impaired Awareness and Employment Outcome after Traumatic Brain Injury. Journal of Head Trauma Rehabilitation, 13(5), 52–61. https://doi.org/10.1097/00001199-199810000-00007

Sherer, M., Hart, T., Nick, T. G., Whyte, J., Thompson, R. N., & Yablon, S. A. (2003). Early impaired self-awareness after traumatic brain injury. Archives of Physical Medicine and Rehabilitation, 84(2), 168–176. https://doi.org/10.1053/apmr.2003.50045

Simmond, M., & Fleming, J. M. (2003). Occupational therapy assessment of self-awareness following traumatic brain injury. In British Journal of Occupational Therapy. https://doi.org/10.1177/030802260306601003

Simmonite, M., Bates, A. T., Groom, M. J., Jackson, G. M., Hollis, C., & Liddle, P. F. (2012). Error processing-associated event-related potentials in schizophrenia

and unaffected siblings. International Journal of Psychophysiology, 84(1), 74–79. https://doi.org/10.1016/j.ijpsycho.2012.01.012

Solbakk, A. K., Funderud, I., Løvstad, M., Endestad, T., Meling, T., Lindgren, M., Knight, R. T., & Krämer, U. M. (2014). Impact of orbitofrontal lesions on electrophysiological signals in a stop signal task. Journal of Cognitive Neuroscience, 26(7), 1528–1545. https://doi.org/10.1162/jocn_a_00561

Sommer, M. A. (2003). The role of the thalamus in motor control. In Current Opinion in Neurobiology. https://doi.org/10.1016/j.conb.2003.10.014

Steinhauser, M., & Andersen, S. K. (2019). Rapid adaptive adjustments of selective attention following errors revealed by the time course of steady-state visual evoked potentials. NeuroImage, 186, 83–92. https://doi.org/10.1016/j.neuroimage.2018.10.059

Steinhauser, M., & Yeung, N. (2010). Decision processes in human performance monitoring. Journal of Neuroscience, 30(46), 15643–15653. https://doi.org/10.1523/JNEUROSCI.1899-10.2010

Steinhauser, M., & Yeung, N. (2012). Error awareness as evidence accumulation: effects of speed-accuracy trade-off on error signaling. Frontiers in Human Neuroscience, 6. https://doi.org/10.3389/fnhum.2012.00240

Stemmer, B., Segalowitz, S. J., Dywan, J., Panisset, M., & Melmed, C. (2007). The error negativity in nonmedicated and medicated patients with Parkinson's disease. Clinical Neurophysiology, 118(6), 1223–1229. https://doi.org/10.1016/j.clinph.2007.02.019

Toglia, J. P. (2011). The dynamic interactional model of cognition in cognitive rehabilitation. In Cognition, occupation, and participation across the life span: Neuroscience, neurorehabilitation, and models of intervention in occupational therapy (3rd ed.).

Trahan, E., Pépin, M., & Hopps, S. (2006). Impaired Awareness of Deficits and Treatment Adherence Among People With Traumatic Brain Injury or Spinal Cord Injury. Journal of Head Trauma Rehabilitation, 21(3), 226–235. https://doi.org/10.1097/00001199-200605000-00003

Tunc, S., Baginski, N., Lubs, J., Bally, J. F., Weissbach, A., Baaske, M. K., Tadic, V., Brüggemann, N., Bäumer, T., Beste, C., & Münchau, A. (2019). Predictive coding and adaptive behavior in patients with genetically determined cerebellar ataxia—A neurophysiology study. NeuroImage: Clinical, 24(November), 102043. https://doi.org/10.1016/j.nicl.2019.102043

Uddin, L. Q. (2015). Salience processing and insular cortical function and dysfunction. Nature Reviews Neuroscience, 16(1), 55–61. https://doi.org/10.1038/nrn3857

Ullsperger, M., Danielmeier, C., & Jocham, G. (2014). Neurophysiology of performance monitoring and adaptive behavior. Physiological Reviews, 94(1), 35–79. https://doi.org/10.1152/physrev.00041.2012

Ullsperger, M., Harsay, H. A., Wessel, J. R., & Ridderinkhof, K. R. (2010). Conscious perception of errors and its relation to the anterior insula. Brain Structure and Function, 214(5–6), 629–643. https://doi.org/10.1007/s00429-010-0261-1

Ullsperger, M., & von Cramon, D. Y. (2006). The Role of Intact Frontostriatal Circuits in Error Processing. Journal of Cognitive Neuroscience, 18(4), 651–664. https://doi.org/10.1162/jocn.2006.18.4.651

Ullsperger, M., von Cramon, D. Y., & Müller, N. G. (2002). Interactions of focal cortical lesions with error processing: Evidence from event-related brain potentials. Neuropsychology, 16(4), 548–561. https://doi.org/10.1037/0894-4105.16.4.548

Vallet, W., Neige, C., Mouchet-Mages, S., Brunelin, J., & Grondin, S. (2021). Response-locked component of error monitoring in psychopathy: A systematic review and meta-analysis of error-related negativity/positivity. In Neuroscience and Biobehavioral Reviews. https://doi.org/10.1016/j.neubiorev.2021.01.004

van Veen, V., & Carter, C. S. (2002). The tinning of action-monitoring processes in the anterior cingulate cortex. Journal of Cognitive Neuroscience, 14(4), 593–602. https://doi.org/10.1162/08989290260045837

Verleger, R., Schroll, H., & Hamker, F. H. (2013). The unstable bridge from stimulus processing to correct responding in Parkinson's disease. Neuropsychologia, 51(13), 2512–2525. https://doi.org/10.1016/j.neuropsychologia.2013.09.017

Warren, C., Seer, C., Lange, F., Kopp, B., & Müller-Vahl, K. (2020). Neural correlates of performance monitoring in adult patients with Gilles de la Tourette syndrome: A study of event-related potentials. Clinical Neurophysiology, 131(3), 597–608. https://doi.org/10.1016/j.clinph.2019.11.019

Weinberg, A., Dieterich, R., & Riesel, A. (2015). Error-related brain activity in the age of RDoC: A review of the literature. International Journal of Psychophysiology, 98(2), 276–299. https://doi.org/10.1016/j.ijpsycho.2015.02.029

Wessel, J. R. (2018). An adaptive orienting theory of error processing. Psychophysiology, 55(3), e13041. https://doi.org/10.1111/psyp.13041

Wessel, J. R., Danielmeier, C., & Ullsperger, M. (2011). Error awareness revisited: Accumulation of multimodal evidence from central and autonomic nervous systems. Journal of Cognitive Neuroscience, 23(10), 3021–3036. https://doi.org/10.1162/jocn.2011.21635

Wessel, J. R., Klein, T. A., Ott, D. V. M., & Ullsperger, M. (2014). Lesions to the prefrontal performance-monitoring network disrupt neural processing and adaptive behaviors after both errors and novelty. Cortex, 50, 45–54. https://doi.org/10.1016/j.cortex.2013.09.002

Willemssen, R., Müller, T., Schwarz, M., Falkenstein, M., & Beste, C. (2009). Response Monitoring in De Novo Patients with Parkinson's Disease. PLoS ONE, 4(3), e4898. https://doi.org/10.1371/journal.pone.0004898

Willemssen, R., Müller, T., Schwarz, M., Hohnsbein, J., & Falkenstein, M. (2008). Error processing in patients with Parkinson's disease: The influence of medication state. Journal of Neural Transmission, 115(3), 461–468. https://doi.org/10.1007/s00702-007-0842-1

Wingfield, A. (1968). Effects of Frequency on Identification and Naming of Objects. The American Journal of Psychology, 81(2), 226. https://doi.org/10.2307/1421267

Wiswede, D., Münte, T. F., Goschke, T., & Rüsseler, J. (2009). Modulation of the error-related negativity by induction of short-term negative affect. Neuropsychologia, 47(1), 83–90. https://doi.org/10.1016/j.neuropsychologia.2008.08.016

Wolff, M., & Vann, S. D. (2019). The cognitive thalamus as a gateway to mental representations. Journal of Neuroscience. https://doi.org/10.1523/JNEUROSCI.0479-18.2018

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. In Psychological Review (Vol. 111, Issue 4, pp. 931–959). https://doi.org/10.1037/0033-295X.111.4.931

Zirnheld, P. J., Carroll, C. A., Kieffaber, P. D., O'Donnell, B. F., Shekhar, A., & Hetrick, W. P. (2004). Haloperidol impairs learning and error-related negativity in humans. Journal of Cognitive Neuroscience, 16(6), 1098–1112. https://doi.org/10.1162/0898929041502779

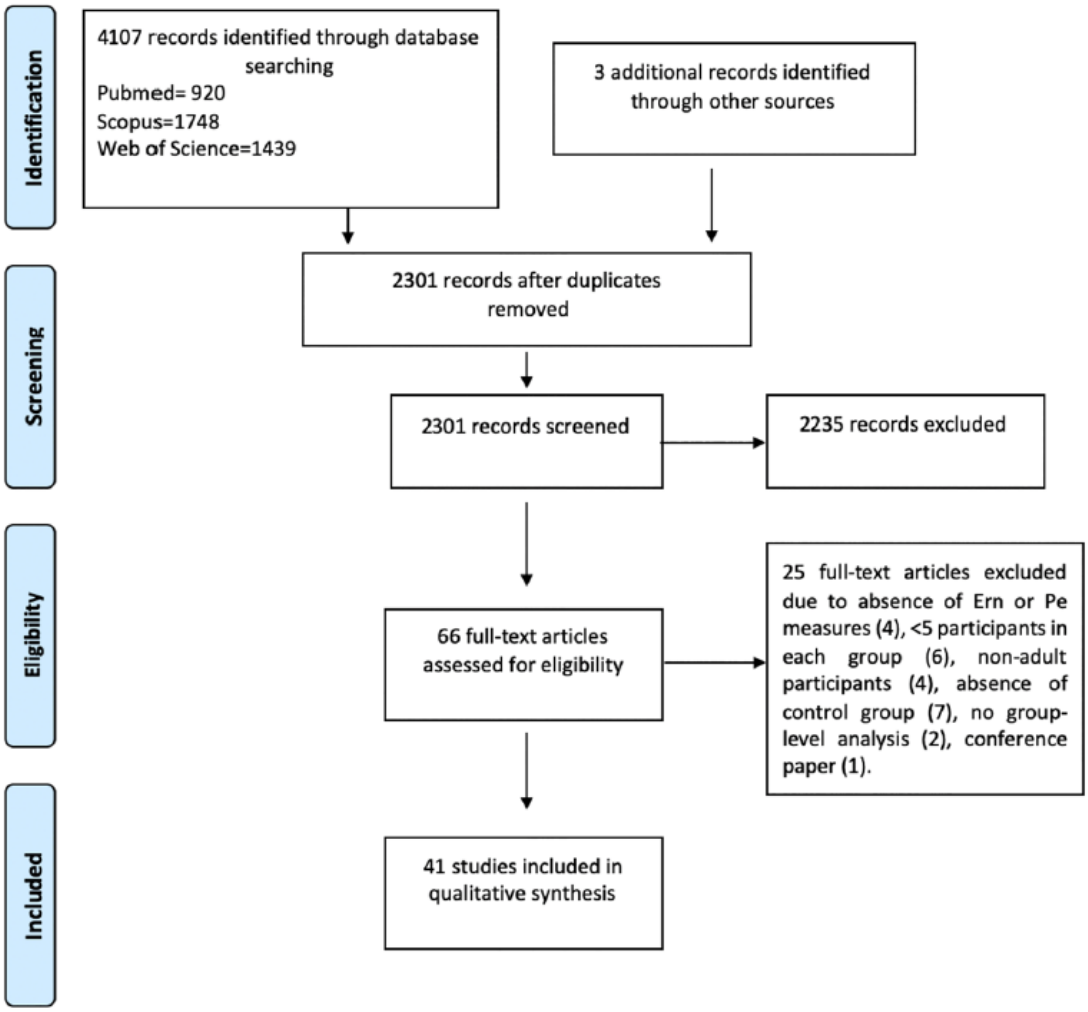

Figure 1. PRISMA flow diagram of articles selection process.

Table 1. The quality assessment criteria for included studies

		Judgment (yes/no/unclear)
Sampling	Was the study design appropriate to answer the research question? Was the sampling method appropriate? Was the control group comparable to the experimental group?	
Measurement	Was the experimental task design suitable? Were ERP measures reported in sensor-space, from 10-20, 10-10, or 10-5 coordinate systems? Were measures derived from an active system, or a high-quality passive system with appropriate shielding.? Were potential confounding variables measured?	
Data Analysis	Were EEG time series subject to appropriate pre-processing, and rigorously combed for artefacts by either manual methods, automated methods, or by Blind Source Separation techniques? Was the statistical analysis appropriate?	

Table 2. Results of individual studies.

Acc, accuracy; RT, reaction times; PES, post-error slowing; Ec, error correction; Ea, error awareness; PEA, post-error accuracy; HC, healthy controls; YHC, young healthy controls; OHC, old healthy controls; EDSS, Expanded Disability Status Scale; MMSS, Multiple Sclerosis Severity Score; EP, executive performance; med, medicated; nonmed, non-medicated; on-med, on medication; off-med, off medication; PTA, post-traumatic amnesia.

Study	*Neurological condition (Sample)*	*Task*	*Behavioral performance*	*ERPs*		
				Amplitude	*Latency*	*Correlations*
Mathalon et al, 2003	Alzheimer's disease (12 AD, 10 YHC, 10 OHC)	Picture-Name Verification Task	Acc: AD<OHC RT: AD>OHC	ERN: AD<OHC; Pe: AD=OHC	nr	-
Ito et al, 2005	Alzheimer's disease (16 AD, 15 HC)	Lexical Decision Paradigm	Acc: AD<HC RT: AD>HC Ec: AD<HC	ERN: AD< HC; Pe: AD<HC	ERN AD >HC, PE AD>HC	-
Johannes et al, 2002	Tourette's syndrome (10 TS, 10 HC)	Oddball	Acc: TS=HC RT: TS=HC	ERN: TS> HC	ERN: TS=HC	-
Schuller et al, 2018	Tourette's syndrome (15 TS, 15 HC)	Stop Signal Task	Acc: TS=HC RT: TS=HC	ERN: TS> HC; Pe: TS<HC	nr	no significant correlations with neuroepileptic medication or clinical parameter

Warren et al, 2020	Tourette's syndrome (23 TS, 27 HC)	Flanker Task	Acc: TS=HC RT: TS=HC	ERN: TS>HC	nr	no significant correlations with patients' clinical characteristics.
Lopez-Gongora et al, 2015	Multiple Sclerosis (27 MS, 31 HC)	Flanker Task with inhibition of response variant ("Stop task")	Acc: MS =HC RT: MS =HC PES: MS=HC	ERN: MS>HC	nr	ERN amplitude negatively correlated with time since last relapse, positively with EDSS and MSSS
Seer et al, 2017	Amyotrophic Lateral Sclerosis (18 ALS, 19 HC)	Flanker Task	Acc: ALS=HC RT: ALS=HC	ERN: ALS=HC; lowEP-ALS<highEP-ALS; lowEP-ALS<lowEP-HC; lowEP-HC=highEP-HC	nr	ERN amplitude was negatively correlated with EP in ALS
Falkenstein et al, 2001	Parkinson's disease (13 PD, 13 HC)	Flanker Task; Simon-type Task; Go/NoGo Task	Acc: PD=HC for Task 1,2, and 3 RT: PD>HC for Task 1,2, and 3 Ec: PD<HC for Task 1 and 2	ERN: PD< HC in Task 1, 2, and 3	ERN PD=HC in Task; 1 PD<HC in Task 2,3	-
Holroyd et al, 2002	Parkinson's disease (9 PD, 9 HC)	Flanker Task	Acc: PD=HC RT: PD=HC Ec: PD=HC	ERN: PD=HC;	nr	-
Falkenstein et al, 2005	Parkinson's disease (13 PD, 13 HC)	Flanker Task; Simon-type Task; Go/NoGo Task	Acc: PD=HC for Task 1,2, and 3 RT: PD>HC for Task 1,2, and 3 Ec: PD<HC for Task 1 and 2	Pe: PD=HC in Task 1 and 2, PD<HC in Task 3	nr	-
Ito et al, 2006	Parkinson's disease (17 PD, 15 HC)	Lexical Decision Paradigm	Acc: PD<HC RT: PD>HC	ERN: PD<HC; Pe: PD<HC	ERN: PD=HC; Pe PD=HC	-

Stemmer et al, 2007	Parkinson's disease (9 med PD, 9 nonmed PD, 14 HC)	Flanker Task	Acc: med PD=nonmed PD=HC RT: med PD=nonmed PD=HC PES: med PD=nonmed PD=HC	ERN: med PD =nonmed PD; med PD <HC; nonmed PD<HC	ERN PD=HC	-
Willemssen et al, 2008	Parkinson's disease (18 PD, 18 HC)	Flanker Task	Acc: PD=HC RT: PD=HC	ERN: on-med PD=off-med PD; off-med PD<HC	nr	-
Willemssen et al, 2009	Parkinson's disease (14 PD, 14 HC)	Flanker Task	Acc: PD=HC RT: PD>HC	ERN: PD<HC	ERN PD=HC	ERN amplitude positively correlated with BDI scores in PD
Beste et al, 2009	Parkinson's disease and Hungtinton's disease (17 medPD, 17 nonmedPD, 15 HD, 15 pHD, 15 YHC, 17 OHC)	Flanker Task	Acc: no group differences RT: med PD, nonmed PD=OHC but >pHD, HD, YHC; OHC>pHD, YHC; YHC< all groups but =pHD	ERN: pHD=YHC; pHD> all groups; YHC> med PD, nonmed PD and HD; HD= med PD and nonmed PD; OHC> both PD and HD, OHC<YHC	ERN no differences across groups	-
Verleger et al, 2013	Parkinson's disease (12 PD, 12 HC)	Flanker Task	Acc: PD=HC RT: PD=HC	ERN: PD=HC	ERN PD=HC	-
Rustamov et al, 2014	Parkinson's disease (20 PD, 20 HC)	Flanker Task	Acc: PD=HC (PD<HC in shift trials) RT: PD>HC	ERN: PD<HC	nr	-
Seer et al, 2017	Parkinson's disease (13 PD, 13 HC)	Flanker Task	Acc: PD, on.med PD<HC RT: PD=on-med PD=HC	ERN: PD<HC; on-med PD< HC; off-med PD=HC; PD on-med< PD off-med	nr	ERN amplitude was correlated with apathy, depression, health status, psychiatric status and schizotypal traits in PD

Beste et al, 2006	Huntington's disease (11 HD, 12 HC)	Flanker Task	Acc: HD=HC RT: HD>HC PES: HD=HC	ERN: HD<HC	ERN HD=HC	ERN amplitude was negatively associated with CAG-index
Beste et al, 2007	Huntington's disease (11 HD, 9 HC)	Flanker Task	Acc: pHD=HC RT: pHD=HC PES: pHD=HC Ec: pHD=HC	ERN: pHD=HC	nr	-
Beste et al, 2008	Huntington's disease (9 HD, 12 pHD)	Flanker Task	Acc: HD=pHD RT: HD>pHD PES: HD=pHD Ec: HD=pHD	ERN: HD<pHD; Pe: HD=pHD	nr	ERN amplitude was correlated with medial frontal gyrus GMW in HD
Peterburs et al, 2012	Cerebellar lesion (8 CL, 22 HC)	Anti-saccade Task	Acc: CL=HC RT: CL>HC PES: CL=HC Ec: CL=HC Ea: CL=HC	ERN: CL< HC; Pe: CL>HC	ERN CL=HC; Pe CL<HC	-
Tunc et al, 2019	Cerebellar ataxia (23 CA, 29 HC)	Flanker Task	Acc: CA=HC RT: CA>HC	ERN: CA=HC; Pe: CA<HC	nr	Pe amplitude was inversely associated with dystonia severity
Peterburs et al, 2015	Cerebellar degenaration (16 CD, 16 HC)	Anti-saccade Task	Acc: CD<HC	ERN: CD<HC; Pe: CD=HC	nr	ERN amplitude was positively correlated with grey matter volume in right lobule V and left lobule VIIb/VIIIa; Pe was

						positively correlated with Crus I.
Peterburs et al, 2011	Thalamic lesion (6 ThL, 28 HC)	Anti-saccade Task	Acc: ThL<HC RT: ThL >HC for error trials, while ThL=HC for correct trials Ea: ThL<HC Ec ThL=HC	ERN: TL<HC	nr	-
Seifert et al, 2011	Thalamic lesion (15 ThL, 16 HC)	Flanker Task	Acc: ThL=HC RT: ThL>HC Ea: ThL<HC Ec: ThL=HC PES: ThL<HC	ERN: TL<HC; Pe: TL<HC	nr	-
Ullsperger & Cramon, 2006	Basal ganglia lesion and lateral prefrontal lesion (9 BG, 7 LPFC, 9 HC for BG, 7 HC for LPFC)	Flanker Task	Acc: BG=HC RT: BG>HC, LPFC>HC Ec: BG=HC PES: BG=HC	ERN: BG<HC, LPFC<HC; Pe: BG<HC, LPFC<HC.	ERN BG=HC, LPFC=HC; Pe nr	-
Gehring & Knight, 2000	Lateral prefrontal cortex lesion (6 PFC, 10 YHC, 10 OHC)	Letter Discrimination Task	Acc: PFC=YHC=OHC RT: PFC>OHC>YHC Ec: PFC<OHC; PFC=YHC PES: PFC=YHC=OHC	ERN: PFC=OHC=YHC	nr	no significant correlations

Ullsperger et al, 2002	Lateral prefrontal cortex, orbitofrontal and temporal lesion (7 LPFC, 6 OFC, 6 TL, 9 YHC, 9 OHC)	Flanker Task	Acc: LPFC=OHC; OFC=YHC; TL=YHC RT: LPFC>OHC; OFC=YHC; TL>YHC	ERN: LPFC<OHC; OBC=YHC; TL=YHC; Pe: LPFC<OHC; OFC=YHC; TL=YHC	ERN:LPFC=OHC; OBC=YHC; TL=YHC Pe. nr	-
Wessel et al, 2014	Lateral prefrontal cortex lesion (8 LPFC, 8 HC)	Flanker Task + Novelty/Oddball Part	Acc: LPFC<HC RT: LPFC=HC Ec: LPFC<HC PES: LPFC=HC	ERN: LPFC<HC	nr	-
Solbakk et al, 2014	Orbitofrontal lesion (12 OFC, 14 HC)	Stop Signal Task	Acc: OFC=HC RT: OFC=HC PES: OFC=HC	ERN: OF<HC; Pe: OF>HC	nr	-
Maier et al, 2015	Anterior cingulate cortex lesion (7 rACC, 7 BDC, 7 HC)	Flanker Task	Acc: rACC,BDC>HC; rACC=BDC RT: rACC>HC; rACC=BDC Ea: rACC=HC=BDC Ec: rACC=HC=BDC PES: rACC=HC=BDC PEA: rACC < HC, BDC	ERN: ACC<HC, ACC< BDC; Pe: ACC= HC, ACC= BDC	Pe: ACC= BDC, ACC=HC	-
Niessen et al., 2020	Left hemisphere lesion (17 LH, 24 HC)	Go/NoGo Task	Acc: LH=HC RT: LH=HC Ea: LH=HC PES: LH=HC	ERN: LH=HC; Pe: LH=HC	ERN: LH=HC; Pe: LH=HC	ERN latency was correlated with lesion size

Larson et al, 2007	Traumatic brain injury (19 TBI, 21 HC)	Stroop Task	Acc: TBI=HC RT: TBI>HC	ERN: TBI<HC; Pe: TBI<HC	ERN: TBI=HC; Pe: TBI=HC	-
Pontifex et al, 2009	Traumatic brain injury (30 TBI, 36 HC)	Flanker Task	Acc: TBI<HC RT: TBI>HC only for incongruent trials PES: TBI=HC PEA: TBI=HC	ERN: TBI<HC; Pe: TBI=HC	nr	ERN amplitude was negatively correlated with number of incidents
Larson et al, 2009	Traumatic brain injury (20 TBI, 20 HC)	Stroop Task	Acc: TBI=HC RT: TBI>HC	ERN: TBI<HC; Pe: TBI=HC	ERN: TBI=HC	ERN amplitude was inversely correlated with negative affect in TBI
Larson et al, 2012	Traumatic brain injury (36 TBI, 46 HC)	Stroop Task	Acc: TBI=HC RT: TBI=HC PES: TBI=HC PEA: TBI=HC	ERN: TBI=HC; Pe: TBI=HC	nr	ERN latency was associated with fewer prior TBIs; Pe amplitude associated with length of PTA, and negatively associated with time since injury
De Beaumont et al, 2013	Traumatic brain injury (14 TBI, 14 HC for task1; 18 TBI, 21 HC for task2)	Visual search modified with oddball condition; Visual short-term memory task	Acc: TBI=HC for task 1 and 2	ERN: TBI<HC; Pe: TBI=HC (for both tasks)	nr	ERN amplitude negatively correlated with number of concussion (for both tasks)
Logan et al, 2015	Traumatic brain injury (19 TBI, 37 HC)	Error Awareness Task	Acc: TBI=HC (including aware and unaware errors) RT: TBI=HC	Pe: TBI=HC	nr	-

			Ea: HC but not TBI showed increased awareness over the course of the task			
Olson et al, 2018	Traumatic brain injury (25 TBI, 22 HC)	Flanker Task	Acc: TBI=HC RT: TBI=HC PES: TBI=HC PEA: TBI=HC	ERN: TBI>HC; Pe: TBI=HC	nr	-
Shen et al, 2019	Traumatic brain injury (18 TBI, 18 HC)	Stop Signal Task	Acc: TBI=HC RT: TBI=HC	ERN: TBI=HC; Pe: TBI=HC	ERN: TBI=HC; Pe: TBI>HC	Pe peak latency was negatively correlated with probability of inhibition and Pe amplitude was positively correlated with probability of inhibition.

Article 3

Lenzoni, S., Sumich, A., & Mograbi, D. (Submitted). Domain specificity of error monitoring: an ERP study in young and older adults. Preprint available at https://psyarxiv.com/vfgmk/

Abstract

Metacognition refers to the ability to monitor and control one's cognitive processes which plays an important role in decision-making throughout the lifespan. It is still debated whether metacognitive abilities decline with age. Recent neuroimaging evidence suggests the existence of domain-specific mechanisms underlying metacognition and it is possible that age-related decline follows domain-specific trajectories across cognitive functions. Event-related potential (ERP) research has identified neural markers of performance monitoring. The error-related negativity (ERN) and the error positivity (Pe) are electrophysiological correlates of error detection and error awareness, respectively. However, ERP differences across cognitive domains have not been yet investigated. In the current study, thirty-eight young adults and thirty-seven older adults completed a classic Flanker Task (perceptual) and an adapted memory-based version during EEG recordings. Univariate and multivariate analyses were conducted to explore domain-specific mechanisms of error monitoring ERPs and age group differences. No difference in ERN amplitude was found between young and older adults and across domains. ERN peaked earlier in the perceptual domain than the memory domain. ΔERN was larger for the memory domain than the perceptual domain. Pe was smaller in older adults but similar across domains. Memory Pe peaked earlier in young adults than older adults. ΔPe was larger for perceptual than memory flanker. During the task, ERN decreased in young but not in older adults. Memory Pe decreased in young adults but increased in older adults while no significant change in perceptual Pe was found. Multivariate analyses of whole scalp data support cross domain differences. These findings suggest that error monitoring may rely on domain-specific mechanisms. Moreover, we speculate that reduced error awareness may be associated with sensory decline in aging, as indicated by learning effects specific to the memory domain.

Keywords

Error monitoring; ERP; ERN; Pe; aging; metacognition

1. Introduction

Successful evaluation of one's actions is crucial for learning and for implementing behavioural adjustments to optimize performance. Metacognition, often defined as "thinking about thinking", refers to the ability to reflect on, monitor, and control one's own cognitive processes (Dunlosky & Metcalfe, 2009; Flavell, 1979; Fleming et al., 2012). Efficient metacognitive processes play an important role in promoting learning, educational achievements, and decision-making across the life span (Bryce et al., 2015; Efrati et al., 2021; Laghi et al., 2020; Moses-Payne et al., 2021; Ohtani & Hisasaka, 2018; Perry et al., 2019; Roebers et al., 2014; Weil et al., 2013). Poor metacognitive competences are associated with dysfunctional behaviours in neurological and psychiatric disorders (Bertrand et al., 2016; Brune et al., 2011; Hallam et al., 2020; Lenzoni et al., 2020; Mograbi et al., 2009; Palmieri et al., 2021; Rogier et al., 2021; Seow et al., 2021; Sun et al., 2017). However, the extent to which such abilities decline with older age remains unclear.

Convergent evidence from neuroimaging research indicated the ventral and posterior regions of the medial prefrontal cortex (mPFC), including the anterior cingulate cortex (ACC) and insular regions as central nodes of an extended metacognition network, underlying self-evaluation and self-monitoring (Fleming & Dolan, 2012; Metcalfe & Schwartz, 2016; Qiu et al., 2018; Vaccaro & Fleming, 2018). Thus, metacognitive abilities might be expected to follow cognitive decline trajectories commonly associated to frontal lobe impairment (Li et al., 2015; McDonald et al., 2018; Onoda et al., 2012). However, previous research has shown mixed findings. Some studies suggest that metacognition is preserved in older age (e.g., Halamish et al., 2011; Hertzog & Dunlosky, 2011; Sanders & Berry, 2020) while other research reported marked differences between young and older adults (Bender & Raz, 2012; Huff et al., 2011; Perrotin et al., 2006; Soderstrom et al., 2012; Souchay et al., 2007; Toth et al., 2011; Wong et al., 2012).

Recent conceptualizations of the neural architecture of metacognition postulated the existence of domain-specific processes, as supported by evidence of distinct neural substrates of metamemory and metaperception (for a review see Rouault et al., 2018; Seow et al., 2021; Vaccaro & Fleming, 2018). Limited aging research has investigated metacognition across domains. Zakrzewski et al. (2021) explored

metacognition for different types of memory processes and reported intact metacognitive efficiency in older adults, despite a decline in performance, as compared to young adults. Moreover, they found a difference in metacognitive abilities between item recognition and associative memory across age groups. Furthermore, one study showed that perceptual but not memory metacognitive efficiency decreased with age, suggesting that age-related changes in metacognitive abilities are characterized by domain-specific trajectories (Palmer et al., 2014).

Neural markers of self-monitoring have been extensively investigated using electroencephalography (EEG), which allows to assess time-locked neurophysiological changes during task performance, for example by quantifying event-related potentials (ERPs; Luck, 2014). The error-related negativity (ERN; Falkenstein et al., 1991; Gehring et al., 1993) is a negative deflection occurring between 0 and 100ms after error commission with higher amplitude at frontocentral sites. ERN amplitude is typically higher compared to activity following correct responses, the correct-related negativity (CRN or correct-trial ERN; Falkenstein et al., 2000). The neural source of ERN has been localized in the ACC (e.g. Brázdil et al., 2005; Debener, 2005; Dehaene et al., 1994; Reinhart & Woodman, 2014; van Veen & Carter, 2002), possibly conveying error prediction signals via the dopaminergic system (Holroyd & Coles, 2002) and it is thought to reflect post-response conflict (Botvinick et al., 2001; Yeung et al., 2004), or mismatch between expected and actual responses (Dehaene, 2018; Falkenstein et al., 2000). Although different computational models have been proposed, the ERN can be considered as index of fast (pre-conscious) monitoring processes promoting adaptive behaviours (Weinberg et al., 2015).

The error positivity (Pe; Falkenstein et al., 2001; Overbeek et al., 2005) is a positive component occurring between 200-400ms after an error, typically recorded at midline posterior sites after errors. Similar to the ERN, Pe amplitude is larger for errors than for correct trials. The neural origin of the Pe is less clear, considering that only a few studies have attempted localising it, suggesting a possible role of insula (Dhar et al., 2011), ACC (Herrmann et al., 2004), and posterior-cingulate/precuneus (O'Connell et al., 2007). Previous research found a relationship between Pe and conscious perception of errors (Endrass et al., 2007; Murphy et al., 2012; Nieuwenhuis et al., 2001). According to the Accumulation

Account, Pe reflects error awareness, which emerges from a post-decisional process of evidence accumulation about the erroneous response (Kirschner et al., 2021; Steinhauser & Yeung, 2010, 2012; Ullsperger et al., 2010; Wessel et al., 2011). Moreover, Pe amplitude was shown to be associated with confidence levels about error commission (Boldt & Yeung, 2015), and behavioural adjustments (Desender et al., 2019), thus strengthening the idea that Pe may track a metacognitive decision variable (Desender et al., 2021).

