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EEG markers of cognitive performance in bipolar disorder – A systematic review

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ABSTRACT

Background: People with bipolar disorder (BD) may experience impairing cognitive deficits, even in remission. Electroencephalographic (EEG) measures can depict neurophysiological activity with high temporal resolution. They could therefore be an adequate method to pinpoint the cognitive impairments in BD, facilitating understanding of when exactly the cognitive processing is disrupted and what neurophysiological systems are involved. In the absence of a previous literature examination, this systematic review aimed to synthesize the evidence of associations between EEG and cognitive measures to identify electrophysiological markers of cognitive performance in BD.

Methods: A systematic search across PubMed, EMBASE, APA PsycInfo and Cochrane Library until November 2023 was undertaken to identify studies in which a direct correlation between any continuous EEG measure and any continuous cognitive measure in participants with BD was reported. A narrative synthesis approach was used to present the identified correlations, across five cognitive (attention and processing speed, working memory, episodic memory, executive function, and intellectual capacity) and four EEG domains (event-related potentials (ERP), spectral, connectivity and other measures).

Results: A total of 16 articles describing 15 studies were included in the review. Six studies identified significant correlations. Most significant correlations were reported between ERP measures and attention and processing speed performance, several between ERP measures and executive functioning and one within the working memory and the intellectual capacity domain respectively. However, most of the identified significant correlations were conflicting within (different measures or mood states) and across studies with no consistent significant correlation across studies. The majority of identified correlations were non-significant.

Conclusions: As yet no robust EEG markers of cognitive performance in people with BD are known. This review highlights the heterogeneity in measures and participant characteristics between studies and the need for standardization. Further studies with homogeneous methods and participant groups may help to establish consistent associations.

1. Introduction

Bipolar disorder (BD) is a chronic mental health condition that significantly affects cognitive functioning in a substantial proportion of patients. Individuals with BD often experience persisting cognitive impairments, such as difficulties with memory, attention, or executive function (Robinson et al., 2006; Bourne et al., 2013). These impairments can persist in remission (Tsapekos et al., 2021), are directly linked to lower psychosocial functioning and quality of life and may even pose a risk for affective relapse (O'Donnell et al., 2017; Sanchez-Moreno et al., 2018). Notably, difficulties can be seen in both "hot" (emotional processing) as well as "cold" (non-emotional) cognition (Douglas et al.,

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2020). Cognitive trajectories in BD may differ between individuals. A proposed model suggests three discrete cognitive subtypes: The first group evidences no cognitive decline and performance that is comparable to healthy individuals throughout adulthood; the second group experiences decline after illness onset but quickly reaches plateau, potentially due to neurotoxic effects from repeated mood episodes and incomplete recovery; the third group is considered more "neurodevelopmental" characterised by lower premorbid IQ and widespread global cognitive impairments, that may further decline after illness onset, similar but typically less severe to patterns seen in schizophrenia patients (Millett and Burdick, 2021).

The exact neurobiological underpinnings of cognitive impairments remain unclear and may differ between subgroups. As mentioned, disease progression as well as episode burden may play a role, as might age of onset, certain medication or somatic and psychiatric comorbidities that impact cognition (Cullen et al., 2016; Gogia et al., 2022; Wingo et al., 2009). The presence of cognitive difficulties in remission might even suggest that cognition is a core component related to the pathophysiology of BD (Morsel et al., 2018), especially in the "neurodevelopmental" subgroup. Therefore, it is important to better understand the neural underpinnings of these deficits to be able to effectively target them with novel treatments and ultimately decrease their burden. In addition, this would help to examine similarities and differences between cognitive subgroups within BD and between diagnostic categories, especially with schizophrenia and major depression, as it is currently unknown whether shared manifestations of cognitive dysfunction reflect shared neurobiological pathways or whether the sources of impairments differ between disorders (Kessing and Miskowiak, 2018).

One widely used method to measure neurophysiological activity is the electroencephalogram (EEG). An EEG directly measures brain activity, specifically electrical activity, with electrodes attached to the scalp (Teplan, 2002) and can therefore provide a real-time reflection of cognitive processing and its changes in BD. The number of electrodes can vary from one to approximately 32 electrodes for low density to up to 256 electrodes for high density EEGs, with more channels resulting in increasing spatial resolution (Stoyell et al., 2021). An EEG reflects mostly cortical activity and has high temporal resolution that can adequately depict fast changes in neuronal networks (Nunez, 1981) and therefore increase our understanding of functional alterations in mental disorders. It can be divided into resting state measures (where participants are typically instructed to look at a fixation point or close their eyes) as well as task-based measures (where participants perform various kinds of tasks, such as oddball or Stroop tasks). Task based measures enable event related potential (ERP) analyses, time-frequency (spectral) analyses and connectivity analyses. ERP analyses allow to observe different electrophysiological components (e.g., P1, N2; see Sur and Sinha (2009) for an overview), that represent specific cognitive processing stages occurring during a task (Nunez, 1981) and are therefore directly linked to cognitive function. For example, P1 reflects an early process of (visual) perception occurring 50–150 msec after the presentation of a (visual) stimulus (Desmedt et al., 1983). N2 is a negative ERP deflection that occurs in tasks involving attention 200–350 msec after stimulus presentation (Folstein and Van Petten, 2008). Time-frequency analyses can describe the dynamics of frequency, power and phase even when signals do not occur at the same time across trials (non-phase-locked) (Morales and Bowers, 2022), while connectivity analyses aim to characterize the patterns of interaction within and between different brain regions (Chiarion et al., 2023). Research has shown abnormalities in these EEG measures in BD participants (Basar and Guntekin, 2013; Özerdem et al., 2013). For example, P3 amplitudes are commonly found to be reduced in BD patients across mood phases (Morsel et al., 2018; Wada et al., 2019), while evoked and event-related theta responses are significantly decreased in the frontoparietal networks in euthymia compared to control subjects (Lu et al., 2022).

It is important to pinpoint the nature of cognitive abnormalities in BD to be able to understand when exactly cognitive processing is disrupted. Testing cognition in BD can be challenging, as heterogeneity between the aforementioned subgroups and between mood states can be substantial and influence performance (Kurtz and Gerraty, 2009; Martinez-Aran and Vieta, 2015). EEG measures as adjunctions in cognition studies could add value in helping establish cognitive disruptions with greater accuracy, especially if the neurobiological correlates of the EEG signal are understood. For example, among others, different impairments in neurotransmitter channels and receptors, as well as GABA signalling, have been identified as the main molecular contributors to abnormalities in low-frequency brain oscillations (Lu et al., 2022). Identifying EEG markers of cognitive performance could therefore assist with a better understanding of the pathophysiology, prognosis and inform treatment strategies (e.g., psychopharmacology, cognitive remediation therapy (CRT)). Previous literature reviews have separately looked into cognitive ERPs (Morsel et al., 2018), brain oscillations (Basar et al., 2013; Basar and Guntekin, 2013; Özerdem et al., 2013) and cognitive dysfunction (Kessing and Miskowiak, 2018) in BD and other related mental disorders. Lu et al. (2022) investigated the association between brain oscillations and cognitive performance in BD, though not in a systematic fashion, and found various examples of deviant brain oscillations and how they might be connected to cognition, such as associations with decreased prefrontal cortical grey matter, decreased hippocampal volume, or aberrant GABAergic neurotransmission signalling. Moreover, a recent systematic review by Perez-Ramos et al. (2024) has compiled evidence on the correlation of neuroimaging, physiological, genetic, and peripheral biomarkers with cognition in different mood states in BD. The included studies reported several significant associations between neuroimaging and peripheral biomarkers with cognition across mood states. Regarding neuroimaging biomarkers, they identified multiple studies reporting hypoactivation in the prefrontal and parietal cortex in tasks involving executive functioning or working memory during acute mood episodes, but most other associations were only reported by single studies. This review proposed that the identification of biomarkers for cognition across mood states in BD is crucial for preventing mood episodes and cognitive deterioration and their findings highlight core regions in the search for them. However, the authors highlighted the need for greater consistency in paradigms and techniques employed to better compare and interpret the results.

