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Predictive coding in neuropsychiatric disorders: A systematic transdiagnostic review

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ABSTRACT

The predictive coding framework postulates that the human brain continuously generates predictions about the environment, maximizing successes and minimizing failures based on prior experiences and beliefs. This PRISMA-compliant systematic review aims to comprehensively and transdiagnostically examine the differences in predictive coding between individuals with neuropsychiatric disorders and healthy controls. We included 72 articles including case-control studies investigating predictive coding as the primary outcome and reporting behavioral, neuroimaging, or electrophysiological findings. Thirty-three studies investigated predictive coding in the schizophrenia spectrum, 33 in neurodevelopmental disorders, 5 in mood disorders, 4 in neurocognitive disorders, 1 in post-traumatic stress disorder, and 1 in substance use disorders. Oddball and oddball-like paradigms were most frequently used to quantify predictive coding performance. Evidence showed heterogeneous impairments in the predictive coding abilities of the brain across neuropsychiatric disorders, particularly in schizophrenia and autism. Patients within the schizophrenia spectrum showed a consistent pattern of impaired non-social predictive coding. Conversely, predictive coding deficits were more selective for social cues in the autism spectrum. Predictive coding impairments were correlated with clinical symptom severity. These findings underscore the potential utility of predictive coding as a framework for understanding cognitive dysfunctions in the neuropsychiatric population, even though more evidence is needed on underexplored conditions, also considering potential confounders such as medication use and sex/gender. The potential role of predictive coding as a determinant of treatment response may also be considered to tailor personalized interventions.

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1. Introduction

The predictive coding framework is a highly influential theory in cognitive neuroscience postulating that the human brain encodes a model of the world in order to enable predictions from sensory stimuli (Clark, 2013). In this framework, priors represent the internal beliefs derived from past experience and knowledge (Aitchison and Lengyel, 2017). Priors are constantly used as a reference point to shape expectations, i.e., predictions, towards the future (Lange et al., 2018). The generation of reliable, heuristic predictions is obtained by maximizing the learning of statistical regularities and minimizing the probability of prediction errors (Friston, 2005; Rao and Ballard, 1999).

Predictive coding is enabled by a complex wiring of cortical loops. While feedforward projections send bottom-up sensory inputs and predictive errors from lower to higher brain regions, feedback processing sends top-down inferences. Working in tandem, these processes allow the brain to continuously infer and update its internal models, minimizing the amount of energy required to adapt to an everchanging environment (Bastos et al., 2012; Friston, 2010). Predictions are organized hierarchically in the predictive coding framework, from higher to lower levels (Huang and Rao, 2011; Mehta, 2001; Rao and Ballard, 1998; 1999; Spratling, 2008, 2010; Vuust et al., 2009). Lower-level predictions involve immediate and specific expectations about basic sensory details (e.g., predicting the continuation of a movement in a visual scene or the next sound in a sequence) and are typically processed in sensory areas. Mid-level predictions involve categorizing and integrating sensory information within a context (e.g., recognizing familiar faces or anticipating a note in a melody) and are processed in temporo-parietal regions. Higher-level predictions are abstract and context-dependent, involve higher cognitive processes (e.g., inferring social responses or understanding a rule in a task and anticipating subsequent stimuli), and are processed in frontal regions. Interestingly, the encoding of prediction errors into brain areas has been recently characterized: a recent meta-analysis highlights a consistent correspondence between specific brain areas and the domain-specificity of the tasks (Corlett et al., 2022).

Previous literature has reported that predictive abilities may be impaired in several psychiatric disorders. For example, it has been suggested that feedback and feedforward processing related to the accuracy of prior beliefs and sensory perceptions contribute to the formation of delusions and hallucinations in individuals with psychosis (Kirihara et al., 2020; Sterzer et al., 2018). According to the predictive coding framework, both these symptoms stem from imbalances between the precision of prior beliefs and the precision of sensory data, so that, for example, excessive expectations over sensory stimuli may lead to misattribute thoughts as false perceptions (Humpston and Broome, 2020; Horga and Abi-Dargham, 2019). Cognitive biases have been also specifically linked to the experience of aberrant salience, a dysfunction of the attentional processes wherein individuals allocate their perceptual and cognitive resources to identify and ascribe significance to the most pertinent environmental stimuli within a given context. Clinically, aberrant salience assumes a pivotal role in the genesis and persistence of psychotic symptoms (Aloi et al., 2024; Pugliese et al., 2022, 2024). Additionally, it has been argued that individuals with autism spectrum disorder (ASD) have a broader perception of priors than healthy controls, leading to a deficit in flexibility (Pellicano and Burr, 2012; Van Boxtel and Lu, 2013; Van de Cruys et al., 2014). It has also been postulated that individuals with depression underestimate or neglect positive information and focus on or overestimate negative information, leading to a negative prediction bias (Kube et al., 2020).