Domain-specific mechanisms underlying ERN and Pe have not been yet investigated. It has been previously discussed that the presence of task-specific effects may limit our understanding of self-monitoring neurophysiology in clinical populations (Lenzoni et al., 2022; Mathews et al., 2012; Riesel, 2019). However, limited research employed multiple experimental paradigms and explored performance monitoring across task. For example, error-related ERPs were observed to vary across tasks in undergraduate students (Flanker, Stroop, Go/NoGo; Riesel et al., 2013), as a function of obsessive-compulsive symptomatology (Flanker, Probabilistic learning; Gründler et al., 2009), and in children and adolescents (Flanker, Go/NoGo; Meyer et al., 2014). Nonetheless, it could be argued that task dissociations do not reflect patters of self-monitoring impairments, but instead they may be a by-product of diverse experimental procedures that can impact ERPs findings, such as differences in task difficulty (Falkenstein, 2004; Hoffmann & Falkenstein, 2010; Johannes et al., 2002; Pailing & Segalowitz, 2004), instructions (S. E. Morris et al., 2006), or number of trials (Fischer et al., 2017)

Several studies found that ERN amplitude is reduced in older adults (Beste et al., 2009; Dywan et al., 2008; Endrass et al., 2012a; Eppinger & Kray, 2011; Falkenstein, Hoormann, et al., 2001; Harty et al., 2017; Herbert et al., 2011; Hoffmann & Falkenstein, 2011; Mathalon et al., 2003; Mathewson et al., 2005; Schreiber et al., 2011; Themanson et al., 2006; Thurm et al., 2020; West, 2004). However, other studies reported that ERN was comparable between young and older adults (Capuana et al., 2012; Clawson et al., 2017; Eppinger et al., 2008; Larson et al., 2016; Pietschmann, Endrass, & Kathmann, 2011; Pietschmann, Endrass, Czerwon, et al., 2011; Thurm et al., 2013) and one study found larger ERN in older adults (Staub et al., 2014). Fewer studies reported the CRN, showing larger

(Larson et al., 2016; Schreiber et al., 2011), smaller (Eppinger et al., 2007; Harty et al., 2017; Mathalon et al., 2003) and comparable (Clawson et al., 2017; Endrass et al., 2012a; Falkenstein, Hoormann, et al., 2001; Pietschmann, Endrass, Czerwon, et al., 2011; Staub et al., 2014; Thurm et al., 2013) amplitude in older adults as compared to young adults. Finally, limited research explored aging effects on Pe, that was found to be attenuated in older adults (Capuana et al., 2012; Clawson et al., 2017; Larson et al., 2016; Mathewson et al., 2005; Thurm et al., 2020). However, one study showed no differences in Pe between young and older adults (Mathalon et al., 2003) and another study did not clarify the effect of age on Pe (Staub et al., 2014). One study investigated task effects in aging, showing smaller ERN and Pe in older as compared to young adults, and that ERN was comparable between Flanker and Source Monitoring Tasks, while Pe was larger in the Flanker Task (Mathewson et al., 2005). Critically, elucidating whether age-related changes in performance monitoring depend on domain-specific trajectories may throw light upon the current literature.

The aim of the current study was to i) investigate whether it is possible to differentiate neurophysiological markers of performance monitoring across cognitive domains; ii) explore whether age-related changes (if any) occur at a global level (domain-general) or are specific to certain cognitive domains (domain-specific). To this end, a group of young and older adults performed two versions of the Flanker Task (Eriksen & Eriksen, 1974): the classic arrow version (perceptual domain) and an adapted memory version that was developed to the test the domain-specificity hypothesis.

2.Methods

2.1 Participants

Forty-two younger adults and 41 older adults were recruited through Psychology Division Research participation schemes at Nottingham Trent University. Inclusion criteria were normal/corrected vision and fluency in English. Participants were excluded if they have history of neurological and/or psychiatric disorders. Four participants were excluded for current diagnosis of psychiatric disorders and 4 participants were excluded from the analyses because they had a low error rate in

at least one of the experimental tasks (number of errors <5). The final enrolment included 38 younger adults (24 females, 14 males) between the ages of 19-34 years (M=22.45, SD= 4.38) and 37 older adults (23 females, 14 males) between the ages of 60-90 years (M=70.95, SD=10.56). The two groups had similar sex ratios ($\chi 2(1)$ <.01, p=.929) and educational levels (W=572, p=.141). All participants provided written consent and all procedures were approved by Nottingham Trent University College of Business, Law and Social Sciences Ethics Committee.

2.2 Experimental Tasks

Participants completed two versions of the Eriksen Flanker Task (Eriksen & Eriksen, 1974): i) the classic arrow version exploring performance monitoring in perceptual decision; ii) a modified version developed to investigate performance monitoring in memory decisions. The tasks were created using PsychoPy2 (v1.90.1; Peirce et al., 2019). All stimuli were 2D icons generated by Freepik (www.flaticon.com). All stimuli were displayed on a white background of a 19'' computer monitor displaying 1,600 × 900 pixels at 60 Hz, approximately 60cm from participants' forehead. The order of the Flanker Tasks was counterbalanced to control for possible effects of learning and fatigue.

2.2.1 Perceptual Flanker Task

In each trial, participants were presented with five horizontal arrows stimuli either pointing all to the same directions (i.e., congruent; <<<<<, >>>>>), or with the central arrow pointing to the opposite direction than the others (i.e., incongruent, <<><<, >><>>). Participants were asked to identify by button press whether the central arrow (target) was pointing to the left or to the right and were instructed to respond as quickly and as accurately as possible, while ignoring the direction of the other arrows (flankers). Participants completed 12 practice trials and 6 blocks of 96 trials for the actual task. In each block half of the trials were congruent and half of the trials were incongruent. For both congruent and incongruent conditions, in half of the trials the target was pointing to the left and in the other half to the right. At the end of each block participants were asked to rate how confident were about their performance on a scale between 1 and 5. Each set of stimuli filled 2.46° of visual angle vertically and 12.36° horizontally. Stimuli were preceded by a fixation cross

(500ms), were randomly presented for 100ms, and participants had 1200msec to respond. ITI varied between 500-900msec.

2.2.2 Memory Flanker Task

In the learning phase, participants memorize four icons (mushroom, chicken, love heart, and shoe): i) each of the four icons is presented at the centre of the screen for 2 seconds (2.46°-2.46°); ii) participants are asked to recall the four icons; iii) participants perform a recognition task in which they see eight icons, one by one (2.46°-2.46°), and they have to decide by button press whether they have just seen the icon or not; iv) the four icons are displayed one last time asking participant to try to remember them. The experimental phase (actual task) takes place 20mins following the end of the learning phase to ensure transfer in long-term memory. Before the beginning of the task, participants were asked to recall the four icons they were asked to remember in the learning phase to ensure that retrieval issues would not bias task execution and memory monitoring. In each trial, participants were presented with five icons that could be either all the same (i.e., congruent), or with the central icon being different from the other four icons (i.e., incongruent). Participants were asked to identify by button press whether the central icon (target) was old (one of the four icons memorized in the learning phase) or new and were instructed to respond as quickly and as accurately as possible, while ignoring other icons (flankers). Participants completed 12 practice trials and 6 blocks of 96 trials for the actual task. In each block half of the trials were congruent and half of the trials were incongruent. For both congruent and incongruent conditions, in half of the trials the target was an old icon to the left and in the other a new icon. At the end of each block participants were asked to rate how confident they were about their performance on a scale between 1 and 5. Each set of stimuli filled 2.46° of visual angle vertically and 12.36° horizontally. Stimuli were preceded by a fixation cross (500ms), were randomly presented for 100ms, and participants had 1200msec to respond. ITI varied between 500-900ms. Stimuli were classified as belonging to four categories: A) animals and food B) objects and symbols. To avoid the possibility that the interference effects in incongruent trials could be caused by physical similarities, rather than old/new effects, target and flanker stimuli were

different in colour and shape. Moreover, to avoid semantic relatedness in incongruent trials, in each trial, target and flanker stimuli were chosen from different categories.

2.3 EEG recordings, preprocessing and ERP extraction

A BioSemi Active II system (Biosemi, Amsterdam, The Netherlands) was used to record continuous EEG. Recordings were taken from 64 active scalp electrodes based on the 10/20 system and 2 external electrodes placed on the right and left mastoids. Data were sampled at 2048 Hz, digitized at 24 bits and referenced online with a CMS/DRL feedback loop. Electrodes off-set was kept within the absolute value of 20 μV. EEGLAB (Delorme & Makeig, 2004) and MATLAB (Mathworks, Natick, Massachusetts, USA) were used for off-line analyses. Data were downsampled to 256 Hz and processed through a 0.1 Hz high pass filter and a 30 Hz low-pass filter. Data were re-referenced to average mastoids. Bad channels were removed and interpolated. Epochs of 1200ms (200ms baseline before response and 1000ms after) were extracted. Independent component analysis (ICA) was used to remove ocular artifacts. The interval between -200ms and 0ms was chosen for baseline correction, as it was showed to be associated with large effect sizes (Clayson et al., 2021) and good internal consistency (Klawohn et al., 2020). Epochs exceeding 100 μV and -100 μV were removed. Response-locked ERPs were averaged separately for each type of response (correct responses and errors). The ERN/CRN was quantified as mean amplitude in the interval 0- 80ms at Fz, F1, F2, FCz, FC1, and FC2 and the Pe/Pc was quantified as mean amplitude in the interval 200-400ms at CPz, CP1, CP2, Pz, P1, and P2. Latencies were extracted at the maximal values in the selected intervals (most negative peak for ERN/CRN and most positive peak for Pe/Pc). ΔERN and ΔPe (error-correct) were also calculated because difference scores are commonly used to isolate an error-specific activity.

2.4 Statistical analysis

All analyses were performed using R (R Core Team, 2020). Trials with RTs lower than 200ms were excluded. Accuracy was calculated as percentage of correct

responses. Post-error slowing (PES) was calculated using the mean-based correct robust measurement approach, as it reduces bias in interference tasks (Derrfuss et al., 2022). Mean confidence judgments were calculated averaging single block ratings. Mixed ANOVA was used to test differences in accuracy, RTs, PES, and confidence judgments.

Multilevel models (MLM) were used to explore task and group differences in ERPs. MLM present multiple advantages for ERP analysis, such as robustness to missing trials and unbalanced designs, inclusion of categorical and continuous variables as independent variables and electrodes as random factors rather than predictors (Volpert-Esmond et al., 2021), as well as and the possibility to explore trial-to-trial variations and within task changes in ERPs (Volpert-Esmond et al., 2018). Maximal model structures included all random slopes and their interaction by participant (Barr, Levy, Scheepers, & Tily, 2013). Response (correct, error), Domain (perceptual, memory) and Group (younger, older) were entered as predictors. Fixed effects were effect coded (categorical variables; -0.5,0.5). In the case of convergence problems, models would include random slopes but not their interactions. The models included electrodes as crossed random factor. In order to explore neurophysiological variations that occur during the task, we reproduced the approach used by Volpert-Esmond and colleagues (2018) and used to examine ERN and Pe changes as function of number of errors. Errors trials were sequentially numbered (i.e., error 1 is the first error regardless trial number). In order to explore the relationship between ERPs and confidence about performance, mean confidence was used as fixed effect. Mean confidence was grand-mean centered (Enders & Tofighi, 2007). Participants and electrodes were included as random factors, and domain was allowed to vary by participant. To fit the models, lme4 package (Bates et al., 2014) was used and lmerTest (Kuznetsova et al., 2017) was used to calculate p-values using Satterthwaite's degrees of freedom. Interactions were tested using post-hoc tests adjusting with Tukey's correction for multiple comparisons for categorical variables and simple slope analysis for continuous variables.

2.5 Multivariate Pattern Analysis (MVPA)

A multivariate pattern analysis (MVPA) was applied on the raw EEG data using the ADAM toolbox (Fahrenfort et al., 2018). MVPA is more sensitive to brain response patterns as compared to univariate analysis, which is usually based on averaged ERP waveforms (Grootswagers et al., 2017; Hebart & Baker, 2018), and can be used to quantify differences across experimental conditions without a priori channel selection (Fahrenfort et al., 2017). EEG epochs time-locked to response were classified according to task domain (perceptual, memory) within response correctness (error, correct). A backward decoding model was used to perform a leave-one-out cross-validated multivariate classification analysis. The linear discriminant classifier was trained on 90% of the data and tested on 10% of the data for each participant, across all electrodes. The area under the receiver operating characteristics curve (AUC; Bradley, 1997) was used as classifier accuracy. AUC is a metric derived from signal detection theory (Wickens, 2010), which is obtained by plotting the cumulative true positive rates against the cumulative false positive rates, and varies between 0 and 1, where 0.5 indicates chance performance and 1 indicates maximum classification accuracy. Group analyses were performed using two-sided t-tests against chance accuracy across subjects. Cluster-based permutation testing was used to control for multiple comparisons.

3. Results

3.1 Behavioural performance

Descriptive statistics of behavioural performance are summarised in Table 1. A 2 (Group: Younger, Older) x 2 (Domain: Perceptual, Memory) mixed ANOVAs with repeated measures on the task domain was conducted to explore differences in accuracy. There was no main effect of Group, $F_{(1,73)} = 0.70$, $p = .405$, $\eta^2 = .01$, no main effect of Domain, $F_{(1,73)} = 0.50$, $p = .482$, $\eta^2 < .01$, no interaction $F_{(1,73)} = 0.23$, $p = .627$, $\eta^2 < .01$, thus suggesting that younger and older adults performance was comparable in both task domains. A 2 (Group: Younger, Older) x2 (Domain: Perceptual, Memory) x 2(Congruency: Congruent, Incongruent) mixed ANOVA with repeated measures on Domain and Congruency was conducted to explore differences in RTs. There was a main effect of group, with younger adults responding more quickly than older adults, $F_{(1,73)} = 88.02$, $p < .001$,

η^2 = .49. There was a main effect of domain, with longer RTs for the memory domain compared to the perceptual domain, $F_{(1,73)} = 112.24$, $p < .001$, $\eta^2 = .20$. There was also a main effect of Congruency, with longer RTs for incongruent trials than for congruent trials, $F_{(1,73)} = 667.96$, $p < .001$, $\eta^2 = .14$. The Group x Congruency interaction was significant, $F_{(1,73)} = 22.54$, $p < .001$, $\eta^2 < .01$. Pairwise comparisons confirmed congruency effects for younger ($p < .001$) and older adults ($p < .001$), with longer RTs in older adults for congruent ($p < .001$) and incongruent ($p < .001$). There was also a significant Domain x Congruency interaction, $F_{(1,73)} = 247.54$, $p < .001$, $\eta^2 = .03$. Congruency effects were present in both perceptual ($p < .001$) and memory domains ($p < .001$), with longer RTs in the memory domain in congruent ($p < .001$) and incongruent ($p < .001$) trials. These findings suggest that congruency effects (differences between congruent and incongruent RTs trials) were stronger in older as compared to younger adults and in the perceptual domain than the memory domain. The Group x Domain and Group x Domain x Congruency interactions were not statically significant. A 2 (Group: Younger, Older) x 2 (Domain: Perceptual, Memory) mixed ANOVA revealed no main effect of group, $F_{(1,65)} = 1.16$, $p = .288$, $\eta^2 < .01$, no main effect of task Domain, $F_{(1,65)} = 0.00$, $p = .992$, $\eta^2 < .01$, no interaction between Group and Domain, $F_{(1,65)} = 0.79$, $p = .377$, $\eta^2 < .01$, on PES, indicating that younger and older adults had similar post-error behaviours across task domains. A 2 (Group: Younger, Older) x 2 (Domain: Perceptual, Memory) mixed ANOVA revealed a main effect of age group on confidence judgments, $F_{(1,72)} = 6.66$, $p = .012$, $\eta^2 = 0.07$, with older adults rating higher their performance as compared to younger adults. Main effect of task domain and the Group x Domain interaction were not statistically significant.

PLEASE INSERT TABLE 1 HERE.

In order to explore whether there were cross domain differences in how behavioural performance varied within-task in accuracy, 2 (Group: Younger, Older) x 2 (Domain: Perceptual, Memory) x 6 (Block:1,2,3,4,5,6) mixed ANOVA were conducted on accuracy, RTs and confidence. For accuracy, there was a Group x

Block interaction, $F_{(3.37,245.98)} = 3.19$, $p = .020$, $\eta^2 = .01$. Post-hoc tests did not reveal any significant differences. Main effects of Group, Domain, and Block, the other 2-way interactions and the 3-way interaction were not statistically significant. For RTs, there was a main effect of Group, $F_{(1,73)} = 92.32$, $p < .001$, $\eta^2 = .47$, with slower responses for older adults than young adults. There were also main effects of Domain, $F_{(1,73)} = 127.78$, $p < .001$, $\eta^2 = .20$, and of Block, $F_{(2.87,209.81)} = 21.66$, $p < .001$, $\eta^2 = .02$, which were qualified by a Domain x Block interaction, $F_{(3.60,262.52)} = 21.40$, $p < .001$, $\eta^2 = .02$. Post-hoc tests confirmed that in all Blocks, RTs were longer for the memory domain (all *p*s < .001), and revealed, that RTs did not change across block for the perceptual domain, but for memory RTs were longer in Block 1 as compared to all the other Blocks in the memory domain (all *p*s <.001), did not change from Block 2 to Block 5, and then decreased from Block 5 to Block 6 ($p < .001$), suggesting that participants became faster during the course of the task for memory only (see Figure 1). The other 2-way interactions and the 3-way interaction were not statistically significant. For confidence ratings, there was a main effect of Group, $F_{(1,72)} = 6.66$, $p = .012$, $\eta^2 = .04$, with older adults reporting higher confidence about performance than young adults. There was also a Group x Block interaction, $F_{(4.32,310.89)} = 2.35$, $p = .049$, $\eta^2 < .01$. However, post-hoc tests did not reveal any significant differences.

PLEASE INSERT FIGURE 1 HERE

3.2 ERPs

Grand-average waveforms as function of age group and task domain are presented in Figure 2. Mean ERN/CRN and Pe/Pc amplitude and latency at each electrode are presented in Supplementary Table 1.

PLEASE INSERT FIGURE 2 HERE

Model estimates for ERN/CRN amplitude can be found in Supplementary Table 2. There was a main effect of Response, *b* = 4.91, 95% CI [3.67. 6.15], *t* (69.29) = 7.41, *p* < .001, with larger amplitude (more negative) for errors than correct responses. There was a main effect of Domain, *b* = 0.80, 95% CI [0.18. 1.42], *t* (73.00) =2.57, *p* = .012, with larger amplitude for perceptual domain than memory domain. The main effects were qualified by a Response x Domain interaction, *b* = 1.14, 95% CI [0.18. 2.11], *t* (72.99) = 2.37, *p* = .020. Post-hoc tests revealed larger amplitude for errors compared to correct responses in both task domains (all *p*s < .001) and larger CRN amplitude for the perceptual domain than the memory domain (*p* < .001). There was no main effect of group, and the other 2-way interactions and the 3-way interaction were not statistically significant.

MLM for ΔERN revealed a main effect of Domain, *b*= -1.14, 95% CI [-2.11. 0.18], *t* (73.00) = -2.37, *p* = .020, with larger ΔERN for the memory domain than the perceptual domain (Figure 7). The main effect of Group and the Group x Response interaction were not statistically significant.

PLEASE INSERT FIGURE 3 HERE

Model estimates for ERN/CRN latency are displayed in Supplementary Table 3. There were main effects of Domain, *b*=1.69, 95% CI [0.52. 2.85], *t* (72.99) = 2.88, *p* = .005, and a main effect of Group, *b* = 2.10, 95% CI [0.25. 3.94], *t* (73.00) = 2.27, *p* = .026. The main effect of Domain was qualified by a Response x Domain interaction, *b* = -5.17, 95% CI [-5.80. -4.54], *t* (1568.00) = -16.10, *p* < .001. Post-hoc test revealed that ERN peaked earlier in the perceptual domain than the memory domain (*p* < .001), but there was no difference for correct trials (*p* = .455). Moreover. only in the perceptual domain. ERN peaked earlier than CRN (*p* = .005). There was also a Response x Group interaction, *b* = -3.43, 95% CI [-6.63. -0.23], *t* (73.01) = -2.14, *p* = .036. Young adults ERN peaked earlier than older adults (*p* = .021), but there was no difference for CRN (*p* = .988). Moreover, for young adults only, ERN peaked earlier than CRN (*p* = .030).

Model estimates for Pe/Pc amplitude are displayed in Supplementary Table 4. There was a main effect of Response, *b* = -8.31, 95% CI [-9.63. -6.98], *t* (73.00) = -12.51, *p* < .001, with higher amplitude (more positive) for errors than correct responses. and a main effect of Group, *b*=-2.81, 95% CI [-4.03.-1.60], *t* (73.00) = -4.61, *p* < .001, with reduced amplitude in older adults as compared to young adults. There was a Response x Domain interaction, *b* = 2.98, 95% CI [1.88 – 4.09], t (73.00) = 5.38, *p* < .001. Memory Pc was larger than Perceptual Pc (*p* < .001), but Pe was comparable across task domains (*p* = .115).

MLM for ΔPe difference wave revealed a main effect of Domain, *b* = -2.98, 95% CI [-4.09 – 1.88], *t* (73.00) = -5.38, *p* < .001, with larger ΔPe for the perceptual domain than the memory domain (Figure 7). The main effect of Group and the Domain x Group interaction was not statistically significant.

PLEASE INSERT FIGURE 4 HERE

Model estimates for Pe/Pc latency are summarised in Table 5. No main effect was statistically significant. There was a Response x Domain interaction, *b* = 2.81, 95% CI [1.25. 4.37], *t* (1568.00) = 3.53, *p* < .001, and a Domain x Group interaction, *b* = 7.18, 95% CI [1.54. 12.83], *t* (73.00) = 2.54, *p*=.013, that were qualified by statistically significant a three-way interaction Response x Domain x Group, *b* = -8.38, 95% CI [-11.14. -5.26], *t* (1568.00) = -5.26, *p* < .001. Memory Pe peaked earlier than Perceptual Pe in young adults (*p* = .004). Memory Pe peaked earlier in young as compared to older adults (*p* = .021).

PLEASE INSERT FIGURE 5 HERE

ERN trial-level analysis revealed a main effect of Error Number, and the Error Number x Domain and the Error Number x Group interactions were statistically significant (Table 2). Simple slope analysis indicated a significant decrease in ERN only for the memory domain (*b* = 0.04, *p* < .001), while the slope for the perceptual

domain was not statistically significant. Moreover, there was a significant ERN decrease in young adults (b = 0.04, p <.001), while the slope for older adults was not statistically significant (Figure 6). Pe trial-level analysis revealed a main effect of Error Number, a main effect of Domain, and a main effect of Group. There were also an Error Number x Domain interaction and an Error Number x Group interaction. Main effects and interactions were qualified by an Error Number x Domain x Group interaction. Follow-up slope analysis indicated a significant decrease of memory Pe in young adults (b = -0.02, p = .005) and a significant increase in memory Pe in older adults (b = 0.09, p < .001), while the other slopes were not statistically significant (Figure 6).

PLEASE INSERT TABLE 2 HERE

PLEASE INSERT FIGURE 6 HERE

MLM for the association between ERPs and confidence are summarised in Table 3. ERN was associated with higher confidence ratings as indicated by the main effect of Confidence. However, the association did not vary in relation to task domain or age group. No association between Pe and confidence was found.

PLEASE INSERT TABLE 3 HERE

3.3 MVPA

MVPA showed that the two conditions (task domains) could be successfully decoded from one another in young and older adults. for correct responses and errors (see Figure 7). Importantly, when the classifier was trained to discriminate between perceptual correct responses and memory correct responses, decoding accuracy was significantly above chance in the entire epoch, for both young and older adults. Similarly, decoding performance for error trials was above chance for

almost in the entire epoch (young adults: 0-996ms, $p < .001$; older adults: -199- -137ms, p=.008, -129 - -82ms, p = .019, -35-35ms, $p = .007$, 43-996ms, $p < .001$).

PLEASE INSERT FIGURE 7 HERE

4. Discussion

The current study aimed at exploring domain-specific mechanisms underlying error monitoring ERPs in young and older adults. Our analyses revealed distinct brain responses to both errors and correct responses between perceptual and memory domains, in line with recent evidence supporting domain specificity of neural correlates of metacognitive processes (Rouault et al., 2018; Vaccaro & Fleming, 2018). Furthermore, this study investigated the effect of age on error monitoring and whether age-related changes may occur at a global or specific level. Our findings are in contrast with the idea of generalized decline in performance monitoring but instead cross-domain differences in within-task activity suggest the presence of age-related changes in mechanisms underlying error awareness. Moreover, distinct patterns of response-related neural activity were identified in both young and older adults.

Behavioural performance was similar across domains, as shown by comparable accuracy and PES, while responses were slower in the memory domain, because of retrieval-related delay during task performance. Nonetheless, it was possible to detect ERPs differences across domains. Overall, mean perceptual ERN and CRN were larger, and perceptual ERN peaked earlier as compared to the memory domain. However, ΔERN was larger during the memory flanker. This may suggest that response monitoring is generally more efficient for lower order representation involved in perceptual decisions while it is possible that sensitivity to errors is higher for the memory domain because of greater cognitive effort needed to retrieve and compare mnemonic representations or because of higher salience of memory errors. Memory Pc was larger than perceptual Pc, memory Pe peaked earlier than perpetual Pe in young adults, but Pe amplitude was comparable across domains. However, ΔPe was larger for the perceptual domain, thus highlighting marked

error-specific differences. In line with the Accumulation Account, Pe reflects error awareness emerging from a processes of evidence accumulation about error commission (Desender et al., 2021; Kirschner et al., 2021; Steinhauser & Yeung, 2010, 2012; Ullsperger et al., 2010; Wessel et al., 2011). Crucially, availability of sensory information during perceptual decisions may lead to stronger post-decisional evidence about response outcomes, and consequently to enhanced error awareness. Indeed, dissociations between ERN and Pe findings are not surprising, as they were previously shown to represent two independent systems of performance monitoring (di Gregorio et al., 2018; Overbeek et al., 2005).

Moreover, trial-based analyses provided further support to the domain specificity hypothesis. First, within-task changes of ERN and Pe were found to be specific to the memory Flanker Task, while perceptual ERPs tended to be more stable during the course of the task. Second, multivariate analyses with no a priori region of interest confirmed the presence of distinct patterns of neural activity between perceptual and memory domains. Crucially, this does not imply that the neural origin of the ERN and Pe varies by cognitive domain. In fact, the neural source of error monitoring has been consistently shown to be localized in the ACC (Brázdil et al., 2005; Debener, 2005; Dehaene et al., 1994; Reinhart & Woodman, 2014; van Veen & Carter, 2002) and to be common across different tasks (Mathewson et al., 2005). Instead, it is possible that domain-specific activity derives from discrete neural activation within the ACC, or within a more widespread overlapping neural network, in line with recent a fMRI-MVPA study on neural correlates of metacognition across domains (Morales et al., 2018). Further research combining EEG and fMRI should attempt to decode regional and network activity associated with performance monitoring ERPs across cognitive domains. Taken together, the findings support models of cognitive awareness which postulated the existence of local and global processes of performance monitoring contributing to the emergence of self-awareness (R. G. Morris & Mograbi, 2013), and are in line with recent neuroanatomical models in which metacognitive functions are believed to rely on domain-specific and -general hubs (Seow et al., 2021).

Consistently with previous research, older adults were slower but, overall, their performance did not differ from young adults (Beste et al., 2009; Endrass et al., 2012; Harty et al., 2017; Schreiber et al., 2011). No group differences were found

for ERN/CRN amplitude, and CRN latency, while ERN latencies were longer for older than for young adults. In older adults, Pe/Pc amplitude was reduced, and memory Pe latencies were longer, thus suggesting a decline in processes underlying error awareness despite efficient implicit error detection. ERN and Pe results replicate the most recent studies on aging using a Flanker Task (Clawson et al., 2017; Larson et al., 2016), while inconsistent with previous research with lower sample size ($n \leq 20$; Beste et al., 2009; Hoffmann & Falkenstein, 2011; Mathewson et al., 2005; Nieuwenhuis et al., 2002; Schreiber et al., 2011) size as highlighted by Larson et al. (2016). Thus, these findings suggest that aging is not characterized by a general decline in monitoring mechanisms (Thurm et al., 2020). In particular, older adults seem to be less aware of error commission and this could be a result of difficulties with accumulating evidence about response outcomes. Evidence accumulation processes are thought to integrate inputs from multiple systems such as cognitive, sensory, proprioceptive, and interoceptive (Desender et al., 2021; Ullsperger et al., 2010). Age-related sensorimotor decline encompasses a series of changes in sensory encoding and, integration, that are likely to diminish the quality of the evidence used in decisional processes (McGovern et al., 2018) which may lead to impaired error awareness in older adults.

When exploring within-task changes, ERN amplitude decreased throughout task performance in young adults but not in older adults. It has been proposed that ERN attenuation may reflect reduction in error salience or motivation (Volpert-Esmond et al., 2018), which seem to be reflected in a slight decrease of accuracy over time in young adults. Interestingly, memory Pe decreased in young adults and increased in older adults as function of number of errors, while perceptual Pe remained stable during the memory Flanker Task performance, suggesting that improvement in conscious processing of errors may be specific to memory in older adults because of learning effects in monitoring mnemonic representations. A similar phenomenon was not observed for perceptual decisions that are based on tracking stimuli sensory properties and their expectations (Summerfield & de Lange, 2014), possibly because of age-related sensory deficits (McGovern et al., 2018). Therefore, older adults may benefit from using elaborative alternative strategies (Zakrzewski et al., 2021), based on tracking non-sensory evidence to monitor performance and boost error awareness. Hence, Pe may be a useful marker for assessing cognitive status

across domains in patients with cognitive impairment or affected by neurological deficits and, and for implementing tailored training or rehabilitation programs (Lenzoni et al., 2022) based on successful compensatory mechanisms that allow error-based (re)learning (Ownsworth et al., 2017).

Furthermore, domain-specific changes during memory monitoring may reflect increased motivational significance. Pe has been previously associated with affective responses and error salience (Overbeek et al., 2005), and it has been proposed that the salience network, which is involved in processing of motivationally or personally relevant information, plays a crucial role in the emergence of error awareness (Ullsperger et al., 2010). Memory concerns are common among older adults, experiencing phenomena such as "dementia worry" (Kessler et al., 2012), or fear of forgetting, thus increasing personal relevance of memory failures and memory performance in aging (Reese & Cherry, 2004). Consequently, changes in Pe during memory performance may be mediated by increasing frustration or emotional reactivity as more errors are committed.

Self-reported confidence about performance was found to be similar across domains and stable during the task performance. Older adults reported higher confidence than young adults, despite similar behavioural performance, suggesting that older adults may overestimate their abilities in line with previous research (Cauvin et al., 2019; Dodson et al., 2007; Hansson et al., 2008; Hertzog et al., 2021). Overall, ERN but not Pe was found to be associated with performance confidence. These findings may seem to diverge from previous evidence on the relation between error awareness and Pe (Desender et al., 2021; Murphy et al., 2012; Nieuwenhuis et al., 2001). However, in the current study participants were asked to rate their performance at the end of each block, therefore confidence judgments refer to the global performance rather than response correctness. Nonetheless, the association between larger ERN and higher confidence may suggest that ERN reflects indirect effects of trait-like characteristics of error monitoring, in line with previous research describing the association with anxiety and depression (Clayson et al., 2020; Weinberg et al., 2015), while Pe is more likely to reflect trial-based metacognitive processes (Desender et al., 2021). However, including confidence ratings after each trial in study is more appropriate for tasks with smaller number of trials because of time-related issues such as attention and fatigue. Future research should investigate

performance monitoring and individual differences in relation to both global and trial-level measures of confidence (e.g., metacognitive efficiency; Fleming, 2017; Maniscalco & Lau, 2012) to confirm this dissociation.

Another potential methodological limitation is the use of different stimuli in the two version of the Flanker Task. The perceptual Flanker includes only symbol-like stimuli (i.e., arrows) while the memory adapted version employs more complex stimuli like objects and animals as well as symbols. However, investigating old/new effects in ERPs tasks with larger number of trials require a large number of stimuli. Moreover, one of the strengths of the study is investigating error monitoring in two domains of the same task, while previous research compared very different experimental paradigms, based on different cognitive processes (e.g., Go/NoGo vs Flanker Task or source monitoring vs Flanker Task) and with different tasks characteristic that my bias findings (Falkenstein, 2004; Fischer et al., 2017; Hoffmann & Falkenstein, 2010; Johannes et al., 2002; S. E. Morris et al., 2006; Pailing & Segalowitz, 2004). Furthermore, current research on domain-specificity of metacognitive abilities in perceptual and memory domains has employed a similar approach (McWilliams et al., 2022).

In summary, the current study demonstrates the existence of domain-specific mechanisms underlying performance monitoring. It was found that implicit processes of performance monitoring were preserved in older adults with an age-related decline in error awareness, as reflected by reduced Pe. Moreover, we speculated that within-task Pe changes may reflect domain-specific compensatory strategies to overcome sensory deficits in older age. Our findings provide relevant insights into neurophysiological bases of self-monitoring and have relevant implications for clinical assessment and intervention of domain-specific cognitive impairments.

