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The event-related potential components across psychiatric disorders: a systematic review and network meta-analysis

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OBJECTIVE: This study aims to compare and rank event-related potentials (ERP) components among healthy individuals and patients with various psychiatric disorders, including schizophrenia (SCZ), bipolar disorder (BD), major depressive disorder (MDD), anxiety disorder (AD), and obsessive-compulsive disorder (OCD).

METHODS: A comprehensive search was conducted in PubMed, Embase, PsycINFO, and Web of Science up to March 20th, 2024. Original studies reporting ERP component data in patients with psychiatric disorders were included. Network meta-analyses were conducted to compare ERP components, including P300, mismatch negative (MMN), P50, N1, N2, P1, and P2.

RESULTS: From 47,391 citations, 687 studies were included, with sample sizes ranging from 893 to 16,879. Compared to healthy controls (HC), significant reduction in amplitudes and latency delay of P300, and reduced MMN amplitude were observed in SCZ, BD, and MDD. Additionally, SCZ displayed significant abnormalities in all P50 components. Reductions in N1, N2, and P2 amplitudes were noted in SCZ, with increased N2 latencies in SCZ and BD. The most significant difference across psychosis in EPR components was the MMN amplitude and ERN amplitude between OCD and SCZ. Finally, the composite ERP-score suggested SCZ rank the first in ERP abnormalities. Most primary outcomes were stable in sensitivity analyses.

CONCLUTION: The findings suggested that SCZ exhibited pronounced electrophysiological abnormalities, particularly in the P300 and P50 components. Moreover, the results provided a comprehensive overview of the varying electrophysiological profiles across different psychoses. These insights support the potential of ERPs as neurophysiological markers reflecting the neural dysfunctions associated with these disorders. Prospero registration: CRD42022361158

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INTRODUCTION

Psychiatric disorders, including schizophrenia (SCZ), bipolar disorder (BD), major depressive disorder (MDD), anxiety disorders (AD), obsessive-compulsive disorder (OCD) and so on, present significant challenges to individuals and place a substantial burden on global healthcare systems [1]. While these disorders differ in their diagnostic criteria, they often share overlapping clinical features, such as delusions, hallucinations, and cognitive impairments, which complicate accurate diagnosis and effective treatment [2]. Event-related potentials (ERP), non-invasive electrophysiological markers derived from electroencephalography (EEG), offer a promising method for investigating the neural and cognitive processes underlying these disorders [3]. ERPs measure brain activity in response to specific sensory, cognitive, or motor events, providing real-time insights with high temporal resolution [4, 5].

In psychiatric research, ERP abnormalities provide insights into the neural mechanisms underlying cognitive dysfunctions commonly observed in these disorders. For example, reduced P300 amplitudes are often linked to impairments in attention and working memory [6, 7], while abnormalities in mismatch negativity (MMN) reflect deficits in automatic auditory processing [8]. These components not only reveal the neurophysiological mechanisms of psychiatric disorders but also show potential for distinguishing different conditions based on disorder-specific ERP alterations. However, emerging evidence suggests that ERPs exhibit potential as biomarkers for differentiating psychiatric conditions based on distinct neurophysiological patterns. For example, reduced P300 amplitudes are commonly observed in SCZ, while alterations in N170 are more pronounced in MDD, reflecting differences in cognitive and emotional processing deficits [9, 10]. These patterns enhance our understanding of the neural underpinnings of psychiatric disorders, while ERPs simultaneously serve as valuable tools for investigating their neurophysiological basis and refining diagnostic classification.

Understanding the neurophysiological mechanisms underlying psychiatric disorders presents a significant challenge in neuroscience. Despite advances in ERP research, findings on the electrophysiological differences across psychiatry disorders remain inconsistent, often due to variations in study design and small

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sample sizes. This variability underscores the need for a comprehensive data synthesis to clarify how ERP components differ across disorders compared to healthy control (HC). A network meta-analysis (NMA) provides a distinct advantage in addressing these challenges by synthesizing findings from studies with varying designs and sample characteristics. Network meta-analysis improves statistical efficiency and result accuracy by integrating direct and indirect comparisons. It can rank intervention measures and identify evidence gaps, while evaluating consistency and inconsistency. It has broad applicability and provides strong support for decision-making. By employing this approach, we systematically compare and rank ERP components across various psychiatric disorders. This integration of findings aims to provide a definitive ranking of electrophysiological characteristics, offering insights that may guide future research and clinical strategies.

METHODS

Search strategy and selection criteria

We conducted a systematic review and network meta-analysis according to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA, https://www.prisma-statement.org/citing-prisma-2020) guidelines (Appendix 1). The protocol was registered in PROSPERO (https://www.crd.york.ac.uk/prospero/, CRD42022361158, Appendix 2). Four electronic bibliographic databases were searched: PubMed, Web of Science, Embase and PsycINFO. The search strategy utilized Medical Subject Headings (MeSH) and keywords appropriate to each database. The detailed search strategy was described in Appendix 3.

Selection criteria based on the PICOS framework included: Population (P): Adult patients diagnosed with SCZ, BD, MDD, OCD, and AD were included. Individuals under 18 years of age were excluded because of the ongoing developmental changes in brain structure and function that could confound the results, as their psychiatric conditions may still be evolving. Similarly, individuals over 65 years were excluded because age-related cognitive decline and multiple comorbidities may complicate the interpretation of event-related potential (ERP) findings. These factors can significantly impact both the amplitude and latency of ERP components. To reduce these confounding variables, studies involving adolescents (under 18 years) and older adults (over 65 years) were not included in this review. Interventions (I): Not applicable. Comparators (C): Healthy adults. Outcomes (O): The primary outcomes were the amplitude and latency of ERP components. Study design (S): Observational studies comparing patients with psychiatry disorders to HC or comparing two or more psychiatric disorders were included. Language was restricted to English. Studies published since inception to March 20th, 2024 were considered for inclusion, with searches conducted in two phases: from inception to Sep 20th 2020, and from Sep 21st 2020 to Mar 20th 2024. The two-phase search strategy is detailed in Appendix 3.

Two reviewers (ZL and JG) completed the screening and recording independently, and they could not interfere with each other's decisions. When there are different opinions between the two authors, the third author (WY) clarified and made a final decision. Study authors were contacted by email in case of missing or unclear information. Two reviewers independently extracted data and input the data into electronic forms using Microsoft Excel 2019, including information on study, disease, sample size, time-window, paradigm, stimulus type, and other relevant details.

Outcomes

The ERP components analyzed including P300, P3a, P3b, MMN, P50, N1, N2, P1, P2, N170, N400, CNV, ERN, and LPP. The amplitude and latency were extracted, so did to the P50 ratio and difference. In cases where multiple study arms involved the same disorder, we combined the means and standard deviations (SDs) following methods described in the Cochrane Handbook (https://training.cochrane.org/handbook/current).

Data analysis

All the statistical actions were conducted on the R software and Stata, *gemtc* and *rjags* package of R software and *network* package of Stata were applied. The random-effects model was selected to account for the anticipated heterogeneity across studies, considering differences in sample populations, ERP measurement protocols, and study designs. This model was used to estimate the standardized mean difference (SMD) with 95% confidence intervals (CIs), as it accommodates between-study variance and provides more generalizable estimates. To assess heterogeneity, we used the I^2 statistic, with values above 50% indicating substantial variation. Additionally, the node-split method was employed to evaluate inconsistencies between direct and indirect evidence.

The sensitivity analyses were performed to examine the sources of heterogeneity. We conducted the network meta-analyses again after excluding the studies with high or very high risk. Funnel plot was used to assess the publication bias. In an unbiased scenario, the plot should resemble a symmetrical inverted funnel shape, where smaller studies with less precision scatter widely at the bottom and larger studies with higher precision are concentrated near the top.