A plethora of specific tasks have been designed to observe the clinical effects of altered predictive coding. The most widely used are oddball and oddball-like paradigms, consisting of frequent standard identical stimuli and infrequent deviant stimuli that differ from the standard ones in one or more aspects (e.g., duration, tone, image, location, etc.). While standard stimuli tend to elicit fewer brain responses each time they are repeated, infrequent, deviant – and therefore “odd” - stimuli are perceived as salient. However, it is still unclear which are the most widely used and generalizable tasks (see Methods section for a more detailed explanation).

In the past decades, several narrative reviews explored the role of predictive coding in neuropsychiatric disorders focusing on a specific diagnostic cluster (Pellicano and Burr, 2012; Sterzer et al., 2018). However, to the best of our knowledge, there are no reviews systematically focused on predictive coding tasks among individuals with neuropsychiatric conditions. Given the growing interest in predictive coding in clinical research, this systematic review aims to comprehensively summarize the findings on this framework in neuropsychiatric disorders.

2. Materials and methods

2.1. *Search strategy*

We performed a systematic review following the PRISMA statement guidelines (Page et al., 2021) and the TRANSD recommendation to improve transdiagnostic research in psychiatry (Fusar-Poli, 2019; Fusar-Poli, Solmi, et al., 2019). The protocol was registered in Open Science Framework OSF (2024) platform (https://osf.io/v3wdk/). We searched the Web of Knowledge (all databases) and PsycINFO from inception to October 5, 2023. The complete search string has been reported in the **Supplementary Materials**.

2.2. *Study selection process*

We selected English studies published in peer-reviewed journals meeting the following inclusion criteria:

1) Participants: individuals of any age and sex diagnosed with a mental disorder according to validated international diagnostic criteria (e.g., Diagnostic and Statistical Manual of Mental Disorders [DSM], International Classification of Diseases [ICD]).
2) Control group: individuals without a diagnosis of neuropsychiatric disorder.
3) Sample size: at least 10 participants for each group.
4) Study design: case-control studies in which there was at least one group diagnosed with a neuropsychiatric disorder and in which the controls were healthy participants.

Exclusion criteria were:

1) Studies not written in English, conference abstracts, theses, dissertations, short papers/letters.
2) Studies in which participants were not diagnosed with validated diagnostic criteria (e.g., self-report and web surveys).
3) Studies in which predictive coding was not the primary focus.

Each study was assessed for inclusion by at least two researchers, with disagreements solved by consultation with a third reviewer.

2.3. *Data extraction*

Two researchers independently extracted information based on the inclusion/exclusion criteria.

Data were extracted in a pre-piloted form which included the following information: 1) Study characteristics: first author, year, country, study design; 2) Participant characteristics: neuropsychiatric diagnosis, sample size, mean age, age range (SD), proportion of males, neuropsychiatric comorbidities, intelligence quotient (IQ) and assessment methods, medications and dosages; 3) Measures and tasks used to assess predictive coding.