References

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2014). Fitting Linear Mixed-Effects Models using lme4. Journal of Statistical Software. http://arxiv.org/abs/1406.5823

Bender, A. R., & Raz, N. (2012). Age-Related differences in recognition memory for items and associations: Contribution of individual differences in working memory and metamemory. Psychology and Aging, 27(3). https://doi.org/10.1037/a0026714

Bertrand, E., Landeira-Fernandez, J., & Mograbi, D. C. (2016). Metacognition and Perspective-Taking in Alzheimer's Disease: A Mini-Review. Frontiers in Psychology, 7(NOV). https://doi.org/10.3389/fpsyg.2016.01812

Beste, C., Willemssen, R., Saft, C., & Falkenstein, M. (2009). Error processing in normal aging and in basal ganglia disorders. Neuroscience, 159(1), 143–149. https://doi.org/10.1016/j.neuroscience.2008.12.030

Boldt, A., & Yeung, N. (2015). Shared neural markers of decision confidence and error detection. Journal of Neuroscience, 35(8), 3478–3484. https://doi.org/10.1523/JNEUROSCI.0797-14.2015

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. Psychological Review, 108(3), 624–652. https://doi.org/10.1037/0033-295X.108.3.624

Bradley, A. P. (1997). The use of the area under the ROC curve in the evaluation of machine learning algorithms. Pattern Recognition, 30(7). https://doi.org/10.1016/S0031-3203(96)00142-2

Brázdil, M., Roman, R., Daniel, P., & Rektor, I. (2005). Intracerebral Error-Related Negativity in a Simple Go/NoGo Task. Journal of Psychophysiology, 19(4), 244–255. https://doi.org/10.1027/0269-8803.19.4.244

Brune, M., Dimaggio, G., & H. Lysaker, P. (2011). Metacognition and Social Functioning in Schizophrenia: Evidence, Mechanisms of Influence and Treatment Implications. Current Psychiatry Reviews, 7(3). https://doi.org/10.2174/157340011797183210

Bryce, D., Whitebread, D., & Szűcs, D. (2015). The relationships among executive functions, metacognitive skills and educational achievement in 5 and 7 year-old children. Metacognition and Learning, 10(2), 181–198. https://doi.org/10.1007/s11409-014-9120-4

Capuana, L. J., Dywan, J., Tays, W. J., & Segalowitz, S. J. (2012). Cardiac workload and inhibitory control in younger and older adults. Biological Psychology, 90(1), 60–70. https://doi.org/10.1016/j.biopsycho.2012.02.018

Cauvin, S., Moulin, C. J. A., Souchay, C., Kliegel, M., & Schnitzspahn, K. M. (2019). Prospective Memory Predictions in Aging: Increased Overconfidence in Older Adults. Experimental Aging Research, 45(5). https://doi.org/10.1080/0361073X.2019.1664471

Clawson, A., Clayson, P. E., Keith, C. M., Catron, C., & Larson, M. J. (2017). Conflict and performance monitoring throughout the lifespan: An event-related potential (ERP) and temporospatial component analysis. Biological Psychology, 124, 87–99. https://doi.org/10.1016/j.biopsycho.2017.01.012

Clayson, P. E., Baldwin, S. A., Rocha, H. A., & Larson, M. J. (2021). The data-processing multiverse of event-related potentials (ERPs): A roadmap for the optimization and standardization of ERP processing and reduction pipelines. NeuroImage, 245. https://doi.org/10.1016/j.neuroimage.2021.118712

Clayson, P. E., Carbine, K. A., & Larson, M. J. (2020). A registered report of error-related negativity and reward positivity as biomarkers of depression: P-Curving the evidence. International Journal of Psychophysiology, 150, 50–72. https://doi.org/10.1016/j.ijpsycho.2020.01.005

Debener, S. (2005). Trial-by-Trial Coupling of Concurrent Electroencephalogram and Functional Magnetic Resonance Imaging Identifies the Dynamics of Performance Monitoring. Journal of Neuroscience, 25(50), 11730–11737. https://doi.org/10.1523/JNEUROSCI.3286-05.2005

Dehaene, S. (2018). The Error-Related Negativity, Self-Monitoring, and Consciousness. Perspectives on Psychological Science, 13(2), 161–165. https://doi.org/10.1177/1745691618754502

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. Psychological Science. https://doi.org/10.1111/j.1467-9280.1994.tb00630.x

Delorme, A., & Makeig, S. (2004). EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. Journal of Neuroscience Methods, 134(1), 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009

Derrfuss, J., Danielmeier, C., Klein, T. A., Fischer, A. G., & Ullsperger, M. (2022). Unbiased post-error slowing in interference tasks: A confound and a simple solution. Behavior Research Methods, 54(3). https://doi.org/10.3758/s13428-021-01673-8

Desender, K., Boldt, A., Verguts, T., & Donner, T. H. (2019). Confidence predicts speed-accuracy tradeoff for subsequent decisions. ELife, 8. https://doi.org/10.7554/eLife.43499

Desender, K., Ridderinkhof, K. R., & Murphy, P. R. (2021). Understanding neural signals of post-decisional performance monitoring: An integrative review. ELife, 10. https://doi.org/10.7554/eLife.67556

Dhar, M., Wiersema, J. R., & Pourtois, G. (2011). Cascade of neural events leading from error commission to subsequent awareness revealed using EEG source imaging. PLoS ONE, 6(5), 19–22. https://doi.org/10.1371/journal.pone.0019578

di Gregorio, F., Maier, M. E., & Steinhauser, M. (2018). Errors can elicit an error positivity in the absence of an error negativity: Evidence for independent systems of human error monitoring. NeuroImage, 172(September 2017), 427–436. https://doi.org/10.1016/j.neuroimage.2018.01.081

Dodson, C. S., Bawa, S., & Krueger, L. E. (2007). Aging, metamemory, and high-confidence errors: A misrecollection account. Psychology and Aging, 22(1). https://doi.org/10.1037/0882-7974.22.1.122

Dunlosky, J., & Metcalfe, J. (2009). Metacognition. Sage Publications.

Dywan, J., Mathewson, K. J., Choma, B. L., Rosenfeld, B., & Segalowitz, S. J. (2008). Autonomic and electrophysiological correlates of emotional intensity in

older and younger adults. Psychophysiology, 45(3), 389–397. https://doi.org/10.1111/j.1469-8986.2007.00637.x

Efrati, Y., Kolubinski, D. C., Marino, C., & Spada, M. M. (2021). Modelling the contribution of metacognitions, impulsiveness and thought suppression to behavioural addictions in adolescents. International Journal of Environmental Research and Public Health, 18(7). https://doi.org/10.3390/ijerph18073820

Enders, C. K., & Tofighi, D. (2007). Centering predictor variables in cross-sectional multilevel models: A new look at an old issue. Psychological Methods, 12(2), 121–138. https://doi.org/10.1037/1082-989X.12.2.121

Endrass, T., Reuter, B., & Kathmann, N. (2007). ERP correlates of conscious error recognition: Aware and unaware errors in an antisaccade task. European Journal of Neuroscience, 26(6), 1714–1720. https://doi.org/10.1111/j.1460-9568.2007.05785.x

Endrass, T., Schreiber, M., & Kathmann, N. (2012a). Speeding up older adults: Age-effects on error processing in speed and accuracy conditions. Biological Psychology, 89(2), 426–432. https://doi.org/10.1016/j.biopsycho.2011.12.005

Endrass, T., Schreiber, M., & Kathmann, N. (2012b). Speeding up older adults: Age-effects on error processing in speed and accuracy conditions. Biological Psychology, 89(2), 426–432. https://doi.org/10.1016/j.biopsycho.2011.12.005

Eppinger, B., & Kray, J. (2011). To Choose or to Avoid: Age Differences in Learning from Positive and Negative Feedback. Journal of Cognitive Neuroscience, 23(1), 41–52. https://doi.org/10.1162/jocn.2009.21364

Eppinger, B., Kray, J., Mecklinger, A., & John, O. (2007). Age differences in task switching and response monitoring: Evidence from ERPs. Biological Psychology, 75(1). https://doi.org/10.1016/j.biopsycho.2006.12.001

Eppinger, B., Kray, J., Mock, B., & Mecklinger, A. (2008). Better or worse than expected? Aging, learning, and the ERN. Neuropsychologia, 46(2), 521–539. https://doi.org/10.1016/j.neuropsychologia.2007.09.001

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. Perception & Psychophysics, 16(1), 143–149. https://doi.org/10.3758/BF03203267

Fahrenfort, J. J., Grubert, A., Olivers, C. N. L., & Eimer, M. (2017). Multivariate EEG analyses support high-resolution tracking of feature-based attentional selection. Scientific Reports, 7(1). https://doi.org/10.1038/s41598-017-01911-0

Fahrenfort, J. J., van Driel, J., van Gaal, S., & Olivers, C. N. L. (2018). From ERPs to MVPA using the Amsterdam Decoding and Modeling toolbox (ADAM). Frontiers in Neuroscience, 12(JUL). https://doi.org/10.3389/fnins.2018.00368

Falkenstein, M. (2004). Errors, Conflicts, and the Brain: A Review of the Contributions to the Error Conference, Dortmund 2003. Journal of Psychophysiology, 18(4).

Falkenstein, M., Hielscher, H., Dziobek, I., Schwarzenau, P., Hoormann, J., Sundermann, B., & Hohnsbein, J. (2001). Action monitoring, error detection, and the basal ganglia: An ERP study. NeuroReport, 12(1), 157–161. https://doi.org/10.1097/00001756-200101220-00039

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. Electroencephalography and Clinical Neurophysiology, 78(6), 447–455. https://doi.org/10.1016/0013-4694(91)90062-9

Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: A tutorial. Biological Psychology. https://doi.org/10.1016/S0301-0511(99)00031-9

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (2001). Changes of error-related ERPs with age. Experimental Brain Research, 138(2), 258–262. https://doi.org/10.1007/s002210100712

Fischer, A. G., Klein, T. A., & Ullsperger, M. (2017). Comparing the error-related negativity across groups: The impact of error- and trial-number differences. Psychophysiology, 54(7), 998–1009. https://doi.org/10.1111/psyp.12863

Flavell, J. H. (1979). Metacognition and cognitive monitoring: A new area of cognitive-developmental inquiry. American Psychologist, 34(10). https://doi.org/10.1037/0003-066X.34.10.906

Fleming, S. M. (2017). HMeta-d: hierarchical Bayesian estimation of metacognitive efficiency from confidence ratings. Neuroscience of Consciousness, 2017(1). https://doi.org/10.1093/nc/nix007

Fleming, S. M., & Dolan, R. J. (2012). The neural basis of metacognitive ability. In Philosophical Transactions of the Royal Society B: Biological Sciences (Vol. 367, Issue 1594). https://doi.org/10.1098/rstb.2011.0417

Fleming, S. M., Dolan, R. J., & Frith, C. D. (2012). Metacognition: Computation, biology and function. In Philosophical Transactions of the Royal Society B: Biological Sciences (Vol. 367, Issue 1594). https://doi.org/10.1098/rstb.2012.0021

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A Neural System for Error Detection and Compensation. Psychological Science, 4(6), 385–390. https://doi.org/10.1111/j.1467-9280.1993.tb00586.x

Grootswagers, T., Wardle, S. G., & Carlson, T. A. (2017). Decoding dynamic brain patterns from evoked responses: A tutorial on multivariate pattern analysis applied to time series neuroimaging data. Journal of Cognitive Neuroscience, 29(4). https://doi.org/10.1162/jocn_a_01068

Gründler, T. O. J., Cavanagh, J. F., Figueroa, C. M., Frank, M. J., & Allen, J. J. B. (2009). Task-related dissociation in ERN amplitude as a function of obsessive-compulsive symptoms. Neuropsychologia, 47(8–9), 1978–1987. https://doi.org/10.1016/j.neuropsychologia.2009.03.010

Halamish, V., McGillivray, S., & Castel, A. D. (2011). Monitoring One's Own Forgetting in Younger and Older Adults. Psychology and Aging, 26(3). https://doi.org/10.1037/a0022852

Hallam, B., Chan, J., Gonzalez Costafreda, S., Bhome, R., & Huntley, J. (2020). What are the neural correlates of meta-cognition and anosognosia in Alzheimer's disease? A systematic review. In Neurobiology of Aging (Vol. 94). https://doi.org/10.1016/j.neurobiolaging.2020.06.011

Hansson, P., Rönnlund, M., Juslin, P., & Nilsson, L. G. (2008). Adult Age Differences in the Realism of Confidence Judgments: Overconfidence, Format Dependence, and Cognitive Predictors. Psychology and Aging, 23(3). https://doi.org/10.1037/a0012782

Harty, S., Murphy, P. R., Robertson, I. H., & O'Connell, R. G. (2017). Parsing the neural signatures of reduced error detection in older age. NeuroImage, 161, 43–55. https://doi.org/10.1016/j.neuroimage.2017.08.032

Hebart, M. N., & Baker, C. I. (2018). Deconstructing multivariate decoding for the study of brain function. In NeuroImage (Vol. 180). https://doi.org/10.1016/j.neuroimage.2017.08.005

Herbert, M., Eppinger, B., & Kray, J. (2011). Younger but Not Older Adults Benefit from Salient Feedback during Learning. Frontiers in Psychology, 2(AUG). https://doi.org/10.3389/fpsyg.2011.00171

Herrmann, M. J., Römmler, J., Ehlis, A.-C., Heidrich, A., & Fallgatter, A. J. (2004). Source localization (LORETA) of the error-related-negativity (ERN/Ne) and positivity (Pe). Cognitive Brain Research, 20(2), 294–299. https://doi.org/10.1016/j.cogbrainres.2004.02.013

Hertzog, C., Curley, T., & Dunlosky, J. (2021). Are age differences in recognition-based retrieval monitoring an epiphenomenon of age differences in memory? Psychology and Aging, 36(2). https://doi.org/10.1037/pag0000595

Hertzog, C., & Dunlosky, J. (2011). Metacognition in later adulthood: Spared monitoring can benefit older adults' self-regulation. In Current Directions in Psychological Science (Vol. 20, Issue 3). https://doi.org/10.1177/0963721411409026

Hoffmann, S., & Falkenstein, M. (2010). Independent component analysis of erroneous and correct responses suggests online response control. Human Brain Mapping, 31(9), 1305–1315. https://doi.org/10.1002/hbm.20937

Hoffmann, S., & Falkenstein, M. (2011). Aging and error processing: Age related increase in the variability of the error-negativity is not accompanied by increase in response variability. PLoS ONE, 6(2). https://doi.org/10.1371/journal.pone.0017482

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. Psychological Review. https://doi.org/10.1037/0033-295X.109.4.679

Huff, M. J., Meade, M. L., & Hutchison, K. A. (2011). Age-related differences in guessing on free and forced recall tests. Memory, 19(4). https://doi.org/10.1080/09658211.2011.568494

Johannes, S., Wieringa, B. M., Nager, W., Müller-Vahl, K. R., Dengler, R., & Münte, T. F. (2002). Excessive action monitoring in Tourette syndrome. Journal of Neurology, 249(8), 961–966. https://doi.org/10.1007/s00415-002-0657-9

Kessler, E. M., Bowen, C. E., Baer, M., Froelich, L., & Wahl, H. W. (2012). Dementia worry: A psychological examination of an unexplored phenomenon. European Journal of Ageing, 9(4). https://doi.org/10.1007/s10433-012-0242-8

Kirschner, H., Humann, J., Derrfuss, J., Danielmeier, C., & Ullsperger, M. (2021). Neural and behavioral traces of error awareness. Cognitive, Affective and Behavioral Neuroscience, 21(3). https://doi.org/10.3758/s13415-020-00838-w

Klawohn, J., Meyer, A., Weinberg, A., & Hajcak, G. (2020). Methodological choices in event-related potential (ERP) research and their impact on internal consistency reliability and individual differences: An examination of the error-related negativity (ERN) and anxiety. Journal of Abnormal Psychology, 129(1). https://doi.org/10.1037/abn0000458

Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2017). lmerTest Package: Tests in Linear Mixed Effects Models. Journal of Statistical Software, 82(13). https://doi.org/10.18637/jss.v082.i13

Laghi, F., Pompili, S., Bianchi, D., Lonigro, A., & Baiocco, R. (2020). Dysfunctional metacognition processes as risk factors for drunkorexia during adolescence. Journal of Addictive Diseases, 38(3). https://doi.org/10.1080/10550887.2020.1756709

Larson, M. J., Clayson, P. E., Keith, C. M., Hunt, I. J., Hedges, D. W., Nielsen, B. L., & Call, V. R. A. (2016). Cognitive control adjustments in healthy older and younger adults: Conflict adaptation, the error-related negativity (ERN), and

evidence of generalized decline with age. Biological Psychology, 115, 50–63. https://doi.org/10.1016/j.biopsycho.2016.01.008

Lenzoni, S., Baker, J., Sumich, A. L., & Mograbi, D. C. (2022). New insights into neural networks of error monitoring and clinical implications: a systematic review of ERP studies in neurological diseases. Reviews in the Neurosciences, 33(2), 161–179. https://doi.org/10.1515/revneuro-2021-0054

Lenzoni, S., Morris, R. G., & Mograbi, D. C. (2020). The Petrified Self 10 Years After: Current Evidence for Mnemonic anosognosia. Frontiers in Psychology, 11. https://doi.org/10.3389/fpsyg.2020.00465

Li, H. J., Hou, X. H., Liu, H. H., Yue, C. L., Lu, G. M., & Zuo, X. N. (2015). Putting age-related task activation into large-scale brain networks: A meta-analysis of 114 fMRI studies on healthy aging. In Neuroscience and Biobehavioral Reviews (Vol. 57). https://doi.org/10.1016/j.neubiorev.2015.08.013

Luck, S. J. (2014). An Introduction to the Event-Related Potential Technique, second edition. In The MIT Press.

Maniscalco, B., & Lau, H. (2012). A signal detection theoretic approach for estimating metacognitive sensitivity from confidence ratings. Consciousness and Cognition, 21(1). https://doi.org/10.1016/j.concog.2011.09.021

Mathalon, D. H., Bennett, A., Askari, N., Gray, E. M., Rosenbloom, M. J., & Ford, J. M. (2003). Response-monitoring dysfunction in aging and Alzheimer's disease: an event-related potential study. Neurobiology of Aging, 24(5), 675–685. https://doi.org/10.1016/S0197-4580(02)00154-9

Mathews, C. A., Perez, V. B., Delucchi, K. L., & Mathalon, D. H. (2012). Error-related negativity in individuals with obsessive-compulsive symptoms: Toward an understanding of hoarding behaviors. Biological Psychology, 89(2), 487–494. https://doi.org/10.1016/j.biopsycho.2011.12.018

Mathewson, K. J., Dywan, J., & Segalowitz, S. J. (2005). Brain bases of error-related ERPs as influenced by age and task. Biological Psychology, 70(2), 88–104. https://doi.org/10.1016/j.biopsycho.2004.12.005

McDonald, A. P., D'Arcy, R. C. N., & Song, X. (2018). Functional MRI on executive functioning in aging and dementia: A scoping review of cognitive tasks. In Aging Medicine (Vol. 1, Issue 2). https://doi.org/10.1002/agm2.12037

McGovern, D. P., Hayes, A., Kelly, S. P., & O'Connell, R. G. (2018). Reconciling age-related changes in behavioural and neural indices of human perceptual decision-making. Nature Human Behaviour, 2(12). https://doi.org/10.1038/s41562-018-0465-6

McWilliams, A., Bibby, H., Steinbeis, N., David, A. S., & Fleming, S. M. (2022, February 24). Age-related decreases in global metacognition are independent of local metacognition and task performance. PsyArXiv.

Metcalfe, J., & Schwartz, B. L. (2016). The ghost in the machine: Self-reflective consciousness and the neuroscience of metacognition. Oxford Handbook of Metamemory.

Meyer, A., Bress, J. N., & Proudfit, G. H. (2014). Psychometric properties of the error-related negativity in children and adolescents. Psychophysiology, 51(7), 602–610. https://doi.org/10.1111/psyp.12208

Mograbi, D. C., Brown, R. G., & Morris, R. G. (2009). Anosognosia in Alzheimer's disease – The petrified self. Consciousness and Cognition, 18(4), 989–1003. https://doi.org/10.1016/j.concog.2009.07.005

Morales, J., Lau, H., & Fleming, S. M. (2018). Domain-general and domain-specific patterns of activity supporting metacognition in human prefrontal cortex. Journal of Neuroscience, 38(14), 3534–3546. https://doi.org/10.1523/JNEUROSCI.2360-17.2018

Morris, R. G., & Mograbi, D. C. (2013). Anosognosia, autobiographical memory and self knowledge in Alzheimer's disease. Cortex, 49(6), 1553–1565. https://doi.org/10.1016/j.cortex.2012.09.006

Morris, S. E., Yee, C. M., & Nuechterlein, K. H. (2006). Electrophysiological analysis of error monitoring in schizophrenia. Journal of Abnormal Psychology, 115(2), 239–250. https://doi.org/10.1037/0021-843X.115.2.239

Moses-Payne, M. E., Habicht, J., Bowler, A., Steinbeis, N., & Hauser, T. U. (2021). I know better! Emerging metacognition allows adolescents to ignore false advice. Developmental Science, 24(5). https://doi.org/10.1111/desc.13101

Murphy, P. R., Robertson, I. H., Allen, D., Hester, R., & O'Connell, R. G. (2012). An electrophysiological signal that precisely tracks the emergence of error awareness. Frontiers in Human Neuroscience, 6(MARCH 2012), 1–16. https://doi.org/10.3389/fnhum.2012.00065

Nieuwenhuis, S., Richard Ridderinkhof, K., Blom, J., Band, G. P. H., & Kok, A. (2001). Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. Psychophysiology, 38(5), 752–760. https://doi.org/10.1017/S0048577201001111

Nieuwenhuis, S., Ridderinkhof, K. R., Talsma, D., Coles, M. G. H., Holroyd, C. B., Kok, A., & van der Molen, M. W. (2002). A computational account of altered error processing in older age: Dopamine and the error-related negativity. Cognitive, Affective, & Behavioral Neuroscience, 2(1), 19–36. https://doi.org/10.3758/CABN.2.1.19

O'Connell, R. G., Dockree, P. M., Bellgrove, M. A., Kelly, S. P., Hester, R., Garavan, H., Robertson, I. H., & Foxe, J. J. (2007). The role of cingulate cortex in the detection of errors with and without awareness: A high-density electrical mapping study. European Journal of Neuroscience, 25(8), 2571–2579. https://doi.org/10.1111/j.1460-9568.2007.05477.x

Ohtani, K., & Hisasaka, T. (2018). Beyond intelligence: a meta-analytic review of the relationship among metacognition, intelligence, and academic performance. Metacognition and Learning, 13(2). https://doi.org/10.1007/s11409-018-9183-8

Onoda, K., Ishihara, M., & Yamaguchi, S. (2012). Decreased functional connectivity by aging is associated with cognitive decline. Journal of Cognitive Neuroscience, 24(11). https://doi.org/10.1162/jocn_a_00269

Overbeek, T. J. M., Nieuwenhuis, S., & Ridderinkhof, K. R. (2005). Dissociable components of error processing: On the functional significance of the Pe vis-à-vis the ERN/Ne. In Journal of Psychophysiology (Vol. 19, Issue 4, pp. 319–329). https://doi.org/10.1027/0269-8803.19.4.319

Ownsworth, T., Fleming, J., Tate, R., Beadle, E., Griffin, J., Kendall, M., Schmidt, J., Lane-Brown, A., Chevignard, M., & Shum, D. H. K. (2017). Do People With Severe Traumatic Brain Injury Benefit From Making Errors? A Randomized Controlled Trial of Error-Based and Errorless Learning. Neurorehabilitation and Neural Repair, 31(12), 1072–1082. https://doi.org/10.1177/1545968317740635

Pailing, P. E., & Segalowitz, S. J. (2004). The effects of uncertainty in error monitoring on associated ERPs. Brain and Cognition, 56(2 SPEC. ISS.), 215–233. https://doi.org/10.1016/j.bandc.2004.06.005

Palmer, E. C., David, A. S., & Fleming, S. M. (2014). Effects of age on metacognitive efficiency. Consciousness and Cognition, 28(1). https://doi.org/10.1016/j.concog.2014.06.007

Palmieri, S., Mansueto, G., Ruggiero, G. M., Caselli, G., Sassaroli, S., & Spada, M. M. (2021). Metacognitive beliefs across eating disorders and eating behaviours: A systematic review. In Clinical Psychology and Psychotherapy (Vol. 28, Issue 5). https://doi.org/10.1002/cpp.2573

Peirce, J., Gray, J. R., Simpson, S., MacAskill, M., Höchenberger, R., Sogo, H., Kastman, E., & Lindeløv, J. K. (2019). PsychoPy2: Experiments in behavior made easy. Behavior Research Methods, 51(1), 195–203. https://doi.org/10.3758/s13428-018-01193-y

Perrotin, A., Isingrini, M., Souchay, C., Clarys, D., & Taconnat, L. (2006). Episodic feeling-of-knowing accuracy and cued recall in the elderly: Evidence for double dissociation involving executive functioning and processing speed. Acta Psychologica, 122(1). https://doi.org/10.1016/j.actpsy.2005.10.003

Perry, J., Lundie, D., & Golder, G. (2019). Metacognition in schools: what does the literature suggest about the effectiveness of teaching metacognition in schools? Educational Review, 71(4), 483–500. https://doi.org/10.1080/00131911.2018.1441127

Pietschmann, M., Endrass, T., Czerwon, B., & Kathmann, N. (2011). Aging, probabilistic learning and performance monitoring. Biological Psychology, 86(1), 74–82. https://doi.org/10.1016/j.biopsycho.2010.10.009

Pietschmann, M., Endrass, T., & Kathmann, N. (2011). Age-related alterations in performance monitoring during and after learning. Neurobiology of Aging, 32(7), 1320–1330. https://doi.org/10.1016/j.neurobiolaging.2009.07.016

Qiu, L., Su, J., Ni, Y., Bai, Y., Zhang, X., Li, X., & Wan, X. (2018). The neural system of metacognition accompanying decision-making in the prefrontal cortex. PLOS Biology, 16(4), e2004037. https://doi.org/10.1371/journal.pbio.2004037

R Core Team. (2020). R Core Team 2020. R: A Language and Environment for Statistical Computing.

Reese, C. M., & Cherry, K. E. (2004). Practical memory concerns in adulthood. International Journal of Aging and Human Development, 59(3). https://doi.org/10.2190/8PA5-8KVB-EMNQ-BFKY

Reinhart, R. M. G., & Woodman, G. F. (2014). Causal Control of Medial-Frontal Cortex Governs Electrophysiological and Behavioral Indices of Performance Monitoring and Learning. Journal of Neuroscience, 34(12), 4214–4227. https://doi.org/10.1523/JNEUROSCI.5421-13.2014

Riesel, A. (2019). The erring brain: Error-related negativity as an endophenotype for OCD-A review and meta-analysis. Psychophysiology, 56(4), e13348. https://doi.org/10.1111/psyp.13348

Riesel, A., Weinberg, A., Endrass, T., Meyer, A., & Hajcak, G. (2013). The ERN is the ERN is the ERN? Convergent validity of error-related brain activity across different tasks. Biological Psychology, 93(3), 377–385. https://doi.org/10.1016/j.biopsycho.2013.04.007

Roebers, C. M., Krebs, S. S., & Roderer, T. (2014). Metacognitive monitoring and control in elementary school children: Their interrelations and their role for test performance. Learning and Individual Differences, 29. https://doi.org/10.1016/j.lindif.2012.12.003

Rogier, G., Beomonte Zobel, S., Morganti, W., Ponzoni, S., & Velotti, P. (2021). Metacognition in gambling disorder: A systematic review and meta-analysis. In Addictive Behaviors (Vol. 112). https://doi.org/10.1016/j.addbeh.2020.106600

Rouault, M., McWilliams, A., Allen, M. G., & Fleming, S. M. (2018). Human Metacognition Across Domains: Insights from Individual Differences and Neuroimaging. In Personality Neuroscience (Vol. 1). https://doi.org/10.1017/pen.2018.16

Sanders, E., & Berry, J. (2020). Evidence for an Age-Related Positivity Effect in Metacognitive Confidence Judgments. Innovation in Aging, 4(Supplement_1). https://doi.org/10.1093/geroni/igaa057.1170

Schreiber, M., Pietschmann, M., Kathmann, N., & Endrass, T. (2011). ERP correlates of performance monitoring in elderly. Brain and Cognition, 76(1), 131–139. https://doi.org/10.1016/j.bandc.2011.02.003

Seow, T. X. F., Rouault, M., Gillan, C. M., & Fleming, S. M. (2021). How Local and Global Metacognition Shape Mental Health. In Biological Psychiatry (Vol. 90, Issue 7). https://doi.org/10.1016/j.biopsych.2021.05.013

Soderstrom, N. C., McCabe, D. P., & Rhodes, M. G. (2012). Older adults predict more recollective experiences than younger adults. Psychology and Aging, 27(4). https://doi.org/10.1037/a0029048

Souchay, C., Moulin, C. J. A., Clarys, D., Taconnat, L., & Isingrini, M. (2007). Diminished episodic memory awareness in older adults: Evidence from feeling-of-knowing and recollection. Consciousness and Cognition, 16(4). https://doi.org/10.1016/j.concog.2006.11.002

Staub, B., Doignon-Camus, N., Bacon, É., & Bonnefond, A. (2014). Age-related differences in the recruitment of proactive and reactive control in a situation of sustained attention. Biological Psychology, 103(1), 38–47. https://doi.org/10.1016/j.biopsycho.2014.08.007

Steinhauser, M., & Yeung, N. (2010). Decision processes in human performance monitoring. Journal of Neuroscience, 30(46), 15643–15653. https://doi.org/10.1523/JNEUROSCI.1899-10.2010

Steinhauser, M., & Yeung, N. (2012). Error awareness as evidence accumulation: effects of speed-accuracy trade-off on error signaling. Frontiers in Human Neuroscience, 6. https://doi.org/10.3389/fnhum.2012.00240

Summerfield, C., & de Lange, F. P. (2014). Expectation in perceptual decision making: Neural and computational mechanisms. In Nature Reviews Neuroscience (Vol. 15, Issue 11). https://doi.org/10.1038/nrn3838

Sun, X., Zhu, C., & So, S. H. W. (2017). Dysfunctional metacognition across psychopathologies: A meta-analytic review. In European Psychiatry (Vol. 45). https://doi.org/10.1016/j.eurpsy.2017.05.029

Themanson, J. R., Hillman, C. H., & Curtin, J. J. (2006). Age and physical activity influences on action monitoring during task switching. Neurobiology of Aging, 27(9), 1335–1345. https://doi.org/10.1016/j.neurobiolaging.2005.07.002

Thurm, F., Antonenko, D., Schlee, W., Kolassa, S., Elbert, T., & Kolassa, I. T. (2013). Effects of aging and mild cognitive impairment on electrophysiological correlates of performance monitoring. Journal of Alzheimer's Disease, 35(3), 575–587. https://doi.org/10.3233/JAD-121348

Thurm, F., Li, S.-C., & Hämmerer, D. (2020). Maturation- and aging-related differences in electrophysiological correlates of error detection and error awareness. Neuropsychologia, 143, 107476. https://doi.org/10.1016/j.neuropsychologia.2020.107476

Toth, J. P., Daniels, K. A., & Solinger, L. A. (2011). What you know can hurt you: Effects of age and prior knowledge on the accuracy of judgments of learning. Psychology and Aging, 26(4). https://doi.org/10.1037/a0023379

Ullsperger, M., Harsay, H. A., Wessel, J. R., & Ridderinkhof, K. R. (2010). Conscious perception of errors and its relation to the anterior insula. Brain Structure and Function, 214(5–6), 629–643. https://doi.org/10.1007/s00429-010-0261-1

Vaccaro, A. G., & Fleming, S. M. (2018). Thinking about thinking: A coordinate-based meta-analysis of neuroimaging studies of metacognitive judgements. Brain and Neuroscience Advances, 2, 239821281881059. https://doi.org/10.1177/2398212818810591

van Veen, V., & Carter, C. S. (2002). The tinning of action-monitoring processes in the anterior cingulate cortex. Journal of Cognitive Neuroscience, 14(4), 593–602. https://doi.org/10.1162/08989290260045837

Volpert-Esmond, H. I., Merkle, E. C., Levsen, M. P., Ito, T. A., & Bartholow, B. D. (2018). Using trial-level data and multilevel modeling to investigate within-task change in event-related potentials. Psychophysiology, 55(5), e13044. https://doi.org/10.1111/psyp.13044

Volpert-Esmond, H. I., Page-Gould, E., & Bartholow, B. D. (2021). Using multilevel models for the analysis of event-related potentials. International Journal of Psychophysiology, 162. https://doi.org/10.1016/j.ijpsycho.2021.02.006

Weil, L. G., Fleming, S. M., Dumontheil, I., Kilford, E. J., Weil, R. S., Rees, G., Dolan, R. J., & Blakemore, S. J. (2013). The development of metacognitive ability in adolescence. Consciousness and Cognition, 22(1). https://doi.org/10.1016/j.concog.2013.01.004

Weinberg, A., Dieterich, R., & Riesel, A. (2015). Error-related brain activity in the age of RDoC: A review of the literature. International Journal of Psychophysiology, 98(2), 276–299. https://doi.org/10.1016/j.ijpsycho.2015.02.029

Wessel, J. R., Danielmeier, C., & Ullsperger, M. (2011). Error awareness revisited: Accumulation of multimodal evidence from central and autonomic nervous systems. Journal of Cognitive Neuroscience, 23(10), 3021–3036. https://doi.org/10.1162/jocn.2011.21635

West, R. (2004). The Effects of Aging on Controlled Attention and Conflict Processing in the Stroop Task. Journal of Cognitive Neuroscience, 16(1), 103–113. https://doi.org/10.1162/089892904322755593

Wickens, T. D. (2010). Elementary signal detection theory. In Elementary Signal Detection Theory. https://doi.org/10.1093/acprof:oso/9780195092509.001.0001

Wong, J. T., Cramer, S. J., & Gallo, D. A. (2012). Age-related reduction of the confidence-accuracy relationship in episodic memory: Effects of recollection quality and retrieval monitoring. Psychology and Aging, 27(4). https://doi.org/10.1037/a0027686

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. Psychological Review, 111(4), 931–959. https://doi.org/10.1037/0033-295X.111.4.931

Zakrzewski, A. C., Sanders, E. C., & Berry, J. M. (2021). Evidence for Age-Equivalent and Task-Dissociative Metacognition in the Memory Domain. Frontiers in Psychology, 12. https://doi.org/10.3389/fpsyg.2021.630143

Table 1. Descriptive statistics, mean (SD).