We applied the ROBINS-E tool (Risk of Bias in Non-randomized Studies - of Exposures) to assess the risk of bias [11]. Discrepancies were resolved through discussion, and if needed, a third author. The risk of bias was marked in both articles and survey results.

The ERP-score was developed to systematically rank ERP abnormalities across psychiatric disorders relative to HC, providing a quantitative measure of electrophysiological differences. We defined the HC were assigned a score of zero, and non-significant differences relative to HC also marked as zero. The score psychiatric disorders ranged from −5 to +5, based on the hierarchy observed in the network meta-analysis. For example, if the order of abnormality was SCZ > BD > MDD > HC > OCD > AD (with all comparations to HC were significant), the ERP-score was 3, 2, 1, 0, −1, and −2, respectively. While the score itself does not serve as a diagnostic tool, it can highlight ERP components that are most sensitive to differences across psychiatric conditions.

The detailed statistical methods were described in Appendix 4.

RESULTS

Search and study selection

The search identified 47,391 citations, including 30,044 unique records, and 4077 full-text articles were retrieved after the exclusion of 25,908 records on the basis of the titles and abstracts. We screened these 4077 full-text articles and included 687 studies (Fig. 1).

P300 component: amplitude and latency analysis

A total of 225 studies with 16,879 participants presented usable data on P300 amplitude (Studies per arm: AD, 20; BD, 28; HC, 224; MDD, 33; OCD, 16; SCZ, 156; model ratio = 0.999, $I^2 = 0.07\%$), the fitted model has great stability. SCZ, BD, and MDD were associated with a significant reduction in P300 amplitude compared to HC. Additionally, the P300 amplitude in SCZ was significantly lower than in OCD and AD. For P300 latency, 173 studies with 12,984 participants were analyzed (Studies per arm: AD, 16; BD, 20; HC, 172; MDD, 22; OCD, 14; SCZ, 121; model ratio = 1.069, $I^2 = 7\%$). SCZ, BD, and MDD showed a significant delay in P300 latency compared to HC. Moreover, the P300 latency in SCZ, BD and MDD was significantly longer than that in OCD, with SCZ exhibiting significantly longer latency than AD (Fig. 2A; Appendix 5, Tables S1–S2).

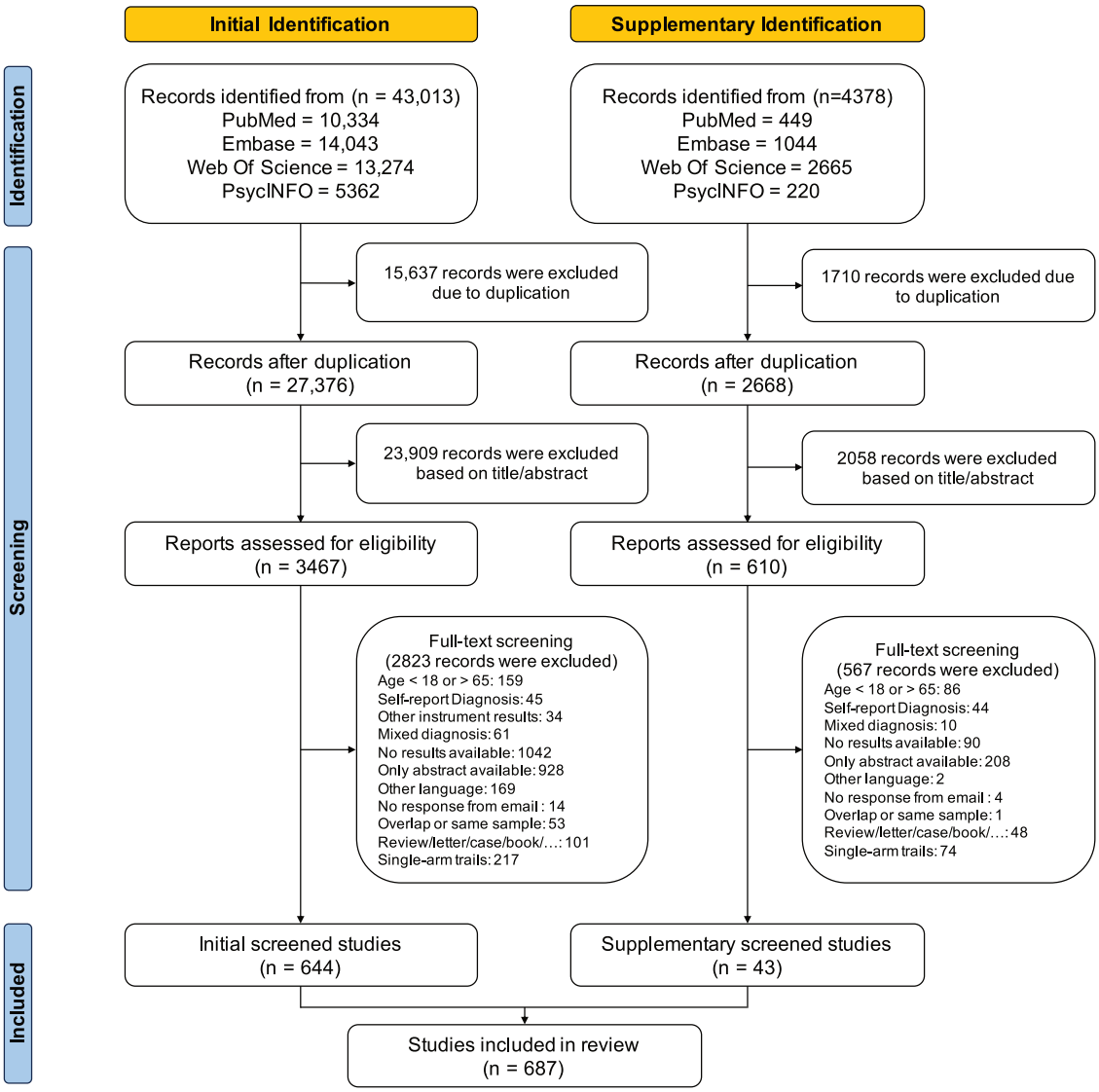


Fig. 1 PRISMA flow diagram. Searches conducted in two phases, initial searching was from inception to Sep 20th 2020, and supplementary searching was from Sep 21st 2020 to Mar 20th 2024.

P3a and P3b: cognitive and task-related variations

A total of 40 studies involving 5939 participants reported usable data for P3a amplitude (Studies per arm: BD, 7; HC, 40; MDD, 5; OCD, 1; SCZ, 31; model ratio = 1.224, $I^2 = 19\%$). SCZ, BD, and MDD showed a significant reduction in P3a amplitude compared to HC. Additionally, 22 studies with 1900 participants provided usable results for P3a latency (Studies per arm: BD, 4; HC, 22; MDD, 5; OCD, 1; SCZ, 14; model ratio = 0.945, $I^2 = 0\%$). Specifically, only the P3a latency in SCZ was significantly longer than in HC (Fig. 2B; Appendix 5, Tables S3–S4).