Taken together, EEG is a direct measure of neural activity with high temporal resolution and therefore provides sensitive markers of physiological and pathological brain states, also during cognitive processing. Moreover, research has identified abnormalities in both EEG as well as cognitive measures in BD and is beginning to determine the underlying biological mechanisms behind the EEG measures. Thus, examining EEG measures from a cognitive neuroscience perspective in a systematic manner could lead to a fruitful overview of EEG-cognition correlates that shed light on the biological underpinnings of cognitive deficits in BD. It could also yield important insights into their treatment, as well as into similarities and differences between diagnostic categories and cognitive subtypes. Moreover, currently employed EEG and cognitive performance measures are very heterogeneous, which impedes interpretability. Consistent correlations would help to identify meaningful EEG measures that can be included in cognitive studies to standardize future trials and harmonize results. To our knowledge, no systematic review has examined correlations between performance on specific cognitive tasks and EEG measures. As such, this review aimed to synthesize the evidence on the association between EEG and cognitive measures in BD, by identifying all studies that reported a direct correlation between any continuous EEG measure (e.g., amplitude) and any continuous, validated cognitive measure in participants with BD.

2. Methods

2.1. Registration

This review was preregistered with the International Prospective Register of Systematic Reviews (PROSPERO; registration identifier CRD42022373097) and followed the PRISMA 2020 (Page et al., 2021) guidelines.

2.2. Search strategy

Systematic searches were conducted in PubMed, EMBASE, APA PsycInfo and Cochrane Library using the following search terms: ("bipolar" OR "manic" OR "mania") AND ("cognition" OR "cognitive" OR "neuropsychological" OR "motor" OR "sensory perception" OR "visual processing" OR "attention" OR "language" OR "memory" OR "learning" OR "executive function*") AND ("biomarker" OR "marker") AND ("EEG" OR "electrophysiolog*"). See supplementary material (supplement 1) for complete search strings. There was no restriction on publication period, up to the date of the last search in November 2023. In addition, relevant reviews and articles, and the reference lists of included studies were manually searched for additional eligible studies.

Duplicate records were removed manually using the Rayyan open-source review management software (Ouzzani et al., 2016). Two reviewers (AVT and PC) independently assessed all retrieved records for inclusion in Rayyan. Initially, reviewers screened article titles and abstracts, and potentially eligible records were retrieved in full. In the second stage, full texts of all potentially eligible articles were examined. Any disagreement was solved through discussion. In case of ongoing discrepancies, the senior authors (RS and PR) were consulted. The final set of included studies was agreed upon within the complete study team.

2.3. Study eligibility

All study designs in human subjects with a diagnosis of BD (cross-sectional, case-control, cohort, randomized controlled trials (RCTs), quasi-experimental) were included in the review. Any in vitro studies, animal studies, study protocols, conference abstracts, (systematic) reviews, posters, or unpublished studies were excluded. However, efforts were made to identify potential full papers when protocols, abstracts, or posters seemed to align with the focus of this review, and authors were contacted twice to inquire for full published articles. Only studies available in English or German were included.

2.4. Participant criteria

We included data from patients with a validated diagnosis of BD (BD-I, BD-II, BD-NOS), regardless of mood state. Their mean age needed to be at least 18 years. Outcomes needed to be reported separately for the BD group in case of mixed populations.

2.5. Outcomes

The main objective of this review was to examine associations between the performance on any cognitive task assessing any cognitive domain and any parameter measured by an EEG. Thus, each study needed to report on the following two outcomes, and their correlation:

1. The first main outcome was cognitive performance, measured on any neurocognitive test or an extensive neurocognitive assessment, as a continuous measure. This could be a score on a single scale measuring any cognitive domain, a test score, or an average score from a comprehensive assessment. Studies that used diagnoses of cognitive impairment or dementia were excluded, as well as studies employing only subjective assessments of cognition (i.e., patient- or clinician-report). Any outcomes assessed with tasks on "hot"/emotional cognition (e.g., gaze-discrimination tasks or affective Go/No-Go tasks) were excluded as well.
2. The second was any continuous parameter (e.g., density, frequency) derived from a wake recording by scalp EEG. Other neuroimaging techniques (e.g., functional magnetic resonance imaging (fMRI), positron emission tomography (PET), magnetoencephalography (MEG)) were excluded.

2.6. Data extraction

One author (AVT) completed the data extraction, while all extracted data was checked independently by another author (PC). Inconsistencies were solved through discussion. The following information was collected using a standardized form: article information (including study and authors' name, and location), methodological information (including study design and participant information) and outcome information (including cognitive task and EEG paradigm employed, and correlation). When studies included different patient groups, only the data for the BD group was extracted. In case the same data has been published multiple times, the most comprehensive source was used and additional information from other publications added. If there was a sample overlap between multiple studies, we extracted the data from both and denoted the overlap.

2.7. Risk of bias assessment

The AXIS (Downes et al., 2016) tool was used for the risk of bias (RoB) assessment. AXIS is a critical appraisal tool designed to assess the quality of cross-sectional studies. It contains a series of questions concerning the content of the introduction, methods, results, and discussion section, as well as on conflicts of interest and ethical approval. When studies employed a longitudinal design, the longitudinal component was disregarded since this review only focuses on cross-sectional associations. All ratings were performed by two researchers independently (AVT and PC) and all discrepancies were solved through discussion.

2.8. Strategy for data synthesis

A narrative synthesis approach was used to present the identified correlations between cognitive tasks and EEG measures in BD participants, following the Synthesis without Meta-analysis (SWiM; (Campbell et al., 2020)) guidelines. Firstly, we grouped the identified correlations by sorting the cognitive tasks into five overarching cognitive domains: 1) attention and processing speed, containing all tasks assessing attention, psychomotor speed, processing speed, and information processing, 2) working memory, including all tasks measuring working memory, 3) episodic memory, comprising all tasks on verbal and visual memory, 4) executive function, including all tasks assessing executive function, verbal fluency, problem solving, and response inhibition, and 5) intellectual capacity, encompassing premorbid IQ, and overall cognitive performance. For each task, we used the cognitive domain that was reported as being the domain of interest in the original paper. If no cognitive domain was specified, we chose the domain most in line with the cognitive test used based on discussion between authors.

We then grouped the correlations based on the EEG measure by 1) ERP measures, 2) spectral measures, 3) connectivity measures, and 4) other measures (see Table 1 for an overview of all included EEG measures that were employed in the studies).

2.9. Changes to procedures since protocol publication

The protocol stated planned use of the Newcastle-Ottawa Scale (Wells et al., 2000) to assess risk of bias. However, the AXIS tool (Downes et al., 2016) was subsequently deemed to be more appropriate as the Newcastle-Ottawa scale focuses mainly on the selection and comparability of the cases and controls or cohorts, whereas the AXIS is

Table 1
Overview and definitions of EEG Measures.