		Younger adults	**Older Adults**
Accuracy (%)	**Perceptual**	0.89 (0.07)	0.90 (0.10)
	Memory	0.89 (0.05)	0.91 (0.07)
RTs (ms)	**Perceptual, Incongruent**	0.48 (0.06)	0.63 (0.09)
	Perceptual, Congruent	0.42 (0.05)	0.54 (0.07)
	Memory, Incongruent	0.53 (0.07)	0.67 (0.07)
	Memory, Congruent	0.51 (0.06)	0.63 (0.07)
PES (ms)	**Perceptual**	0.05 (0.04)	0.07 (0.07)
	Memory	0.06 (0.05)	0.07 (0.05)
Confidence	**Perceptual**	3.31 (0.92)	3.54 (0.86)
	Memory	3.19 (0.94)	3.64 (0.83)

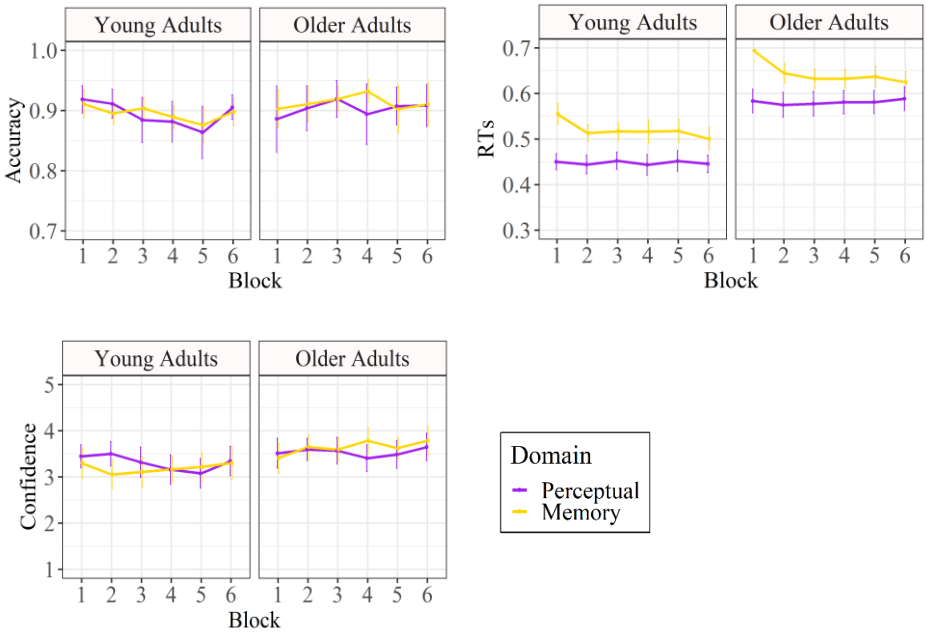

Figure 1. Behavioural performance during the experimental tasks. Top left: mean accuracy rate (%). Top right: mean reaction times (sec). Bottom left: mean confidence ratings.

RTs, reaction times

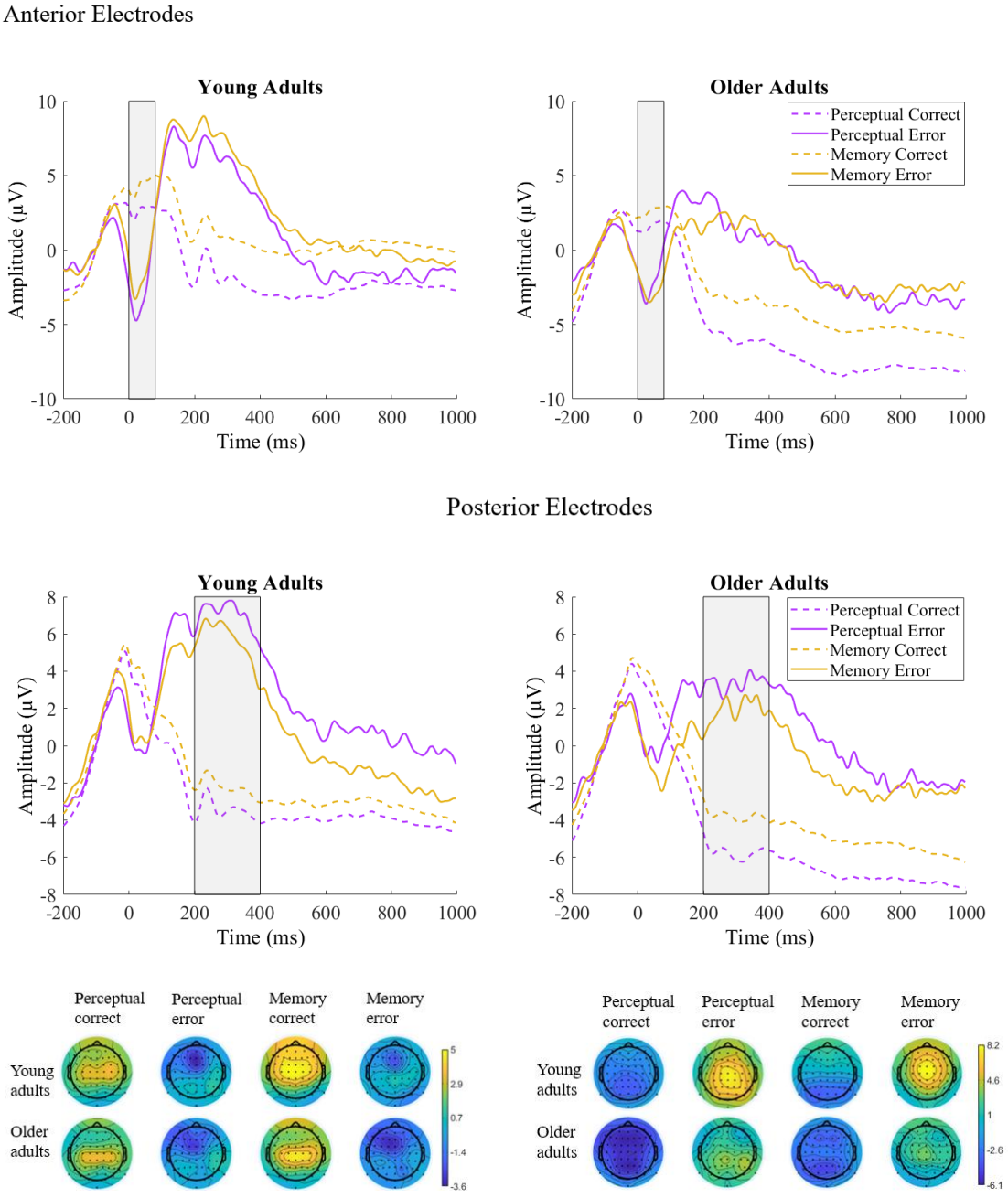

Figure 2. Top plot, Grand-average waveforms for ERN/CRN as average activity over anterior electrodes (F1, F2, FCz, FC1, FC2, FC2) and error positivity (Pe) s as average activity over posterior electrodes (P1, P2, Pz, CP1, CP2, CPz). Bottom plot, topographical distribution for response type in each domain between 0 and 80ms (left) and 200-400ms (right)

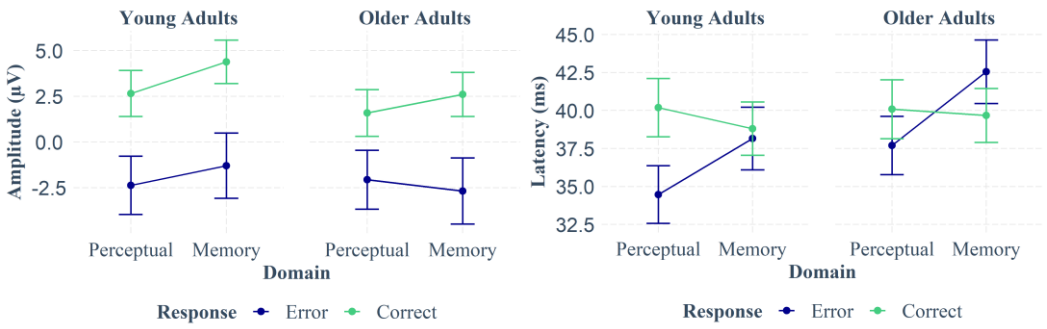

Figure 3. Model estimates and confidence intervals for ERN/CRN amplitude and latency by group and domain.

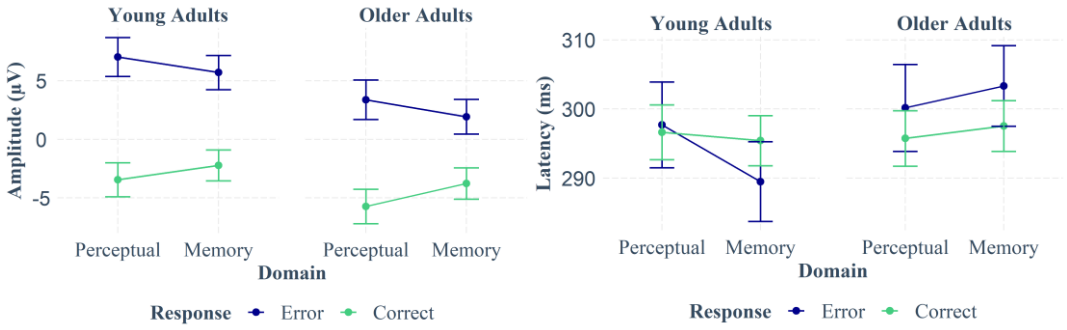

Figure 4. Model estimates and confidence intervals for Pe/Pc amplitude and latency by group and domain

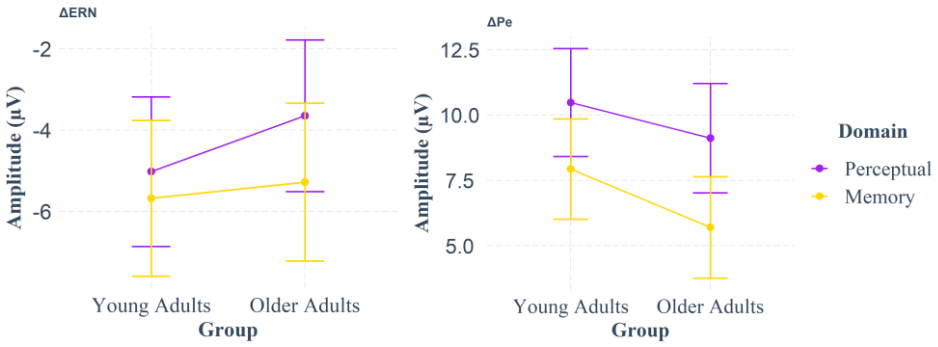

Figure 5. Model estimates and confidence intervals for ΔERN and ΔPe by group and domain.

Table 2. Model estimates for ERN and Pe as function of number of errors.

ERN. error-related negativity; Pe. error positivity; 95% CI, 95% confidence interval

	ERN				
	b	*95% CI*	*df*	*t*	*p*
Intercept	-2.64	-3.86 – -1.42	68.54	-4.31	**<.001**
Error Number	0.03	0.02 – 0.04	27019.40	5.13	**<.001**
Domain	-0.29	-1.21 – 0.64	90.64	-0.62	.539
Group	0.22	-2.02 – 2.47	75.67	0.20	.844
Error Number x Domain	0.03	0.01 – 0.05	21053.51	2.70	**.007**
Error Number x Group	-0.03	-0.05 – -0.01	27019.40	-2.45	**.014**
Domain x Group	-1.21	-3.06 – 0.63	90.64	-1.31	.195
Error Number x Domain x Group	0.01	-0.04 – 0.05	21053.51	0.34	.733

	Pe				
	b	*95% CI*	*df*	*t*	*p*
Intercept	4.34	3.30 – 5.39	54.44	8.34	**<.001**
Error Number	0.01	0.00 – 0.03	27347.50	2.46	**.014**
Domain	-1.78	-3.05 – -0.51	82.67	-2.79	**.006**
Group	-4.33	-6.17 – -2.49	77.53	-4.69	**<.001**
Error Number x Domain	0.05	0.02 – 0.07	25713.54	3.86	**<.001**
Error Number x Group	0.05	0.03 – 0.07	27347.50	4.17	**<.001**
Domain x Group	-1.93	-4.47 – 0.61	82.67	-1.51	**.**134
Error Number x Domain x Group	0.13	0.09 – 0.18	25713.54	5.73	**<.001**

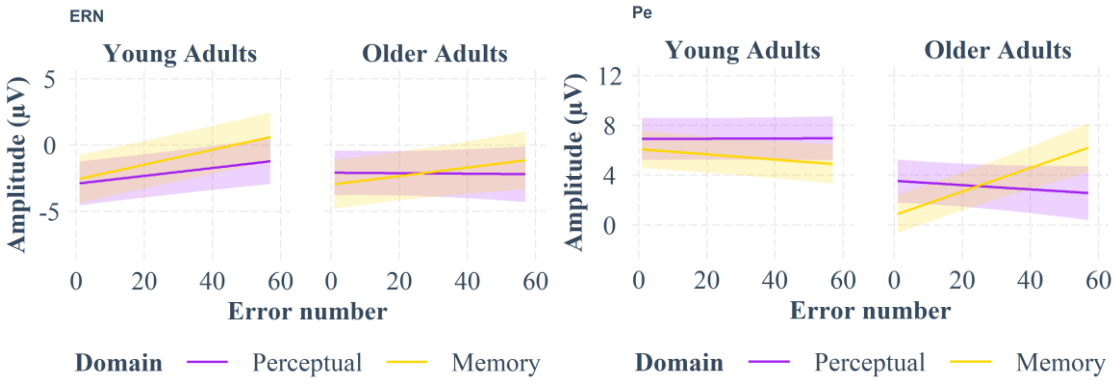

Figure 6. Slopes associated with change in ERN amplitude (left) and Pe amplitude (right) during the task plotted by group and task domain.

Table 3. Model estimates for ERN and Pe in relation to confidence

ERN. error-related negativity; Pe. error positivity; 95% CI, 95% confidence interval

	ERN				
	b	*95% CI*	*df*	*t*	*p*
Intercept	-2.15	-3.36 – -0.93	72.85	-3.51	**.001**
Confidence	-1.39	-2.55 – -0.24	126.28	-2.38	**.019**
Domain	0.16	-0.75 – 1.07	69.87	0.35	.728
Group	0.06	-2.21 – 2.33	75.70	0.05	.957
Confidence x Domain	-1.10	-2.59 – 0.39	73.90	-1.47	.145
Confidence x Group	0.21	-2.10 – 2.53	126.28	0.18	.856
Domain x Group	-0.87	-2.70 – 0.95	69.87	-0.96	.342
Confidence x Domain x Group	-0.24	-3.22 – 2.74	73.90	-0.16	.871

	Pe				
	b	*95% CI*	*df*	*t*	*P*
Intercept	4.34	3.29 – 5.38	60.47	8.29	**<.001**
Confidence	0.40	-0.86 – 1.66	134.51	0.63	.532
Domain	-1.44	-2.71 – -0.18	73.25	-2.27	**.026**
Group	-3.73	-5.61 – -1.85	75.50	-3.96	**<.001**
Confidence x Domain	-0.68	-2.70 – 1.33	86.68	-0.67	.502
Confidence x Group	1.79	-0.73 – 4.31	134.51	1.40	.162
Domain x Group	0.19	-2.35 – 2.73	73.25	0.15	.882
Confidence x Domain x Group	-1.45	-5.49 – 2.58	86.68	-0.72	.475

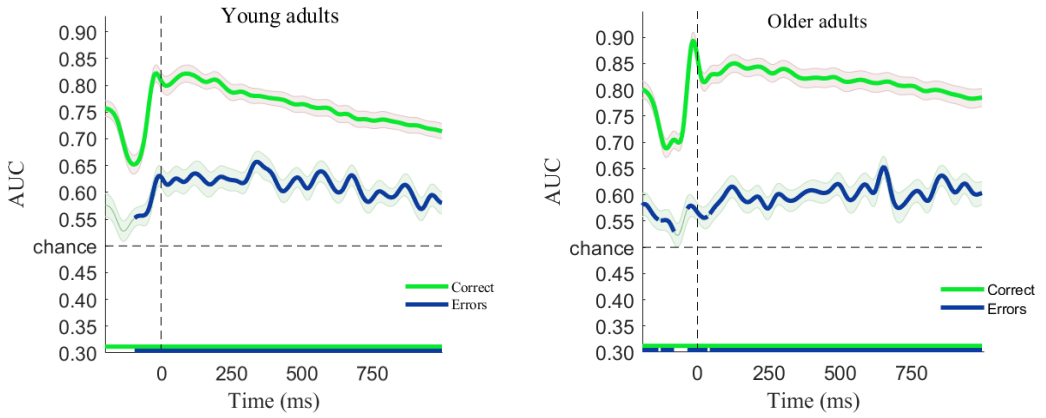

Figure 7. Decoding performance (AUC) for domain contrasts. Chance-level decoding=0.5. Colour bars below indicate significant differences.

Article 4

Lenzoni, S., Sumich, A., & Mograbi, D. (In preparation). Electrophysiology of memory monitoring: the role of sensory processing in age-related changes in error awareness

Abstract

Aging is associated with a decline in conscious processing of errors. The error positivity (Pe) is a response-locked potential reflecting error awareness. It has been recently hypothesized that reduced Pe in older adults may be associated with age-related sensory decline. The present study aimed at investigating neurophysiological mechanisms associated with reduced error awareness in older adults. A group of 38 young adults and 37 younger adults completed a memory monitoring task during EEG recordings. Stimulus-locked potentials were quantified to understand whether different stages of stimulus processing, including sensory and mnemonic mechanisms are associated with reduced error awareness in aging. Specifically, N1, P2 and N2 were extracted to track visual sensory processing while FN400 and LPC, reflecting familiarity and recollection, respectively were used as indexes of memory processes. Pe, N1 were reduced and P2 was larger in older adults and LPC was found to be comparable between age groups. N2 was modulated by the old-new effect in young but not in the older group and FN400 was during incongruent trials in older adults. There was no association between N1 and Pe. Larger P2 was associated with larger P2 in older adults only. Larger N2 and FN400 were associated with larger Pe in young adults and with smaller Pe in older adults. Larger LPC was associated with larger Pe in both young and older adults. Overall, these findings showed that reduced error awareness in older adults is associated with impairments in perceptual processing of stimuli.

Keywords

Error awareness; ERP; Pe; aging; metacognition; metamemory

1. Introduction

The ability to monitor performance, including detecting committed errors, is crucial for learning and to improve or correct behaviours (Ullsperger et al., 2014). Healthy aging has been associated with a decline in error awareness (Hämmerer et al., 2014; Harty et al., 2013; Sim et al., 2020). Poor appreciation of errors in everyday life activities such as cooking, driving, or taking medications has obvious negative consequences, including engagement in dangerous behaviours and failure in medication management (Cooper et al., 2005; Cosentino et al., 2011; Cotrell et al., 2006; Starkstein et al., 2007; Sunderaraman & Cosentino, 2017). Moreover, impaired self-awareness has been associated with poor clinical outcomes and increased caregiver burden (Starkstein, 2014) and may be an index of functional decline in aging (Arora et al., 2021). Therefore, it is crucial to understand mechanisms underlying error awareness in older adults in order to design effective tools to counteract it.

Electrophysiological research has investigated neural markers of performance monitoring, such as a positive component peaking between 200 and 400ms after error commission at centro-parietal sites, named error positivity (Pe; Falkenstein et al., 2001; Overbeek et al., 2005). The Pe has been shown to reflect error awareness (Boldt & Yeung, 2015; Desender et al., 2019, 2021; Endrass et al., 2007; Murphy et al., 2012; Nieuwenhuis et al., 2001), potentially emerging from processes of evidence accumulation (Desender et al., 2021). According to the Evidence Accumulation account, error awareness emerges from the integration of multimodal inputs, such as cognitive, sensory, proprioceptive, and interoceptive signals (Steinhauser & Yeung, 2010, 2012; Wessel et al., 2011). In line with source localization findings (Dhar et al., 2011; Herrmann et al., 2004; O'Connell et al., 2007), a candidate network underlying error awareness includes anterior cingulate cortex, anterior insula and somatosensory regions (Ullsperger et al., 2010).

A growing body of evidence has shown that Pe is reduced in older adults as compared to a young group (Capuana et al., 2012; Clawson et al., 2017; Larson et al., 2016; Mathewson et al., 2005; Thurm et al., 2020). Furthermore, Lenzoni et al. (Submitted) investigated domain-specificity of performance monitoring in aging employing a perceptual and a memory version of the flanker task (Eriksen &

Eriksen, 1974). The findings showed smaller Pe in older adults in both task domains, indicating a general decline in error awareness. However, domain-specific dynamics were observed when examining Pe changes as function of number of errors. Specifically, perceptual Pe was observed to remain stable during the task in young and older adults, while memory Pe was found to decrease in young adults and increase in older adults. Possible interpretations of these findings are that: i) age-related sensory decline contribute to reduced error awareness in older adults through non-efficient use of sensory evidence about error commission; and ii) error awareness improves in the memory task because of learning effects that allow older adults to rely on non-sensory information, such as more complex representation like name-picture association, that can be used to monitor mnemonic processes.

It is well known that visual sensory/perceptual decline is associated with cognitive functioning in aging (for review, see Li & Lindenberger, 2002; Roberts & Allen, 2016). ERP evidence offered further insights into the interplay between perception and cognition in older adults. The N1 peaks between 150 and 200ms at posterior sites and it is commonly associated with visual discrimination, modulated by top-down attentional processes (Hillyard & Anllo-Vento, 1998; Vogel & Luck, 2000). N1 has been to show to be diminished in older age, reflecting the attenuation of early visual sensory processing (Čeponiene et al., 2008; Wiegand et al., 2014). Moreover, frontal P2 has been found to be enhanced in older adults than young adults and this increase may represent an over recruitment of attentional resources (Staub et al., 2014, 2015) to retrieve task relevant information, such as stimulus-response mapping (Finke et al., 2011; Gajewski et al., 2018). Additionally, the N2 is a higher-level visual processing component reflecting conflict detection in interference tasks (Larson et al., 2014). N2 has been found to be smaller in older adults for incongruent trials, which suggests an age-related decline in conflict monitoring and adaptation (Hsieh & Fang, 2012; Larson et al., 2016).

ERP research employing recognition memory paradigms has focused on differences in neural activity between old (studied) and new (non-studied) items (old/new effect; Rugg & Curran, 2007). The FN400 is a midfrontal negative potential, peaking between 300 and 500ms after stimulus onset, which is typically more positive for old than new items (Ally & Budson, 2007; Curran, 2000; Friedman & Johnson, 2000). The late positive complex (LPC) is a later posterior component

occurring between 500 and 800ms, which is larger for old than new items (Curran, 2000; Rugg et al., 1998). According to the dual-process theory, recognition memory relies on two dissociable processes: familiarity, a fast process that occur without rich contextual remembering, and recollection, a slower process that allows retrieval of item-specific information (Parks & Yonelinas, 2007; Yonelinas, 2002). Previous research has demonstrated that FN400 and LPC represents familiarity and recollection, respectively (Allan et al., 1998; Ally, Waring, et al., 2008; Ally & Budson, 2007; Curran, 2000; Curran & Cleary, 2003; Curran & Doyle, 2011; Nyhus & Curran, 2009; Rugg & Curran, 2007). In picture-based tasks, these mnemonic processes were shown to be intact in healthy aging (Ally et al., 2009; Ally, Waring, et al., 2008; James et al., 2016). However, experimental manipulations of stimuli perceptual properties such as object colour (Dulas & Duarte, 2013), rotation (Ally, Simons, et al., 2008), and spatial plausibility (Bridger et al., 2017) have been shown to affect recollection in older adults, as reflected by reduced late parietal effects. To the best of our knowledge, the association between sensory and mnemonic representations and error awareness has not been yet investigated.

The aim of the current study was to understand whether different stages of stimulus processing, including sensory and mnemonic mechanisms may be associated with error awareness. To this end, young and older participants completed a memory flanker task (Lenzoni et al., Submitted). First, we aimed at replicating findings of previous research using this novel paradigm. Specifically, we expected that decreased sensory processing in older adults as reflected by smaller N1, P2, and N2, but spared memory processes (i.e., no differences in FN400 and LPC between young and older adults). Then, we aimed at exploring whether age-related sensory decline contributes to error awareness. Therefore, we expected that N1, P2, and N2 were predictors of Pe amplitude in young adults but not in older adults, while FN400 and LPC were positively associated with Pe in young and older adults, indicating a relation between stronger memory representations and higher error awareness in both groups.

2.Methods

2.1 Participants

38 younger adults (24 females, 14 males) between the ages of 19-34 years (M = 22.4, SD = 4.4) and 37 older adults (23 females, 14 males) between the ages of 60-90 years (M = 70.9, SD = 10. 6) were recruited through Psychology Division Research participation schemes at Nottingham Trent University. Inclusion criteria were normal/corrected vision and fluency in English. Participants were excluded if they have history of neurological and/or psychiatric disorders. The two groups had similar sex ratios ($\chi 2$ (1) < .01, p = .929) and educational levels (W = 572, p = .141). All participants provided written consent and all procedures were approved by Nottingham Trent University College of Business, Law and Social Sciences Ethics Committee.

2.2 Memory Flanker Task

Participants completed a memory version of the Eriksen flanker task (Lenzoni et al., Submitted). The task was created using PsychoPy2 (v1.90.1; Peirce et al., 2019). All stimuli were 2D icons generated by Freepik (www.flaticon.com). All stimuli were displayed on a white background of a 19'' computer monitor (approximately 60cm from participants forehead). In the learning phase, participants memorize four icons (mushroom, chicken, love heart, and shoe): i) each of the four icons is presented at the centre of the screen for 2 seconds; ii) participants are asked to recall the four icons; iii) participants perform a recognition task in which they see eight icons, one by one, and they have to decide by button press whether they have just seen the icon or not; iv) the four icons are displayed at the center of the screen for 2 seconds. The experimental phase took place 20mins following the end of the learning phase to ensure transfer in long-term memory. Before the beginning of the task, participants were asked to recall the four icons they were asked to remember in the learning phase to ensure that retrieval issues would not bias task execution and memory monitoring. In each trial, participants were presented with five icons that could be either all the same (i.e., congruent), or with the central icon being different from the other four icons (i.e., incongruent). Participants were asked to identify by button press whether the central icon (target) was old (one of the four icons memorized in the learning phase) or new and were instructed to respond as quickly and as accurately as possible, while ignoring other icons (flankers).

Participants completed 12 practice trials and 6 blocks of 96 trials for the actual task. In each block half of the trials were congruent and half of the trials were incongruent. For both congruent and incongruent conditions, in half of the trials the target was an old icon and in the other half it was a new icon. At the end of each block participants were asked to rate how confident were about their performance on a scale between 1 and 5. Stimuli were preceded by a fixation cross (500ms), were randomly presented for 100ms, and participants had 1200msec to respond. ITI varied between 500-900ms. Each set of stimuli filled 2.46° of visual angle vertically and 12.36° horizontally. Stimuli were classified as belonging to four categories: A) animals and food B) objects, and symbols. Target and flanker stimuli were different in colour and shape to avoid that the interference effect in incongruent trials could be caused by physical similarities, rather than by old/new effect. Moreover, in each trial, target and flanker stimuli were chosen from different categories, to avoid semantic relatedness effects in incongruent trials. A graphical representation of trials is displayed in Supplementary Material.

2.3 EEG recordings, preprocessing and ERP extraction

A BioSemi Active II system (Biosemi, Amsterdam, The Netherlands) was used to record continuous EEG. Recordings were taken from 64 active scalp electrodes based on the 10/20 system and 2 external electrodes placed on the right and left mastoids. Data were sampled at 2048 Hz, digitized at 24 bits and referenced online with a CMS/DRL feedback loop. Electrodes off-set was kept within the absolute value of 20 µV. EEGLAB (Delorme & Makeig, 2004) and MATLAB (Mathworks, Natick, Massachusetts, USA) were used for off-line analyses. Data were downsampled to 256 Hz and processed through a 0.1 Hz high pass filter and a 30 Hz low-pass filter. Data were re-referenced to average mastoids. Bad channels were removed and interpolated. Data were segmented in epochs, and independent component analysis (ICA) was used to remove ocular artifacts. Epochs exceeding -100 µV and 100 µV were removed.

Stimulus-locked epochs were extracted in a temporal window from 400ms prior to the stimulus presentation to 1200ms following stimulus presentation. The interval between 400ms and 0ms was chosen for baseline correction. Stimulus-locked ERPs

were averaged separately for each condition (old congruent, old incongruent, new congruent, new incongruent). Following visual inspection of participant data, grand-averages and scalp maps, specific time windows for young and older adults were selected to extract ERPs. N1 was quantified as mean amplitude at Cz, C1, C2, CPz, CP1, CP2 in the 70-140ms interval for all participants. P2 was quantified as mean amplitude at Fz, F1, F2, FCZ, FC1, FC2, in the 140-210ms interval for young adults and un the 140-250ms interval for older adults. N2 was quantified as mean amplitude at Fz, F1, F2, FCZ, FC1, FC2, in the 210-300ms interval for young adults and in the 250-330ms interval for older adults. FN400 was as mean amplitude at Fz, F1, F2, FCZ, FC1, FC2, in the 300-450ms interval for young adults and in the 330-450ms interval for older adults. LPC was as mean amplitude at CPz, CP1, CP2, Pz, P1, and P2, in the 430-670ms interval for young adults and in the 450-730ms interval for older adults.

Response-locked epochs were extracted in a temporal window from -200ms prior button presss and 1000ms following button press. The interval between -200ms and 0ms was chosen for baseline correction. ERPs were averaged separately for each type of response (correct responses and errors) and the Pe was quantified as mean amplitude in the interval 200-400ms at CPz, CP1, CP2, Pz, P1, and P2.

2.4 Statistical analysis

All analyses were performed using R (R Core Team, 2020). Accuracy was calculated as percentage of correct responses in each condition. A 2x2x2 mixed-design ANOVA, with group (young and older adults) as a between-subjects factor, and familiarity (old or new) and congruency (congruent and incongruent) as within-subjects factors was conducted to test differences in accuracy and RTs. Multilevel models (MLM) were used to explore differences in ERPs. MLM present multiple advantages for ERP analysis, such as robustness to missing trials and unbalanced designs, inclusion of categorical and continuous variables as independent variables and electrodes as random factors rather than predictors (Volpert-Esmond et al., 2021) and the possibility to explore trial-to-trial variations and within task changes in ERPs (Volpert-Esmond et al., 2018). Maximal model structures including all random slopes and their interaction by participant (Barr, Levy, Scheepers, & Tily,

2013). In the case of convergence problems, models would include random slopes but not their interactions. The models included electrodes as nested random factor (or crossed random factor in case of convergence issues). Fixed effects were effect coded (categorical variables; -0.5,0.5). Stimulus-locked ERPs were entered grand-mean centered (Enders & Tofighi, 2007) as predictors of separate models exploring their association with Pe amplitude. All ERP amplitudes were calculated as average region of interest at each trial. Participants and trials were included as random factors. To fit the models, lme4 package (Bates et al., 2014) was used and lmerTest (Kuznetsova et al., 2017) was used to calculate p-values using Satterthwaite's degrees of freedom. Interactions were tested using post-hoc tests adjusting with Tukey's correction for multiple comparisons for categorical variables and simple slope analysis for continuous variables.

3.Results

3.1 Behavioural performance

3.1.1 Accuracy

There was a main effect of Familiarity, $F_{(1,73)} = 25.44$, $p < .001$, $\eta^2 = .040$, and a main effect of Congruency, $F_{(1,73)} = 156.38$, $p < .001$, $\eta^2 = .20$, The main effects were qualified by a Familiarity x Congruency interaction, $F_{(1,73)} = 9.67$, $p = .003$, $\eta^2 < .01$. Post-hoc comparisons revealed that accuracy was higher for new congruent images than old congruent images ($p = .002$), for new incongruent than old incongruent ($p < .001$), for old congruent than old incongruent ($p < .001$), and for new congruent than new incongruent ($p < .001$), suggesting that the congruency effect was slightly larger for old images. The main effect of Group, the Group x Congruency interaction, the Group x Familiarity interaction and the Group x Familiarity x Congruency interaction were not statistically significant, suggesting that young and older adults had similar accuracy rates.

3.1.2 RTs

There were main effects of Group, $F_{(1,73)} = 72.36$, $p < .001$, $\eta^2 = .48$ of Familiarity, $F_{(1,73)} = 108.25$, $p < .001$, $\eta^2 = .06$, and of Congruency, $F_{(1,73)} = 304.70$, $p < .001$,

η^2 = .05. The main effects were qualified by 2-way interactions. The Group x Congruency interaction was statistically significant, $F_{(1,73)} = 32.09$, $p < .001$, $\eta^2 = .006$. Follow-up tests revealed that older adults were slower than young adults for both congruent and incongruent images and that responses were slower for incongruent conditions in both groups (all *p*s<.001), suggesting that the congruency effect was larger in older adults. The Familiarity x Congruency interaction was statistically significant, $F_{(1,73)} = 14.42$, $p < .001$, $\eta^2 < .01$. Post-hoc test indicated that responses were slower in incongruent trials than congruent trials for both old and new images (all *p*s < .001), and that responses for old images were slower than new images for both congruency conditions (all *p*s < .001).

The Group x Familiarity interaction and the Group x Familiarity x Congruency interaction were not statistically significant.

PLEASE INSERT TABLE 1 HERE

3.2 ERPs

3.2.1 Pe

All ERP mean amplitudes at channel level are summarised in Supplementary Table 1. Full MLM are reported in Supplementary Table 2. There was a main effect of Response Type, *b* = -6.82, 95% CI [-0.55,1.37], *t* (73.00) = -9.85, *p* < .001, with larger Pe for errors as compared to correct responses, and a main effect of Group, *b* = -2.26, 95% CI [-4.02, -1.31], *t (*73.00) = -3.91, *p* < .001, with larger Pe in young adults than older adults. The Response Type x Group interaction was not statistically significant.

PLEASE INSERT FIGURE 1 HERE

3.2.2 N1

N1 amplitude was larger in young adults as compared to older adults as indicated by the main effect of Group. *b* = 1.60, 95% CI [0.87, 2.32], *t* (73.00) = 4.40 *p* <.001. There was Familiarity x Congruency interaction, *b* = 0.66, 95% CI [0.31-1.00], *t*

(73.00) = 3.83 p < .001. Post-hoc tests revealed that N1 amplitude was larger for old incongruent images than old congruent images (p < .001), and larger for new congruent images than old congruent images (p = .002). The main effect of Familiarity, the main effect of Congruency, the Familiarity x Group interaction, the Congruency x Group interaction and the Familiarity x Congruency x Group interaction were not statistically significant. No evidence of an association between N1 and Pe was found.

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PLEASE INSERT FIGURE 2 HERE

3.2.3 P2

P2 amplitude was larger in older adults than young adults as indicated by the main effect of Group, b = 4.22, 95% CI [2.41, 6.04], t (73.01) = 4.63, p < .001. There was a main effect of Congruency, b = -0.69, 95% CI [-0.89, -0.48], t (73.00) = -6.71, p <. 001, which was qualified by a Familiarity x Congruency interaction, b = *0.48*, 95% CI [0.07,0.90], t (73.01) = 2.31, p = .024. Post-hoc tests revealed that P2 amplitude was larger in congruent than incongruent trials for old images (p < .001) and new images (p = .015), suggesting that the congruency effect was larger for old images than new images. The main effect of Familiarity, the Familiarity x Group interaction, the Congruency x Group interaction, and the Familiarity x Congruency x Group interaction were not statistically significant.