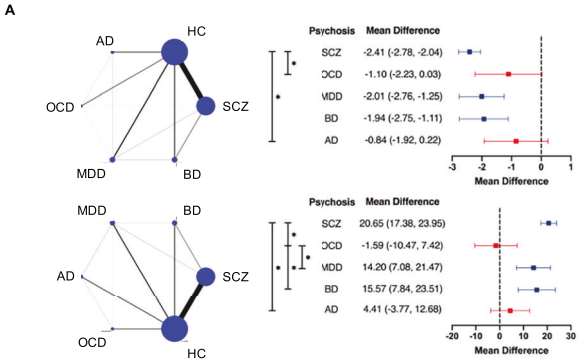
A total of 37 studies Involving 5000 participants provided usable data for P3b amplitude (Studies per arm: AD, 2; BD, 5; HC, 36; MDD, 8; OCD, 1; SCZ, 24; model ratio = 1.022, $I^2 = 3\%$). SCZ, BD, and MDD showed a significant reduction in P3b amplitude compared to HC and OCD, whereas OCD exhibited significantly higher P3b amplitude than HC. Furthermore, 22 studies with 1822 participants reported usable data for P3b latency (Studies per arm: AD, 2; BD, 4; HC, 21; MDD, 5; OCD, 2; SCZ, 11; model ratio = 1.041, $I^2 = 6\%$). SCZ and BD demonstrated a significant delay in P3b latency compared to MDD, OCD, and HC (Fig. 2C; Appendix 5, Tables S5–S6).

MMN: sensory and cognitive deviance detection

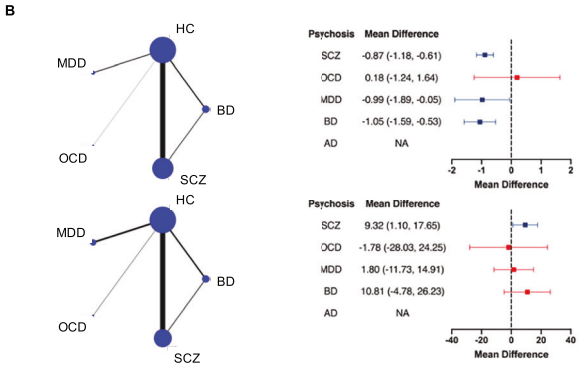
Totally, 118 studies comprising 10,895 participants presented usable results for MMN amplitude (Studies per arm: BD, 15; HC, 116; MDD, 10; OCD, 2; SCZ, 104; model ratio = 0.994, $I^2 = 0\%$). SCZ, BD, and MDD related to significant reduction in MMN amplitude compared to HC, while OCD had significantly higher MMN amplitude than HC. Besides, MMN amplitude was significantly lower in SCZ, BD, and MDD compared to OCD. Moreover, 66 studies with 5576 participants presented usable data for MMN latency (Studies per arm: AD, 1; BD, 7; HC, 64; MDD, 9; OCD, 2; SCZ, 55; model ratio = 1.030, $I^2 = 4\%$). There was no difference in MMN latency between psychiatric disorders and HC, even across psychiatric disorders (Fig. 2D; Appendix 5, Tables S7–S8).

P50 component: sensory gating and filtering mechanisms

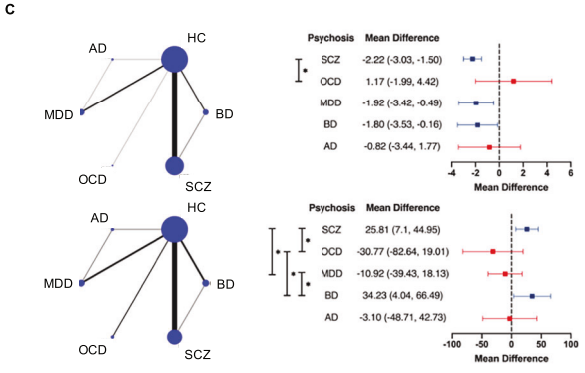
A total of 95 studies involving 10,391 participants provided usable data for P50 S1 amplitude (Studies per arm: AD, 4; BD, 9; HC, 94; MDD, 2; OCD, 2; SCZ, 86; model ratio = 1.014, $I^2 = 2\%$). Only the P50 S1 amplitude of SCZ was significantly lower than HC. For P50 S1 latency, 51 studies with 5412 participants reported usable results (Studies per arm: AD, 2; BD, 6; HC, 51; MDD, 1; OCD, 1; SCZ,



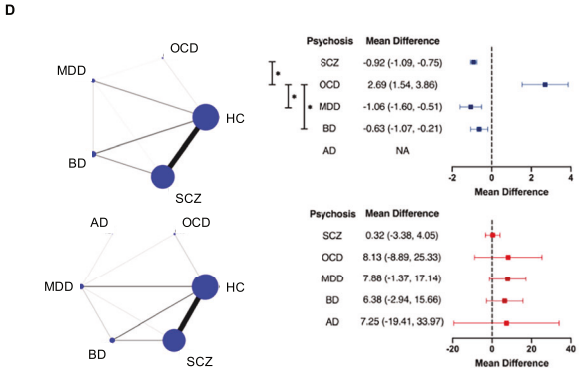
SCZ	5.07 (-3.07, 13.07)	6.42 (-1.41, 14.15)	**22.28 (12.66, 31.65)**	**16.25 (7.42, 24.98)**	**20.65 (17.38, 23.95)**
-0.47 (-1.34, 0.38)	**BD**	1.34 (-9.24, 11.81)	**17.19 (5.27, 29.09)**	11.23 (-0.25, 22.47)	**15.57 (7.84, 23.51)**
-0.40 (-1.24, 0.43)	0.07 (-1.00, 1.15)	**MDD**	**15.80 (4.56, 27.18)**	9.80 (-0.47, 19.99)	**14.20 (7.08, 21.47)**
-1.31 (-2.51, -0.12)	-0.84 (-2.23, 0.56)	-0.91 (-2.24, 0.46)	**OCD**	-5.97 (-17.65, 5.46)	-1.59 (-10.47, 7.42)
-1.57 (-2.70, -0.43)	-1.09 (-2.45, 0.24)	-1.16 (-2.44, 0.10)	-0.25 (-1.74, 1.24)	**AD**	4.41 (-3.77, 12.68)
-2.41 (-2.78, -2.04)	**-1.94 (-2.75, -1.11)**	**-2.01 (-2.76, -1.25)**	-1.10 (-2.23, 0.03)	-0.84 (-1.92, 0.22)	**HC**



SCZ	-1.40 (-18.44, 15.35)	7.56 (-7.92, 23.39)	11.29 (-16.47, 38.45)	NA	**9.32 (1.10, 17.65)**
0.17 (-0.38, 0.72)	**BD**	9.04 (-11.52, 29.61)	12.65 (-18.16, 42.78)	NA	10.81 (-4.78, 26.23)
0.12 (-0.89, 1.05)	-0.06 (-1.14, 0.97)	**MDD**	3.58 (-25.98, 32.92)	NA	1.80 (-11.73, 14.91)
-1.05 (-2.56, 0.38)	-1.23 (-2.79, 0.31)	-1.18 (-2.85, 0.58)	**OCD**	NA	-1.78 (-28.03, 24.25)
NA	NA	NA	NA	**AD**	NA
-0.87 (-1.18, -0.61)	**-1.05 (-1.59, -0.53)**	**-0.99 (-1.89, -0.05)**	0.18 (-1.24, 1.64)	NA	**HC**



SCZ	-8.33 (-44.34, 25.46)	**36.78 (2.59, 70.90)**	**56.70 (3.79, 112.38)**	29.21 (-20.23, 78.55)	**25.81 (7.10, 44.95)**
-0.41 (-2.17, 1.35)	**BD**	**45.07 (4.28, 88.53)**	**65.26 (7.36, 126.06)**	37.56 (-16.59, 93.41)	**34.23 (4.04, 66.49)**
-0.30 (-1.93, 1.35)	0.10 (-2.12, 2.36)	**MDD**	19.88 (-36.84, 79.45)	-7.74 (-51.49, 36.38)	-10.92 (-39.43, 18.13)
-3.40 (-6.77, -0.16)	-2.99 (-6.71, 0.60)	-3.09 (-6.69, 0.36)	**OCD**	-27.66 (-95.00, 37.77)	-30.77 (-82.64, 19.01)
-1.40 (-4.12, 1.32)	-0.99 (-4.11, 2.11)	-1.10 (-3.71, 1.48)	2.00 (-2.06, 6.16)	**AD**	-3.10 (-48.71, 42.73)
-2.22 (-3.03, -1.50)	**-1.80 (-3.53, -0.16)**	**-1.92 (-3.42, -0.49)**	1.17 (-1.99, 4.42)	-0.82 (-3.44, 1.77)	**HC**