	EEG measure	Definition & associated neural process
ERP measures	P50 amplitude and latency	• Calculated as average electric response around 50 ms post-stimulus onset • Positive polarity • Associated with sensory gating, i.e. pre-attentional filtering of sensory information
	N1 amplitude and latency	• Calculated as average electric response around 80–120 ms post-stimulus onset • Negative Polarity • Associated with early sensory processing of auditory, visual, or somatosensory stimuli, thought to reflect the detection and initial processing of stimulus features
	Mismatch negativity (MMN) amplitude and latency	• Generated when a divergent (e.g., in frequency or duration) stimulus occurs during a stream of standard stimuli • Defined as the difference between the ERP waveform of the standard and deviant stimuli • Negative polarity • Thought to represent an automated, pre-attentive, cerebral process for change detection that indicates auditory, sensory, memory, and context-dependent information processing
	P2 amplitude and latency	• Typically peaks around 100–250 ms • Positive polarity • Associations largely unknown
	N2 amplitude, difference amplitude and latency	• Typically peaks around 180–325 ms • Negative polarity • Typically associated with identification and distinction of deviant stimuli • Difference amplitude: peak nontarget amplitude subtracted from peak target amplitude (in tasks where targets and nontargets are presented)
	P3 amplitude and latency	• Generated by infrequent but task-relevant stimuli • Typically peaks around 300 ms • Positive polarity • Associated with cognitive processes involved in stimulus categorization and evaluation as an indicator of selective attention and memory updating • May be divided into subcomponents (P3a and P3b, see below)
	P3a amplitude and latency	• Usually elicited by distractor stimuli • Represents an orienting response
	P3b amplitude and latency	• Reflects task-relevant attentional mechanisms to the target stimulus
	Span endogenous negativity (SEN) amplitude	• Typically peaks around 160–360 ms • Negative component • Associated with visual search
	Error-related negativity (ERN)	• Typically peaks around 50–150 ms following an error • Negative polarity
	Error-related positivity (Pe)	• Thought to be related to motivational significance of error and post-error adjustments in behavioural performance • Typically peaks around 150–400 ms following an error • Positive polarity • Thought to be associated with error-awareness and post-error adjustments in behavioural performance
	Conflict-related negativity (N450)	• Typically peaks around 400–500 ms following the onset of a deviant stimulus • Negative polarity • Follows onset of conflict stimulus
	Stimulus-locked lateralized readiness potential (S-LRP)	• Captures motor cortex activity thought to be related to stimulus response preparation/ activation
Spectral measures	(Oscillatory) delta, alpha, and beta frequency band activity/ response	• Relative (%) frequency band power compared to baseline in delta (around 0.1–4.0 Hz), alpha (around 7–14 Hz), and beta (around 15–30 Hz) band in response to a stimulus or in resting states
	Delta, theta, alpha, and beta frequency band power	• Power/frequency (V^2/Hz) spectrum of EEG in delta, theta (around 4–7 Hz), alpha and beta frequency bands
Connectivity measures	Global and theta band connectivity strength	• Depicts a quantitative index of synchrony between neural assemblies in the global (1–70 Hz) and theta band, and thus estimates its functional connectivity
	Gamma frequency band coherence	• Depicts the synchronicity or coupling between signals in the gamma (around 30–100 Hz) frequency band and thus its functional connectivity
	Event-related and sensory-evoked gamma coherence	• Event-related gamma coherence depicts functional connectivity in the gamma frequency band after application of pure sensory signals • Sensory-evoked gamma coherence depicts functional connectivity in the gamma frequency band after stimulation by a sensory signal loaded with a cognitive task
Other	Spectral Entropy modulation	• Quantifies global regularity of EEG signal: high spectral entropy indicates more uniform distribution of spectral content (a highly random signal), low spectral entropy indicates a spectrum whose power is condensed to a narrower frequency range (a more regular signal) • Spectral entropy values can be assessed immediately before or after a target stimulus in a task

more fitting for observational studies without comparison group. Secondly, we decided to include studies that measured IQ, general intelligence, or general intellectual ability, as long as they used a continuous scale and reported a correlation with an EEG measure, since those could be counted towards the cognitive domain of intellectual capacity.

3. Results

3.1. Selection and inclusion of studies

The PRISMA-flowchart (Fig. 1) summarizes the study identification and inclusion process. The systematic search generated 2008 records (1256 after duplicates were removed). Title and abstract screening resulted in 162 potentially eligible articles, which were reviewed in full. Overall, 16 articles reporting on 15 studies were included in the review.

3.2. Characteristics of included studies

The most relevant study characteristics of all included studies are presented in Table 2. Across the 15 studies, 364 participants with BD were included, with a mean BD sample size of 26 (*SD*=11, range 10–60). The majority of studies were conducted within Europe (n = 9; 60 %), followed by trials from the USA (n = 5; 33 %). Studies conducted in Istanbul were counted towards European studies. Participants were mostly diagnosed with BD-I (n = 7; 47 %) and in a euthymic mood state (n = 5; 33 %). It should be noted that there is potential sample overlap between two studies (Atagün et al., 2014; Özerdem et al., 2011), so exact numbers might differ slightly. Additional study characteristics (control groups, total sample size, diagnostic tool used, EEG task employed) can be found in the supplementary material (supplement 2).

3.3. Risk of bias assessment

Table 3 depicts the risk of bias assessment according to AXIS (Downes et al., 2016). Risk of bias assessments for each individual study can be found in the supplementary material (supplement 3). Overall, most studies provided a clear aim, used appropriate methodology, described consistent results, a justifiable discussion, and included a statement about conflict of interest and ethical approval. However, no study gave a justification of their sample size. AXIS does not provide a numerical scale for assessing the quality of each study. Still, as most studies reported on most required domains, we rated the included studies as having low to moderate risk of bias overall.

3.4. Characteristics of participants

Table 4 summarizes the most important participant characteristics. Participants mean age was 35 years (*SD*=9), mean age of onset at 25 years (*SD*=3.8) and had a mean BD illness duration of 11 years (*SD*=2.9). Most studies (n = 7; 47 %) did not exclude participants with a psychiatric comorbidity, or only restricted certain comorbidities, such as (DSM IV) axis I comorbidities, such as alcohol, or substance abuse (n = 5; 33 %). Moreover, most studies (n = 11; 73 %) included participants who were receiving medication. Additional participant characteristics (ethnicity, education level) can be found in the supplementary material (supplement 2).