P2 amplitude was found to be associated with Pe, as indicated by a significant P200 X Group x Response interaction, b = -021, 95% CI [-0.34, -0.09], t (17469.12) = -3.40, p = .001. Follow-up analyses revealed a positive association between P2 and correct-trial Pe in young adults, b = 0.03, SE = 0.01, z = 2.01, p=0.044, and a negative association between P2 and correct-trial Pe in older adults, b = -0.03, SE= 0.01, z= 2.05, p=0.041. P2 was associated with Pe in older adults, b = 0.13, SE = 0.045, z = 2.65, p = 0.008, while the association between P2 and Pe in young adults was not statistically significant.

PLEASE INSERT FIGURE 3 HERE

3.2.4 N2

There were main effects of Familiarity, *b* = -0.85, 95% CI [-11.19, -0.51], *t* (73.00) = -5.00, *p* < .001, and of Congruency, *b* = -1.52, 95% CI [-1.79, -1.24], *t* (73.00) = -10.91, *p* <.001. The Familiarity x Congruency interaction was statistically significant, *b* = -0.65, 95% CI [-1.12, -0.19], *t* (73.00) = -2.82, *p* = .006. Post-hoc tests indicated that N2 amplitude was larger in incongruent than congruent conditions for old (*p* < .001) and new images (*p* = .001). N2 amplitude was larger for new images than old images in incongruent trials (*p*<.001) but not in congruent trials (*p* = .144), indicating that the familiarity effect was specific to incongruent trials. The Familiarity x Group interaction was statistically significant, *b* = 0.94, 95% CI [0.26,1.62], *t* (73.00) = 2.75, *p* = .007. Post-hoc comparisons indicated that N2 was larger for new than old images in young adults (*p* < .001) but there was no difference between old and new images in older adults (*p* = .398), suggesting that the familiarity effect was specific to young adults. Moreover, no difference in N2 was found between young and older adults for both old (*p* = .888) and new images (*p* = .398). Finally, the Congruency x Group interaction was significant, *b* = 0.66, 95% CI [0.11,1.22], *t* (73.00) = 2.39, *p* = .020). Post-hoc tests revealed that N2 was larger in incongruent trials than congruent trials in young and older adults (*p*s = .001). There was no difference in N2 amplitude between young and older adults in congruent (*p* = .827) and incongruent trials (*p* = .473). The main effect of Group and the Familiarity x Congruency x Group interaction were not statistically significant.

N2 amplitude was found to be associated with Pe, as indicated by the N2 X Group interaction, *b* = 0.15, 95% CI [0.10, 0.21], *t* (17524.03) = 5.47, *p* < .001, and the N2 x Group x Response interaction, *b* = -0.28, 95% CI [-0.38, -0.17], *t* (17473.71) = -5.08, *p* < .001. Larger N2 was associated with larger correct-trial Pe, *b* = -0.02, SE = 0.01, z = -2.34, *p* = 0.019, and larger Pe in young adults, *b* = -0.15, SE = 0.03, z = -4.45, *p* < .001. In older adults, larger N2 was associated with lower Pe, *b* = 0.14, SE = 0.04, z = 3.47, *p* = 0.001, while no significant association was found between N2 and correct-trial Pe.

PLEASE INSERT FIGURE 4 HERE

3.2.5 FN400

There was a main effect of Familiarity, *b* = -2.70 95% CI [-3.19, -2.22], *t* (73.00) = -11.92, *p* < .001, indicating FN400 was larger new images than old images. There was a main effect of Congruency, *b* = -1.54, 95% CI [-1.85, -1.23], *t* (72.99) = -9.99, *p* < .001, and a main effect of Group, *b* = 3.27, 95% CI [0.66,5.87], *t* (73.01) = -11.92, *p* = .015. These were qualified by the Congruency x Group, *b* = 1.06, 95% CI [0.45, 1.68], *t* (72.99) = 3.44 *p* = .001. Post-hoc comparisons indicated that FN400 amplitude was larger in incongruent than congruent trials in both young and older adults (*p*s < .001). FN400 in incongruent trials was larger in young adults than older adults (*p* = .003) while the difference in congruent trials was not statistically significant (*p* = .178). The Familiarity x Congruency, the Familiarity x Group, and the Familiarity x Congruency x Group interactions were not statistically significant.

FN400 amplitude was found to be associated with Pe, as indicated by the FN400 X Group interaction, *b* = 0.10, 95% CI [0.05, 0.15], *t* (17525.41)= 3.88, *p* < .001, the FN400 x Response interaction, *b* = -0.09, 95% CI [-0.14, -0.04], *t* (17472.28)= -3.66, *p* < .001, and the FN400 X Group x Response interaction, *b* = -0.22, 95% CI [-0.32, -0.12], *t (*17471.80)= -4.21, *p* < .001. Follow-up analyses indicated that larger FN400 was associated with larger Pe in young adults *b* = -0.07, SE = 0.03, z= -2.34, *p* = .019, and with smaller Pe older adults, *b* = 0.14, SE = 0.04, z = 3.57, *p* < .001. Larger FN400 was associated with larger correct-trial Pe in both young *b* = -0.06, SE = 0.01, z = -5.83, *p* < .001, and older adults, *b* = -0.06, SE = 0.01, z= -6.03, *p* < .001.

PLEASE INSERT FIGURE 5 HERE

3.2.6 LPC

LPC amplitude was larger for old images than new images as indicated by the main effect of Familiarity, *b* = -2.70, 95% CI [-3.67, -2.77], *t* (73.01) = -14.20, *p* < .001. Moreover, LPC was larger in congruent than incongruent trials, as indicated by the

main effect of Congruency, *b* = -1.29, 95% CI [-1.61, -0.96], *t* (73.00) = -7.92, *p* < .001. The Familiarity x Congruency x Group interaction, *b* = -1.19, 95% CI [-2.22, -0.15], *t* (73.01) = -2.29, *p* = .025. Post-hoc tests revealed that LPC was larger for old than new images in congruent and incongruent trials, and in congruent than incongruent trials for both old and new images in in young and older adults (all *p*s < .001). There was no difference between young and older adults in any condition. The main effect of Group and the 2-way interactions were not statistically significant.

LPC amplitude was associated with Pe, as indicated by the main effect of LPC, *b* = -0.15, 95% CI [0.12,0.17], *t* (17519.07) = -11.37, p < .001. The LPC x Group interaction was statistically significant, *b* = -0.10, 95% CI [-0.15, -0.05], *t* (17519.16) = -3.89, *p* < .001, indicating that larger LPC was associated with larger Pe in young, *b* = 0.20, SE=0.02, z = 12.94, *p* < .001, and older adults, *b* = 0.10, SE = 0.02, z = 4.63, *p* < .001. However, this association was stronger in young adults than older adults, *b* = -0.10, SE = 0.02, z = 3.90, p < .001. Moreover, there was a LPC x Response interaction, *b* = -0.28, 95% CI [-0.33, -0.23], *t* (17474.07) = -10.94, *p* < .001. Follow-up analyses indicated that larger LPC was associated with larger Pe, *b* = 0.28, SE = 0.02, z = 11.62, *p* < .001, but the association between LPC and correct trial-Pe was not statistically significant, *b* = 0.01, SE = 0.01, z = 1.33, *p* =. 183.

PLEASE INSERT FIGURE 6 HERE

4. Discussion

The purpose of this study was to elucidate mechanisms underlying decreased error awareness in aging during memory monitoring. First, we expected to replicate age effects on ERPs in a novel memory version of the flanker task. In line with previous research, Pe, N1 and P2 showed marked differences between young and older adults and LPC was found to be comparable between age groups. The effect of congruency on N2 in older adults was not replicated, but instead it was found that familiarity modulated N2 in young but not in the older group. Moreover, FN400 was reduced by interference effects during incongruent trials in older adults. The findings

partially supported our second hypothesis. Although there was no association between N1 and Pe, larger N2 were associated with larger Pe in young adults, indicating that the efficient conflict processing was related to higher error awareness. Interestingly, inverse associations were found in older adults, thus indicating alteration of related processes in aging. Larger P2 was associated with larger Pe in older adults only, suggesting that overrecruitment of visual attentional resources contributed to higher error awareness. In line with our hypothesis, larger LPC was associated with larger Pe in both young and older adults. However, FN400 findings were aligned to N2 findings. Overall, the study's findings suggests that reduced error awareness in older adults is associated with alteration of sensory processing.

Behaviourally, young and older adults were similar in accuracy and older adults were overall slower, as frequently reported in error monitoring studies (e.g., Beste et al., 2009; Endrass et al., 2012; Harty et al., 2017; Hoffmann & Falkenstein, 2011; Themanson et al., 2006). However, ERP analyses highlighted differences in neurophysiological processes during memory monitoring. Age-related changes in N1 and P2 are in line with other ERP studies (Čeponiene et al., 2008; Staub et al., 2014, 2015; Wiegand et al., 2014), providing further evidence of decline in early visual processes with age. In the present study, N2 was smaller in young adults when the target stimulus was an old image, indicating lower conflict for studied material, while in older adults N2 did not differ between old and new images. In contrast, previous research using a classic arrow version of the flanker task reported that N2 was smaller in older adults for incongruent trials (Hsieh & Fang, 2012; Larson et al., 2016). N2 reflects stimulus-related conflict monitoring activity generated in the anterior cingulate cortex (Carter et al., 1998; Ladouceur et al., 2007; van Veen & Carter, 2002; Wang et al., 2005; Yeung et al., 2004). N2 amplitude has been shown to be sensitive to manipulation of distractors properties (e.g., Danielmeier et al., 2009; Forster et al., 2011; Yeung et al., 2007) and dissociations between N2 and error-related negativity (ERN) suggested that N2 alterations are associated with attending irrelevant stimulus information, rather than deficits related to anterior cingulate dysfunction (Yeung & Cohen, 2006). Similarly, our previous study showed that ERN was not reduced in the same sample of older adults (Lenzoni et al., Submitted). This suggests that older adults have difficulties

linked to processing task-irrelevant information (i.e., flanker stimuli) and therefore experience similar level of conflict for familiar and unfamiliar conditions.

With the regard to memory ERPs, old/new effects were recorded in both age groups, and FN400 in incongruent trials was smaller in older adults relative to young adults while no difference was found for LPC, suggesting that reduced familiarity-related brain activity but intact recollection. The dissociation between the neurophysiological processes underlying memory ERPs has already been previously reported (e.g., Addante, Ranganath, & Yonelinas, 2012; Addante, Ranganath, Olichney, et al., 2012). Moreover, simultaneous EEG-fMRI evidence suggests that FN400 amplitude is associated with activity in the right dorsolateral prefrontal cortex and intraparietal sulcus while LPC is linked to hippocampus, parahippocampal and retrosplenial cortex activity (Hoppstädter et al., 2015). The study's findings are in contrast with research showing that both processes in picture-based tasks are preserved in aging (Ally et al., 2009; Ally, Waring, et al., 2008; James et al., 2016) and that experimental manipulations of picture features affect LPC (Ally, Simons, et al., 2008; Bridger et al., 2017; Dulas & Duarte, 2013). However, it has been highlighted that brain contribution to familiarity-related brain signals rely on task characteristics (Bastin et al., 2019). Unlike past research, the task used in the current study included the use of distractors. Faster and more efficient perceptual abilities allow young adults to extract relevant target stimuli properties while ignoring flanker stimuli. Importantly, this process is facilitated by the fact that colour and shape of target and flanker stimuli always differ in incongruent trials. Instead, older adults are less likely to filter irrelevant sensory input, attending distractors in incongruent trials, because of a top-down suppression deficit in early visual processes (Clapp & Gazzaley, 2012; Gazzaley et al., 2005, 2008), thus suggesting that perceptual interference may affect familiarity processes in aging. Overall, high performance levels and LPC findings are in support of intact mnemonic processes in older adults.

In line with previous studies, we found evidence of an age-related decline in error awareness, as indicated by smaller Pe in the older group (Capuana et al., 2012; Clawson et al., 2017; Larson et al., 2016; Mathewson et al., 2005; Thurm et al., 2020). Pe has recently defined as metacognitive decision variable, reflecting a post-response process of evidence accumulation about error commission (Desender et

al., 2021). In the current study we observed that early sensory ERPs and mnemonic processes were associated with Pe, supporting the hypothesis that error awareness emergence through the integration of multimodal inputs about the response correctness (Steinhauser & Yeung, 2010, 2012; Wessel et al., 2011). Interestingly, dissociated patterns of association between P2, N2, and FN400, and Pe were recorded for young and older adults. The positive association in young adults suggest the efficient sensory processing was associated with higher error awareness. In the older group, larger ERP amplitudes were instead associated with reduced error awareness, suggesting that larger P2, N2, and FN400 may index dysfunctional processes in aging. For example, larger P2 has been linked to a compensatory mechanism in older adults, who need greater allocation of cognitive resources as reflected by frontal overrecruitment (Staub et al., 2014). Therefore, our findings may indicate that compensatory mechanisms implemented to increase visual attentive processes may contribute to error awareness in older adults. Therefore, it is possible that N2 and FN400 dysfunctional mechanism may be resulting from increased cognitive load. In the case of N2, it is also possible that the effect is driven by larger amplitude for studied images, indicating that impaired conflict adaption may lead to a reduction in error awareness. As expected, the association between LPC and Pe was recorded in both age groups, suggesting that efficient recollection of mnemonic representation can be reliably used as evidence of error responses by young and older adults. Past research investigating verbal processing suggests that late parietal effects reflect conscious access to semantic information (Rohaut et al., 2015; Sergent et al., 2005; van Gaal et al., 2014), thus strengthening the idea that this process may contribute to conscious response monitoring and error awareness.

It is important to note that the errors elicited by the task do not represent memory failures and do not include lures (identifying as “old” item an image that was previously presented at test but not in the learning phase), because the study was designed to avoid biases due to lack of recollection on Pe findings. Therefore, future studies investigating memory monitoring should include a larger subset of stimuli to recognise at test and explore related mechanisms of error awareness and investigate the interplay between sensory and memory processes in population with memory disorders to corroborate this study’s findings. Another limitation is the cross-sectional design. Further research is needed to explore the association

between longitudinal changes in error monitoring and cognitive decline. Specifically, it is important to elucidate whether compensatory strategies for sensory decline could improve error awareness.

In conclusion, the study's findings offer novel evidence of age-related neural changes in memory monitoring, highlighting the importance of sensory decline in the emergence of error awareness. Furthermore, this line of research has relevant implication for the conceptualization of new tools to improve error monitoring and self-awareness in individuals affected by cognitive decline or neurological conditions. Importantly, sensory stimulation has become a popular therapeutic tool with promising results on clinical outcomes (Jain & Ramakrishnan, 2020; Maseda et al., 2018; Padilla & Domina, 2016; Sánchez et al., 2013; Yang et al., 2021). Further developments of these programs should extend the assessment of cognitive abilities and the use of biomarkers such as ERPs to explore their benefits on error detection, awareness, and correction to improve everyday life behaviours.

References

Addante, R. J., Ranganath, C., Olichney, J., & Yonelinas, A. P. (2012). Neurophysiological evidence for a recollection impairment in amnesia patients that leaves familiarity intact. Neuropsychologia, 50(13). https://doi.org/10.1016/j.neuropsychologia.2012.07.038

Addante, R. J., Ranganath, C., & Yonelinas, A. P. (2012). Examining ERP correlates of recognition memory: Evidence of accurate source recognition without recollection. NeuroImage, 62(1). https://doi.org/10.1016/j.neuroimage.2012.04.031

Allan, K., Wilding, E. L., & Rugg, M. D. (1998). Electrophysiological evidence for dissociable processes contributing to recollection. Acta Psychologica, 98(2–3). https://doi.org/10.1016/s0001-6918(97)00044-9

Ally, B. A., & Budson, A. E. (2007). The worth of pictures: Using high density event-related potentials to understand the memorial power of pictures and the dynamics of recognition memory. NeuroImage, 35(1). https://doi.org/10.1016/j.neuroimage.2006.11.023

Ally, B. A., McKeever, J. D., Waring, J. D., & Budson, A. E. (2009). Preserved frontal memorial processing for pictures in patients with mild cognitive impairment. Neuropsychologia, 47(10). https://doi.org/10.1016/j.neuropsychologia.2009.03.015

Ally, B. A., Simons, J. S., McKeever, J. D., Peers, P. v., & Budson, A. E. (2008). Parietal contributions to recollection: Electrophysiological evidence from aging and patients with parietal lesions. Neuropsychologia, 46(7). https://doi.org/10.1016/j.neuropsychologia.2008.02.026

Ally, B. A., Waring, J. D., Beth, E. H., McKeever, J. D., Milberg, W. P., & Budson, A. E. (2008). Aging memory for pictures: Using high-density event-related potentials to understand the effect of aging on the picture superiority effect. Neuropsychologia, 46(2). https://doi.org/10.1016/j.neuropsychologia.2007.09.011

Arora, C., Frantz, C., & Toglia, J. (2021). Awareness of Performance on a Functional Cognitive Performance-Based Assessment Across the Adult Lifespan. Frontiers in Psychology, 12. https://doi.org/10.3389/fpsyg.2021.753016

Bastin, C., Besson, G., Simon, J., Delhaye, E., Geurten, M., Willems, S., & Salmon, E. (2019). An Integrative Memory model of recollection and familiarity to understand memory deficits. Behavioral and Brain Sciences. https://doi.org/10.1017/S0140525X19000621

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2014). Fitting Linear Mixed-Effects Models using lme4. Journal of Statistical Software. http://arxiv.org/abs/1406.5823

Beste, C., Willemssen, R., Saft, C., & Falkenstein, M. (2009). Error processing in normal aging and in basal ganglia disorders. Neuroscience, 159(1), 143–149. https://doi.org/10.1016/j.neuroscience.2008.12.030

Boldt, A., & Yeung, N. (2015). Shared neural markers of decision confidence and error detection. Journal of Neuroscience, 35(8), 3478–3484. https://doi.org/10.1523/JNEUROSCI.0797-14.2015

Bridger, E. K., Kursawe, A. L., Bader, R., Tibon, R., Gronau, N., Levy, D. A., & Mecklinger, A. (2017). Age effects on associative memory for novel picture pairings. Brain Research, 1664. https://doi.org/10.1016/j.brainres.2017.03.031

Capuana, L. J., Dywan, J., Tays, W. J., & Segalowitz, S. J. (2012). Cardiac workload and inhibitory control in younger and older adults. Biological Psychology, 90(1), 60–70. https://doi.org/10.1016/j.biopsycho.2012.02.018

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. Science, 280(5364). https://doi.org/10.1126/science.280.5364.747

Čeponiene, R., Westerfield, M., Torki, M., & Townsend, J. (2008). Modality-specificity of sensory aging in vision and audition: Evidence from event-related potentials. Brain Research, 1215. https://doi.org/10.1016/j.brainres.2008.02.010

Clapp, W. C., & Gazzaley, A. (2012). Distinct mechanisms for the impact of distraction and interruption on working memory in aging. Neurobiology of Aging, 33(1). https://doi.org/10.1016/j.neurobiolaging.2010.01.012

Clawson, A., Clayson, P. E., Keith, C. M., Catron, C., & Larson, M. J. (2017). Conflict and performance monitoring throughout the lifespan: An event-related potential (ERP) and temporospatial component analysis. Biological Psychology, 124, 87–99. https://doi.org/10.1016/j.biopsycho.2017.01.012

Cooper, C., Carpenter, I., Katona, C., Schroll, M., Wagner, C., Fialova, D., & Livingston, G. (2005). The AdHOC study of older adults' adherence to medication in 11 countries. American Journal of Geriatric Psychiatry, 13(12). https://doi.org/10.1097/00019442-200512000-00006

Cosentino, S., Metcalfe, J., Cary, M. S., de Leon, J., & Karlawish, J. (2011). Memory awareness influences everyday decision making capacity about medication management in Alzheimer's disease. International Journal of Alzheimer's Disease. https://doi.org/10.4061/2011/483897

Cotrell, V., Wild, K., & Bader, T. (2006). Medication management and adherence among cognitively impaired older adults. Journal of Gerontological Social Work, 47(3–4). https://doi.org/10.1300/J083v47n03_03

Curran, T. (2000). Brain potentials of recollection and familiarity. Memory and Cognition, 28(6). https://doi.org/10.3758/BF03209340

Curran, T., & Cleary, A. M. (2003). Using ERPs to dissociate recollection from familiarity in picture recognition. Cognitive Brain Research, 15(2). https://doi.org/10.1016/S0926-6410(02)00192-1

Curran, T., & Doyle, J. (2011). Picture superiority doubly dissociates the ERP correlates of recollection and familiarity. Journal of Cognitive Neuroscience, 23(5). https://doi.org/10.1162/jocn.2010.21464

Danielmeier, C., Wessel, J. R., Steinhauser, M., & Ullsperger, M. (2009). Modulation of the error-related negativity by response conflict. Psychophysiology, 46(6), 1288–1298. https://doi.org/10.1111/j.1469-8986.2009.00860.x

Delorme, A., & Makeig, S. (2004). EEGLAB: an open-source toolbox for analysis of single-trial EEG dynamics including independent component analysis. Journal of Neuroscience Methods, 134(1), 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009

Desender, K., Boldt, A., Verguts, T., & Donner, T. H. (2019). Confidence predicts speed-accuracy tradeoff for subsequent decisions. ELife, 8. https://doi.org/10.7554/eLife.43499

Desender, K., Ridderinkhof, K. R., & Murphy, P. R. (2021). Understanding neural signals of post-decisional performance monitoring: An integrative review. ELife, 10. https://doi.org/10.7554/eLife.67556

Dhar, M., Wiersema, J. R., & Pourtois, G. (2011). Cascade of neural events leading from error commission to subsequent awareness revealed using EEG source imaging. PLoS ONE, 6(5). https://doi.org/10.1371/journal.pone.0019578

Dulas, M. R., & Duarte, A. (2013). The influence of directed attention at encoding on source memory retrieval in the young and old: An ERP study. Brain Research, 1500. https://doi.org/10.1016/j.brainres.2013.01.018

Enders, C. K., & Tofighi, D. (2007). Centering predictor variables in cross-sectional multilevel models: A new look at an old issue. Psychological Methods, 12(2), 121–138. https://doi.org/10.1037/1082-989X.12.2.121

Endrass, T., Reuter, B., & Kathmann, N. (2007). ERP correlates of conscious error recognition: Aware and unaware errors in an antisaccade task. European Journal of Neuroscience, 26(6), 1714–1720. https://doi.org/10.1111/j.1460-9568.2007.05785.x

Endrass, T., Schreiber, M., & Kathmann, N. (2012). Speeding up older adults: Age-effects on error processing in speed and accuracy conditions. Biological Psychology, 89(2), 426–432. https://doi.org/10.1016/j.biopsycho.2011.12.005

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. Perception & Psychophysics, 16(1), 143–149. https://doi.org/10.3758/BF03203267

Falkenstein, M., Hielscher, H., Dziobek, I., Schwarzenau, P., Hoormann, J., Sundermann, B., & Hohnsbein, J. (2001). Action monitoring, error detection, and the basal ganglia: An ERP study. NeuroReport, 12(1), 157–161. https://doi.org/10.1097/00001756-200101220-00039

Finke, M., Barceló, F., Garolera, M., Cortiñas, M., Garrido, G., Pajares, M., & Escera, C. (2011). Impaired preparatory re-mapping of stimulus-response associations and rule-implementation in schizophrenic patients-The role for differences in early processing. Biological Psychology, 87(3). https://doi.org/10.1016/j.biopsycho.2011.04.006

Forster, S. E., Carter, C. S., Cohen, J. D., & Cho, R. Y. (2011). Parametric manipulation of the conflict signal and control-state adaptation. Journal of Cognitive Neuroscience, 23(4). https://doi.org/10.1162/jocn.2010.21458

Friedman, D., & Johnson, R. (2000). Event-related potential (ERP) studies of memory encoding and retrieval: A selective review. In Microscopy Research and Technique (Vol. 51, Issue 1). https://doi.org/10.1002/1097-0029(20001001)51:1<6::AID-JEMT2>3.0.CO;2-R

Gajewski, P. D., Ferdinand, N. K., Kray, J., & Falkenstein, M. (2018). Understanding sources of adult age differences in task switching: Evidence from behavioral and ERP studies. In Neuroscience and Biobehavioral Reviews (Vol. 92). https://doi.org/10.1016/j.neubiorev.2018.05.029

Gazzaley, A., Clapp, W., Kelley, J., McEvoy, K., Knight, R. T., & D'Esposito, M. (2008). Age-related top-down suppression deficit in the early stages of cortical visual memory processing. Proceedings of the National Academy of Sciences of the United States of America, 105(35). https://doi.org/10.1073/pnas.0806074105

Gazzaley, A., Cooney, J. W., Rissman, J., & D'Esposito, M. (2005). Top-down suppression deficit underlies working memory impairment in normal aging. Nature Neuroscience, 8(10). https://doi.org/10.1038/nn1543

Hämmerer, D., Müller, V., & Li, S. C. (2014). Performance monitoring across the lifespan: Still maturing post-conflict regulation in children and declining task-set monitoring in older adults. In Neuroscience and Biobehavioral Reviews (Vol. 46, Issue P1). https://doi.org/10.1016/j.neubiorev.2014.06.008

Harty, S., Murphy, P. R., Robertson, I. H., & O'Connell, R. G. (2017). Parsing the neural signatures of reduced error detection in older age. NeuroImage, 161, 43–55. https://doi.org/10.1016/j.neuroimage.2017.08.032

Harty, S., O'Connell, R. G., Hester, R., & Robertson, I. H. (2013). Older adults have diminished awareness of errors in the laboratory and daily life. Psychology and Aging, 28(4). https://doi.org/10.1037/a0033567

Herrmann, M. J., Römmler, J., Ehlis, A.-C., Heidrich, A., & Fallgatter, A. J. (2004). Source localization (LORETA) of the error-related-negativity (ERN/Ne) and positivity (Pe). Cognitive Brain Research, 20(2), 294–299. https://doi.org/10.1016/j.cogbrainres.2004.02.013

Hillyard, S. A., & Anllo-Vento, L. (1998). Event-related brain potentials in the study of visual selective attention. Proceedings of the National Academy of Sciences of the United States of America, 95(3). https://doi.org/10.1073/pnas.95.3.781

Hoffmann, S., & Falkenstein, M. (2011). Aging and error processing: Age related increase in the variability of the error-negativity is not accompanied by increase in response variability. PLoS ONE, 6(2). https://doi.org/10.1371/journal.pone.0017482

Hoppstädter, M., Baeuchl, C., Diener, C., Flor, H., & Meyer, P. (2015). Simultaneous EEG–fMRI reveals brain networks underlying recognition memory ERP old/new effects. NeuroImage, 116, 112–122. https://doi.org/10.1016/j.neuroimage.2015.05.026

Hsieh, S., & Fang, W. (2012). Elderly adults through compensatory responses can be just as capable as young adults in inhibiting the flanker influence. Biological Psychology, 90(2). https://doi.org/10.1016/j.biopsycho.2012.03.006

Jain, R., & Ramakrishnan, A. G. (2020). Electrophysiological and Neuroimaging Studies – During Resting State and Sensory Stimulation in Disorders of Consciousness: A Review. In Frontiers in Neuroscience (Vol. 14). https://doi.org/10.3389/fnins.2020.555093

James, T., Strunk, J., Arndt, J., & Duarte, A. (2016). Age-related deficits in selective attention during encoding increase demands on episodic reconstruction

during context retrieval: An ERP study. Neuropsychologia, 86. https://doi.org/10.1016/j.neuropsychologia.2016.04.009

Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2017). lmerTest Package: Tests in Linear Mixed Effects Models. Journal of Statistical Software, 82(13). https://doi.org/10.18637/jss.v082.i13

Ladouceur, C. D., Dahl, R. E., & Carter, C. S. (2007). Development of action monitoring through adolescence into adulthood: ERP and source localization. Developmental Science, 10(6). https://doi.org/10.1111/j.1467-7687.2007.00639.x

Larson, M. J., Clayson, P. E., & Clawson, A. (2014). Making sense of all the conflict: A theoretical review and critique of conflict-related ERPs. International Journal of Psychophysiology, 93(3), 283–297. https://doi.org/10.1016/j.ijpsycho.2014.06.007

Larson, M. J., Clayson, P. E., Keith, C. M., Hunt, I. J., Hedges, D. W., Nielsen, B. L., & Call, V. R. A. (2016). Cognitive control adjustments in healthy older and younger adults: Conflict adaptation, the error-related negativity (ERN), and evidence of generalized decline with age. Biological Psychology, 115, 50–63. https://doi.org/10.1016/j.biopsycho.2016.01.008

Li, K. Z. H., & Lindenberger, U. (2002). Relations between aging sensory/sensorimotor and cognitive functions. Neuroscience and Biobehavioral Reviews, 26(7). https://doi.org/10.1016/S0149-7634(02)00073-8

Maseda, A., Cibeira, N., Lorenzo-López, L., González-Abraldes, I., Buján, A., de Labra, C., & Millán-Calenti, J. C. (2018). Multisensory Stimulation and Individualized Music Sessions on Older Adults with Severe Dementia: Effects on Mood, Behavior, and Biomedical Parameters. Journal of Alzheimer's Disease, 63(4). https://doi.org/10.3233/JAD-180109

Mathewson, K. J., Dywan, J., & Segalowitz, S. J. (2005). Brain bases of error-related ERPs as influenced by age and task. Biological Psychology, 70(2), 88–104. https://doi.org/10.1016/j.biopsycho.2004.12.005

Murphy, P. R., Robertson, I. H., Allen, D., Hester, R., & O'Connell, R. G. (2012). An electrophysiological signal that precisely tracks the emergence of error

awareness. Frontiers in Human Neuroscience, 6(MARCH 2012), 1–16. https://doi.org/10.3389/fnhum.2012.00065

Nieuwenhuis, S., Richard Ridderinkhof, K., Blom, J., Band, G. P. H., & Kok, A. (2001). Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. Psychophysiology, 38(5), 752–760. https://doi.org/10.1017/S0048577201001111

Nyhus, E., & Curran, T. (2009). Semantic and perceptual effects on recognition memory: Evidence from ERP. Brain Research, 1283. https://doi.org/10.1016/j.brainres.2009.05.091

O'Connell, R. G., Dockree, P. M., Bellgrove, M. A., Kelly, S. P., Hester, R., Garavan, H., Robertson, I. H., & Foxe, J. J. (2007). The role of cingulate cortex in the detection of errors with and without awareness: A high-density electrical mapping study. European Journal of Neuroscience, 25(8), 2571–2579. https://doi.org/10.1111/j.1460-9568.2007.05477.x

Overbeek, T. J. M., Nieuwenhuis, S., & Ridderinkhof, K. R. (2005). Dissociable components of error processing: On the functional significance of the Pe vis-à-vis the ERN/Ne. In Journal of Psychophysiology (Vol. 19, Issue 4, pp. 319–329). https://doi.org/10.1027/0269-8803.19.4.319

Padilla, R., & Domina, A. (2016). Effectiveness of sensory stimulation to improve arousal and alertness of people in a coma or persistent vegetative state after traumatic brain injury: A systematic review. American Journal of Occupational Therapy, 70(3). https://doi.org/10.5014/ajot.2016.021022

Parks, C. M., & Yonelinas, A. P. (2007). Moving beyond pure signal-detection models: Comment on wixted (2007). Psychological Review, 114(1). https://doi.org/10.1037/0033-295X.114.1.188

Peirce, J., Gray, J. R., Simpson, S., MacAskill, M., Höchenberger, R., Sogo, H., Kastman, E., & Lindeløv, J. K. (2019). PsychoPy2: Experiments in behavior made easy. Behavior Research Methods, 51(1), 195–203. https://doi.org/10.3758/s13428-018-01193-y

R Core Team. (2020). R Core Team 2020. R: A Language and Environment for Statistical Computing.