SCZ	-6.11 (-15.62, 3.50)	-7.51 (-17.27, 2.23)	-7.80 (-25.42, 9.59)	-6.82 (-33.74, 19.71)	0.32 (-3.38, 4.05)
-0.28 (-0.72, 0.16)	**BD**	-1.46 (-13.29, 10.22)	-1.67 (-21.24, 17.54)	-0.91 (-28.68, 26.94)	6.38 (-2.94, 15.66)
0.14 (-0.43, 0.70)	0.43 (-0.23, 1.07)	**MDD**	-0.24 (-18.47, 17.87)	0.57 (-24.61, 25.56)	7.88 (-1.37, 17.14)
-3.60 (-4.79, -2.44)	**-3.32 (-4.56, -2.10)**	**-3.75 (-4.97, -2.54)**	**OCD**	0.93 (-30.11, 31.66)	8.13 (-8.89, 25.33)
NA	NA	NA	NA	**AD**	7.25 (-19.41, 33.97)
-0.92 (-1.09, -0.75)	**-0.63 (-1.07, -0.21)**	**-1.06 (-1.60, -0.51)**	**2.69 (1.54, 3.86)**	NA	**HC**

Fig. 2 The differences of P300 and MMN across psychiatric disorders. A The differences of P300 on amplitude (top) and latency (bottom). **B** The differences of P3a on amplitude (top) and latency (bottom). **C** The differences of P3b on amplitude (top) and latency (bottom). **D** The differences of MMN on amplitude (top) and latency (bottom). Network plot (left): the node size of the nodes corresponds to the sample size of each psychosis (or HC). Direct between-psychiatric disorders comparisons are linked with a line, and the thickness corresponds to the number of trials evaluating the comparison. Forest plot (middle): the mean differences compared with HC, significant results are labelled in blue, and non-significant results are labelled in red, * refers to significant results across psychiatric disorders. League table (right): the mean differences between-psychiatric disorders comparisons, the left red part represents results of amplitude (ratio), and the right blue part represents results of latency (difference). Cells in bold print indicate significant results. SCZ schizophrenia, BD bipolar disorder, MDD major depressive disorder, OCD Obsessive-compulsive disorder, AD anxiety disorder, HC healthy control, NA not applicable.

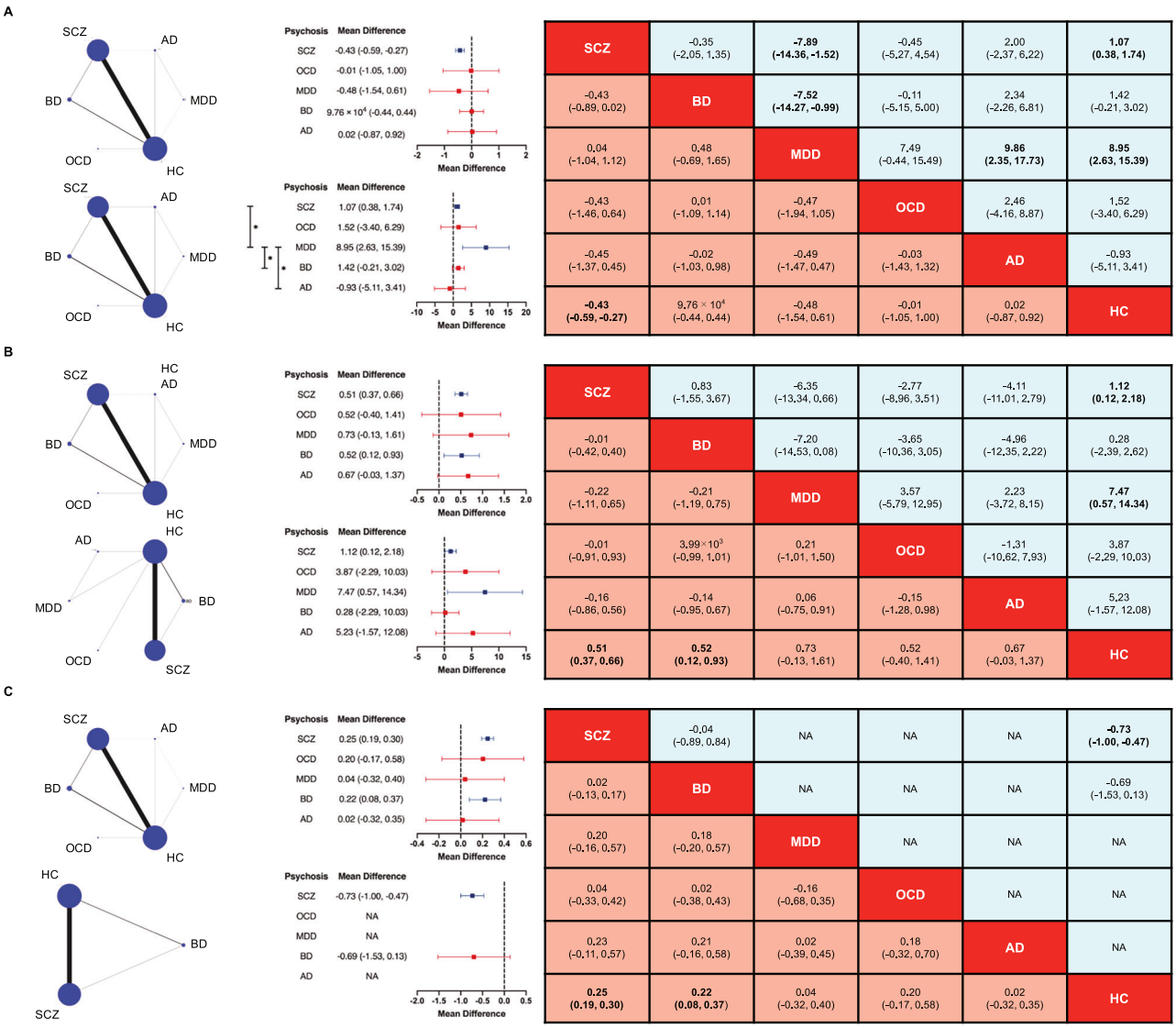


Fig. 3 The differences of P50 across psychiatric disorders. A The differences of P50 S1 on amplitude (top) and latency (bottom). **B** The differences of P50 S2 on amplitude (top) and latency (bottom). **C** The differences of P50 ratio and difference on amplitude (top) and latency (bottom). Network plot (left): the node size corresponds to the sample size of each psychosis (or HC). Direct between-psychiatric disorders comparisons are linked with a line, and the thickness represents the number of trials evaluating the comparison. Forest plot (middle): the mean differences compared with HC, significant results are labelled in blue, and non-significant results are labelled in red, * refers to significant results across psychiatric disorders. League table (right): the mean difference of between-psychiatric disorders comparisons, the left red part represents results of amplitude (ratio), and the right blue part represents results of latency (difference). Cells in bold print indicate significant results. SCZ schizophrenia, BD bipolar disorder, MDD major depressive disorder, OCD Obsessive-compulsive disorder, AD anxiety disorder, HC healthy control, NA not applicable.

45; model ratio = 0.956, $I^2 = 0\%$). SCZ and MDD showed a significant delay in P50 S1 latency compared to HC. The P50 S1 latency of MDD was significantly longer than SCZ, BD and AD (Fig. 3A; Appendix 5, Tables S9–S10).