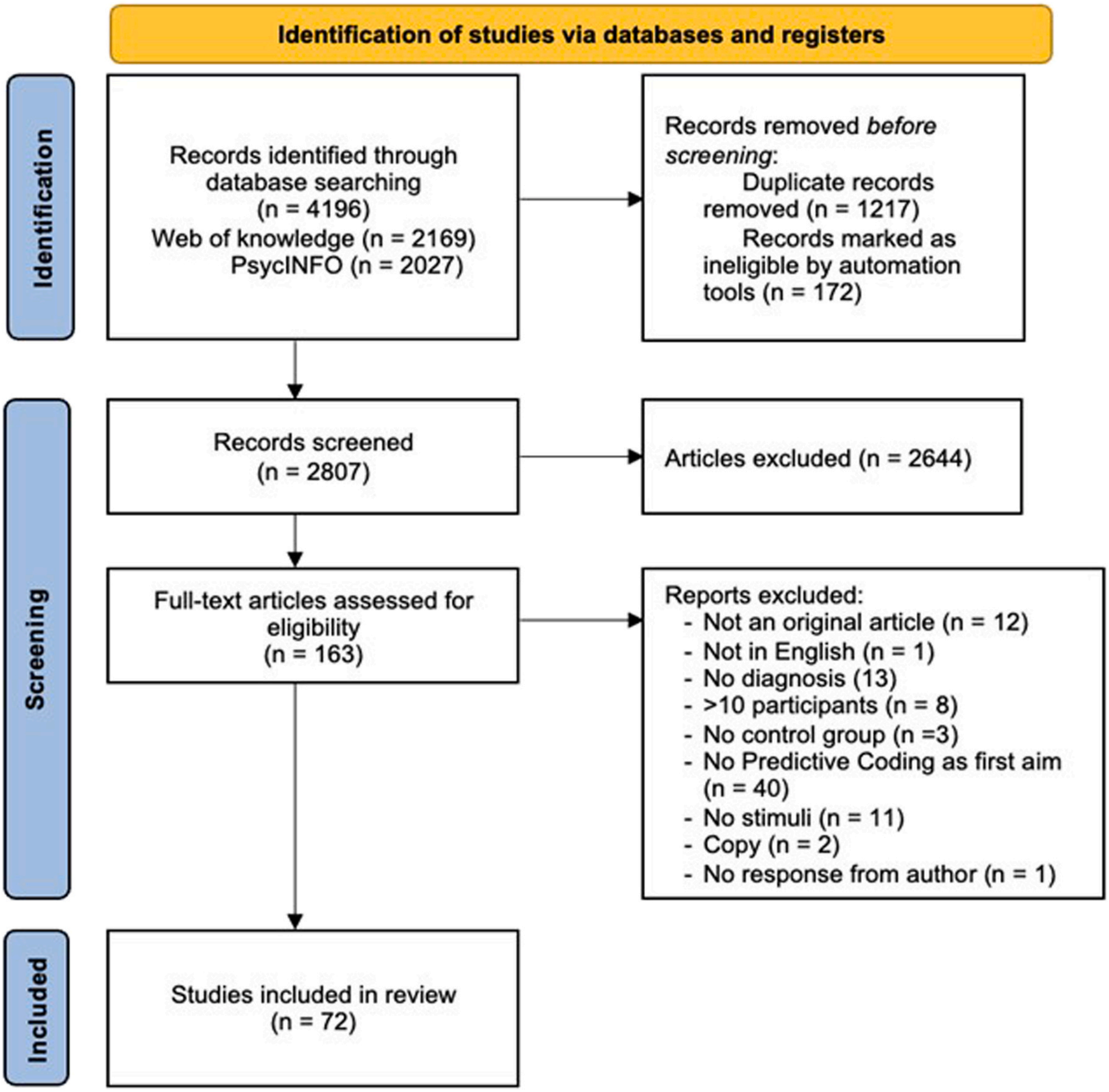

Fig. 1. PRISMA flow-diagram of the study selection process.

2.4. Measures and tasks used to assess predictive coding

Oddball or oddball-like paradigms were the most used tasks to study predictive processes. The classical oddball paradigm consists of frequent standard identical stimuli and infrequent deviant stimuli that differ from the standard ones in one or more aspects (e.g., duration, tone, image, location, etc.). Typically, standard stimuli tend to elicit fewer brain responses each time they are repeated, while infrequent, deviant – and therefore “odd” - stimuli are perceived as salient and evoke event-related potentials (ERPs) usually registered by an electroencephalogram (EEG) (Fong et al., 2020; Li et al., 2019; Steiner et al., 2013).

"Oddball-like" refers to paradigms sharing the main feature of the classic oddball paradigm (i.e., a set of frequent standard stimuli and rare deviants), but may introduce additional features, such as multiple deviants or manipulation of the presentation probabilities of deviant outcomes. This violation of expectations (i.e., the presentation of the deviant stimulus after a set of repeated standard stimuli) elicits a specific ERP called mismatch negativity (MMN) (Bishop and Hardiman, 2010; Garrido et al., 2009; Light et al., 2007), which is considered a measure of predictive error or surprise (den Ouden et al., 2012). The presentation of the deviants can be manipulated by the researcher. Tasks can involve any type of perception, although the most common tasks involve visual and/or auditory sensory channels.