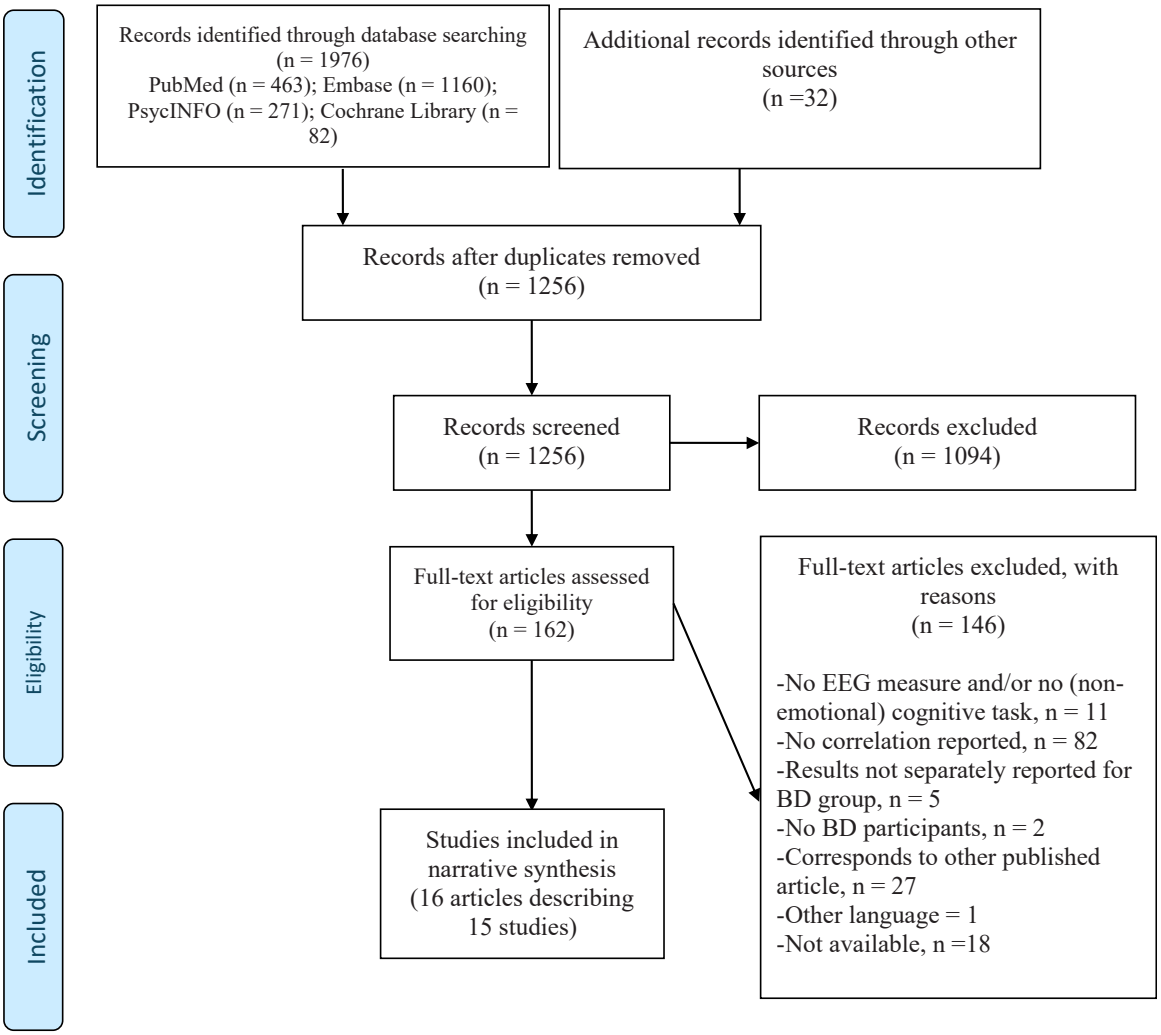

Fig. 1. PRISMA Flowchart of Study Inclusion.

Table 2
Study Characteristics.

Study	Continent	BD Sample Size	Setting	BD-Diagnosis	Mood state
Andersson et al., (2008)	Europe	25	Outpatients	BD-II	All
Atagün et al., (2014)[1]	Europe	22	Outpatients	BD-I/ BD-II	Euthymic
Cea-Canas et al., (2020)[2]	Europe	29	Outpatients	NR	NR
El-Badri et al., (2001)	Europe	29	Outpatients	BD-I	Euthymic
Howells et al., (2018)	Africa	28	Outpatients	BD-I with history of psychosis	NR
Klein et al., (2020)	North-America	26	Outpatients	NR	NR
Kaymak Koca et al., (2022)	Europe	60	In- and Outpatients	NR	All
Li et al., (2021)	Asia	24	Outpatients	BD-I with or without psychotic features	All
Minzenberg et al., (2014)	North-America	26	Outpatients	BD-I with recent (<12months) psychosis onset	Euthymic / subsyndromal
Molina et al., (2020)[2]	Europe	29	Outpatients	BD-I	Euthymic
O'Donnell et al., (2004)	North-America	13	In- and outpatients	BD-I	Manic or mixed
Özerdem et al., (2008)/(2010)[3]	Europe	10	Outpatients	BD-I/ BD-II	Manic or hypomanic
Özerdem et al., (2011)[1]	Europe	20	Outpatients	BD-I/ BD-II	Euthymic
VanMeerten et al., (2016)	North-America	31	Outpatients	NR	NR
Van Voorhis et al., (2019)	North-America	21	Outpatients	BD-I	NR

Note: BD – Bipolar disorder; NR - not reported; [1]partial sample overlap; [2]complete BD sample overlap (but different assessments); [3]complete BD sample overlap and two papers of the same study.

Table 3
Assessment of the studies included in the systematic review according to the appraisal tool for cross-sectional studies (AXIS).

Questions	Yes	No	NR/ NA
1. Were the aims/objectives of the study clear?	16	0	0
2. Was the study design appropriate for the stated aim(s)?	16	0	0
3. Was the sample size justified?	0	16	0
4. Was the target/reference population clearly defined?	10	6	0
5. Was the sample frame taken from an appropriate population base so that it closely represented the target/ reference population under investigation?	5	5	6
6. Was the selection process likely to select subjects/ participants that were representative of the target/ reference population under investigation?	15	0	1
7. Were measures undertaken to address and categorise non-responders?	0	0	16
8. Were the risk factor and outcome variables measured appropriate to the aims of the study?	16	0	0
9. Were the risk factor and outcome variables measured correctly using instruments/measurements that had been trialled, piloted or published previously?	15	1	0
10. Is it clear what was used to determined statistical significance and/or precision estimates?	6	10	0
11. Were the methods sufficiently described to enable them to be repeated?	13	3	0
12. Were the basic data adequately described?	15	1	0
13. Does the response rate raise concerns about non-response bias?	0	0	16
14. If appropriate, was information about non-responders described?	0	0	16
15. Were the results internally consistent?	16	0	0
16. Were the results presented for all the analyses described in the methods?	16	0	0
17. Were the authors' discussions and conclusions justified by the results?	16	0	0
18. Were the limitations of the study discussed?	10	6	0
19. Were there any funding sources or conflicts of interest that may affect the authors' interpretation of the results?	0	11	5
20. Was ethical approval or consent of participants attained?	13	0	3

3.5. Main outcomes

An overview of the assessed cognitive domains, the cognitive measures employed, and the assessed EEG measures can be found in Table 5. Results are summarized in a heatmap (Fig. 2). Note that results in the heatmap were standardised with higher scores indicating better performance in the cognitive tasks. Results in the text are reported as they were originally in articles.

3.5.1. Attention and processing speed

3.5.1.1. ERP measures. Seven studies reported associations between attention and processing speed performance and ERP measures (Andersson et al., 2008; Kaymak Koca et al., 2022; Klein et al., 2020; Li et al., 2021; O'Donnell et al., 2004; Van Voorhis et al., 2019; VanMeerten et al., 2016). The majority of correlations were non-significant, still, nine different significant associations were identified across the seven studies.

For remitted, but not for depressed or manic patients, Kaymak Koca et al. (2022) found significant correlations between attention and processing speed and P50 latency across two electrodes ($r = 0.472$, $p = .036$; $r = 0.537$, $p = .015$), with higher scores indicating worse performance in the cognitive task. Moreover, Donnell et al. (2004) reported a positive correlation ($r = 0.69$, $p = 0.009$) between better attention and N1 amplitude. However, Andersson et al. (2008), Kaymak Koca et al. (2022), and Van Meerten et al. (2016) found no significant correlation. Kaymak Koca et al. (2022) found a strong significant correlation between attention and processing speed and N1 latency ($r = 0.579$, $p = .007$), where higher task scores meant worse performance, for remitted patients, but not for the depressed or manic patient group. Andersson et al. (2008) and O'Donnell et al. (2004) did not find a significant association here. Andersson et al. (2008) identified a significant positive correlation between psychomotor speed and MMN latency ($r = 0.46$, $p = 0.024$), with better performance being associated with longer latencies. Furthermore, Kaymak Koca et al.'s (2022) results showed a negative relationship between attention and processing speed, with higher task scores indicating worse performance, and N2 amplitude for depressed ($r = -0.514$, $p = .029$) and remitted patients ($r = -0.470$, $p = .036$). There was no significant correlation for the manic patient group or in the study by O'Donnell et al. (2004). They also found a negative relationship between attention and processing speed (higher scores indicating worse performance) and N2 latency ($r = -0.462$, $p = 0.05$) for depressed patients, but a positive relationship ($r = 0.481$, $p = .032$) for remitted patients. Again there was no significant correlation for the manic patient group (Kaymak Koca et al., 2022) or in the study by O'Donnell et al. (2004). Li et al. (2021) found that P3a amplitude was significantly positively correlated with the better attention task performance (partial correlation=0.33) and that the scores (B=3.05, SE=1.27, adjusted $p = 0.028$) were significant predictors of the P3a amplitude. Interestingly, there was no association when using another attention task, nor in the study by Kaymak Koca et al. (2022), O'Donnell et al. (2004) or Van Meerten et al. (2016). Klein et al. (2020) found that P3b amplitudes were positively associated with attention task scores ($r = 0.619$, FDR corrected $p < .01$), with higher task scores indicating worse performance. Andersson et al. (2008), however, found

Table 4
Participant characteristics.