A total of, 93 studies with 10,372 participants presented usable data for P50 S2 amplitude (Studies per arm: AD, 4; BD, 9; HC, 92; MDD, 3; OCD, 2; SCZ, 83; model ratio = 1.051, $I^2 = 5\%$). SCZ and BD exhibited a significant increase in P50 S2 amplitude compared to HC. Additionally, 40 studies with 4222 participants provided usable results for P50 S2 latency (Studies per arm: AD, 1; BD, 4; HC, 40; MDD, 1; OCD, 1; SCZ, 35; model ratio = 1.257, $I^2 = 21\%$). SCZ and MDD showed a significant delay in P50 S2 latency compared to HC. (Fig. 3B; Appendix 5, Tables S11–S12).

Totally, 97 studies comprising 11,463 participants reported usable results for P50 ratio (Studies per arm: AD, 2; BD, 12; HC, 97; MDD, 2; OCD, 2; SCZ, 88; model ratio = 1.008, $I^2 = 9\%$). SCZ and BD demonstrated a significant increase in P50 ratio compared to HC. Furthermore, 33 studies involving 4 616 participants presented usable results for P50 difference (Studies per arm: BD, 3; HC, 33; SCZ, 31; model ratio = 1.029, $I^2 = 4\%$). SCZ showed a significant decrease in P50 difference compared to HC (Fig. 3C; Appendix 5, Tables S13–S14).

N1, N2, and P1: early sensory and cognitive responses

A total of 127 studies involving 9593 participants presented usable results for N1 amplitude (Studies per arm: AD, 14; BD, 19; HC, 126; MDD, 20; OCD, 4; SCZ, 88; model ratio = 1.070, $I^2 = 7\%$). SCZ, BD, and MDD were associated with a significant reduction in N1 amplitude compared to HC. Additionally, the N1 amplitude of SCZ was significantly lower than BD, MDD, and OCD, whereas the N1 amplitude of OCD was notably higher than AD, BD, and MDD. Furthermore, 98 studies comprising 6374 participants provided

usable data for N1 latency (Studies per arm: AD, 13; BD, 13; HC, 98; MDD, 17; OCD, 5; SCZ, 63; model ratio = 1.022, $I^2 = 3\%$). The N1 latency of OCD was significantly higher compared to HC and other psychiatric disorders (Fig. 4A; Appendix 5, Tables S15–S16).

Totally, 82 studies with 6001 participants reported usable results for N2 amplitude (Studies per arm: AD, 13; BD, 10; HC, 82; MDD, 17; OCD, 9; SCZ, 45; model ratio = 1.188, $I^2 = 16\%$). Only the N2 amplitude of SCZ was significantly lower than HC. Besides, the N2 amplitude of SCZ was notably lower than BD. Moreover, 69 studies involving 4962 participants presented usable results for N2 latency (Studies per arm: AD, 13; BD, 7; HC, 69; MDD, 14; OCD, 7; SCZ, 39; model ratio = 1.037, $I^2 = 4\%$). SCZ and BD related to significant delay in N2 latency compared to HC. Additionally, the N2 latency of SCZ was longer than OCD and AD, and the N2 latency of BD was also longer than AD (Fig. 4B; Appendix 5, Tables S17–S18).

A total of 38 studies comprising 1985 participants provided usable results for P1 amplitude (Studies per arm: AD, 6; BD, 2; HC, 38; MDD, 5; OCD, 2; SCZ, 25; model ratio = 1.010, $I^2 = 2\%$), and 21 studies with 987 participants presented usable results for P1 latency (Studies per arm: AD, 3; BD, 3; HC, 21; MDD, 4; OCD, 1; SCZ, 12; model ratio = 0.990, $I^2 = 1\%$). There were no differences in P1 components between psychiatric disorders and HC, even across psychiatric disorders (Fig. 4C; Appendix 5, Tables S19–S20).

A total of 86 studies involving 7321 participants presented usable results for P2 amplitude (Studies per arm: AD, 16; BD, 13; HC, 85; MDD, 15; OCD, 5; SCZ, 52; model ratio = 1.087, $I^2 = 8\%$). Only the P2 amplitude of SCZ was significantly lower than HC. Additionally, the P2 amplitude of OCD was also notably higher than SCZ, BD, and MDD. Moreover, 65 studies involving 4724 participants provided usable results for P2 latency (Studies per arm: AD, 11; BD, 8; HC, 65; MDD, 10; OCD, 4; SCZ, 42; model ratio = 0.974, $I^2 = 0\%$). There was no difference in P2 latency between psychiatric disorders and HC, even across psychiatric disorders (Fig. 4D; Appendix 5, Tables S21–S22).

N170 and N400: visual and semantic processing

A total of 31 studies involving 1993 participants presented usable results for N170 amplitude (Studies per arm: AD, 2; BD, 4; HC, 31; MDD, 6; SCZ, 20; model ratio = 0.995, $I^2 = 1\%$). Both SCZ and MDD were associated with a significant decrease in N170 amplitude compared to HC. Additionally, 16 studies comprising 1143 participants reported usable results for N170 latency (Studies per arm: BD, 3; HC, 16; MDD, 4; SCZ, 10; model ratio = 1.015, $I^2 = 4\%$). Only MDD showed a significant delay in N170 latency compared to HC (Appendix 5, Tables S23–S24; Appendix 7, Fig. S1).

Totally, 22 studies with 1143 participants provided usable data for N400 amplitude (Studies per arm: AD, 1; BD, 1; HC, 21; MDD, 3; SCZ, 19; model ratio = 0.975, $I^2 = 0\%$). For N400 latency, 15 studies involving 893 participants presented usable results (Studies per arm: BD, 1; HC, 15; MDD, 1; SCZ, 14; model ratio = 1.124, $I^2 = 14\%$). The SCZ was associated with a significant reduction in N400 amplitude and a significant delay in N400 latency compared to HC (Appendix 5, Tables S25–S26; Appendix 7, Fig. S2).

CNV and ERN

A total of 18 studies with 1297 participants presented usable results for CNV amplitude (Studies per arm: AD, 2; BD, 4; HC, 17; MDD, 9; OCD, 1; SCZ, 9; model ratio = 1.002, $I^2 = 3\%$). AD showed a significant reduction in CNV amplitude compared to HC, and CNV amplitude of BD was significantly higher than AD (Appendix 5, Table S27; Appendix 7, Fig. S3). Additionally, 34 studies involving 2243 participants provided usable results for ERN amplitude (Studies per arm: AD, 8; BD, 2; HC, 34; MDD, 11; OCD, 13; SCZ, 5; model ratio = 1.046, $I^2 = 6\%$). SCZ was associated with a significant reduction in ERN amplitude compared to HC, whereas the OCD and AD showed a significant increase. The ERN amplitude of SCZ was significantly lower than MDD, OCD, and AD (Appendix 5, Table S28; Appendix 7, Fig. S4). Additionally, 18 studies involving 1222 participants presented usable results for LPP amplitude (Studies per arm: AD, 3; HC, 18; MDD, 8; OCD, 3; SCZ, 4; model ratio = 0.943, $I^2 = 0\%$). There were no differences in LPP amplitude between psychiatric disorders and HC, even across psychiatric disorders (Appendix 5, Table S29; Appendix 7, Fig. S5). The latency of CNV, ERN, and LPP did not undergo the network meta-analyses due to the limited number of included studies.