Predictive coding tasks can investigate both socio-emotional and non-social components (Corlett et al., 2022). Specifically, they examine how prior beliefs and expectations generated by the subject’s experience through statistical regularities can predict forthcoming events (e.g., the target stimulus, the behavior of other people, the movement of an object). The task could be deterministic or stochastic. In the former case, the probabilities of outcomes are completely determined by known initial conditions and fixed rules, resulting in predictable and consistent results, while in the latter probabilities are not determined by previous outcomes, leading to a completely random and uncertain state. The other tasks used to study predictive abilities included visual illusions (Kaliuzhna et al., 2019), the presentation of a cue followed by a congruent or incongruent stimulus (Roa Romero et al., 2016), movement prediction following an initial movement (Arthur et al., 2021; Limongi et al., 2018; Scheliga et al., 2022), the size comparison of two consecutive shapes (Sapey-Triomphe et al., 2021), the presentation of an image depicting a set of similar stimuli and an odd one (Van de Cruys et al., 2021), perceptual closure task (i.e., recognition of incomplete or partially obscured stimuli) (Gomez-Pilar et al., 2018), additional singleton task (i.e., the subject is presented with same-color shape items and has to detect a target stimulus in a pattern of same-color distractors;

Table 1
Characteristics of the studies evaluating predictive coding in schizophrenia spectrum disorders (SSD).

First Author	Year	Country	Diagnosis	N SSD (% male)	Mean age ± SD	Comorbidities	Mean IQ ± SD (measure)	Medication	N Controls (% male)	Mean age ± SD	Mean IQ ± SD (measure)	Predictive Coding task
Baldeweg	2015	UK	SCZ	49 (57.14 %)	38 ± 12.5	NR	110.2 ± 8.9 (NART)	Atypical anti-psychotic medications Antidepressants Anticonvulsant Mood stabilisers Antiparkinsonian agents' Chlorpromazine	49 (51.02 %)	36.4 ± 11.5	112.4 ± 13.9 (NART)	Oddball-like*
Cassidy	2018	USA	SSD	10 (62.5 %)	30.6 ± 11.6	None	NR	NR	12 (70.6 %)	29.5 ± 8.4	NR	Oddball-like
Coffman et al.	2017	USA	SCZ	26 (57.69 %)	36.2 ± 7.6	NR	106.2 ± 16 (WASI IQ) 41.7 ± 13.2 (BACS)	NR	26 (42.31 %)	33.3 ± 11.3	105.3 ± 9.1 (WASI IQ) 55.1 ± 11.8 (BACS)	Oddball
Donaldson	2020	New York (US)	SCZ, SAD	116 (62.1 %)	47.3 ± 7.9	NR	NR (WRAT3) NR (WMSR)	Antipsychotics	248 (55.6 %)	50.5 ± 8.8	NR	Oddball*
Donaldson et al.	2023	US	established PSD (including SSD, MD with PS, PSD	131 (57.6 %)	46.6 ± 7.9	NR	0.90 (COWAT – Compository score)	NR	170 (52.4 %)	51.4 ± 9	0.87 (COWAT-Compository score)	Oddball
Dzafic	2021	Australia	SCZ	22 (59 %)	36.82 ± 6.9	SAD (8), DD (1), SSD (1)	105.75 ± 7.42 (WAIS-R)	Chlorpromazine equivalents	22 (54.54 %)	34.09 ± 7.96	107.78 ± 5.83 (WAIS-R)	Oddball*
Farkas et al.	2015	Hungary	SCZ	28 (57.14 %)	37.7 ± 8.4	NR	NR	Clorpromazina	27 (55.56 %)	38.2 ± 10.6	NR	Oddball-like
Fogelson	2014	Israel	SCZ	25 (88 %)	33.1 ± 2.1	None	NR	Chlorpromazine equivalent	25 (88 %)	33.7 ± 2.2	NR	Ad hoc
Ford	2014	California (USA)	SCZ (N = 23) and SAD (N = 3)	26 (76.92)	44.51 ± 12.26		NR	20 Second generation antipsychotic6 First generation antipsychotic	22 (68.18 %)	42.82 ± 13.12	NR	Ad hoc
Gomez-Pilar	2018	Spain	SCZ	35 (57.14 %)	32.68 ± 10.37	NR	BACS scale: 15.79 ± 5.31 (Working memory) 42.45 ± 15.42 (Processing speed) 15.57 ± 3.44 (Executive function) 34.76 ± 11.25 (Verbal memory) 47.34 ± 14.69 (Motor speed) 17.44 ± 6.39	Antipsychotic	51 (45.09 %)	29.31 ± 9.74	BACS scale: 20.67 ± 4 (Working memory) 70 ± 14.1 (Processing speed) 17.18 ± 2.63 (Executive function) 51.61 ± 8.57 (Verbal memory) 72.16 ± 14.11 (Motor speed) 27.89 ± 5.77 (Verbal fluently)	Oddball