Study	% Women	Age	BD Duration	BD Age of Onset	Psychiatric Comorbidities	Medication
Andersson et al., (2008)	80	36.3	NR	22	Allowed	Yes (48 %)
Atagün et al., (2014)[1]	72.7	30.8	10.1	21.9	Not allowed	No
Cea-Canas et al., (2020)[2]	41.4	46.4	15.2	NR	Allowed	Yes
Molina et al., (2020)[2]			15.7			
El-Badri et al., (2001)	65.5	30.7	9.3	20.9	Not allowed	Yes (100 %)
Howells et al., (2018)	42.9	30.5 (Median)	NR	NR	Allowed	Yes
Klein et al., (2020)	19	45.1	NR	NR	Allowed	Yes
Kaymak Koca et al., (2022)	NR	42	NR	28.8	No DSM-IV axis I comorbidities	Yes
Li et al., (2021)	54.2	22.2	NR	NR	Allowed	No
Minzenberg et al., (2014)	19	21.6	NR	NR	Allowed	Yes (86 %)
O'Donnell et al., (2004)	69.2	39.6	14	25.6	No current/lifetime substance use disorder	Yes (62 %)
Özerdem et al., (2008/2010)[3]	40	37.8	7.5	30.3	No DSM-IV axis I comorbidities	Only valproate
Özerdem et al., (2011)[1]	70	32.2	10.6	22.2	No DSM-IV axis I comorbidities	No
VanMeerten et al., (2016)	26.6	44.6	NR	NR	Allowed	NR
Van Voorhis et al., (2019)	23.9	45	NR	NR	No current alcohol or substance dependence	Yes

Note: BD – Bipolar disorder; NR - not reported; Age, BD duration and BD age of onset reported in mean years; Medication indicates whether or not patients were medicated and if yes, what percentage (if reported); [1]partial sample overlap; [2]complete BD sample overlap; [3]complete BD sample overlap and same study.

no significant association here. Lastly, Van Voorhis et al. (2019) found a significant positive correlation between stimulus-locked lateralized readiness potential (S-LRP) onset latencies and attention and processing speed scores ($r = .70$, $p = .001$), with higher task scores indicating worse performance.

In sum, nine significant associations between attention and processing speed with P50, N1, N2, P3a, P3b and S-LRP measures as well as between psychomotor speed and MMN latency were found, although most correlations were non-significant and inconsistent within or across studies.

3.5.1.2. Spectral, connectivity, and other measures. All identified correlations between spectral measures (Atagün et al., 2014; El-Badri et al., 2001; Howells et al., 2018; Özerdem et al., 2008), connectivity measures (Cea-Canas et al., 2020; Özerdem et al., 2011, 2010), and other measures (Molina et al., 2020) with attention and processing speed task scores were non-significant.

3.5.2. Working memory

3.5.2.1. ERP measures. Li et al. (2021) found that MMN amplitude was significantly negatively correlated with working memory (higher scores indicating better performance) (partial correlation =-0.54), with task scores (B=-0.08, SE=0.03, adjusted $p = 0.021$) explaining 15 % of the variance in MMN amplitude, but there was no significant correlation with P3a amplitude.

3.5.2.2. Connectivity and other measures. No significant correlations with working memory and connectivity measures (Cea-Canas et al., 2020; Özerdem et al., 2011) or spectral entropy modulation (Molina et al., 2020) were identified.

3.5.3. Episodic memory

3.5.3.1. ERP, spectral, connectivity, and other measures. There was no significant correlation between episodic memory task scores and ERP (Andersson et al., 2008), spectral (El-Badri et al., 2001), connectivity (Cea-Canas et al., 2020) or other EEG measures (Molina et al., 2020).

3.5.4. Executive functioning

3.5.4.1. ERP measures. Four different studies (Andersson et al., 2008; Kaymak Koca et al., 2022; Minzenberg et al., 2014; Van Voorhis et al., 2019) looked at executive functioning ERP measure correlates. Again, most identified correlations were non-significant. Five correlations were significant:

Kaymak Koca et al. (2022) found that better executive functioning was associated with lower N1 amplitudes across different electrodes (r = -0.611, $p = .006$; r = -0.783, $p < .001$, r = -0.671, $p = .002$) as well as shorter N1 latencies (r = -0.571, $p < .001$, r = -0.5, $p = .008$, and r = -0.492, $p = .008$) for depressed participants, but no significant relationship for manic or euthymic participants. Interestingly, using different executive functioning tasks, Andersson et al. (2008) did not identify a significant correlation here. Moreover, Kaymak Koca et al. (2022) found a moderate positive relationship between response inhibition (with higher scores indicating worse performance) and N1 latencies for remitted patients across two electrodes ($r = 0.453$, $p = .045$; $r = 0.464$, $p = .039$), but not for depressed or manic participants. Furthermore, the same authors identified a moderate positive relationship for N2 amplitudes and response inhibition (higher scores meaning worse performance) in the depressed patient group across two electrodes ($r = 0.468$, $p = 0.043$ and $r = 0.478$, $p = .045$), but not for the remitted or manic group. They also found a strong relationship between response inhibition, with higher scores indicating worse performance, and N2 latency ($r = 0.680$, $p = .001$) for depressed, but not for manic or remitted participants. Lastly, Van Voorhis et al. (2019) found a significant positive correlation between S-LRP onset latencies and response inhibition scores, with higher task scores indicating worse performance ($r = .53$, $p = .019$), although other response inhibition scores from different tests showed a non-significant relationship.

In summary, most correlations were non-significant and findings varied within and between studies. However, five significant associations were identified, including links between better executive functioning and lower N1 amplitudes and shorter N1 latencies, as well as positive correlations between response inhibition deficits and N1 latencies, N2 amplitudes, N2 latency, and S-LRP onset latencies.

3.5.4.2. Spectral, connectivity, and other measures. The identified correlations between spectral (El-Badri et al., 2001), connectivity (Cea-Canas et al., 2020), and other EEG measures (Molina et al., 2020) with scores within the executive functioning domain were all non-significant.

3.5.5. Intellectual capacity

3.5.5.1. ERP measures. Four studies (Andersson et al., 2008; Klein et al., 2020; Li et al., 2021; Minzenberg et al., 2014) examined correlations between intellectual capacity and ERP measures. Most identified associations were non-significant, however, Li et al. (2021) found that intellectual capacity (IQ test score) was significantly correlated with P3a amplitude (partial correlation=0.25) and the score (B=1.35, SE=0.59, adjusted $p = 0.047$) was a significant predictor of P3a amplitude.

Table 5
Summary of cognitive and EEG measures.