Sensitivity analysis and ERP-score

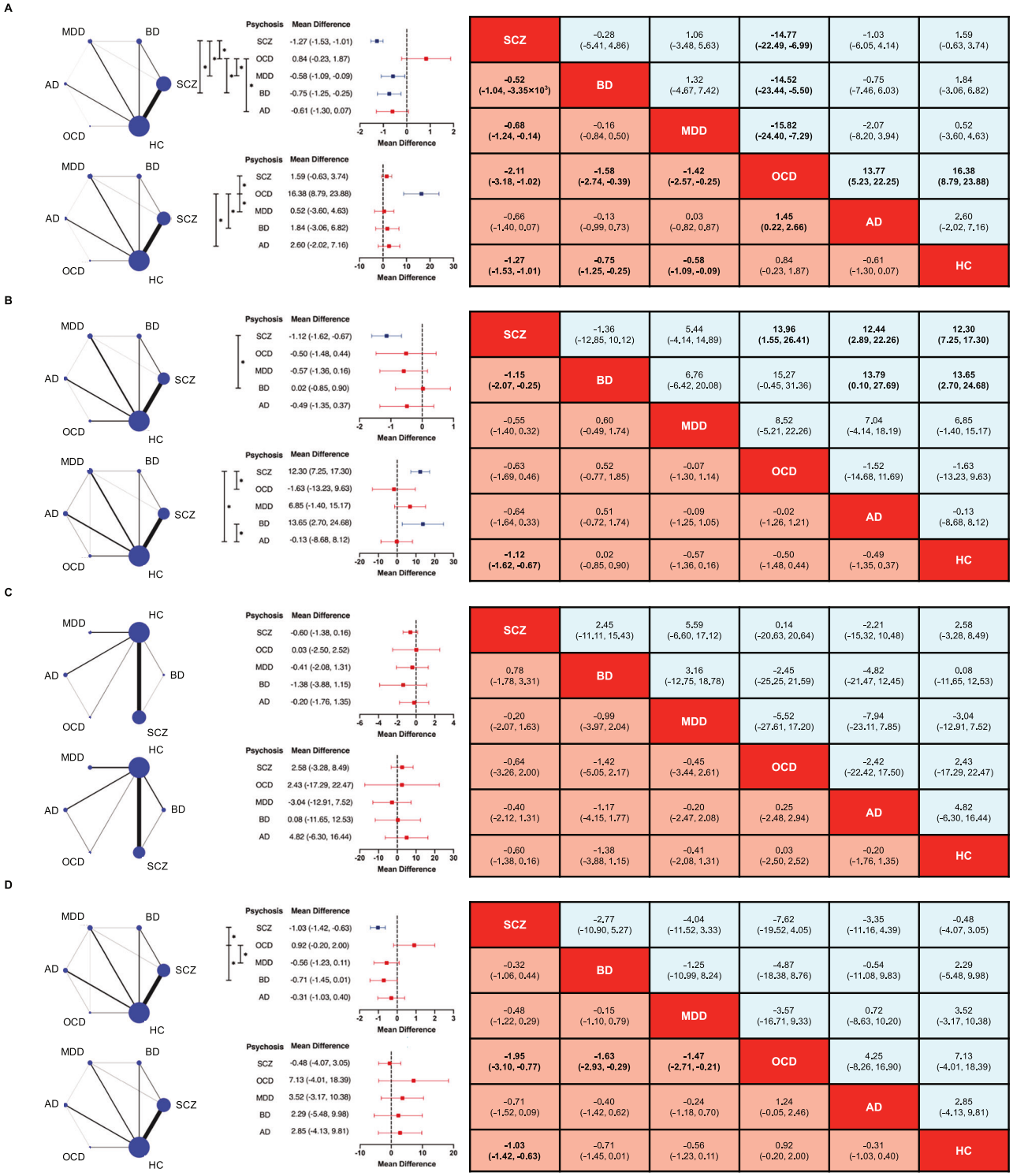
Sensitivity analyses were conducted by excluding studies with an overall high or very high risk of bias (Appendix 6). The stability of results was confirmed for P300 amplitude, P3a amplitude, MMN amplitude and latency, P50 S1 amplitude, P50 S2 amplitude, P50 ratio and difference, N1 latency, N2 amplitude, P1 amplitude and latency, P2 amplitude and latency, N400 amplitude and latency (Appendix 8, Figs. S6–S19). No study with an overall high or very high risk were found for N170 amplitude, N170 latency, and LPP amplitude. Some ERP components became statistically significant after excluding studies with high or very high risk of bias. For example, the difference in P300 latency between SCZ and MDD became significant, as did between BD and AD. Conversely, some significant differences disappeared, such as the difference in P300 latency between MDD and HC, as well as between MDD and OCD. Funnel plots indicated low publication bias for most ERP indices, with the majority of studies concentrated near the top (Appendix 9, Figs. S20–S48).

Finally, an ERP-score was calculated to illustrate electrophysiological characteristics. According to the ERP-score, SCZ exhibited the highest degree of electrophysiological abnormality, the finding persisted even after excluding studies with high or very high risk of bias. In contrast, OCD appeared to have opposite electrophysiological characteristics compared to SCZ, particularly in MMN amplitude and ERN amplitude (Fig. 5).

DISCUSSION

This study presents the first and largest network meta-analysis of ERP components across major psychiatric disorders, incorporating data from 687 studies and 29 ERP components, with participant numbers ranging from 893 to 16,879. Through a systematic search and screening process, we included ERP components related to attention, cognitive processing, sensory gating, and emotion regulation, ensuring a robust characterization of electrophysiological abnormalities. While components such as P300 and MMN have been more extensively studied, our analysis provides a broader perspective on neurophysiological differences across disorders. The findings confirm significant ERP abnormalities across psychiatric disorders, with schizophrenia showing the most severe impairments, particularly in P300 and P50 components, suggesting potential diagnostic value in linking electrophysiological profiles to cognitive dysfunctions. Specifically, BD exhibited reduced amplitudes in both P3a and P3b components, MDD showed significant reductions in P300 amplitude and delays in P3a latency, and OCD demonstrated increased MMN amplitude and delays in P3b latency. These ERP abnormalities not only highlight the distinct neurophysiological profiles of these disorders but also offer potential in guiding personalized treatment approaches by identifying specific sensory and cognitive deficits, which could inform tailored interventions for schizophrenia, bipolar disorder, and major depressive disorder.

The P300, typically evoked by the "oddball" paradigm, comprises two subcomponents: P3a and P3b. P3a is linked to automatic attention processes, responding to novel stimuli and reflecting attention engagement and novelty processing. P3b, associated with conscious processing and working memory update, supports higher cognitive functions like decision-making and stimulus relevance evaluation, and it is instrumental in studying attention, working memory, and decision-making



SCZ	-0.28 (-5.41, 4.86)	1.06 (-3.48, 5.63)	**-14.77 (-22.49, -6.99)**	-1.03 (-6.05, 4.14)	1.59 (-0.63, 3.74)
-0.52 (-1.04, -3.35×10^{3})	**BD**	1.32 (-4.67, 7.42)	**-14.52 (-23.44, -5.50)**	-0.75 (-7.46, 6.03)	1.84 (-3.06, 6.82)
-0.68 (-1.24, -0.14)	-0.16 (-0.84, 0.50)	**MDD**	**-15.82 (-24.40, -7.29)**	-2.07 (-8.20, 3.94)	0.52 (-3.60, 4.63)
-2.11 (-3.18, -1.02)	**-1.58 (-2.74, -0.39)**	**-1.42 (-2.57, -0.25)**	**OCD**	**13.77 (5.23, 22.25)**	**16.38 (8.79, 23.88)**
-0.66 (-1.40, 0.07)	-0.13 (-0.99, 0.73)	0.03 (-0.82, 0.87)	**1.45 (0.22, 2.66)**	**AD**	2.60 (-2.02, 7.16)
-1.27 (-1.53, -1.01)	**-0.75 (-1.25, -0.25)**	**-0.58 (-1.09, -0.09)**	0.84 (-0.23, 1.87)	-0.61 (-1.30, 0.07)	**HC**