(*continued on next page*)

Table 1 (*continued*)

First Author	Year	Country	Diagnosis	N SSD (% male)	Mean age ± SD	Comorbidities	Mean IQ ± SD (measure)	Medication	N Controls (% male)	Mean age ± SD	Mean IQ ± SD (measure)	Predictive Coding task
							(Verbal fluently)					
Horga	2014	USA	SCZ or SAD with AVH	10 (70 %)	44.7 ± 5.55		104.22 ± 7.27 (FSIQ−4) 111 ± 7.2 (FSIQ−2)	Ziprasidone Olanzapine RisperidoneRisperidone Aripiprazole Quetiapine	10 (70 %)	37.5 ± 4.46	113.40 ± 13.33 (FSIQ−4) 115 ± 15.09 (FSIQ−2)	Speech decision-making task
Hua et al.	2023	California (USA)	SCZ early SCZ	89 (58.1 %)	23.01 ± 6.35	NR	NR	Chlorpromazine equivalents (88.76 %)	105 (70.71 %)	22.48 ± 4.24	NR	Oddball-like
Kaliuzhna	2019	AustraliaGermanySwitzerland	SCZ	90 (50 %)	33.3 ± 5.6	NR	NR	Chlorpromazine equivalent.	21 (50 %)	33.7 ± 6.4	NR	Visual illusions
Kort	2017	California, USA	SCZ	34 (76.47 %)	34.68 ± 9.79	None	NR	Chlorpromazione equivalents	33 (75.75 %)	34.21 ± 8.99	NR	Speak/listen experimental paradigm*
Leptourgos	2022	Baltimore (USA)	SCZH	18 (64.7 %)	31.3 ± 9.6	None	101.2 ± 28.5 (WTAR)	Antipsychotic (Atypical/Typical). Other psychotropic medication (Antidepressant, Benzodiazepine, Mood Stabilizer	24 (41.66 %)	34.8 ± 10.3	114.2 ± 13.8 (WTAR)	Conditioned hallucination task
Leptourgos	2022	Baltimore (USA)	SCZNH	17 (72.22 %)	35.5 ± 10.7	None	112.4 ± 9.1 (WTAR)	Antipsychotic (Atypical/ Typica) Other psychotropic medication (Antidepressant, Benzodiazepine, Mood Stabilizer)	24 (41.66 %)	34.8 ± 10.3	114.2 ± 13.8 (WTAR)	Conditioned hallucination task
Limongi	2018	Chile	SCZ	15 (60 %)	41.3 ± 8.44	NR	NR	Olanzapine	15 (53.33 %)	41 ± 6.54	NR	Ad hoc
McCleery	2018	USA	SCZH	16 (69 %)	51.1 ± 10.23	NR	NR	NR	20 (50.0 %)	49.58 ± 8.99	NR	Oddball
McCleery	2018	USA	SCZNH	14 (50 %)	47.29 ± 12.7	NR	NR	NR	20 (50.0 %)	49.58 ± 8.99	NR	Oddball
McCleery et al.	2019	USA	SCZ	43 (67.4 %)	47.81 ± 10.04	NR	NR	NR	30 (60 %)	46.4 ± 8.5	NR	Oddball-like
Neuhaus et al.	2013	Germany	SCZ	22 (54.55 %)	40.67 ± 11.3	Nicotine abuse/ dependence.		Chlorpromazine	24 (54.17 %)	37.96 ± 7.3		Oddball
Okruszek	2018	Poland	SCZ	46 (67.39 %)	33.4 ± 7	None	NR	NR	40 (50 %)	30.2 ± 10.7	NR	Ad hoc
Okruszek	2019	Poland	SCZ	39 (64 %)	35.4 ± 9.9	None	NR	Antipsychotic	22 (55 %)	34.5 ± 11.7	NR	Ad hoc
Rentzsch et al.	2015	Germany	SCZ	25 (60 %)	29.72 ± 7.9	NR	112 ± 18.82 (vocabulary test)	Chlorpromazine equivalent	25 (60 %)	29.52 ± 7.9	116.08 ± 16.4 (vocabulary test)	Oddball-like
Romero	2016	Germany	SCZ	17 (70.58 %)	35.24 ± 7.73	None	245.65 ± 43.31 (BACS)	Chlorpromazine equivalent	17 (76.47 %)	36 ± 8.29	273.47 ± 37.37 (BACS)	Congurent-Incongurent Audio Visual Task
Sauer	2017	GermanyIsraelUnited KingdomNetherlands	SCZ	16 (100 %)	37.38 ± 14.9	NR	NR	Antipsychotic	16 (100 %)	36.38 ± 9.38	NR	Oddball