Roberts, K. L., & Allen, H. A. (2016). Perception and cognition in the ageing brain: A brief review of the short- and long-term links between perceptual and cognitive decline. In Frontiers in Aging Neuroscience (Vol. 8, Issue MAR). https://doi.org/10.3389/fnagi.2016.00039

Rohaut, B., Faugeras, F., Chausson, N., King, J. R., Karoui, I. el, Cohen, L., & Naccache, L. (2015). Probing ERP correlates of verbal semantic processing in patients with impaired consciousness. Neuropsychologia, 66. https://doi.org/10.1016/j.neuropsychologia.2014.10.014

Rugg, M. D., & Curran, T. (2007). Event-related potentials and recognition memory. In Trends in Cognitive Sciences (Vol. 11, Issue 6). https://doi.org/10.1016/j.tics.2007.04.004

Rugg, M. D., Mark, R. E., Walla, P., Schloerscheidt, A. M., Birch, C. S., & Allan, K. (1998). Dissociation of the neural correlates of implicit and explicit memory. Nature, 392(6676). https://doi.org/10.1038/33396

Sánchez, A., Millán-Calenti, J. C., Lorenzo-López, L., & Maseda, A. (2013). Multisensory stimulation for people with dementia: A review of the literature. In American Journal of Alzheimer's Disease and other Dementias (Vol. 28, Issue 1). https://doi.org/10.1177/1533317512466693

Sergent, C., Baillet, S., & Dehaene, S. (2005). Timing of the brain events underlying access to consciousness during the attentional blink. Nature Neuroscience, 8(10). https://doi.org/10.1038/nn1549

Sim, J., Brown, F. L., O'Connell, R. G., & Hester, R. (2020). Impaired error awareness in healthy older adults: an age group comparison study. Neurobiology of Aging, 96. https://doi.org/10.1016/j.neurobiolaging.2020.08.001

Starkstein, S. E. (2014). Anosognosia in Alzheimer's disease: Diagnosis, frequency, mechanism and clinical correlates. Cortex, 61, 64–73. https://doi.org/10.1016/j.cortex.2014.07.019

Starkstein, S. E., Jorge, R., Mizrahi, R., Adrian, J., & Robinson, R. G. (2007). Insight and danger in Alzheimer's disease. European Journal of Neurology, 14(4), 455–460. https://doi.org/10.1111/j.1468-1331.2007.01745.x

Staub, B., Doignon-Camus, N., Bacon, É., & Bonnefond, A. (2014). The effects of aging on sustained attention ability: An ERP study. Psychology and Aging, 29(3). https://doi.org/10.1037/a0037067

Staub, B., Doignon-Camus, N., Marques-Carneiro, J. E., Bacon, É., & Bonnefond, A. (2015). Age-related differences in the use of automatic and controlled processes in a situation of sustained attention. Neuropsychologia, 75. https://doi.org/10.1016/j.neuropsychologia.2015.07.021

Steinhauser, M., & Yeung, N. (2010). Decision processes in human performance monitoring. Journal of Neuroscience, 30(46), 15643–15653. https://doi.org/10.1523/JNEUROSCI.1899-10.2010

Steinhauser, M., & Yeung, N. (2012). Error awareness as evidence accumulation: effects of speed-accuracy trade-off on error signaling. Frontiers in Human Neuroscience, 6. https://doi.org/10.3389/fnhum.2012.00240

Sunderaraman, P., & Cosentino, S. (2017). Integrating the Constructs of Anosognosia and Metacognition: a Review of Recent Findings in Dementia. Current Neurology and Neuroscience Reports, 17(3), 27. https://doi.org/10.1007/s11910-017-0734-1

Themanson, J. R., Hillman, C. H., & Curtin, J. J. (2006). Age and physical activity influences on action monitoring during task switching. Neurobiology of Aging, 27(9), 1335–1345. https://doi.org/10.1016/j.neurobiolaging.2005.07.002

Thurm, F., Li, S.-C., & Hämmerer, D. (2020). Maturation- and aging-related differences in electrophysiological correlates of error detection and error awareness. Neuropsychologia, 143, 107476. https://doi.org/10.1016/j.neuropsychologia.2020.107476

Ullsperger, M., Danielmeier, C., & Jocham, G. (2014). Neurophysiology of performance monitoring and adaptive behavior. Physiological Reviews, 94(1), 35–79. https://doi.org/10.1152/physrev.00041.2012

Ullsperger, M., Harsay, H. A., Wessel, J. R., & Ridderinkhof, K. R. (2010). Conscious perception of errors and its relation to the anterior insula. Brain Structure and Function, 214(5–6), 629–643. https://doi.org/10.1007/s00429-010-0261-1

van Gaal, S., Naccache, L., Meuwese, J. D. I., van Loon, A. M., Leighton, A. H., Cohen, L., & Dehaene, S. (2014). Can the meaning of multiple words be integrated unconsciously? Philosophical Transactions of the Royal Society B: Biological Sciences, 369(1641). https://doi.org/10.1098/rstb.2013.0212

van Veen, V., & Carter, C. S. (2002). The anterior cingulate as a conflict monitor: FMRI and ERP studies. Physiology and Behavior, 77(4–5), 477–482. https://doi.org/10.1016/S0031-9384(02)00930-7

Vogel, E. K., & Luck, S. J. (2000). The visual N1 component as an index of a discrimination process. Psychophysiology, 37(2). https://doi.org/10.1017/S0048577200981265

Volpert-Esmond, H. I., Merkle, E. C., Levsen, M. P., Ito, T. A., & Bartholow, B. D. (2018). Using trial-level data and multilevel modeling to investigate within-task change in event-related potentials. Psychophysiology, 55(5), e13044. https://doi.org/10.1111/psyp.13044

Volpert-Esmond, H. I., Page-Gould, E., & Bartholow, B. D. (2021). Using multilevel models for the analysis of event-related potentials. International Journal of Psychophysiology, 162. https://doi.org/10.1016/j.ijpsycho.2021.02.006

Wang, C., Ulbert, I., Schomer, D. L., Marinkovic, K., & Halgren, E. (2005). Responses of human anterior cingulate cortex microdomains to error detection, conflict monitoring, stimulus-response mapping, familiarity, and orienting. Journal of Neuroscience, 25(3). https://doi.org/10.1523/JNEUROSCI.4151-04.2005

Wessel, J. R., Danielmeier, C., & Ullsperger, M. (2011). Error Awareness Revisited: Accumulation of Multimodal Evidence from Central and Autonomic Nervous Systems. Journal of Cognitive Neuroscience, 23(10), 3021–3036. https://doi.org/10.1162/jocn.2011.21635

Wiegand, I., Töllner, T., Dyrholm, M., Müller, H. J., Bundesen, C., & Finke, K. (2014). Neural correlates of age-related decline and compensation in visual attention capacity. Neurobiology of Aging, 35(9). https://doi.org/10.1016/j.neurobiolaging.2014.02.023

Yang, H., Luo, Y., Hu, Q., Tian, X., & Wen, H. (2021). Benefits in Alzheimer's Disease of Sensory and Multisensory Stimulation. In Journal of Alzheimer's Disease (Vol. 82, Issue 2). https://doi.org/10.3233/JAD-201554

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. Psychological Review, 111(4), 931–959. https://doi.org/10.1037/0033-295X.111.4.931

Yeung, N., & Cohen, J. D. (2006). The impact of cognitive deficits on conflict monitoring predictable dissociations between the error-related negativity and N2. Psychological Science, 17(2), 164–171. https://doi.org/10.1111/j.1467-9280.2006.01680.x

Yeung, N., Ralph, J., & Nieuwenhuis, S. (2007). Drink alcohol and dim the lights: The impact of cognitive deficits on medial frontal cortex function. Cognitive, Affective and Behavioral Neuroscience, 7(4). https://doi.org/10.3758/CABN.7.4.347

Yonelinas, A. P. (2002). The nature of recollection and familiarity: A review of 30 years of research. In Journal of Memory and Language (Vol. 46, Issue 3). https://doi.org/10.1006/jmla.2002.2864

Table 1. Descriptive statistics, mean (SD).

	Young adults	Older adults	Young adults	Older adults
	Accuracy (%)		RTs (sec)	
Old Congruent	0,92 (0,08)	0,93 (0,08)	0,499 (0,058)	0,611 (0,063)
Old Incongruent	0,83 (0,10)	0,86 (0,12)	0,515 (0,066)	0,646 (0,071)
New Congruent	0,95 (0,04)	0,94 (0,06)	0,522 (0,066)	0,643 (0,065)
New Incongruent	0,88 (0,09)	0,89 (0,12)	0,547 (0,069)	0,689 (0,074)

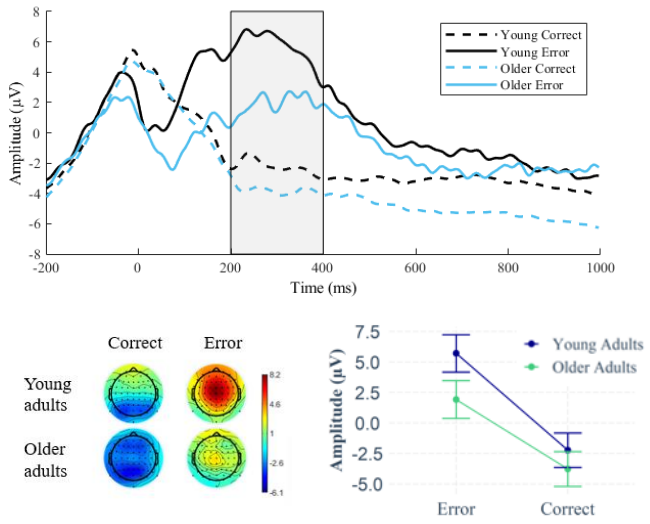

Figure 1. Top plot, Grand-average waveforms for Pe as average activity at CPz, CP1, CP2, Pz, P1, P2; Bottom left, topographical distribution for each response type between 200 and 400; Bottom right, Model estimates and confidence intervals for Pe amplitude.

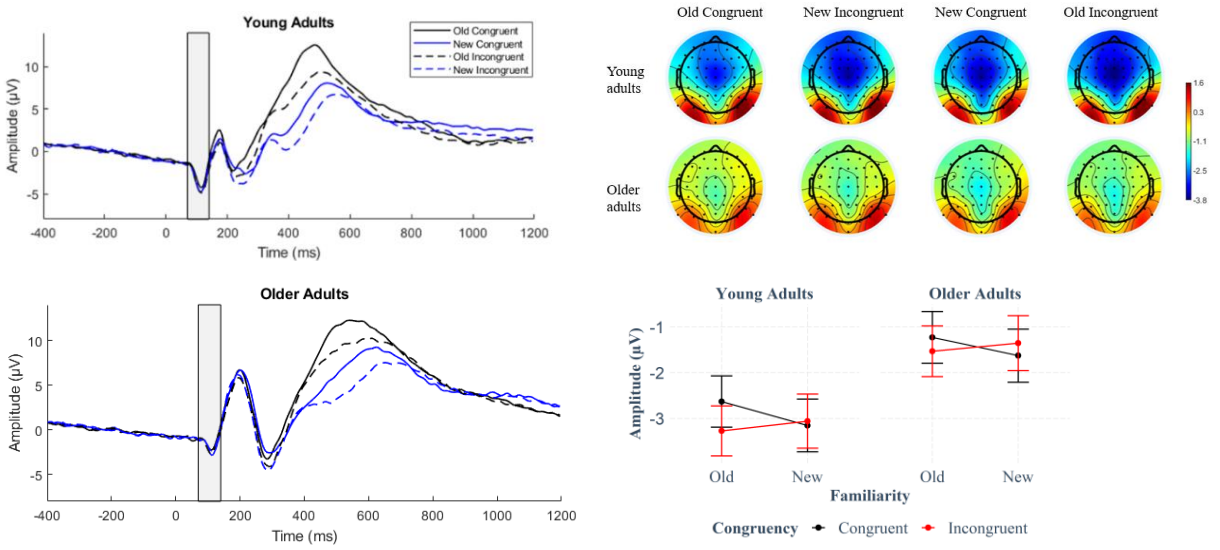

Figure 2. Left plots, Grand-average waveforms for N1 as average activity at Cz, C1, C2, CPz, CP1, CP2; Top right, topographical distribution for each condition between 70 and 140ms; Bottom right, Model estimates and confidence intervals for N1 amplitude.

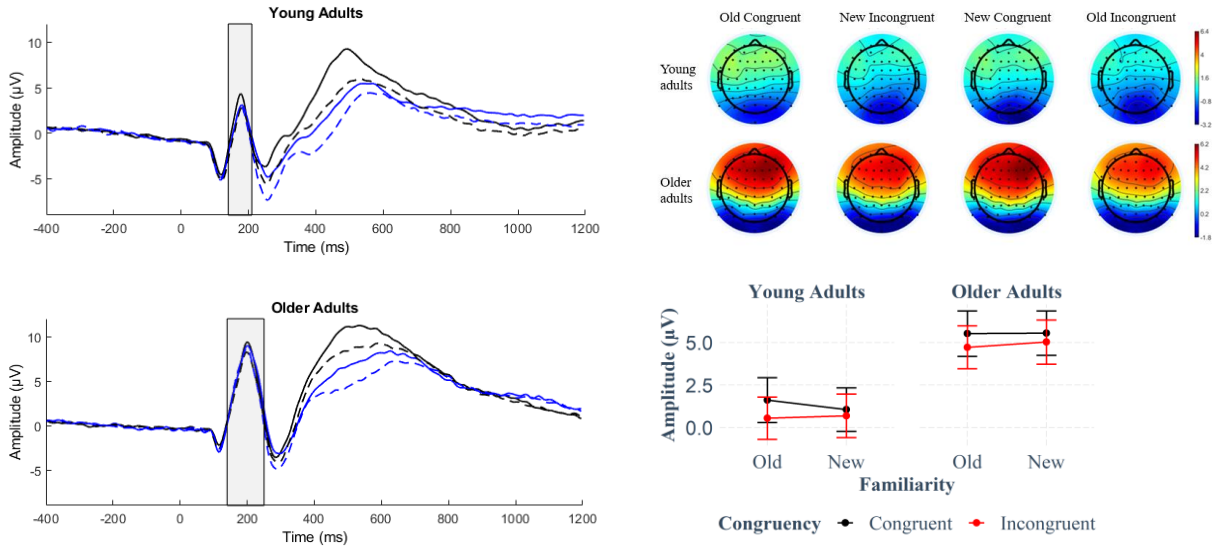

Figure 3. Left plots, Grand-average waveforms for P2 as average activity Fz, F1, F2, FCZ, FC1, FC2; Top right, topographical distribution for each condition between 140 and 210ms for young adults and between 140 and 250ms for older adults; Bottom right, Model estimates and confidence intervals for N1 amplitude.

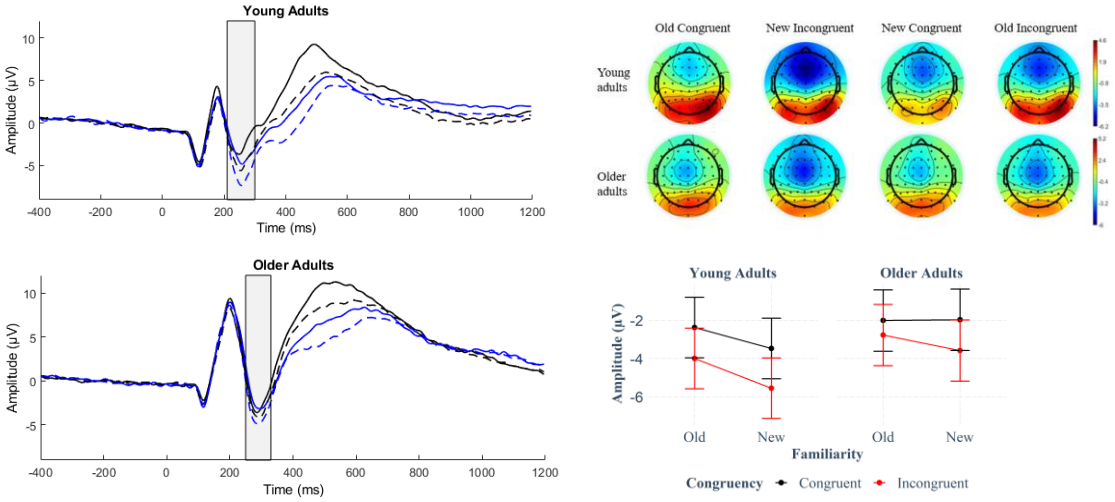

Figure 4. Left plots, Grand-average waveforms for N2 as average activity Fz, F1, F2, FCZ, FC1, FC2; Top right, topographical distribution for each condition between 210 and 300 for young adults and between 250 and 330ms for older adults; Bottom right, Model estimates and confidence intervals for N1 amplitude.

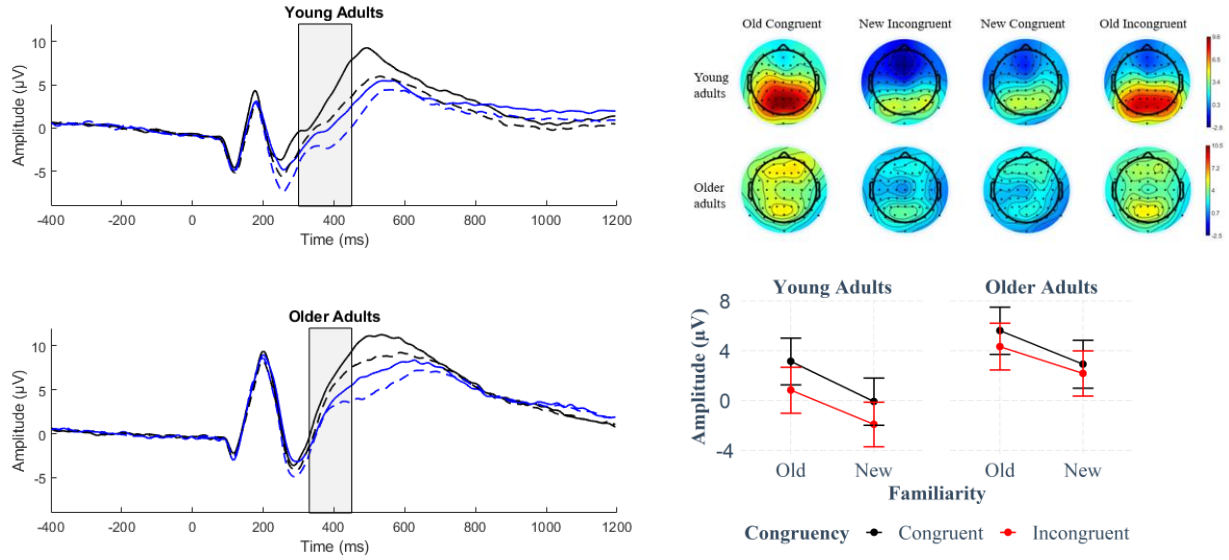

Figure 5. Left plots, Grand-average waveforms for FN400 as average activity Fz, F1, F2, FCZ, FC1, FC2; Top right, topographical distribution for each condition between 300 and 450ms for young adults and between 330 and 450ms for older adults; Bottom right, Model estimates and confidence intervals for N1 amplitude.

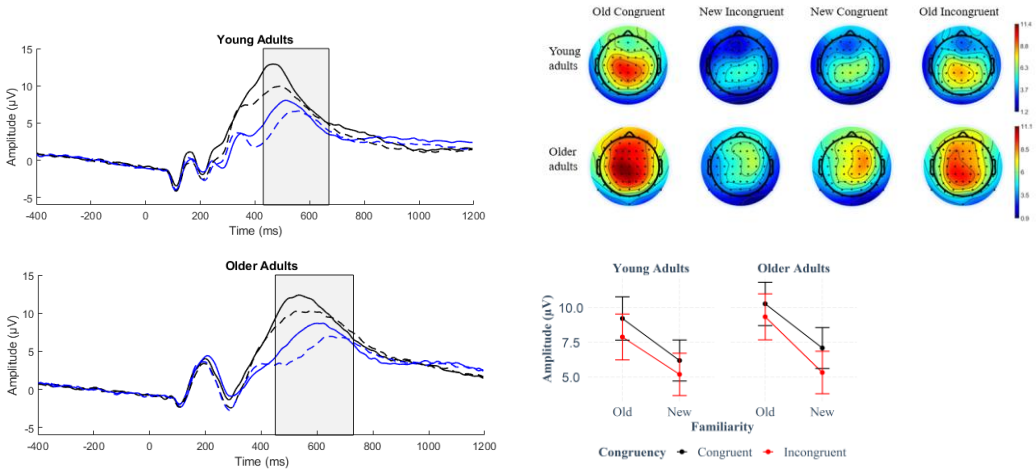

Figure 6. Left plots, Grand-average waveforms for LPC as average activity CPz, CP1, CP2, Pz, P1, and P2; Top right, topographical distribution for each condition between 430 and 670ms for young adults and between 450 and 730ms for older adults; Bottom right, Model estimates and confidence intervals for N1 amplitude.

IV. General Discussion

The general objective of thesis was to investigate the neural correlates of metacognitive awareness. Specifically, this work focused on identifying neural dysfunction associated with deficits of awareness and characterizing neurophysiological mechanisms underlying metacognition in healthy individuals. This research aimed at contributing to a better understanding of biological objective measures of both efficient and impaired metacognitive abilities with direct relevance for clinical assessment and rehabilitation.

The first part of the thesis focused on deficits in metacognitive processes in neurological conditions to determine the neurocognitive substrates of mnemonic and executive anosognosia, as proposed by the CAM (Agnew & Morris, 1998; Hannesdottir & Morris, 2007; R. G. Morris & Hannesdottir, 2004; R. G. Morris & Mograbi, 2013). Article 1 focused on mnemonic anosognosia. The notion of 'Petrified Self' was previously developed in a paper by Mograbi et al. (2009), in which it was hypothesized that memory deficits played a major role in awareness deficits in anosognosia for AD. Lenzoni et al. (2020) reviewed 10 years of research on AD, evaluating the core concepts formulated in the 2009 article. The evidence on retrograde amnesia suggests that hippocampal damage leads to episodic memory retrieval deficits, characterized by a temporal gradient from the early stages of the disease, while semantic memory retrieval subserved by frontal recruitment can be preserved in AD, thus supporting MTT dissociation of neuroanatomical localization of memory sub-processes (Moscovitch, Rosenbaum, et al., 2005; Moscovitch, Westmacott, et al., 2005). Furthermore, anterograde amnesia interferes with transfer and consolidation of new information and, consequently, even in case of efficient behavioural monitoring, information about patient's own abilities cannot be updated, thus supports the idea that AD self-knowledge relies on remote, abstract, and outdated information. The implication of memory dysfunction in AD awareness deficits is also supported by neuroimaging findings showing the association between anosognosia and disrupted connectivity in intra- and inter-regional connectivity in temporal and fronto-temporal structures. Importantly, the findings highlighted the pivotal role of fronto-cingulate structural and functional changes in metacognitive awareness deficits, thus partially supporting the idea that

anosognosia in AD may also arise from monitoring deficits, i.e., executive anosognosia (Mograbi et al., 2009).

Neural correlates of executive anosognosia were investigated in Article 2, which was a systematic review of ERP studies in neurological disorders. The findings are aligned with the CAM formulation of neurocognitive mechanisms underlying comparator mechanisms. ERN is thought to arise from mismatch between competing representations of actual and correct responses (Dehaene, 2018; Falkenstein et al., 1991; Gehring et al., 1993; Scheffers & Coles, 2000), thus mimicking the proposed monitoring process of comparing actual performance and expectations using information stored in the personal database about cognitive abilities. Indeed, Lenzoni et al. (2022) showed that ERN alterations are globally linked to dysfunction of a cortico-subcortical network centred in the ACC, that includes thalamus and basal ganglia functioning, and therefore corroborating the formulation of type I comparator (Mograbi & Morris, 2013; R. G. Morris & Mograbi, 2013). Moreover, type I comparators have modular properties, which means that disruption of local feed-forward mechanisms can lead to domain-specific deficits of cognitive awareness. In Article 2, we speculated that inconsistent findings, characterized by the interaction between lesion localization and task domain in neurological disorders, support the domain specificity hypothesis (Lenzoni et al., 2022). Crucially, Article 3 was the first ERP study exploring domain-specific mechanisms of metacognitive abilities and provided evidence that it is possible to differentiate performance monitoring neural processes across domains in young and older adults (Lenzoni et al., submitted). In line with recent fMRI evidence (Morales et al., 2018), the findings highlight the co-existence of global and local mechanisms of self-monitoring.

Type 2 comparators are thought to function as higher-order secondary processes that, by integrating signals from type 1 comparator mismatch output and several cognitive systems signals, give rise to awareness of deficits. Notably, according to the Evidence Accumulation, error awareness, as indexed by Pe amplitude (Desender et al., 2021), emerges through the integration of ERN mismatch and conflict information, cognitive, sensory, proprioceptive, interoceptive signals (Steinhauser & Yeung, 2010; Ullsperger et al., 2010). The study's results suggest in fact that error awareness deficits arise from regional lesion or disconnection

within a widespread network involving medial prefrontal areas, insula, and somatosensory areas, thus corroborating the evidence on multiple neural sources of Pe (Dhar et al., 2011; Herrmann et al., 2004; O'Connell et al., 2007), and the hypothesis that awareness impairments occur following disrupted integration of neural signals from several systems (Mograbi & Morris, 2013; R. G. Morris & Mograbi, 2013; Ullsperger et al., 2010). This notion is further supported by Article 3 and 4 It was demonstrated that although there is a global decline of error awareness in aging, within-task Pe increase occurred during the memory flanker task and not for the perpetual flanker task in older adults (Lenzoni et al., submitted), thus showing that error awareness emerges through different processes across task domains. We speculated that age-related sensory decline hinders perceptual processing of stimuli properties but learning effects facilitate the use of memory-based alternative strategies, strengthening mnemonic evidence, that cannot be employed during the perceptual flanker task. This idea was supported by Lenzoni et al. (in preparation), showing that in older adults diminished error awareness was associated with dysfunctional sensory processing of perceptual stimuli properties while higher error awareness was associated with efficient recollection neurophysiological processes.

This work not only contributes to our understanding of neurocognitive mechanisms underlying metacognitive awareness but has also relevant clinical implications. As highlighted by Article 2, we found strong evidence that ERN and Pe are associated with clinical factors across several neurological disorders, including symptom severity and psychiatric comorbidities (Lenzoni et al., 2022). Moreover, the findings highlighted that error monitoring ERPs may be predictor of post-traumatic outcomes, supporting the link between self-monitoring impairments and clinical outcomes across several neurological conditions (e.g., Katz et al., 2002; Ownsworth & Fleming, 2005; Starkstein, 2014; Turró-Garriga et al., 2016). Further insight into the clinical relevance of measuring neurophysiological markers of error monitoring can also be found in the second section of this work. Importantly, it was demonstrated that ERN and Pe can be used to explore domain-specific dynamics of metacognitive processes using both between- and within-task changes (Lenzoni et al., submitted). Specifically, the findings suggest that mnemonic processes are preserved in healthy aging and could be used trough appropriate training to

compensate for sensory deficits to efficiently monitor performance (Lenzoni et al., submitted; in preparation). Therefore, the clinical employment of ERN and Pe has crucial assessment and rehabilitation purposes, by defining what processes underlying metacognitive processes are disrupted (e.g., the type of anosognosia), identifying whether these deficits are global or local (i.e., domain-general or domain-specific), characterizing intact cognitive processes that could subserved compensatory mechanisms to improve self-monitoring in tailored training programs, and evaluating treatment effectiveness. Notably, the development of ERPs as biomarkers of cognitive impairments in neurological and psychiatric disorders (Green et al., 2015; Horvath et al., 2018; Luck et al., 2011; Lutz et al., 2021; Müller et al., 2020; Xu et al., 2022) is becoming popular for many reasons. For example, recording EEG is often part of the initial clinical evaluation and differential diagnosis, it is cost effective and non-invasive, therefore preferable when compared to other neuroimaging techniques such as fMRI or PET. Additionally, EEG temporal resolution, adaptability, and portability allow to easy access to equipment and training, and to quickly acquire large amount of brain activity data, which is particularly relevant for averaging purposes in the context of neuroimaging interpretability. Hence, we propose that performance monitoring ERPs can be used as objective measures of metacognitive processes integrity that can be combined with methods based on self-reported evaluations and confidence judgments of task performance and inform the debate on how to best measure metacognitive efficiency (Desender et al., 2022; Fleming & Frith, 2014; Fleming & Lau, 2014).

One of the strengths of this work is the attempt at reconciling theoretical frameworks from different disciplines. Although a number of accounts have been discussed in ERP research to delineate performance monitoring processes (Alexander & Brown, 2011; Dehaene, 2018; Holroyd & Coles, 2002; Overbeek et al., 2005; Steinhauser & Yeung, 2010; Ullsperger et al., 2010; Wessel, 2012, 2018; Wessel et al., 2011; Yeung et al., 2004), rarely these processes are incorporated into a broader understanding of metacognition and self-awareness. In this context, this research contributes to the understanding of domain-specificity of metacognition, exploring electrophysiological similarities and differences underlying metaperception and metamemory, which have been the focus of behavioural and

fMRI research (McWilliams et al., 2022; Morales et al., 2018; Rouault et al., 2018; Vaccaro & Fleming, 2018). Crucially the development of a novel task to study metamemory using EEG methodology was accomplished by adapting the Flanker task, which is the most popular task of performance monitoring research and has been evaluated by several psychometric analyses (Baldwin et al., 2015; Foti et al., 2013; Klawohn et al., 2020; Larson et al., 2010; Meyer et al., 2013, 2014; Olvet & Hajcak, 2009; Pontifex et al., 2010; Riesel et al., 2013; Segalowitz et al., 2010; Weinberg & Hajcak, 2011). One relevant aspect of the Memory Flanker is that matches the classic version not only in the task design (i.e., errors induced by flankers interference) but also in task instructions, number of trials, difficulty (as showed by comparable cross-domain accuracy rates in Article 3), stimulus size and timing, therefore avoid issues that can bias ERP findings (Falkenstein, 2004; Fischer et al., 2017; Hoffmann & Falkenstein, 2010; S. E. Morris et al., 2006; Pailing & Segalowitz, 2004). However, one major drawback of the task design was the confidence judgment measurement. As mentioned in Article 3, participants were asked to indicate how confident they were about performance at the end of each block, thus resulting in six global judgments about performance. Indeed, this prevented investigating trial-level associations between participants' estimations in association with brain activity fluctuations. Moreover, this work presents some general limitations that need to be addressed. The first section consisted in reviews articles and therefore does not investigate experimentally neural correlates of metacognitive awareness in neurological disorders. Critically, it was highlighted that disease severity may play a key role in awareness deficits (Lenzoni et al., 2020, 2022) and information about disease severity was missing in many of the studies included, but also very limited research investigated changes across different disease stages either cross-sectionally or longitudinally. Furthermore, although aging is characterized by cognitive decline and, specifically awareness impairments, the experimental section of this work focused on healthy young and older individuals, and therefore did not provide clinical evidence on the matter.

Hence, these issues should be addressed by future studies. Further research should replicate the findings on domain specificity of self-monitoring ERPs and extend the investigation to neurological disorders include assessment of other cognitive processes, beyond metaperception and metamemory. Based on the results reported,

it is important to understand whether sensory stimulation interventions and/or memory-based training aimed at promoting compensatory strategies can improve error awareness in older adults. Additionally, it is important to evaluate whether blunted Pe can be a predictor of MCI, further elucidating the role of cross domain differences in differentiation between amnestic and non-amnestic MCI, and subsequential conversion to dementia. This line of ERP research would greatly benefit clinical assessment and provide valuable information for patient prognosis and intervention. In this context, research on anosognosia should extend its focus to self-monitoring ERPs, in order to accurately characterize neurophysiological changes underlying metacognitive awareness deficits in AD, especially considering the peculiar heterogeneity of underlying neurodegenerative trajectories. Finally, future adaptations of the memory flanker task should include experimental manipulation to increase difficulty, so that a lower number of trials would allow recording of metacognitive judgments at the end of each trial, while still maintaining an acceptable accuracy rate.

In conclusion, the current work extends our understanding of neurocognitive correlates of metacognitive awareness, contributes to methodological advances in the field of self-monitoring combining principles from multiple disciplines, and, finally, has relevant implications for clinical assessment of anosognosia by offering insights for the development of intervention techniques.

Agnew, S. K., & Morris, R. G. (1998). The heterogeneity of anosognosia for memory impairment in Alzheimer's disease: A review of the literature and a proposed model. Aging & Mental Health, 2(1), 7–19. https://doi.org/10.1080/13607869856876

Alexander, W. H., & Brown, J. W. (2011). Medial prefrontal cortex as an action-outcome predictor. Nature Neuroscience, 14(10), 1338–1344. https://doi.org/10.1038/nn.2921

Allen, G., Barnard, H., McColl, R., Hester, A. L., Fields, J. A., Weiner, M. F., Ringe, W. K., Lipton, A. M., Brooker, M., McDonald, E., Rubin, C. D., & Cullum, C. M. (2007). Reduced hippocampal functional connectivity in Alzheimer disease. Archives of Neurology, 64(10). https://doi.org/10.1001/archneur.64.10.1482

Alzheimer's Association. (2018). 2018 Alzheimer's Disease Facts and Figures. Alzheimers Dement 2018;14(3):367-429. In Alzheimers dement (Vol. 14, Issue 3).