SCZ	-1.36 (-12.85, 10.12)	5.44 (-4.14, 14.89)	**13.96 (1.55, 26.41)**	**12.44 (2.89, 22.26)**	**12.30 (7.25, 17.30)**
-1.15 (-2.07, -0.25)	**BD**	6.76 (-6.42, 20.08)	15.27 (-0.45, 31.36)	**13.79 (0.10, 27.69)**	**13.65 (2.70, 24.68)**
-0.55 (-1.40, 0.32)	0.60 (-0.49, 1.74)	**MDD**	8.52 (-5.21, 22.26)	7.04 (-4.14, 18.19)	6.85 (-1.40, 15.17)
-0.63 (-1.69, 0.46)	0.52 (-0.77, 1.85)	-0.07 (-1.30, 1.14)	**OCD**	-1.52 (-14.68, 11.69)	-1.63 (-13.23, 9.63)
-0.64 (-1.64, 0.33)	0.51 (-0.72, 1.74)	-0.09 (-1.25, 1.05)	-0.02 (-1.26, 1.21)	**AD**	-0.13 (-8.68, 8.12)
-1.12 (-1.62, -0.67)	0.02 (-0.85, 0.90)	-0.57 (-1.36, 0.16)	-0.50 (-1.48, 0.44)	-0.49 (-1.35, 0.37)	**HC**

SCZ	2.45 (-11.11, 15.43)	5.59 (-6.60, 17.12)	0.14 (-20.63, 20.64)	-2.21 (-15.32, 10.48)	2.58 (-3.28, 8.49)
0.78 (-1.78, 3.31)	**BD**	3.16 (-12.75, 18.78)	-2.45 (-25.25, 21.59)	-4.82 (-21.47, 12.45)	0.08 (-11.65, 12.53)
-0.20 (-2.07, 1.63)	-0.99 (-3.97, 2.04)	**MDD**	-5.52 (-27.61, 17.20)	-7.94 (-23.11, 7.85)	-3.04 (-12.91, 7.52)
-0.64 (-3.26, 2.00)	-1.42 (-5.05, 2.17)	-0.45 (-3.44, 2.61)	**OCD**	-2.42 (-22.42, 17.50)	2.43 (-17.29, 22.47)
-0.40 (-2.12, 1.31)	-1.17 (-4.15, 1.77)	-0.20 (-2.47, 2.08)	0.25 (-2.48, 2.94)	**AD**	4.82 (-6.30, 16.44)
-0.60 (-1.38, 0.16)	-1.38 (-3.88, 1.15)	-0.41 (-2.08, 1.31)	0.03 (-2.50, 2.52)	-0.20 (-1.76, 1.35)	**HC**

SCZ	-2.77 (-10.90, 5.27)	-4.04 (-11.52, 3.33)	-7.62 (-19.52, 4.05)	-3.35 (-11.16, 4.39)	-0.48 (-4.07, 3.05)
-0.32 (-1.06, 0.44)	**BD**	-1.25 (-10.99, 8.24)	-4.87 (-18.38, 8.76)	-0.54 (-11.08, 9.83)	2.29 (-5.48, 9.98)
-0.48 (-1.22, 0.29)	-0.15 (-1.10, 0.79)	**MDD**	-3.57 (-16.71, 9.33)	0.72 (-8.63, 10.20)	3.52 (-3.17, 10.38)
-1.95 (-3.10, -0.77)	**-1.63 (-2.93, -0.29)**	**-1.47 (-2.71, -0.21)**	**OCD**	4.25 (-8.26, 16.90)	7.13 (-4.01, 18.39)
-0.71 (-1.52, 0.09)	-0.40 (-1.42, 0.62)	-0.24 (-1.18, 0.70)	1.24 (-0.05, 2.46)	**AD**	2.85 (-4.13, 9.81)
-1.03 (-1.42, -0.63)	-0.71 (-1.45, 0.01)	-0.56 (-1.23, 0.11)	0.92 (-0.20, 2.00)	-0.31 (-1.03, 0.40)	**HC**

Fig. 4 The differences of N1, N2, P1, and P2 across psychiatric disorders. A The differences of N1 on amplitude (top) and latency (bottom). **B** The differences of N2 on amplitude (top) and latency (bottom). **C** The differences of P1 on amplitude (top) and latency (bottom). **D** The differences of P2 on amplitude (top) and latency (bottom). Network plot (left): the node size corresponds to the sample size of each psychosis (or HC). Direct between-psychiatric disorders comparisons are linked with a line, and the thickness represents the number of trials evaluating the comparison. Forest plot (middle): the mean differences compared with HC, significant results are labelled in blue, and non-significant results are labelled in red, * refers to significant results across psychiatric disorders. League table (right): the mean difference of between-psychiatric disorders comparisons, the left red part represents results of amplitude (ratio), and the right blue part represents results of latency (difference). Cells in bold print indicate significant results. SCZ schizophrenia, BD bipolar disorder, MDD major depressive disorder, OCD Obsessive-compulsive disorder, AD anxiety disorder, HC healthy control, NA not applicable.

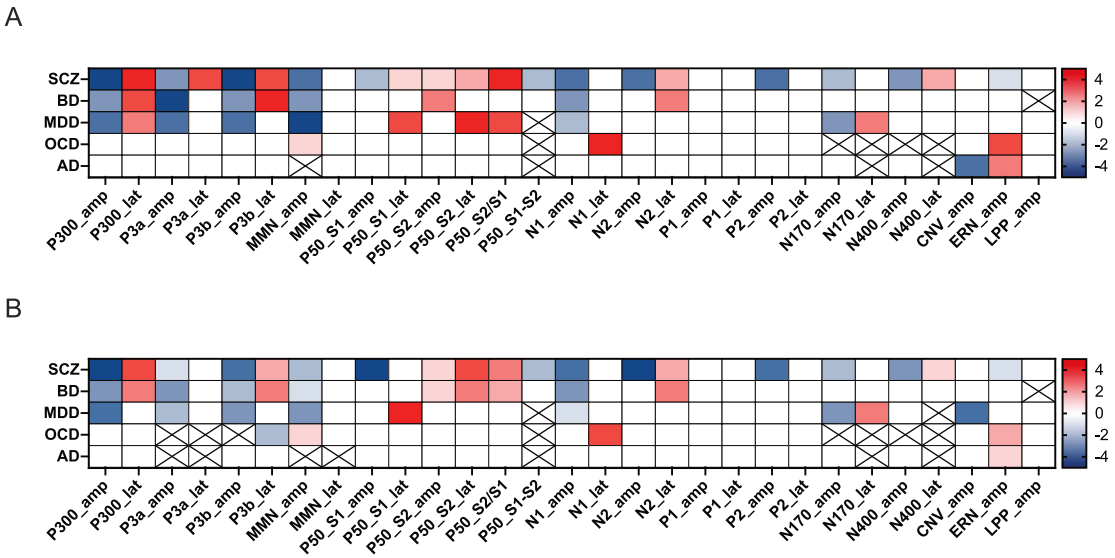


Fig. 5 ERP-score across psychiatric disorders. A Initial ERP-score. **B** ERP-score after removing the studies with high or very high risk of bias. The blue color refers to the ERP components related to significant decrease compared to HC, the red color refers to the ERP components related to significant increase compared to HC. MMN Mismatch Negativity, amp amplitude, lat latency, P50 S2/S1 P50 ratio, P50 S1-S2 P50 difference, SCZ schizophrenia, BD bipolar disorder, MDD major depressive disorder, OCD Obsessive-compulsive disorder, AD anxiety disorder. ×, missing data.

processes. Abnormal P300 responses, such as amplitude reductions and latency delays, are observed in SCZ, BD [12], and MDD [13]. Meta-analyses consistently identify P300 amplitude reduction as a robust biological marker of schizophrenia [14, 15], reflecting both trait-like deficits and state-dependent fluctuations [16, 17]. Moreover, P300 deficits worsen with illness duration, suggesting progressive pathophysiological processes in schizophrenia [16]. Efforts have explored P300 as a potential vulnerability marker for schizophrenia, predicting psychosis onset in at-risk individuals identified through clinical criteria for clinical high risk for psychosis (CHR-P). Studies suggest similarities in P300 deficits between CHR-P individuals and those with schizophrenia [18–21]. Unfortunately, investigations into the relationships between specific schizophrenia symptom domains and P300 have yielded inconsistent results [14]. Our findings indicate P300 deficits are evident across SCZ, BD, and MDD, with SCZ showing the most pronounced deficits. Notably, differences among these psychiatric disorders were generally nonsignificant, except for P3b latency differences between BD and MDD.