Study	Cognitive Measure(s) with Associated Subdomain(s)	EEG Correlates Assessed
Andersson et al., (2008)	1. PASAT *(attention, working memory)* 2. Interference part of SCWT *(executive function)* 3. FAS *(executive function)* 4. CVLT-II *(verbal memory)* 5. ROCFT *(visual memory)* 6. DSST from WAIS-R *(psychomotor speed)* 7. Colour-naming and word-reading part of SCWT *(psychomotor speed)* 8. Vocabulary and Matrices Subtest from WAIS *(premorbid IQ)*	1. MMN latency 2. MMN amplitude 3. N1 amplitude 4. N1 latency 5. P3a amplitude 6. P3a latency 7. P3b amplitude 8. P3b latency
Atagün et al., (2014)	1. Number of errors in auditory oddball task *(attention)*	1. Oscillatory delta response
Cea-Cañas et al., (2020)	1. Overall cognition from WAIS-III and BACS *(intellectual capacity)* 2. BACS *(verbal memory, working memory, motor speed, verbal fluency, processing speed, problem solving)*	1. Global band connectivity strength 2. Theta modulation connectivity strength
El-Badri et al., (2001)	1. DGS from WAIS-R *(verbal memory)* 2. SMTS and DMTS from CANTAB *(visual memory)* 3. FAS *(executive function)* 4. Tower of London from CANTAB *(executive function)* 5. TMT-B from WAIS-R *(psychomotor speed, information processing)* 6. DSST from WAIS-R *(psychomotor speed, information processing)*	1. Delta frequency band power 2. Theta frequency band power 3. Alpha frequency band power 4. Beta frequency band power
Howells et al., (2018)	1. Correct responses in visual continuous performance task *(attention)* 2. Overall response time in visual continuous performance task *(attention, processing speed)* 3. Errors of commission in visual continuous performance task *(attention)* 4. Errors of omission *(attention)*	1. Delta frequency band activity 2. Alpha frequency band activity
Klein et al., (2020)	1. Target detection/ perceptual sensitivity in DS-CPT *(attention)* 2. Rate of false alarms in DS-CPT *(attention)* 3. SimDiff: Subtracting errors for dissimilar nontargets from errors for similar nontargets *(attention)* 4. Vocabulary and block design subtests *(estimated IQ)*	1. N1 amplitude 2. N2 difference amplitude 3. P3b amplitude
Kaymak Koca et al., (2022)	1. FAB total *(executive function)* 2. Stroop interference *(response inhibition)* 3. Stroop total time *(attention, processing speed)*	1. P50 amplitude 2. P50 latency 3. N1 amplitude 4. N1 latency 5. P2 amplitude 6. P2 latency 7. P3 amplitude 8. P3 latency 9. N2 amplitude 10. N2 latency
Li et al., (2021)	1. DMTS from CANTAB *(working memory)* 2. RVP from CANTAB *(sustained attention)* 3. WAIS *(IQ)* 4. CCFT *(IQ)*	1. MMN amplitude 2. P3a amplitude
Minzenberg et al., (2014)	1. PES in Stroop task *(executive function)* 2. PEA in Stroop task *(executive function)* 3. PCS in Stroop task *(executive function)* 4. PCA in Stroop task *(executive function)* 5. WAIS *(IQ)*	1. ERN 2. Pe 3. N450
Molina et al., (2020)	1. BACS *(verbal memory, working memory, motor speed, verbal fluency, processing speed, problem solving)*	1. Spectral entropy modulation
O'Donnell et al. (2004)	1. Accuracy in auditory discrimination task *(attention)*	1. N1 amplitude 2. N1 latency 3. P2 amplitude 4. P2 latency 5. N2 amplitude 6. N2 latency 7. P3 amplitude 8. P3 latency
Özerdem et al., (2008)	1. Number of errors in visual oddball task *(attention)*	1. Alpha response 2. Beta response
Özerdem et al., (2010)	1. Number of errors in visual oddball task *(attention)*	1. Gamma frequency band coherence
Özerdem et al., (2011)	1. Number or errors in visual oddball task *(attention, working memory)*	1. Event-related gamma coherence 2. Sensory evoked gamma coherence
VanMeerten et al., (2016)	1. Accuracy in SOA task *(attention)* 2. Reaction time on correct trials in SOA task *(attention, processing speed)*	1. N1 amplitude 2. P3 amplitude 3. SEN amplitude
Van Voorhis et al., (2019)	1. Go only median reaction time on stop-signal task *(attention, processing speed)* 2. Go-Stop median reaction time on stop-signal task *(response inhibition)* 3. Stop-signal reaction time *(response inhibition)* 4. Adjusted P(r): probability of responding in the presence of a stop signal adjusted for rate of response omissions in the high stop probability condition *(response inhibition)*	1. P300 amplitude (Go only and Go/Stop) 2. S-LRP onset latency (Go-only and Go/Stop)

Note: BACS – Brief Assessment in Cognition in Schizophrenia Scale; CANTAB – Cambridge Neuropsychological Test Automated Battery; CCFT – Cattell Culture Fair Intelligence Test; CPT – Continuous Performance Task; CVLT – California Verbal Learning Test; DGS – Digit Span; DMTS – Delayed Matching to Sample; DS-CPT – Degraded Stimulus Continuous Performance Task; DSST – Digital Symbol Substitution Test; ERN – error-related negativity; FAB – Frontal Assessment Battery; FAS – Phonemic Verbal Fluency Test; NS – not significant; PASAT – Paced Auditory Serial Addition Test; PCS – post-conflict speeding; Pe – error-related positivity; PEA – post-error increase in accuracy; PES – post-error slowing; ROCFT – Rey-Osterreith Complex Figure Test; RVP – Rapid Visual Information Processing; SCWT – Stroop Colour Word Test; SEN – span endogenous negativity; S-LRP – Stimulus-locked lateralized readiness potential; SMTS – Simultaneous Matching to Sample; SOA – Span of Apprehension; TMT – Trail Making Test; WAIS – Wechsler Adult Intelligence Scale; WAIS-R – Wechsler Adult Intelligence Scale- Revised.

Interestingly, for another IQ test score no significant association was identified and Andersson et al. (2008) also did not find a significant correlation between P3a amplitude and intellectual capacity (premorbid IQ).

3.5.5.2. Connectivity measures. Cea-Canas et al. (2020) were unable to find a significant relationship between any connectivity measure score and intellectual capacity.

4. Discussion

Because EEG captures brain activity with high temporal resolution, is a direct measure of neural activity and therefore a good reflection of