Antoine, P., Nandrino, J.-L., & Billiet, C. (2013). Awareness of deficits in Alzheimer's disease patients: Analysis of performance prediction discrepancies. Psychiatry and Clinical Neurosciences, 67(4), 237–244. https://doi.org/10.1111/pcn.12050

Archer, H. A., Edison, P., Brooks, D. J., Barnes, J., Frost, C., Yeatman, T., Fox, N. C., & Rossor, M. N. (2006). Amyloid load and cerebral atrophy in Alzheimer's disease: An 11C-PIB positron emission tomography study. Annals of Neurology, 60(1). https://doi.org/10.1002/ana.20889

Arora, C., Frantz, C., & Toglia, J. (2021). Awareness of Performance on a Functional Cognitive Performance-Based Assessment Across the Adult Lifespan. Frontiers in Psychology, 12. https://doi.org/10.3389/fpsyg.2021.753016

Avondino, E., & Antoine, P. (2016). Heterogeneity of Cognitive Anosognosia and its Variation with the Severity of Dementia in Patients with Alzheimer's Disease. Journal of Alzheimer's Disease, 50(1), 89–99. https://doi.org/10.3233/JAD-150496

Bach, L., & David, A. (2006). Self-awareness after acquired and traumatic brain injury. In Neuropsychological Rehabilitation (Vol. 16, Issue 4). https://doi.org/10.1080/09602010500412830

Baldwin, S. A., Larson, M. J., & Clayson, P. E. (2015). The dependability of electrophysiological measurements of performance monitoring in a clinical sample: A generalizability and decision analysis of the ERN and Pe. Psychophysiology, 52(6), 790–800. https://doi.org/10.1111/psyp.12401

Belleville, S., & Bherer, L. (2012). Biomarkers of Cognitive Training Effects in Aging. Current Translational Geriatrics and Experimental Gerontology Reports, 1(2). https://doi.org/10.1007/s13670-012-0014-5

Bellon, E., Fias, W., & de Smedt, B. (2020). Metacognition across domains: Is the association between arithmetic and metacognitive monitoring domain-specific? PLOS ONE, 15(3), e0229932. https://doi.org/10.1371/journal.pone.0229932

Bhome, R., McWilliams, A., Huntley, J. D., Fleming, S. M., & Howard, R. J. (2019). Metacognition in functional cognitive disorder- a potential mechanism and treatment target. Cognitive Neuropsychiatry, 24(5). https://doi.org/10.1080/13546805.2019.1651708

Boldt, A., & Yeung, N. (2015). Shared neural markers of decision confidence and error detection. Journal of Neuroscience, 35(8), 3478–3484. https://doi.org/10.1523/JNEUROSCI.0797-14.2015

Bottini, G., Magnani, F. G., Salvato, G., & Gandola, M. (2018). Multiple dissociations in patients with disorders of body awareness: Implications for the study of consciousness. Frontiers in Psychology, 9(OCT). https://doi.org/10.3389/fpsyg.2018.02068

Braak, H., Braak, E., & Bohl, J. (1993). Staging of alzheimer-related cortical destruction. European Neurology, 33(6). https://doi.org/10.1159/000116984

Brázdil, M., Roman, R., Daniel, P., & Rektor, I. (2005). Intracerebral Error-Related Negativity in a Simple Go/NoGo Task. Journal of Psychophysiology, 19(4), 244–255. https://doi.org/10.1027/0269-8803.19.4.244

Canzano, L., Scandola, M., Pernigo, S., Aglioti, S. M., & Moro, V. (2014). Anosognosia for apraxia: Experimental evidence for defective awareness of one's own bucco-facial gestures. Cortex, 61. https://doi.org/10.1016/j.cortex.2014.05.015

Chapman, S., Colvin, L. E., Vuorre, M., Cocchini, G., Metcalfe, J., Huey, E. D., & Cosentino, S. (2018). Cross domain self-monitoring in anosognosia for memory loss in Alzheimer's disease. Cortex, 101, 221–233. https://doi.org/10.1016/j.cortex.2018.01.019

Clare, L., Wilson, B. A., Carter, G., Roth, I., & Hodges, J. R. (2002). Assessing awareness in early-stage Alzheimer's disease: Development and piloting of the Memory Awareness Rating Scale. Neuropsychological Rehabilitation, 12(4), 341–362. https://doi.org/10.1080/09602010244000129

Cocchini, G., Beschin, N., Fotopoulou, A., & della Sala, S. (2010). Explicit and implicit anosognosia or upper limb motor impairment. Neuropsychologia, 48(5). https://doi.org/10.1016/j.neuropsychologia.2010.01.019

Cocchini, G., Gregg, N., Beschin, N., Dean, M., & Sala, S. della. (2010). Vata-L: Visual-analogue test assessing anosognosia for language impairment. Clinical Neuropsychologist, 24(8). https://doi.org/10.1080/13854046.2010.524167

Cooper, C., Carpenter, I., Katona, C., Schroll, M., Wagner, C., Fialova, D., & Livingston, G. (2005). The AdHOC study of older adults' adherence to medication in 11 countries. American Journal of Geriatric Psychiatry, 13(12). https://doi.org/10.1097/00019442-200512000-00006

Cosentino, S., Metcalfe, J., Cary, M. S., de Leon, J., & Karlawish, J. (2011). Memory awareness influences everyday decision making capacity about medication management in Alzheimer's disease. International Journal of Alzheimer's Disease. https://doi.org/10.4061/2011/483897

Craig, A. D. (2002). How do you feel? Interoception: The sense of the physiological condition of the body. Nature Reviews Neuroscience, 3(8). https://doi.org/10.1038/nrn894

de Ruijter, N. S., Schoonbrood, A. M. G., van Twillert, B., & Hoff, E. I. (2020). Anosognosia in dementia: A review of current assessment instruments. Alzheimer's and Dementia: Diagnosis, Assessment and Disease Monitoring, 12(1). https://doi.org/10.1002/dad2.12079

Debener, S. (2005). Trial-by-Trial Coupling of Concurrent Electroencephalogram and Functional Magnetic Resonance Imaging Identifies the Dynamics of

Performance Monitoring. Journal of Neuroscience, 25(50), 11730–11737. https://doi.org/10.1523/JNEUROSCI.3286-05.2005

Dehaene, S. (2018). The Error-Related Negativity, Self-Monitoring, and Consciousness. Perspectives on Psychological Science, 13(2), 161–165. https://doi.org/10.1177/1745691618754502

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a Neural System for Error Detection and Compensation. Psychological Science, 5(5), 303–305. https://doi.org/10.1111/j.1467-9280.1994.tb00630.x

Dentakos, S., Saoud, W., Ackerman, R., & Toplak, M. E. (2019). Does domain matter? Monitoring accuracy across domains. Metacognition and Learning, 14(3), 413–436. https://doi.org/10.1007/s11409-019-09198-4

Desender, K., Ridderinkhof, K. R., & Murphy, P. R. (2021). Understanding neural signals of post-decisional performance monitoring: An integrative review. ELife, 10. https://doi.org/10.7554/eLife.67556

Desender, K., Vermeylen, L., & Verguts, T. (2022). Dynamic influences on static measures of metacognition. Nature Communications, 13(1), 4208. https://doi.org/10.1038/s41467-022-31727-0

Dhar, M., Wiersema, J. R., & Pourtois, G. (2011). Cascade of neural events leading from error commission to subsequent awareness revealed using EEG source imaging. PLoS ONE, 6(5), 19–22. https://doi.org/10.1371/journal.pone.0019578

di Gregorio, F., Maier, M. E., & Steinhauser, M. (2018). Errors can elicit an error positivity in the absence of an error negativity: Evidence for independent systems of human error monitoring. NeuroImage, 172(September 2017), 427–436. https://doi.org/10.1016/j.neuroimage.2018.01.081

di Tella, S., Cabinio, M., Isernia, S., Blasi, V., Rossetto, F., Saibene, F. L., Alberoni, M., Silveri, M. C., Sorbi, S., Clerici, M., & Baglio, F. (2021). Neuroimaging Biomarkers Predicting the Efficacy of Multimodal Rehabilitative Intervention in the Alzheimer's Dementia Continuum Pathology. Frontiers in Aging Neuroscience, 13. https://doi.org/10.3389/fnagi.2021.735508

Dourado, M. C. N., Mograbi, D. C., Santos, R. L., Sousa, M. F. ernanda B., Nogueira, M. L., Belfort, T., Landeira-Fernandez, J., & Laks, J. (2014). Awareness of disease in dementia: factor structure of the assessment scale of psychosocial impact of the diagnosis of dementia. Journal of Alzheimer's Disease : JAD, 41(3). https://doi.org/10.3233/JAD-140183

Dromer, E., Kheloufi, L., & Azouvi, P. (2021). Impaired self-awareness after traumatic brain injury: a systematic review. Part 1: Assessment, clinical aspects and recovery. In Annals of Physical and Rehabilitation Medicine (Vol. 64, Issue 5). https://doi.org/10.1016/j.rehab.2020.101468

Dunlosky, J., & Metcalfe, J. (2009). Metacognition. Sage Publications.

Endrass, T., Klawohn, J., Gruetzmann, R., Ischebeck, M., & Kathmann, N. (2012). Response-related negativities following correct and incorrect responses: Evidence from a temporospatial principal component analysis. Psychophysiology, 49(6), 733–743. https://doi.org/10.1111/j.1469-8986.2012.01365.x

Endrass, T., Reuter, B., & Kathmann, N. (2007). ERP correlates of conscious error recognition: Aware and unaware errors in an antisaccade task. European Journal of Neuroscience, 26(6), 1714–1720. https://doi.org/10.1111/j.1460-9568.2007.05785.x

Falkenstein, M. (2004). Errors, Conflicts, and the Brain: A Review of the Contributions to the Error Conference, Dortmund 2003. Journal of Psychophysiology, 18(4).

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. Electroencephalography and Clinical Neurophysiology, 78(6), 447–455. https://doi.org/10.1016/0013-4694(91)90062-9

Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: A tutorial. Biological Psychology. https://doi.org/10.1016/S0301-0511(99)00031-9

Fischer, A. G., Klein, T. A., & Ullsperger, M. (2017). Comparing the error-related negativity across groups: The impact of error- and trial-number differences. Psychophysiology, 54(7), 998–1009. https://doi.org/10.1111/psyp.12863

Fischer, S., Trexler, L. E., & Gauggel, S. (2004). Awareness of activity limitations and prediction of performance in patients with brain injuries and orthopedic disorders. Journal of the International Neuropsychological Society, 10(2). https://doi.org/10.1017/S1355617704102051

Fisher, P. L., & Noble, A. J. (2017). Anxiety and depression in people with epilepsy: The contribution of metacognitive beliefs. Seizure, 50. https://doi.org/10.1016/j.seizure.2017.06.012

Flavell, J. H. (1979). Metacognition and cognitive monitoring: A new area of cognitive-developmental inquiry. American Psychologist, 34(10). https://doi.org/10.1037/0003-066X.34.10.906

Fleming, J. M., Strong, J., & Ashton, R. (1996). Self-awareness of deficits in adults with traumatic brain injury: How best to measure? Brain Injury, 10(1). https://doi.org/10.1080/026990596124674

Fleming, J., Ownsworth, T., Doig, E., Hutton, L., Griffin, J., Kendall, M., & Shum, D. H. K. (2017). The efficacy of prospective memory rehabilitation plus metacognitive skills training for adults with traumatic brain injury: Study protocol for a randomized controlled trial. Trials, 18(1). https://doi.org/10.1186/s13063-016-1758-6

Fleming, J., & Strong, J. (1995). Self-Awareness of Deficits following Acquired Brain Injury: Considerations for Rehabilitation. British Journal of Occupational Therapy, 58(2). https://doi.org/10.1177/030802269505800204

Fleming, S. M., & Dolan, R. J. (2012). The neural basis of metacognitive ability. In Philosophical Transactions of the Royal Society B: Biological Sciences (Vol. 367, Issue 1594). https://doi.org/10.1098/rstb.2011.0417

Fleming, S. M., Dolan, R. J., & Frith, C. D. (2012). Metacognition: Computation, biology and function. In Philosophical Transactions of the Royal Society B: Biological Sciences (Vol. 367, Issue 1594). https://doi.org/10.1098/rstb.2012.0021

Fleming, S. M., & Frith, C. D. (2014). The cognitive neuroscience of metacognition. In The Cognitive Neuroscience of Metacognition (Vol. 9783642451904). Springer-Verlag Berlin Heidelberg. https://doi.org/10.1007/978-3-642-45190-4

Fleming, S. M., & Lau, H. C. (2014). How to measure metacognition. In Frontiers in Human Neuroscience (Vol. 8, Issue JULY). https://doi.org/10.3389/fnhum.2014.00443

Foti, D., Kotov, R., & Hajcak, G. (2013). Psychometric considerations in using error-related brain activity as a biomarker in psychotic disorders. Journal of Abnormal Psychology, 122(2). https://doi.org/10.1037/a0032618

Gainotti, G. (2018). Anosognosia in degenerative brain diseases: The role of the right hemisphere and of its dominance for emotions. Brain and Cognition, 127, 13–22. https://doi.org/10.1016/j.bandc.2018.08.002

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A Neural System for Error Detection and Compensation. Psychological Science, 4(6), 385–390. https://doi.org/10.1111/j.1467-9280.1993.tb00586.x

Gerretsen, P., Chung, J. K., Shah, P., Plitman, E., Iwata, Y., Caravaggio, F., Nakajima, S., Pollock, B. G., & Graff-Guerrero, A. (2017). Anosognosia g pIS an independent py g predictor of conversion y From mild cognitive impairment to Alzheimer's disease and is associated with reduced brain metabolism. Journal of Clinical Psychiatry, 78(9). https://doi.org/10.4088/JCP.16m11367

Green, D. L., Payne, L., Polikar, R., Moberg, P. J., Wolk, D. A., & Kounios, J. (2015). P50: A candidate ERP biomarker of prodromal Alzheimer's disease. Brain Research, 1624. https://doi.org/10.1016/j.brainres.2015.07.054

Gross, J. J. (2013). Emotion regulation: Taking stock and moving forward. Emotion, 13(3), 359–365. https://doi.org/10.1037/a0032135

Haggard, P. (2017). Sense of agency in the human brain. Nature Reviews Neuroscience, 18(4), 196–207. https://doi.org/10.1038/nrn.2017.14

Hämmerer, D., Müller, V., & Li, S. C. (2014). Performance monitoring across the lifespan: Still maturing post-conflict regulation in children and declining task-set monitoring in older adults. In Neuroscience and Biobehavioral Reviews (Vol. 46, Issue P1). https://doi.org/10.1016/j.neubiorev.2014.06.008

Hannesdottir, K., & Morris, R. G. (2007). Primary and secondary anosognosia for memory impairment in patients with Alzheimer's disease. Cortex, 43(7). https://doi.org/10.1016/S0010-9452(08)70698-1

Harty, S., O'Connell, R. G., Hester, R., & Robertson, I. H. (2013). Older adults have diminished awareness of errors in the laboratory and daily life. Psychology and Aging, 28(4). https://doi.org/10.1037/a0033567

Herrmann, M. J., Römmler, J., Ehlis, A.-C., Heidrich, A., & Fallgatter, A. J. (2004). Source localization (LORETA) of the error-related-negativity (ERN/Ne) and positivity (Pe). Cognitive Brain Research, 20(2), 294–299. https://doi.org/10.1016/j.cogbrainres.2004.02.013

Hester, R., Foxe, J. J., Molholm, S., Shpaner, M., & Garavan, H. (2005). Neural mechanisms involved in error processing: A comparison of errors made with and without awareness. NeuroImage, 27(3), 602–608. https://doi.org/10.1016/j.neuroimage.2005.04.035

Hoffmann, S., & Falkenstein, M. (2010). Independent component analysis of erroneous and correct responses suggests online response control. Human Brain Mapping, 31(9), 1305–1315. https://doi.org/10.1002/hbm.20937

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. Psychological Review, 109(4), 679–709. https://doi.org/10.1037/0033-295X.109.4.679

Holroyd, C. B., & Yeung, N. (2012). Motivation of extended behaviors by anterior cingulate cortex. In Trends in Cognitive Sciences. https://doi.org/10.1016/j.tics.2011.12.008

Horvath, A., Szucs, A., Csukly, G., Sakovics, A., Stefanics, G., & Kamondi, A. (2018). EEG and ERP biomarkers of Alzheimer's disease: A critical review. In Frontiers in Bioscience - Landmark (Vol. 23, Issue 2). https://doi.org/10.2741/4587

Huntley, J. D., Fleming, S. M., Mograbi, D. C., Bor, D., Naci, L., Owen, A. M., & Howard, R. (2021). Understanding Alzheimer's disease as a disorder of consciousness. Alzheimer's and Dementia: Translational Research and Clinical Interventions, 7(1). https://doi.org/10.1002/trc2.12203

Jacobsen, H. B., Aasvik, J. K., Borchgrevink, P. C., Landrø, N. I., & Stiles, T. C. (2016). Metacognitions are associated with subjective memory problems in individuals on sick leave due to chronic fatigue. Frontiers in Psychology, 7(MAY). https://doi.org/10.3389/fpsyg.2016.00729

Jahn, H. (2013). Memory loss in Alzheimer's disease. Dialogues in Clinical Neuroscience, 15(4), 445–454. https://doi.org/10.31887/DCNS.2013.15.4/hjahn

Jatoi, M. A., Kamel, N., Malik, A. S., Faye, I., & Begum, T. (2014). A survey of methods used for source localization using EEG signals. In Biomedical Signal Processing and Control (Vol. 11, Issue 1). https://doi.org/10.1016/j.bspc.2014.01.009

Katz, N., Fleming, J., Keren, N., Lightbody, S., & Hartman-Maeir, A. (2002). Unawareness and/or denial of disability: Implications for occupational therapy intervention. Canadian Journal of Occupational Therapy, 69(5). https://doi.org/10.1177/000841740206900504

Kennett, R. (2012). Modern electroencephalography. Journal of Neurology, 259(4). https://doi.org/10.1007/s00415-012-6425-6

Kirschstein, T., & Köhling, R. (2009). What is the source of the EEG? Clinical EEG and Neuroscience, 40(3). https://doi.org/10.1177/155005940904000305

Klawohn, J., Meyer, A., Weinberg, A., & Hajcak, G. (2020). Methodological choices in event-related potential (ERP) research and their impact on internal consistency reliability and individual differences: An examination of the error-related negativity (ERN) and anxiety. Journal of Abnormal Psychology, 129(1). https://doi.org/10.1037/abn0000458

Koltai, D. C., Welsh-Bohmer, K. A., & Schmechel, D. E. (2001). Influence of anosognosia on treatment outcome among dementia patients. Neuropsychological Rehabilitation, 11(3–4). https://doi.org/10.1080/09602010042000097

la Foresta, S. , Messina, S. , Faraone, C. , Pistorino, G. , Vita, G. , Lunetta, C. , & Quattropani, M. C. (2015). Conceptualizing the relations between metacognition and executive functions in Amyotrophic Lateral Sclerosis (ALS) patients' caregivers. A preliminary study. Mediterranean Journal of Clinical Psychology, 3(3).

Larson, M. J., Baldwin, S. A., Good, D. A., & Fair, J. E. (2010). Temporal stability of the error-related negativity (ERN) and post-error positivity (Pe): The role of number of trials. Psychophysiology, 47(6). https://doi.org/10.1111/j.1469-8986.2010.01022.x

Lenzoni, S., Baker, J., Sumich, A. L., & Mograbi, D. C. (2022). New insights into neural networks of error monitoring and clinical implications: a systematic review of ERP studies in neurological diseases. Reviews in the Neurosciences, 33(2), 161–179. https://doi.org/10.1515/revneuro-2021-0054

Lenzoni, S., Morris, R. G., & Mograbi, D. C. (2020). The Petrified Self 10 Years After: Current Evidence for Mnemonic anosognosia. Frontiers in Psychology, 11. https://doi.org/10.3389/fpsyg.2020.00465

Lou, H. C., Changeux, J. P., & Rosenstand, A. (2017). Towards a cognitive neuroscience of self-awareness. In Neuroscience and Biobehavioral Reviews (Vol. 83). https://doi.org/10.1016/j.neubiorev.2016.04.004

Luck, S. J. (2014). An Introduction to the Event-Related Potential Technique, second edition. In The MIT Press.

Luck, S. J., Mathalon, D. H., O'Donnell, B. F., Hämäläinen, M. S., Spencer, K. M., Javitt, D. C., & Uhlhaas, P. J. (2011). A Roadmap for the Development and Validation of Event-Related Potential Biomarkers in Schizophrenia Research. Biological Psychiatry, 70(1), 28–34. https://doi.org/10.1016/j.biopsych.2010.09.021

Lutz, M. C., Kok, R., & Franken, I. H. A. (2021). Event-related potential (ERP) measures of error processing as biomarkers of externalizing disorders: A narrative review. International Journal of Psychophysiology, 166. https://doi.org/10.1016/j.ijpsycho.2021.06.002

Maier, F., & Prigatano, G. P. (2017). Impaired Self-Awareness of Motor Disturbances in Parkinson's Disease. In Archives of Clinical Neuropsychology (Vol. 32, Issue 7). https://doi.org/10.1093/arclin/acx094

Maniscalco, B., & Lau, H. (2012). A signal detection theoretic approach for estimating metacognitive sensitivity from confidence ratings. Consciousness and Cognition, 21(1). https://doi.org/10.1016/j.concog.2011.09.021

Marcel, A. J., Tegnér, R., & Nimmo-Smith, I. (2004). Anosognosia for plegia: specificity, extension, partiality and disunity of bodily unawareness. Cortex; a Journal Devoted to the Study of the Nervous System and Behavior, 40(1), 19–40. https://doi.org/10.1016/s0010-9452(08)70919-5

Mazancieux, A., Souchay, C., Casez, O., & Moulin, C. J. A. (2019). Metacognition and self-awareness in Multiple Sclerosis. Cortex, 111, 238–255. https://doi.org/10.1016/j.cortex.2018.11.012

McWilliams, A., Bibby, H., Steinbeis, N., David, A. S., & Fleming, S. M. (2022, February 24). Age-related decreases in global metacognition are independent of local metacognition and task performance. PsyArXiv.

Meghdadi, A. H., Karic, M. S., McConnell, M., Rupp, G., Richard, C., Hamilton, J., Salat, D., & Berka, C. (2021). Resting state EEG biomarkers of cognitive decline associated with Alzheimer's disease and mild cognitive impairment. PLoS ONE, 16(2 February). https://doi.org/10.1371/journal.pone.0244180

Metcalfe, J., & Schwartz, B. L. (2016). The ghost in the machine: Self-reflective consciousness and the neuroscience of metacognition. Oxford Handbook of Metamemory.

Meyer, A., Bress, J. N., & Proudfit, G. H. (2014). Psychometric properties of the error-related negativity in children and adolescents. Psychophysiology, 51(7), 602–610. https://doi.org/10.1111/psyp.12208

Meyer, A., Riesel, A., & Proudfit, G. H. (2013). Reliability of the ERN across multiple tasks as a function of increasing errors. Psychophysiology, 50(12). https://doi.org/10.1111/psyp.12132

Migliorelli, R., Teson, A., Sabe, L., Petracca, G., Petracchi, M., Leiguarda, R., & Starkstein, S. E. (1995). Anosognosia in Alzheimer's disease: A study of associated factors. Journal of Neuropsychiatry and Clinical Neurosciences, 7(3). https://doi.org/10.1176/jnp.7.3.338

Mihaljcic, T., Haines, T. P., Ponsford, J. L., & Stolwyk, R. J. (2017). Investigating the relationship between reduced self-awareness of falls risk, rehabilitation engagement and falls in older adults. Archives of Gerontology and Geriatrics, 69. https://doi.org/10.1016/j.archger.2016.11.003

Mograbi, D. C., Brown, R. G., & Morris, R. G. (2009). Anosognosia in Alzheimer's disease – The petrified self. Consciousness and Cognition, 18(4), 989–1003. https://doi.org/10.1016/j.concog.2009.07.005

Mograbi, D. C., Brown, R. G., Salas, C., & Morris, R. G. (2012). Emotional reactivity and awareness of task performance in Alzheimer's disease. Neuropsychologia, 50(8), 2075–2084. https://doi.org/10.1016/j.neuropsychologia.2012.05.008

Mograbi, D. C., Huntley, J., & Critchley, H. (2021). Self-awareness in Dementia: a Taxonomy of Processes, Overview of Findings, and Integrative Framework. In Current Neurology and Neuroscience Reports (Vol. 21, Issue 12). https://doi.org/10.1007/s11910-021-01155-6

Mograbi, D. C., & Morris, R. G. (2013). Implicit awareness in anosognosia: Clinical observations, experimental evidence, and theoretical implications. Cognitive Neuroscience, 4(3–4), 181–197. https://doi.org/10.1080/17588928.2013.833899

Mograbi, D. C., & Morris, R. G. (2018). Anosognosia. Cortex, 103, 385–386. https://doi.org/10.1016/j.cortex.2018.04.001

Montine, T. J., Phelps, C. H., Beach, T. G., Bigio, E. H., Cairns, N. J., Dickson, D. W., Duyckaerts, C., Frosch, M. P., Masliah, E., Mirra, S. S., Nelson, P. T., Schneider, J. A., Thal, D. R., Trojanowski, J. Q., Vinters, H. v., & Hyman, B. T. (2012). National institute on aging-Alzheimer's association guidelines for the neuropathologic assessment of Alzheimer's disease: A practical approach. Acta Neuropathologica, 123(1). https://doi.org/10.1007/s00401-011-0910-3

Morales, J., Lau, H., & Fleming, S. M. (2018). Domain-general and domain-specific patterns of activity supporting metacognition in human prefrontal cortex. Journal of Neuroscience, 38(14), 3534–3546. https://doi.org/10.1523/JNEUROSCI.2360-17.2018

Morin, A. (2006). Levels of consciousness and self-awareness: A comparison and integration of various neurocognitive views. Consciousness and Cognition, 15(2). https://doi.org/10.1016/j.concog.2005.09.006

Morin, A. (2011). Self-awareness part 1: Definition, measures, effects, functions, and antecedents. Social and Personality Psychology Compass, 5(10). https://doi.org/10.1111/j.1751-9004.2011.00387.x

Mormino, E. C., Kluth, J. T., Madison, C. M., Rabinovici, G. D., Baker, S. L., Miller, B. L., Koeppe, R. A., Mathis, C. A., Weiner, M. W., Jagust, W. J., & Alzheimer's Disease Neuroimaging Initiative. (2009). Episodic memory loss is related to hippocampal-mediated beta-amyloid deposition in elderly subjects. Brain : A Journal of Neurology, 132(Pt 5), 1310–1323. https://doi.org/10.1093/brain/awn320

Morris, R. G., & Hannesdottir, K. (2004). Loss of "awareness" in Alzheimer's Disease. In The Cognitive Neuropsychology of Alzheimer's Disease (pp. 275–296).

Morris, R. G., & Kopelman, M. D. (1986). The Memory Deficits in Alzheimer-type Dementia: A Review. The Quarterly Journal of Experimental Psychology Section A, 38(4). https://doi.org/10.1080/14640748608401615

Morris, R. G., & Mograbi, D. C. (2013). Anosognosia, autobiographical memory and self knowledge in Alzheimer's disease. Cortex, 49(6), 1553–1565. https://doi.org/10.1016/j.cortex.2012.09.006

Morris, S. E., Yee, C. M., & Nuechterlein, K. H. (2006). Electrophysiological analysis of error monitoring in schizophrenia. Journal of Abnormal Psychology, 115(2), 239–250. https://doi.org/10.1037/0021-843X.115.2.239

Moscovitch, M., Rosenbaum, R. S., Gilboa, A., Addis, D. R., Westmacott, R., Grady, C., McAndrews, M. P., Levine, B., Black, S., Winocur, G., & Nadel, L. (2005). Functional neuroanatomy of remote episodic, semantic and spatial memory: a unified account based on multiple trace theory. Journal of Anatomy, 207(1), 35–66. https://doi.org/10.1111/j.1469-7580.2005.00421.x

Moscovitch, M., Westmacott, R., Gilboa, A., Addis, D. R., Rosenbaum, R. S., Viskontas, I., Priselac, S., Svoboda, E., Ziegler, M., Black, S., Gao, F., Grady, C., Freedman, M., Köhler, S., Leach, L., Levine, B., McAndrews, M. P., Nadel, L., Proulx, G., … Winocur, G. (2005). Hippocampal Complex Contribution to Retention and Retrieval of Recent and Remote Episodic and Semantic Memories: Evidence from Behavioral and Neuroimaging Studies of Healthy and Brain-

Damaged People. In Dynamic Cognitive Processes (pp. 333–380). Springer-Verlag. https://doi.org/10.1007/4-431-27431-6_14

Müller, A., Vetsch, S., Pershin, I., Candrian, G., Baschera, G.-M., Kropotov, J. D., Kasper, J., Rehim, H. A., & Eich, D. (2020). EEG/ERP-based biomarker/neuroalgorithms in adults with ADHD: Development, reliability, and application in clinical practice. The World Journal of Biological Psychiatry, 21(3), 172–182. https://doi.org/10.1080/15622975.2019.1605198

Murphy, P. R., Robertson, I. H., Allen, D., Hester, R., & O'Connell, R. G. (2012). An electrophysiological signal that precisely tracks the emergence of error awareness. Frontiers in Human Neuroscience, 6(MARCH 2012), 1–16. https://doi.org/10.3389/fnhum.2012.00065

Nelson, T. O. (1984). A comparison of current measures of the accuracy of feeling-of-knowing predictions. Psychological Bulletin, 95(1). https://doi.org/10.1037/0033-2909.95.1.109

Nieuwenhuis, S., Richard Ridderinkhof, K., Blom, J., Band, G. P. H., & Kok, A. (2001). Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. Psychophysiology, 38(5), 752–760. https://doi.org/10.1017/S0048577201001111

Nuzzo, D., Picone, P., Caruana, L., Vasto, S., Barera, A., Caruso, C., & di Carlo, M. (2014). Inflammatory mediators as biomarkers in brain disorders. Inflammation, 37(3). https://doi.org/10.1007/s10753-013-9780-2

O'Connell, R. G., Dockree, P. M., Bellgrove, M. A., Kelly, S. P., Hester, R., Garavan, H., Robertson, I. H., & Foxe, J. J. (2007). The role of cingulate cortex in the detection of errors with and without awareness: A high-density electrical mapping study. European Journal of Neuroscience, 25(8), 2571–2579. https://doi.org/10.1111/j.1460-9568.2007.05477.x

Olvet, D. M., & Hajcak, G. (2009). Reliability of error-related brain activity. Brain Research, 1284, 89–99. https://doi.org/10.1016/j.brainres.2009.05.079

Overbeek, T. J. M., Nieuwenhuis, S., & Ridderinkhof, K. R. (2005a). Dissociable components of error processing: On the functional significance of the Pe vis-à-vis

the ERN/Ne. In Journal of Psychophysiology (Vol. 19, Issue 4, pp. 319–329). https://doi.org/10.1027/0269-8803.19.4.319

Overbeek, T. J. M., Nieuwenhuis, S., & Ridderinkhof, K. R. (2005b). Dissociable Components of Error Processing. Journal of Psychophysiology, 19(4), 319–329. https://doi.org/10.1027/0269-8803.19.4.319

Ownsworth, T., & Fleming, J. (2005). The relative importance of metacognitive skills, emotional status, and executive function in psychosocial adjustment following acquired brain injury. Journal of Head Trauma Rehabilitation, 20(4). https://doi.org/10.1097/00001199-200507000-00004

Ownsworth, T., Fleming, J., Tate, R., Beadle, E., Griffin, J., Kendall, M., Schmidt, J., Lane-Brown, A., Chevignard, M., & Shum, D. H. K. (2017). Do People With Severe Traumatic Brain Injury Benefit From Making Errors? A Randomized Controlled Trial of Error-Based and Errorless Learning. Neurorehabilitation and Neural Repair, 31(12), 1072–1082. https://doi.org/10.1177/1545968317740635

Ownsworth, T., McFarland, K., & Young, R. McD. (2000). Development and Standardization of the Self-Regulation Skills Interview (SRSI): A New Clinical Assessment Tool for Acquired Brain Injury. The Clinical Neuropsychologist, 14(1), 76–92. https://doi.org/10.1076/1385-4046(200002)14:1;1-8;FT076

Pailing, P. E., & Segalowitz, S. J. (2004). The error-related negativity as a state and trait measure: Motivation, personality, and ERPs in response to errors. Psychophysiology, 41(1), 84–95. https://doi.org/10.1111/1469-8986.00124

Parnetti, L., Gaetani, L., Eusebi, P., Paciotti, S., Hansson, O., El-Agnaf, O., Mollenhauer, B., Blennow, K., & Calabresi, P. (2019). CSF and blood biomarkers for Parkinson's disease. In The Lancet Neurology (Vol. 18, Issue 6). https://doi.org/10.1016/S1474-4422(19)30024-9

Poil, S. S., de Haan, W., van der Flier, W. M., Mansvelder, H. D., Scheltens, P., & Linkenkaer-Hansen, K. (2013). Integrative EEG biomarkers predict progression to Alzheimer's disease at the MCI stage. Frontiers in Aging Neuroscience, 5(OCT). https://doi.org/10.3389/fnagi.2013.00058

Pontifex, M. B., Scudder, M. R., Brown, M. L., O'Leary, K. C., Wu, C. T., Themanson, J. R., & Hillman, C. H. (2010). On the number of trials necessary for

stabilization of error-related brain activity across the life span. Psychophysiology, 47(4). https://doi.org/10.1111/j.1469-8986.2010.00974.x

Prigatano, G. P. (1996). Neuropsychological rehabilitation after brain injury: Scientific and professional issues. Journal of Clinical Psychology in Medical Settings, 3(1). https://doi.org/10.1007/BF01989285

Prigatano, G. P., & Sherer, M. (2020). Impaired Self-Awareness and Denial During the Postacute Phases After Moderate to Severe Traumatic Brain Injury. Frontiers in Psychology, 11. https://doi.org/10.3389/fpsyg.2020.01569

Reed, B. R., Jagust, W. J., & Coulter, L. (1993). Anosognosia in Alzheimer's disease: Relationships to depression, cognitive function, and cerebral perfusion. Journal of Clinical and Experimental Neuropsychology, 15(2). https://doi.org/10.1080/01688639308402560

Reinhart, R. M. G., & Woodman, G. F. (2014). Causal Control of Medial-Frontal Cortex Governs Electrophysiological and Behavioral Indices of Performance Monitoring and Learning. Journal of Neuroscience, 34(12), 4214–4227. https://doi.org/10.1523/JNEUROSCI.5421-13.2014

Riesel, A., Weinberg, A., Endrass, T., Meyer, A., & Hajcak, G. (2013). The ERN is the ERN is the ERN? Convergent validity of error-related brain activity across different tasks. Biological Psychology, 93(3), 377–385. https://doi.org/10.1016/j.biopsycho.2013.04.007

Robertson, K., & Schmitter-Edgecombe, M. (2015). Self-awareness and traumatic brain injury outcome. Brain Injury, 29(7–8), 848–858. https://doi.org/10.3109/02699052.2015.1005135

Rouault, M., McWilliams, A., Allen, M. G., & Fleming, S. M. (2018). Human Metacognition Across Domains: Insights from Individual Differences and Neuroimaging. In Personality Neuroscience (Vol. 1). https://doi.org/10.1017/pen.2018.16

Scandola, M., Canzano, L., Avesani, R., Leder, M., Bertagnoli, S., Gobbetto, V., Aglioti, S. M., & Moro, V. (2021). Anosognosia for limb and bucco-facial apraxia as inferred from the recognition of gestural errors. Journal of Neuropsychology, 15(1). https://doi.org/10.1111/jnp.12203

Scheffers, M. K., & Coles, M. G. H. (2000). Performance monitoring in a confusing world: Error-related brain activity, judgments of response accuracy, and types of errors. Journal of Experimental Psychology: Human Perception and Performance, 26(1), 141–151. https://doi.org/10.1037/0096-1523.26.1.141

Segalowitz, S. J., Santesso, D. L., Murphy, T. I., Homan, D., Chantziantoniou, D. K., & Khan, S. (2010). Retest reliability of medial frontal negativities during performance monitoring. Psychophysiology, 47(2), 260–270. https://doi.org/10.1111/j.1469-8986.2009.00942.x