MMN reflects the automatic response to changes in auditory stimuli, typically in an oddball paradigm where a sequence of standard sounds is occasionally interrupted by deviant sounds differing in acoustic properties. MMN occurs pre-attentively, indicating it doesn't require conscious attention, but rather signals the automatic detection of auditory pattern deviations. Abnormal MMN responses suggest deficits in sensory memory, auditory perception, and automatic detection processes [22]. MMN deficits are a trait feature of schizophrenia, with meta-analyses confirming amplitude reductions in SCZ [15, 23–25]. Similar reductions have been observed in MDD [26] and BD [27]. In our study, MMN amplitude reductions were evident in SCZ, BD, and MDD, with MDD exhibiting the lowest amplitudes. Previous research indicates BD shows moderate MMN impairment compared to the pronounced deficits seen in schizophrenia. However, differences among these disorders were not statistically significant. Interestingly, MMN amplitudes differed significantly between OCD and HC, with a reversal in effect direction compared to the three psychiatric disorders. While MMN impairments are well-documented in schizophrenia and other psychiatric disorders, the extent of impairment varies across studies, likely due to differences in stimulus properties and participant characteristics. A more systematic investigation of these factors could offer deeper insights into the specificity of MMN abnormalities.

The P50 is primarily used to assess sensory gating (P50 ratio), which filters unnecessary or repetitive stimuli, crucial for normal cognitive function. In schizophrenia, robust P50 suppression deficits are observed, associated with difficulty in filtering irrelevant information [15, 28]. Similar abnormalities occur in conditions like BD [29]. In our studies, SCZ showed deficits in all P50 components (amplitude, latency of P50 S1 and S2, P50 ratio and difference). MDD exhibited delayed latency of P50 S1 and S2, while BD showed reduced P50 S2 amplitude and ratio deficit.

Abnormal N1 and P1 amplitude or latency indicate sensory processing deficits, seen in SCZ [30, 31]. N2 relates to attention, conflict monitoring, and cognitive control, while P2 reflects higher-order cognitive processing beyond sensory encoding [32]. N1, N2, P1, P2 can elucidate how cognitive processes alter with training and experience [33, 34]. According to our findings, SCZ displayed severe deficits in N1, N2, and P2 amplitudes and delayed N1 latency.

N170 is linked to face perception [35], SCZ and MDD exhibited deficits in our study. N400 indicates semantic processing in language comprehension [36], showing deficits in SCZ in our study. CNV studies anticipatory attention [37], the CNV deficits only occurred in AD in our study. ERN detects errors and cognitive control initiation [38]. However, the number of studies analyzing these indices in the current research is relatively small. Future research could benefit from expanding the number of studies for reanalysis. Abnormal ERN components have been observed in various clinical populations, including AD [39], OCD [40], and MDD [41]. In this study, abnormal ERN occurred in SCZ, AD, and OCD. LPP reflects allocation of attention to emotionally salient stimuli [42]. There was no difference in LPP amplitude between psychiatric disorders and HC.

To ensure a comprehensive synthesis of ERP abnormalities in psychiatric disorders, we systematically reviewed existing studies. While our study includes a broad range of ERP components, the sample size of some components is insufficient. Specifically, P300 and MMN were more commonly studied, whereas ERN and CNV had relatively fewer available samples. The insufficient sample size may influence the generalizability of our findings, while it reflects the current state of related research. Future studies should expand

investigations into underrepresented ERP components to enhance the robustness of findings. To provide a more comprehensive characterization of electrophysiological abnormalities, we calculated the ERP-score based on our network meta-analysis results. This composite index reflects the overall magnitude of ERP deviations across cognitive domains and may help identify impaired cognitive processes in psychiatric disorders. However, its clinical utility remains to be fully established, requiring further validation to optimize component weightings and assess its potential for aiding diagnosis and treatment monitoring. Beyond individual ERP components, future studies integrating ERP with neuroimaging techniques such as fMRI or MEG may offer deeper insights into the neural circuits underlying these electrophysiological disruptions. Multimodal approaches could help link ERP alterations to dysfunctional brain networks, advancing our understanding of the pathophysiology of psychiatric disorders. Longitudinal investigations of ERP changes could further elucidate illness progression and support the development of early detection and intervention strategies for at-risk populations.

Our analysis encountered several limitations. Firstly, significant between-study heterogeneity was observed. The calculation of mean values across recorded scalp locations may have influenced outcomes, as previous meta-analyses have noted larger effect sizes in studies reporting ERP components from a single fixed electrode [13]. Moreover, the lack of longitudinal data limits our ability to infer causal relationships between ERP abnormalities and psychiatric disorders. Medication status, comorbidities, and age differences between disorder groups could also be potential confounding factors that may affect ERP findings. For example, medication usage may alter neurophysiological responses, and comorbid conditions such as anxiety or substance abuse might exacerbate or mask ERP abnormalities. Furthermore, age-related differences in brain function could account for some of the observed variability in ERP findings across disorders. Although we attempted to minimize these influences by adhering to strict inclusion criteria, their potential impact cannot be entirely ruled out, and future studies should aim to systematically control for these confounders. Finally, some study arms included a limited number of publications, which may affect the generalizability of our findings.

In summary, this meta-analysis highlights the potential of ERP components in characterizing electrophysiological abnormalities across psychiatric disorders. SCZ exhibits the highest degree of electrophysiological abnormality, but BD and MDD also demonstrate significant impairments. Although these ERP abnormalities provide valuable insights into the neurophysiological basis of psychiatric disorders, their clinical applicability remains an area for further investigation. Future studies could explore how ERP deficits correlate with specific clinical symptoms, such as cognitive impairment, emotional dysregulation, or symptom severity. Additionally, while ERP abnormalities are not yet suitable as diagnostic markers at the individual level, they may serve as biomarkers for treatment monitoring, assisting in the refinement of therapeutic strategies based on electrophysiological profiles. The distinct electrophysiology patterns identified across disorders highlight the potential of ERPs for precision diagnosis and tailored interventions. However, addressing methodological inconsistencies and conducting more longitudinal studies will be critical for translating these findings into clinical practice.

DATA AVAILABILITY

All data in this network meta-analysis was publicly available.

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AUTHOR CONTRIBUTIONS

WY and ZL obtained funding for the study and designed it. ZL and JG set up the database. ZL, JG, JS, YS, YZ, Y.YZ, YL, ZK, XF, GZ, RY, and YZ screened the literature search, acquired reports of relevant trials, selected included studies, and extracted data. ZL and JG contacted the authors of records for additional information. ZL and JG did all statistical analyses. ZL, JG, JS, YS, YZ, Y.YZ and WY analysed and interpreted the data. ZL drafted the report. All authors critically reviewed the report for important intellectual content and approved the final submitted version.

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COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

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