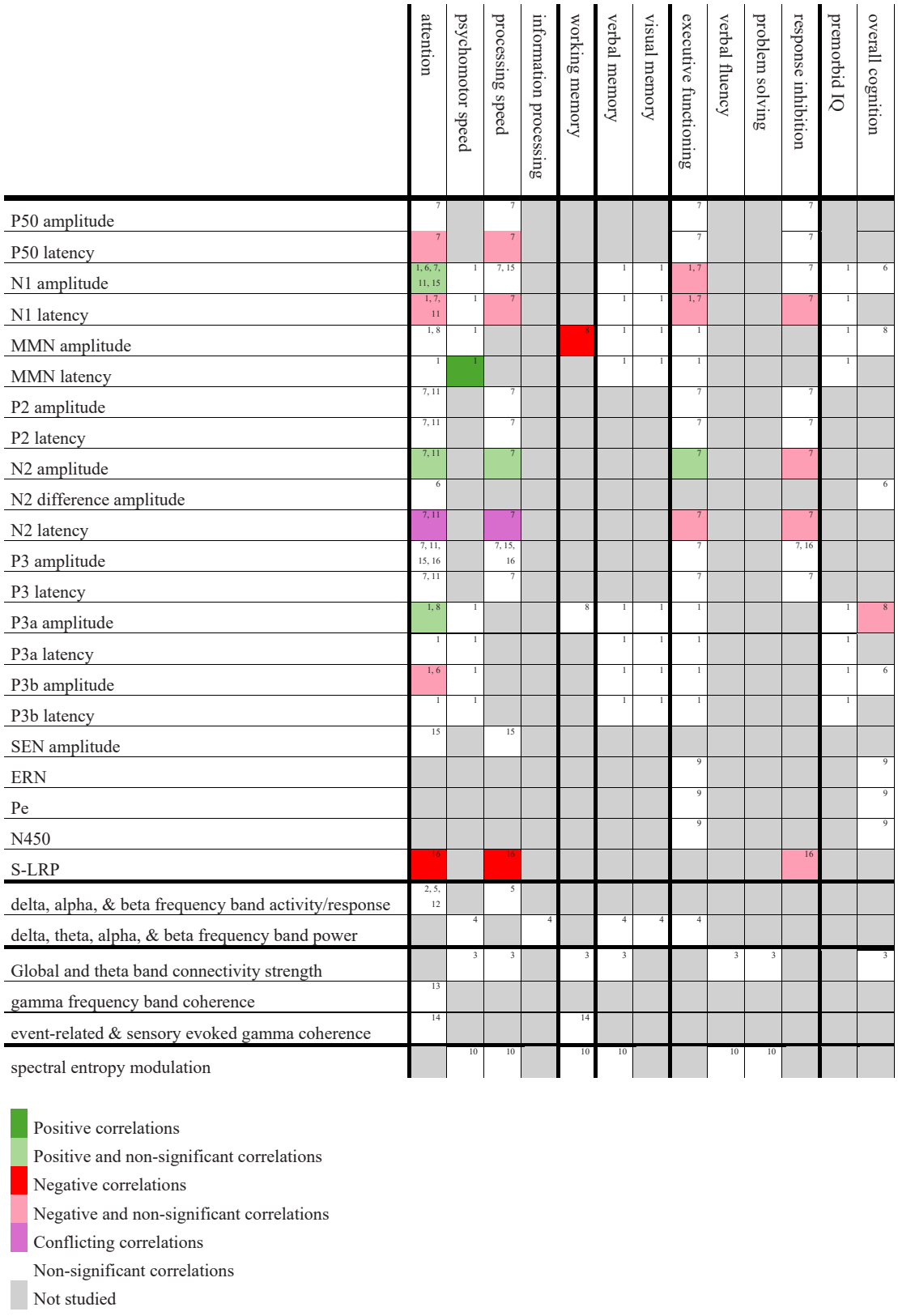

Fig. 2. Results Summary. *Note:* ERN – error-related negativity; MMN – mismatch negativity; Pe – error-related positivity; SEN – span endogenous negativity; S-LRP – stimulus-locked lateralized readiness potential; numbers refer to citations: 1 – Andersson et al. (2008); 2 – Atagün et al. (2014); 3 – Cea-Cañas et al. (2020); 4 – El-Badri et al. (2001); 5 – Howells et al. (2018); 6 – Klein et al. (2020); 7 – Kaymak Koca et al. (2022); 8 – Li et al. (2021); 9 – Minzenberg et al. (2014); 10 – Molina et al. (2020); 11 – O'Donnell et al. (2004); 12 – Özerdem et al. (2008); 13 – Özerdem et al. (2010); 14 – Özerdem et al. (2011); 15 – VanMeerten et al. (2016); 16 – Van Voorhis et al. (2019).

cognitive processing, and both EEG abnormalities as well as cognitive deficits are common in patients with BD, examining correlations between EEG and cognitive measures can be a fruitful way to better understand the biological mechanisms behind cognitive dysfunction in BD. In this systematic review, we identified 16 articles presenting 15 studies examining EEG - markers of cognitive performance in BD. To our knowledge, this is the first systematic review examining the association between EEG measures and cognitive tasks assessing multiple cognitive domains providing the first overview of established EEG-cognition correlates in BD. Most reported correlations were not significant. However, 16 significant associations within six studies were identified (Andersson et al., 2008; Kaymak Koca et al., 2022; Klein et al., 2020; Li et al., 2021; O'Donnell et al., 2004; Van Voorhis et al., 2019), mostly in the attention and processing speed and the executive functioning domain. There was only one significant correlation in the working memory and the intellectual capacity domain, respectively. No significant correlations were found for episodic memory tasks.

Significant correlations within the attention and processing speed domain were with P50 latency, N1 amplitude, N1 latency, MMN latency, N2 amplitude, N2 latency, P3a amplitude, P3b amplitude, and S-LRP onset latency. However, only three of these correlations had no contradictory results (i.e., a result in the opposite direction or a non-significant result) within one (e.g., across cognitive tasks or mood states) or between several studies, though none of these three had been assessed in multiple studies. These were the correlations of MMN latency with psychomotor speed score (Andersson et al., 2008) and S-LRP onset latency with attention and processing speed scores (Van Voorhis et al., 2019).

Longer MMN latency was associated with better performance in two psychomotor speed tasks, namely the Digital Symbol Substitution Test and the colour-naming and word-reading parts of the Stroop Colour Word Test (Andersson et al., 2008). The MMN reflects an information processing stage prior to voluntary control of attention (Fitzgerald and Todd, 2020; see Table 1), so it is possible that too rapid stimulus processing could result into errors. Notably, previous research in schizophrenia has been inconclusive and found prolonged MMN latency to be associated with both poorer (Toyomaki et al., 2008) and enhanced cognitive performance (Kargel et al., 2014). MMN indices have been associated with clinical and functional outcomes. For example, Kim et al. (2020) found significantly reduced MMN amplitudes in patients with BD compared to healthy controls which were associated with impaired social functioning. The exact neural underpinnings of the MMN are under debate and research often relies on animal models (Ross and Hamm, 2020). EEG-fMRI coupling data suggests two propagations with the MMN being generated in the auditory cortex and then advancing to the motor areas and the inferior frontal gyrus, respectively (Li et al., 2019). Indeed, there is evidence for altered connectivity patterns between these regions during auditory oddball tasks in children at high risk for schizophrenia and BD (Larsen et al., 2024). As such, MMN abnormalities could be an indication for connectivity impairments in BD and medication enhancing connectivity, such as 5-HT_4 receptor agonists (de Cates et al., 2023), might be suitable candidates to improve cognition and social functioning. Moreover, research suggests that MMN abnormalities might be associated with a disturbed frontal glutamate system and increased levels of combined glutamate and glutamine, which highlights the potential of glutamatergic agents in the treatment of BD (Chitty et al., 2013). However, overall research on MMN abnormalities, its correlates and neural underpinnings has been inconclusive and further studies in BD samples need to be conducted to draw firmer conclusions.

Moreover, a higher S-LRP onset latency was associated with a longer reaction time in a stop-signal task (Van Voorhis et al., 2019) in both BD and schizophrenia patients, which has been shown in both healthy controls as well as participants with schizophrenia before (Kappenman et al., 2016; Luck et al., 2009). Interestingly, when controlling for antipsychotic medication, this relationship was no longer significant in the BD group, suggesting a potential impact of medication on response inhibition. They found higher S-LRP latencies in both BD and schizophrenia patients and identified a positive correlation between S-LRP latencies and IQ in the BD group, meaning a higher IQ was associated with more time required for response selection, while correlations in the opposite direction were found for the schizophrenia and control group. This highlights that shared electrophysiological abnormalities can still manifest in different functional outcomes between diagnostic groups. The S-LRP captures motor cortex activity related to stimulus response preparation and activation (see Table 1), so the authors suggest that slower activation (i.e., longer S-LRP onset latency) may result in slower response. However, research on exact neural underpinnings of LRPs as well as their functional and clinical links remains sparse and, again, further BD-specific evidence is needed.

For working memory, higher MMN amplitudes were associated with worse performance on the Delayed Matching to Sample task (Li et al., 2021). As healthy controls exhibited lower amplitudes than the BD group (and generally show better cognitive performance), this result was in line with the authors expectations and previous research (e.g., Kim et al., 2020, see above) and could represent dysfunctional attention processing, as outlined above. Again, further studies are needed to replicate these findings.

For executive functioning, significant correlations were detected with N1 amplitude, N1 latency, N2 amplitude, N2 latency, and S-LRP onset latency, although contradictory or non-significant results were also found for all of them. Similarly, for intellectual capacity, there was a significant correlation with P3 amplitude, but results within this study were not consistent.