Seltzer, B., Vasterling, J. J., Yoder, J., & Thompson, K. A. (1997). Awareness of Deficit in Alzheimer's Disease: Relation to Caregiver Burden. The Gerontologist, 37(1), 20–24. https://doi.org/10.1093/geront/37.1.20

Sheline, Y. I., & Raichle, M. E. (2013). Resting state functional connectivity in preclinical Alzheimer's disease. In Biological Psychiatry (Vol. 74, Issue 5). https://doi.org/10.1016/j.biopsych.2012.11.028

Sherer, M., Bergloff, P., Boake, C., High, W., & Levin, E. (1998). The Awareness Questionnaire: Factor structure and internal consistency. Brain Injury, 12(1). https://doi.org/10.1080/026990598122863

Sherer, M., Bergloff, P., Levin, E., High, W. M., Oden, K. E., & Nick, T. G. (1998). Impaired Awareness and Employment Outcome after Traumatic Brain Injury. Journal of Head Trauma Rehabilitation, 13(5), 52–61. https://doi.org/10.1097/00001199-199810000-00007

Sherer, M., Hart, T., Nick, T. G., Whyte, J., Thompson, R. N., & Yablon, S. A. (2003). Early impaired self-awareness after traumatic brain injury. Archives of Physical Medicine and Rehabilitation, 84(2), 168–176. https://doi.org/10.1053/apmr.2003.50045

Sim, J., Brown, F. L., O'Connell, R. G., & Hester, R. (2020). Impaired error awareness in healthy older adults: an age group comparison study. Neurobiology of Aging, 96. https://doi.org/10.1016/j.neurobiolaging.2020.08.001

Solís-Vivanco, R., Rodríguez-Violante, M., Rodríguez-Agudelo, Y., Schilmann, A., Rodríguez-Ortiz, U., & Ricardo-Garcell, J. (2015). The P3a wave: A reliable

neurophysiological measure of Parkinson's disease duration and severity. Clinical Neurophysiology, 126(11). https://doi.org/10.1016/j.clinph.2014.12.024

Souchay, C. (2007). Metamemory in Alzheimer's Disease. Cortex, 43(7), 987–1003. https://doi.org/10.1016/S0010-9452(08)70696-8

Spalletta, G., Piras, F., Piras, F., Sancesario, G., Iorio, M., Fratangeli, C., Cacciari, C., Caltagirone, C., & Orfei, M. D. (2014). Neuroanatomical correlates of awareness of illness in patients with amnestic mild cognitive impairment who will or will not convert to Alzheimer's disease. Cortex, 61, 183–195. https://doi.org/10.1016/j.cortex.2014.10.010

Starkstein, S. E. (2014). Anosognosia in Alzheimer's disease: Diagnosis, frequency, mechanism and clinical correlates. Cortex, 61, 64–73. https://doi.org/10.1016/j.cortex.2014.07.019

Steinhauser, M., & Yeung, N. (2010). Decision processes in human performance monitoring. Journal of Neuroscience, 30(46), 15643–15653. https://doi.org/10.1523/JNEUROSCI.1899-10.2010

Steinhauser, M., & Yeung, N. (2012). Error awareness as evidence accumulation: effects of speed-accuracy trade-off on error signaling. Frontiers in Human Neuroscience, 6. https://doi.org/10.3389/fnhum.2012.00240

Strimbu, K., & Tavel, J. A. (2010). What are biomarkers? Current Opinion in HIV and AIDS, 5(6), 463–466. https://doi.org/10.1097/COH.0b013e32833ed177

Tabert, M. H., Albert, S. M., Borukhova-Milov, L., Camacho, Y., Pelton, G., Liu, X., Stern, Y., & Devanand, D. P. (2002). Functional deficits in patients with mild cognitive impairment: Prediction of AD. Neurology, 58(5). https://doi.org/10.1212/WNL.58.5.758

Therriault, J., Ng, K. P., Pascoal, T. A., Mathotaarachchi, S., Kang, M. S., Struyfs, H., Shin, M., Benedet, A. L., Walpola, I. C., Nair, V., Gauthier, S., & Rosa-Neto, P. (2018). Anosognosia predicts default mode network hypometabolism and clinical progression to dementia. Neurology, 90(11), e932–e939. https://doi.org/10.1212/WNL.0000000000005120

Turró-Garriga, O., Garre-Olmo, J., Reñé-Ramírez, R., Calvó-Perxas, L., Gascón-Bayarri, J., & Conde-Sala, J. L. (2016). Consequences of Anosognosia on the Cost of Caregivers' Care in Alzheimer's Disease. Journal of Alzheimer's Disease, 54(4). https://doi.org/10.3233/JAD-160419

Turró-Garriga, O., Garre-Olmo, J., Vilalta-Franch, J., Conde-Sala, J. L., de Gracia Blanco, M., & López-Pousa, S. (2013). Burden associated with the presence of anosognosia in Alzheimer's disease. International Journal of Geriatric Psychiatry, 28(3), 291–297. https://doi.org/10.1002/gps.3824

Tuthill, J. C., & Azim, E. (2018). Proprioception. Current Biology, 28(5), R194–R203. https://doi.org/10.1016/j.cub.2018.01.064

Ullsperger, M., Harsay, H. A., Wessel, J. R., & Ridderinkhof, K. R. (2010). Conscious perception of errors and its relation to the anterior insula. Brain Structure and Function, 214(5–6), 629–643. https://doi.org/10.1007/s00429-010-0261-1

Vaccaro, A. G., & Fleming, S. M. (2018). Thinking about thinking: A coordinate-based meta-analysis of neuroimaging studies of metacognitive judgements. Brain and Neuroscience Advances, 2, 239821281881059. https://doi.org/10.1177/2398212818810591

van Veen, V., & Carter, C. S. (2002). The tinning of action-monitoring processes in the anterior cingulate cortex. Journal of Cognitive Neuroscience, 14(4), 593–602. https://doi.org/10.1162/08989290260045837

Villalobos, D., Bilbao, Á., López-Muñoz, F., & Pacios, J. (2019). Improving Self-awareness after Acquired Brain Injury Leads to Enhancements in Patients' Daily Living Functionality. Brain Impairment. https://doi.org/10.1017/BrImp.2019.10

Vuorre, M., & Metcalfe, J. (2022). Measures of relative metacognitive accuracy are confounded with task performance in tasks that permit guessing. Metacognition and Learning, 17(2). https://doi.org/10.1007/s11409-020-09257-1

Webber, H. E., Lopez-Gamundi, P., Stamatovich, S. N., de Wit, H., & Wardle, M. C. (2021). Using pharmacological manipulations to study the role of dopamine in human reward functioning: A review of studies in healthy adults. In Neuroscience and Biobehavioral Reviews (Vol. 120). https://doi.org/10.1016/j.neubiorev.2020.11.004

Weinberg, A., Dieterich, R., & Riesel, A. (2015). Error-related brain activity in the age of RDoC: A review of the literature. International Journal of Psychophysiology, 98(2), 276–299. https://doi.org/10.1016/j.ijpsycho.2015.02.029

Weinberg, A., & Hajcak, G. (2011). Longer term test-retest reliability of error-related brain activity. Psychophysiology, 48(10). https://doi.org/10.1111/j.1469-8986.2011.01206.x

Wessel, J. R. (2012). Error awareness and the error-related negativity: Evaluating the first decade of evidence. Frontiers in Human Neuroscience, 6(APRIL 2012), 1–16. https://doi.org/10.3389/fnhum.2012.00088

Wessel, J. R. (2018). An adaptive orienting theory of error processing. Psychophysiology, 55(3), e13041. https://doi.org/10.1111/psyp.13041

Wessel, J. R., Danielmeier, C., & Ullsperger, M. (2011a). Error awareness revisited: Accumulation of multimodal evidence from central and autonomic nervous systems. Journal of Cognitive Neuroscience, 23(10), 3021–3036. https://doi.org/10.1162/jocn.2011.21635

Wessel, J. R., Danielmeier, C., & Ullsperger, M. (2011b). Error Awareness Revisited: Accumulation of Multimodal Evidence from Central and Autonomic Nervous Systems. Journal of Cognitive Neuroscience, 23(10), 3021–3036. https://doi.org/10.1162/jocn.2011.21635

Xu, H., Gu, L., Zhang, S., Wu, Y., Wei, X., Wang, C., Xu, Y., & Guo, Y. (2022). N200 and P300 component changes in Parkinson's disease: a meta-analysis. Neurological Sciences. https://doi.org/10.1007/s10072-022-06348-6

Yeo, Y. X., Pestell, C. F., Bucks, R. S., Allanson, F., & Weinborn, M. (2021). Metacognitive knowledge and functional outcomes in adults with acquired brain injury: A meta-analysis. In Neuropsychological Rehabilitation (Vol. 31, Issue 3). https://doi.org/10.1080/09602011.2019.1704421

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. Psychological Review, 111(4), 931–959. https://doi.org/10.1037/0033-295X.111.4.931

Yu, L., Mottola, G., Wilson, R. S., Valdes, O., Bennett, D. A., & Boyle, P. A. (2022). Metamemory and Financial Decision Making in Older Adults Without Dementia. Neuropsychology, 36(1). https://doi.org/10.1037/neu0000773

Appendix A

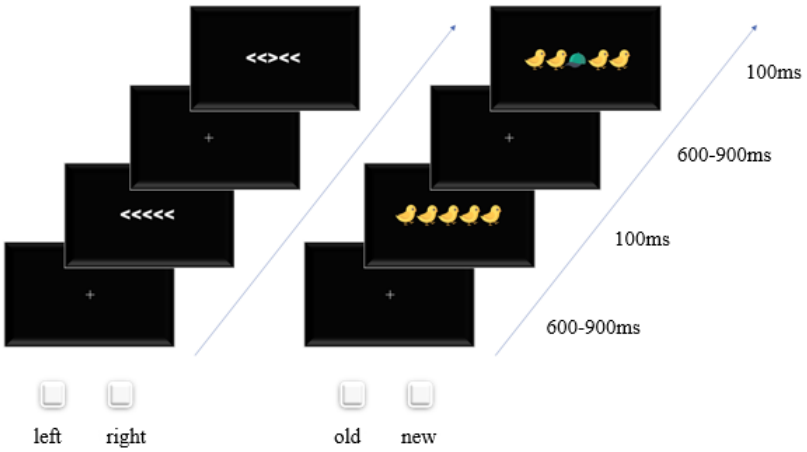

Figure 1. Graphical representation of experimental tasks: perceptual domain (on the left) and memory domain (on the right

Table 1. Mean (SD) response-locked ERP amplitude (μV), and latency (ms) summary data as a function of age group and task domain.

	Amplitude (μV)				**Latency (ms)**			
	Young Adults		Older Adults		Young Adults		Older Adults	
ERN	Perceptual	Memory	Perceptual	Memory	Perceptual	Memory	Perceptual	Memory
F1	-2.77	-1.67	-2.49	-3.11	34.37	38.94	36.67	42.22
	(4.90)	(5.28)	(5.28)	(6.05)	(8.12)	(5.87)	(7.65)	(8.43)
FC1	-1.21	-0.34	-1.83	-2.52	35.23	37.84	38.65	43.91
	(4.58)	(4.91)	(4.02)	(5.17)	(7.61)	(6.24)	(6.93)	(9.87)
Fz	-3.35	-2.1	-2.11	-2.98	35.14	38.55	37.05	42.04
	(5.13)	(5.86)	(5.03)	(5.83)	(7.29)	(6.35)	(7.28)	(9.74)
F2	-2.05	-1.09	-1.77	-2.28	35.04	38.59	37.05	41.75
	(4.91)	(5.61)	(4.48)	(5.15)	(7.03)	(6.04)	(7.18)	(9.14)
FC2	-1.80	-0.8	-1.41	-2.17	33.79	37.46	38.33	42.71
	(5.56)	(6.06)	(4.50)	(5.01)	(6.43)	(6.2)	(7.13)	(10.33)
FCz	-3.01	-1.75	-2.73	-3.00	33.25	37.59	38.46	42.69
	(6.00)	(6.52)	(5.79)	(6.09)	(6.69)	(6.26)	(7.12)	(10.02)
CRN	Perceptual	Memory	Perceptual	Memory	Perceptual	Memory	Perceptual	Memory
F1	2.38	4.17	1.08	2.06	39.7	38.48	39.96	39.83
	(3.64)	(3.50)	(4.04)	(4.01)	(5.54)	(5.25)	(5.73)	(5.56)
FC1	2.77	4.40	1.82	2.66	41.37	40.01	40.93	40.43
	(3.31)	(3.19)	(3.31)	(3.35)	(5.29)	(5.42)	(5.24)	(5.19)
Fz	2.37	4.21	1.26	2.26	39.52	38.16	39.68	39.17
	(3.74)	(3.6)	(4.10)	(3.97)	(5.44)	(5.7)	(6.14)	(4.94)
F2	2.62	4.22	1.29	2.34	39.67	38.35	39.86	39.52
	(3.86)	(3.44)	(3.97)	(3.66)	(5.54)	(5.25)	(5.91)	(4.74)

FC2	3.06	4.65	2.12	3.23	40.88	39.29	40.37	39.89
	(4.15)	(3.71)	(4.05)	(3.74)	(5.87)	(5.84)	(6.23)	(4.97)
FCz	2.76	4.69	1.97	3.06	40.03	38.55	39.72	39.23
	(4.27)	(3.97)	(4.35)	(4.04)	(5.81)	(5.79)	(6.26)	(5.43)

Pe	Perceptual	Memory	Perceptual	Memory	Perceptual	Memory	Perceptual	Memory
CP1	6,95	6,17	3,53	2,7	293,38	287,71	295,72	304,01
	(5,89)	(4,79)	(5,28)	(4,74)	(20,37)	(16,49)	(26,6)	(24,68)
P1	6,44	4,48	3,34	1,22	300,33	291,38	302,33	304,85
	(5,39)	(4,39)	(4,26)	(3,84)	(21,09)	(14,75)	(25,15)	(22,1)
Pz	7,02	5,28	3,34	1,45	300,61	290,09	302,06	303,73
	(5,61)	(4,77)	(4,55)	(4,41)	(23,23)	(15,7)	(25,59)	(20,33)
CPz	7,64	7,04	3,87	2,87	294,97	287,84	299,27	303,11
	(5,98)	(5,22)	(5,59)	(4,84)	(21,83)	(13,79)	(24,31)	(26,26)
CP2	7,18	6,32	3,21	2,37	295,84	288,01	297,58	300,36
	(6,02)	(5,06)	(4,80)	(4,24)	(20,58)	(14,13)	(21,56)	(23)
P2	6,95	4,96	3,01	0,95	301,07	291,9	304,1	303,98
	(5,53)	(4,78)	(4,11)	(4,09)	(22,24)	(15,55)	(23,1)	(24,04)

Pc	Perceptual	Memory	Perceptual	Memory	Perceptual	Memory	Perceptual	Memory
CP1	-3,43	-1,68	-5,52	-3,22	294,47	293,58	293,93	295,33
	(3,70)	(2,82)	(4,94)	(5,07)	(9,87)	(11,16)	(11,66)	(10,48)
P1	-3,58	-3,04	-5,73	-4,40	299,59	297,28	295,91	297,68
	(3,78)	(3,21)	(5,01)	(4,89)	(10,59)	(11,64)	(11,08)	(9,84)
Pz	-3,64	-2,76	-6,11	-4,44	298,19	295,92	296,26	297,92
	(3,86)	(3,2)	(5,22)	(4,9)	(10,52)	(11,22)	(9,97)	(9,29)

CPz	-3,42 (3,92)	-1,56 (2,89)	-5,52 (5,43)	-3,18 (5,31)	293,52 (10,4)	293,72 (10,9)	294,19 (10,76)	296,53 (10,84)
CP2	-3,15 (3,58)	-1,5 (2,72)	-5,51 (5,10)	-3,09 (4,96)	294,95 (11,2)	294,82 (11,08)	295,59 (10,37)	297,9 (10,56)
P2	-3,51 (3,75)	-2,82 (3,26)	-6,01 (5,30)	-4,31 (4,77)	299,08 (10,61)	297,24 (10,45)	298,63 (9,34)	299,85 (9,24)

Table 2. MLM for ERN/CRN amplitude
95% CI. 95% confidence interval; ICC. intraclass correlation coefficient

Intercept-only	**b**	**95% CI**	**df**	**t**	**p**
(Intercept)	1.42	0.67 – 2.18	65.82	3.77	**<.001**

Random effects	**Variance**	**SD**	**ICC**
Subject	11.67	3.41	0.13
Response	52.29	7.23	0.57
Domain	7.87	2.80	0.09
Response x Domain	17.76	4.21	0.19
Channel	0.14	0.38	0.002
Residual	1.19	1.09	

	b	**95% CI**	**df**	**t**	**p**
Intercept	0.36	-0.45 – 1.16	69.29	0.88	.382
Response	4.91	3.67 – 6.15	73.00	7.90	**<.001**
Domain	0.80	0.18 – 1.42	73.00	2.57	**.012**

Group	-0.98	-2.48 – 0.51	73.00	-1.31	.193
Response x Domain	1.14	0.18 – 2.11	72.99	2.37	**.020**
Response x Group	-0.89	-3.36 – 1.59	73.00	-0.72	.477
Domain x Group	-1.20	-2.44 – 0.04	73.00	-1.94	.057
Response x Domain x Group	0.98	-0.95 – 2.90	72.99	1.01	.315

Table 3. MLM for ERN latency
95% CI. 95% confidence interval; ICC. intraclass correlation coefficient

Intercept-only	**b**	**95% CI**	**df**	**t**	**p**
(Intercept)	39.07	38.08 – 40.06	78.99	69.67	**<.001**

Random effects	**Variance**	**SD**	**ICC**
Subject	16.38	4.05	0.15
Response	49.99	7.07	0.47
Domain	25.87	5.09	0.24
Channel	0.15	0.38	0.001
Residual	13.48	3.67	

	b	**95% CI**	**df**	**t**	**p**
Intercept	38.96	37.98 – 39.93	68.99	79.63	**<.001**
Response	1.47	-0.13 – 3.07	73.01	1.83	.071
Domain	1.69	0.52 – 2.85	72.99	2.88	**.005**

Group	2.10	0.25 – 3.94	73.00	2.27	**.026**
Response x Domain	-5.17	-5.80 – -4.54	1568.00	-16.10	**<.001**
Response x Group	-3.43	-6.63 – -0.23	73.01	-2.14	**.036**
Domain x Group	1.07	-1.26 – 3.40	72.99	0.91	.364
Response x Domain x Group	-0.18	-1.44 – 1.08	1568.00	-0.28	.778

Table 4. MLM for Pe/Pc amplitude
95% CI. 95% confidence interval; ICC. intraclass correlation coefficient

Intercept-only	**b**	**95% CI**	**df**	**t**	**p**
(Intercept)	0.48	-0.30 – 1.25	45.19	1.24	.221

Random effects	**Variance**	**SD**	**ICC**
Subject	8.82	2.97	0.06
Response	101.52	10.09	0.65
Domain	13.05	3.61	0.08
Response x Domain	30.22	5.50	0.19
Channel	0.24	0.48	0.001
Residual	1.92	1.38	

	b	**95% CI**	**df**	**t**	**p**
Intercept	0.36	-0.38 – 1.09	39.44	0.99	.332

Response	-8.31	-9.63 – -6.98	73.00	-12.51	**<.001**
Domain	0.10	-0.75 – 0.95	73.00	0.24	.811
Group	-2.81	-4.03 – -1.60	73.00	-4.61	**<.001**
Response*Domain	2.98	1.88 – 4.09	73.00	5.38	**<.001**
Response*Group	1.80	-0.85 – 4.45	73.00	1.36	.179
Domain*Group	0.30	-1.40 – 2.00	73.00	0.35	.726
Response*Domain*Group	0.86	-1.35 – 3.07	73.00	0.78	**.439**

Table 5. MLM for Pe latency
95% CI. 95% confidence interval; ICC. intraclass correlation coefficient

Intercept-only	**b**	**95% CI**	**df**	**t**	**p**
(Intercept)	296.44	293.88 – 299.01	24.46	238.4	**<.001**

Random effects	**Variance**	**SD**	**ICC**
Subject	107.10	10.35	0.16
Response	307.36	17.53	0.48
Domain	148.46	12.18	0.23
Channel	3.69	1.92	0.005
Residual	73.18	8.55	

	b	**95% CI**	**df**	**t**	**p**
Intercept	297.01	294.11 – 299.90	36.65	207.83	**<.001**

Response	-1.34	-5.41 – 2.72	73.00	-0.66	.513
Domain	-1.12	-3.94 – 1.71	73.00	-0.79	.433
Group	4.39	-0.37 – 9.15	73.00	1.84	.070
Response*Domain	2.81	1.25 – 4.37	1568.00	3.53	**<.001**
Response*Group	-7.55	-15.68 – 0.58	73.00	-1.85	.068
Domain*Group	7.18	1.54 – 12.83	73.00	2.54	**.013**
Response*Domain*Group	-8.38	-11.51 – -5.26	1568.00	-5.26	**<.001**

Appendix B

Figure 1. Graphical representation of the experimental task trial.

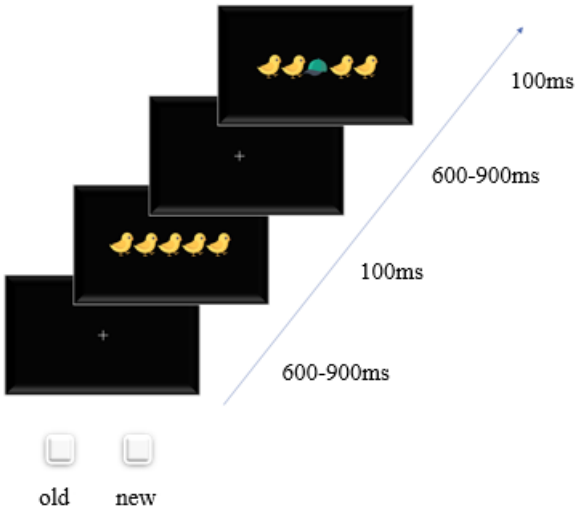

Table 1. Pe amplitude (μV),) summary data at single electrode site as a function of age group, and response correctness. Mean (SD).

	Young adults		Older adults	
	Error	Correct	Error	Correct
CP1	6,17 (4,79)	-1,68 (2,82)	2,7 (4,74)	-3,22 (5,07)
P1	4,48 (4,39)	-3,04 (3,21)	1,22 (3,84)	-4,40 (4,89)
Pz	5,28 (4,77)	-2,76 (3,2)	1,45 (4,41)	-4,44 (4,9)
CPz	7,04 (5,22)	-1,56 (2,89)	2,87 (4,84)	-3,18 (5,31)
CP2	6,32 (5,06)	-1,5 (2,72)	2,37 (4,24)	-3,09 (4,96)
P2	4,96 (4,78)	-2,82 (3,26)	0,95 (4,09)	-4,31 (4,77)

Table 2. Stimulus-locked ERPs amplitude (μV),) summary data at single electrode site as a function of age group, familiarity and congruency. Mean (SD).

N1	Young adults				Older adults			
	Old congruent	Old incongruent	New congruent	New incongruent	Old congruent	Old incongruent	New congruent	New incongruent
C1	-2.751 (8.789)	-3.17 (9.338)	-2.725 (9.086)	-2.271 (8.779)	-1.145 (8.332)	-1.254 (8.27)	-1.414 (8.066)	-1.102 (7.953)
C2	-2.778 (8.904)	-2.76 (8.854)	-3.204 (8.969)	-2.26 (8.66)	-1.044 (8.267)	-1.165 (8.01)	-1.256 (8.433)	-1.104 (7.808)
CP1	-3.034 (9.133)	-2.701 (8.877)	-3.132 (9.005)	-3.641 (9.523)	-1.491 (8.136)	-1.166 (7.937)	-1.165 (8.406)	-1.586 (8.458)
CP2	-3.263 (8.877)	-3.664 (9.34)	-3.243 (9.089)	-2.868 (8.867)	-1.561 (8.298)	-1.85 (8.331)	-1.719 (8.236)	-1.42 (7.936)
CPz	-3.201 (8.874)	-2.916 (8.755)	-3.454 (8.847)	-2.816 (8.814)	-1.442 (8.206)	-1.296 (7.986)	-1.408 (8.304)	-1.33 (7.949)

Cz	-3.349 (9.015)	-2.924 (8.706)	-3.4 (8.823)	-3.787 (9.371)	-1.701 (8.205)	-1.3 (7.979)	-1.262 (8.469)	-1.581 (8.467)

P2	Young adults				Older adults			
	Old congruent	Old incongruent	New congruent	New incongruent	Old congruent	Old incongruent	New congruent	New incongruent
FC1	1.729 (10.527)	0.706 (10.451)	0.796 (10.472)	1.223 (10.45)	5.891 (10.124)	5.085 (9.762)	5.325 (10.051)	5.702 (9.745)
FC3	1.71 (9.66)	0.682 (9.45)	0.91 (9.619)	1.275 (9.559)	5.222 (9.693)	4.492 (9.433)	4.811 (9.741)	5.264 (9.404)
Fz	1.659 (10.755)	0.549 (10.731)	0.772 (10.69)	1.082 (10.723)	6.063 (10.063)	5.073 (9.857)	5.51 (10.112)	5.92 (9.723)
F2	1.674 (10.56)	0.59 (10.43)	0.753 (10.415)	1.127 (10.416)	6.173 (10.027)	5.322 (10.03)	5.614 (10.105)	6.198 (9.759)
FC2	1.281 (10.54)	0.284 (10.344)	0.395 (10.389)	0.737 (10.389)	5.805 (10.086)	4.995 (9.98)	5.174 (10.086)	5.83 (9.697)
FCz	1.354 (10.759)	0.233 (10.687)	0.415 (10.773)	0.802 (10.715)	5.606 (10.211)	4.81 (10.066)	5.079 (10.379)	5.629 (9.994)

N2	Young adults				Older adults			
	Old congruent	Old incongruent	New congruent	New incongruent	Old congruent	Old incongruent	New congruent	New incongruent
FC1	-2.593 (10.943)	-4.145 (11.071)	-3.487 (10.941)	-5.78 (11.542)	-1.741 (11.827)	-2.38 (11.488)	-1.889 (11.498)	-3.435 (11.741)
FC3	-1.395 (10.054)	-2.81 (10.065)	-2.422 (10.084)	-4.104 (10.418)	-1.566 (11.309)	-2.059 (11.113)	-1.421 (11.15)	-2.808 (11.381)
Fz	-2.878 (11.199)	-4.593 (11.344)	-3.932 (11.127)	-6.208 (11.704)	-1.714 (11.825)	-2.663 (11.542)	-1.909 (11.521)	-3.496 (11.584)
F2	-2.617 (11.004)	-4.245 (11.023)	-3.543 (10.818)	-5.805 (11.357)	-1.288 (11.734)	-2.137 (11.59)	-1.353 (11.479)	-3.017 (11.671)
FC2	-2.277 (10.981)	-3.741 (10.946)	-3.409 (10.83)	-5.258 (11.239)	-1.725 (11.86)	-2.593 (11.798)	-1.683 (11.541)	-3.539 (11.664)
FCz	-2.589 (11.208)	-4.346 (11.238)	-3.86 (11.143)	-5.968 (11.669)	-2.621 (12.258)	-3.305 (11.983)	-2.505 (11.959)	-4.301 (12.221)

FN400	Young adults				Older adults			
	Old congruent	Old incongruent	New congruent	New incongruent	Old congruent	Old incongruent	New congruent	New incongruent
FC1	2.506 (12.005)	0.181 (11.688)	-0.628 (11.832)	-2.469 (11.807)	6.117 (13.06)	4.829 (12.783)	3.192 (12.828)	2.409 (12.882)
FC3	4.532 (11.115)	2.199 (10.831)	0.426 (10.939)	-0.99 (10.871)	5.45 (12.325)	4.208 (12.222)	2.46 (12.225)	1.923 (12.269)
Fz	1.873 (12.16)	-0.475 (11.963)	-0.971 (12.057)	-2.9 (12.047)	6.078 (13.031)	4.574 (12.952)	3.262 (12.805)	2.477 (12.767)
F2	2.169 (11.941)	-0.088 (11.685)	-0.255 (11.804)	-2.249 (11.84)	6.15 (12.753)	4.796 (12.689)	3.54 (12.777)	2.707 (12.652)

FC2	3.436 (11.92)	1.393 (11.6)	0.604 (11.947)	-1.211 (11.79)	5.215 (13.033)	4.19 (13.057)	2.936 (12.714)	2.04 (12.662)
FCz	3.443 (12.216)	1.008 (11.985)	-0.288 (12.024)	-2.197 (11.99)	5.053 (13.691)	4.008 (13.4)	2.472 (13.265)	1.597 (13.277)

LPC	Young adults				Older adults			
	Old congruent	Old incongruent	New congruent	New incongruent	Old congruent	Old incongruent	New congruent	New incongruent
CP1	9.671 (12.253)	8.131 (12.167)	6.463 (11.988)	5.225 (12.178)	11.043 (12.037)	9.884 (12.199)	7.276 (12.125)	5.397 (12.399)
P1	8.333 (11.06)	7.487 (11.255)	5.921 (11.065)	4.984 (11.106)	9.731 (11.637)	9.239 (11.842)	6.42 (11.579)	5.036 (11.72)
Pz	9.197 (11.697)	7.903 (11.665)	6.121 (11.523)	5.071 (11.319)	10.308 (11.937)	9.563 (12.345)	7.055 (11.664)	5.268 (11.929)
CPz	9.894 (12.602)	8.307 (12.235)	6.652 (12.286)	5.527 (12.354)	11.029 (12.485)	9.763 (12.689)	7.702 (12.324)	5.737 (12.418)
CP2	9.337 (12.474)	7.714 (12.213)	6.343 (12.142)	5.255 (12.174)	10.443 (12.044)	9.331 (12.382)	7.992 (12.016)	5.97 (12.103)
P2	8.443 (11.436)	7.467 (11.494)	5.411 (11.255)	4.768 (11.328)	9.641 (11.331)	9.036 (11.548)	6.831 (11.162)	5.189 (11.322)

Table 3. MLM for stimulus-locked ERPs amplitude
95% CI. 95% confidence interval.

N1	*b*	*95% CI*	*df*	*t*	*p*
Intercept	-2.23	-2.64 – -1.82	56.09	-10.92	**<.001**
Familiarity	-0.13	-0.31 – 0.04	73.00	-1.51	.136
Congruency	-0.14	-0.30 – 0.01	73.00	-1.88	**.064**
Group	1.60	0.87 – 2.32	73.00	4.40	**<.001**
Familiarity x Congruency	0.66	0.31 – 1.00	73.00	3.83	**<.001**
Familiarity x Group	0.04	-0.31 – 0.39	73.00	0.25	.804
Congruency x Group	0.26	-0.05 – 0.57	73.00	1.69	.095
Familiarity x Congruency x Group	-0.16	-0.84 – 0.52	73.00	-0.47	.642

P2	*b*	*95% CI*	*df*	*t*	*p*
Intercept	3.09	2.18 – 4.00	73.01	6.78	**<.001**
Familiarity	-0.02	-0.22 – 0.18	73.01	-0.22	.829
Congruency	-0.69	-0.89 – -0.48	73.01	-6.71	**<.001**
Group	4.22	2.41 – 6.04	73.01	4.63	**<.001**
Familiarity x Congruency	0.48	0.07 – 0.90	73.01	2.31	**.024**
Familiarity x Group	0.38	-0.03 – 0.78	73.01	1.86	.067
Congruency x Group	0.05	-0.36 – 0.46	73.01	0.24	.811
Familiarity x Congruency x Group	-0.40	-1.24 – 0.43	73.01	-0.96	.339

N2	*b*	*95% CI*	*df*	*t*	*p*
Intercept	-3.21	-4.29 – -2.13	73.00	-5.91	**<.001**
Familiarity	-0.85	-1.19 – -0.51	73.00	-5.00	**<.001**
Congruency	-1.52	-1.79 – -1.24	73.00	-10.91	**<.001**

Group	1.26	-0.90 – 3.43	73.00	1.16	.248
Familiarity x Congruency	-0.65	-1.12 – -0.19	73.00	-2.82	**.006**
Familiarity x Group	0.94	0.26 – 1.62	73.00	2.75	**.007**
Congruency x Group	0.66	0.11 – 1.22	73.00	2.39	**.020**
Familiarity x Congruency x Group	-0.39	-1.31 – 0.54	73.00	-0.83	.408

FN400	*b*	*95% CI*	*df*	*t*	*p*
Intercept	2.13	0.83 – 3.43	73.01	3.26	**.002**
Familiarity	-2.70	-3.19 – -2.22	73.00	-11.16	**<.001**
Congruency	-1.54	-1.85 – -1.23	72.99	-9.99	**<.001**
Group	3.27	0.66 – 5.87	73.01	2.50	**.015**
Familiarity x Congruency	0.50	-0.02 – 1.02	73.01	1.93	.057
Familiarity x Group	0.56	-0.41 – 1.53	73.00	1.16	.252
Congruency x Group	1.06	0.45 – 1.68	72.99	3.44	**.001**
Familiarity x Congruency x Group	0.05	-0.99 – 1.09	73.01	0.10	.923

LPC	*b*	*95% CI*	*df*	*t*	*p*
Intercept	7.59	6.49 – 8.70	76.84	13.64	**<.001**
Familiarity	-3.22	-3.67 – -2.77	73.01	-14.20	**<.001**
Congruency	-1.29	-1.61 – -0.96	73.00	-7.92	**<.001**
Group	0.93	-1.19 – 3.05	73.00	0.87	.385
Familiarity x Congruency	-0.26	-0.78 – 0.25	73.01	-1.01	.315
Familiarity x Group	-0.73	-1.63 – 0.18	73.01	-1.60	.113
Congruency x Group	-0.25	-0.90 – 0.40	73.00	-0.77	.442
Familiarity x Congruency x Group	-1.19	-2.22 – -0.15	73.01	-2.29	**.025**