This review provides evidence that the assessment of correlations should be conceptually meaningful. A lot of the examined correlations were exploratory and hypotheses-driven analyses might yield more fruitful results. If we had identified more consistent correlations, they could have given a first indication which neurological processes could be disrupted, if the neural correlates of the respective EEG measures were known, and which potential correlations warrant more research. Most identified correlations were non-consistent within and across studies and we therefore refrained from further discussing their potential underlying mechanisms. Our review provides preliminary evidence that especially MMN measures and their underlying mechanisms could be a fruitful component for further investigations and treatment targets. However, it needs to be pointed out that, while there were no contradictory results, only single studies found a significant correlation with MMN latency and amplitude, respectively. As yet, neural correlates of EEG measures are currently mostly unknown and further research into the underlying mechanisms and electrophysiological markers of cognition in BD is dearly needed.

In line with the review by Perez-Ramos et al. (2024) on cognitive biomarkers and mood states in BD, our review also highlights the heterogeneity of instruments and participants within and between studies. Many of the identified correlations were not consistent across mood states or cognitive tasks. In most cases, when a positive or negative correlation was identified, the same correlation was not significant or even was in the opposite direction when measured within a different patient subgroup or cognitive task. This may be due to the large number of cognitive and EEG measures employed, the wide range and poor standardisation of EEG acquisition and analysis methods between sites, as well as the heterogeneity in participant characteristics, especially in mood states. Notably, Kaymak Koca et al. (2022) identified most of the significant associations (8 out of 16), and this was the only trial distinguishing between depressed, manic and remitted participants in their analyses. Inconsistent and conflicting findings across mood states underline the potential impact of mood on neurophysiology and cognitive performance and therefore highlight that mood symptoms should be accounted for. For example, they found shorter N2 latency being associated with poorer attention and processing speed performance in depressed patients, but the opposite in remitted, and no correlation for

manic patients. A study by Fridberg et al. (2009) has not found an influence of mood state on N2, while the influence of mood state on cognitive performance has been established (Martínez-Arán et al., 2004), thus further research into the interplay of mood, neurophysiology and cognition could yield important insights into their driving factors. Likewise, variability in cognitive measures needs to be considered. For example, Li et al. (2021) found a significant positive association between P3a amplitude and overall cognition using Wechsler Adult Intelligence Scale, whereas no association was found when overall cognition was measured with the Cattell Culture Fair Intelligence Test. This calls for standardization and harmonization of EEG and cognitive measures across studies. The International Society for Bipolar Disorder (ISBD) developed a battery of cognitive tests for the use in BD research (Yatham et al., 2010) that compiles instruments best suitable to assess cognitive impairments in BD clinical trials. One could envision a similar endeavour for EEG measures that can be used alongside cognitive tests. Such an EEG battery could greatly benefit the harmonization and interpretability of future neurophysiological cognitive research.

The present review has several strengths. To our knowledge, this was the first systematic review on the association of EEG and cognitive measures in BD. We have aimed to map out the landscape in this area and identified large gaps in the knowledge base that have not yet been addressed by any study (the grey boxes in Fig. 2). We further identified a few associations that may provide a crystalising point for future research (especially the dark red and green boxes in Fig. 2). Moreover, we employed broad inclusion criteria, only excluding "hot" cognitive outcomes and neurophysiological measures other than EEG (e.g., MEG) to ensure comparability, so we were able to include as many studies as possible. This way, we could examine a wide range of cognitive domains and assessments as well as EEG measures. Moreover, substantial efforts were made to include all available papers, contacting authors of promising abstracts or posters multiple times and obtaining additional articles through reference screenings.

However, several limitations need to be considered. Firstly, we did not include a control group in our review. Therefore, we do not know whether our findings are specific to BD, given the absence of a comparison group. However, not all studies included a control group, thus we decided against the consideration of control groups for consistency across included studies. Secondly, heterogeneity in methods and participant characteristics between studies was considerable and we were unable to account for it due to the low number of total studies. For example, participants differed in medication status, diagnoses (BD-I, BD-II, some with psychosis), and mood state; different electrode locations were used for the EEG measures, which can affect effect sizes (Arikan et al., 2024), and cognitive domains were assessed with numerous different instruments. Thus, an exact association (i.e., a certain EEG measure with a specific cognitive domain assessed using a particular cognitive test) was never examined across studies, which might have contributed to our findings' inconsistency. Thirdly, we synthesised the results and assigned all employed cognitive tasks to a cognitive domain and all EEG measures to an EEG measure category to the best of our knowledge, though other assignments or categories may certainly have been possible and could have resulted in different conclusions. Lastly, due to increased heterogeneity, we were unable to calculate effect sizes and perform a meta-analysis which should be a future endeavour to better understand the relationship between neurophysiology and cognition in BD.

Future studies should therefore adopt as homogeneous a methodology as possible or control for any possible covariates. EEG and cognitive measures should be chosen based on guidelines whenever available and be used consistently across trials. Additionally, studies should ideally be conducted in homogeneous samples of either remitted or symptomatic (depressed, manic, hypomanic, mixed) groups, or at least account for that in the analyses, to allow insights into the cognition and neurophysiology changes across mood stages. Medication status should be assessed and accounted for whenever possible. Moreover, longitudinal studies might herby be helpful to further understand cognitive trajectories. Once more trials on the association between cognitive performance and EEG measures in BD are available, future reviews should include control groups and a specification of mood states, in order to examine the specific features of these associations in BD compared to healthy controls or other disorders, and of mood status. Furthermore, future research needs to pursue neurophysiological markers for cognition in BD other than traditional EEG measures. For example, sleep EEG assessments are a promising area of research, as specific sleep parameters, e.g., sleep spindles, are associated with cognitive performance in schizophrenia (Au and Harvey, 2020), and preliminary evidence suggests similar patterns in BD (Ritter et al., 2018). Finally, it should be examined how different EEG markers could be utilised to inform treatment strategies. It has previously been suggested to integrate ERPs into clinical practice, where "ERP-oriented cognitive remediation" could allow for targeted cognitive training specifically in those cognitive domains that ERP screenings suggest impairments (Campanella, 2013) and sleep EEG markers could inform the delivery of cognitive interventions in a similar way.

In conclusion, due to the lack of consistent correlations, no robust markers of cognitive performance in BD were identified. Such markers would be helpful to better understand the underlying mechanisms of cognitive performance and potentially to inform future interventions. Future neurophysiology and cognition studies that follow a coherent methodological approach may be able to achieve these goals.

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Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Rebecca Strawbridge reports a relationship with Janssen Pharmaceuticals Inc that includes: speaking and lecture fees. Allan H Young reports a relationship with Flow Neuroscience that includes: consulting or advisory and speaking and lecture fees. Allan H Young reports a relationship with Novartis that includes: consulting or advisory and speaking and lecture fees. Allan H Young reports a relationship with Roche that includes: consulting or advisory and speaking and lecture fees. Allan H Young reports a relationship with Janssen Pharmaceuticals Inc that includes: consulting or advisory, funding grants, and speaking and lecture fees. Allan H Young reports a relationship with Takeda that includes: consulting or advisory and speaking and lecture fees. Allan H Young reports a relationship with Noema pharma that includes: consulting or advisory and speaking and lecture fees. Allan H Young reports a relationship with Compass that includes: consulting or advisory, funding grants, and speaking and lecture fees. Allan H Young reports a relationship with AstraZeneca that includes: consulting or advisory and speaking and lecture fees. Allan H Young reports a relationship with Boehringer Ingelheim that includes: consulting or advisory and speaking and lecture fees. Allan H Young reports a relationship with Eli Lilly that includes: consulting or advisory and speaking and lecture fees. Allan H Young reports a relationship with LivaNova that includes: consulting or advisory, funding grants, and speaking and lecture fees. Allan H Young reports a relationship with Lundbeck that includes: consulting or advisory and speaking and lecture fees. Allan H Young reports a relationship with Sunovion that includes: consulting or advisory and speaking and

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2025.106157.

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