(*continued on next page*)

Table 1 (*continued*)

First Author	Year	Country	Diagnosis	N SSD (% male)	Mean age ± SD	Comorbidities	Mean IQ ± SD (measure)	Medication	N Controls (% male)	Mean age ± SD	Mean IQ ± SD (measure)	Predictive Coding task
Scheliga	2022	Germany	SSD (10 SCZ, 1 SAD, 1 SCZS, 1 PITPS, 1 RSCZ)	17 (64.7 %)	37.35 ± 11.77	Substance abuse (alcohol, cannabis), former manic symptoms, panicsymptoms, obsessive thoughts and actions, or recurrent depressivesymptoms	29.33 ± 6.11 (WSTa) 26.25 ± 10.28 (TMT-A) 58.10 ± 34.73 (TMT-B) 7.65 ± 1.32 (MWT-A) 6.18 ± 1.78 (MWT-B)	Chlorpromazine	23 (69.57 %)	36.22 ± 11.65	32.91 ± 3.84 (WSTa) 21.33 ± 12.81 (TMT-A) 36.96 ± 22.42 (TMT-B) 8.65 ± 1.97 (MWT-A) 8.09 ± 2.07 (MWT-B)	Ad hoc
Schmack	2017	Germany	SCZ	21 (76.19 %)	34.1 ± 6.7	None	105.2 ± 8.8 (WST)	Except one olanzapine	28 (71.43 %)	31.5 ± 7.2	107.4 ± 6.8 (WST)	Motion detection task
Vogel	2018	Germany	SCZ	17 (82.35 %)	31.94 ± 7.5	None	107.24 ± 13.5 (MWTB) 111.18 ± 5.8 (LPS)	NR	18 (83.33 %)	30.67 ± 6.5	112.83 ± 12.9 MWTB 114.28 ± 10.8LPS	Oddball-like
Whitton	2021	Switzerland	SCZ Smoker	34 (82.4 %)	41.65 ± 12.49	None	NR (NART)	Chlorpromazine equivalent, D2 antagonist	129 (37.2 %)	32.02 ± 12.13	NR	Oddball-like*

Legend: AVH: Auditory verbal hallucinations; BACS: Brief Assessment Cognition Schizophrenia; COWAT: Controlled Oral Word Association Test; FSIQ-4: Full-Scale Intelligence Quotient; FSIQ2: full-scale IQ test assessed with two subtests of the Wechsler Abbreviated Scale of Intelligence; LPS: Specific language impairment; MD: mood disorders; MWT: Mehrfachwahl-Wortschatz-Intelligenztest A-B(german version of the multiple-choice vocabulary intelligence test); NART: The National Adult Reading Test; PITPS: psychosis induced through psychoactive substances; PS: psychosis; PSD: psychotic disorder; RSCZ: Residual schizophrenia; SAD: schizo-affective disorder; SCZ: schizophrenia; SCZH: Schizophrenia with hallucinations; SCZNH: Schizophrenia without hallucinations; SCZS: schizophrenia simplex; MT: Trail Making Test; WASI IQ: Wechsler Abbreviated Scales of Intelligence; WAIS-R: Wechsler Adult Intelligence Scale; WMSR: Wechsler Memory Scale; WRAT: Wide Range Achievement Test-3; WST: vocabulary test (Wortschatztest); WTAR: Wechsler Test of Adult Reading.
*This study included also groups of participants with other diagnosis, see Tables 3 and 4.

in addition, a distractor of a different color, called a singleton, may also appear to increase the difficulty) (Allenmark et al., 2021).

Behavioral outcomes can be quantified by measuring reaction times or eye movements and can be conducted in association with neurophysiology and neuroimaging techniques. The most used ones in the articles included in the present review were EEG, magnetoencephalography (MEG), structural and functional magnetic resonance imaging (MRI and fMRI respectively). One study used positron emission tomography (PET).

In EEG, the ERPs studied as components of predictive coding were N100 (N1), N200 (or N2), and P300 (or P3). N1 plays a role in sensory perception such as self-generated speech sound, N2 reflects information processing related to attention (e.g., ignoring deviant stimulus in repetitive stimulus presentation), while P3 reflects the encoding of predictive information: (Ford et al., 2007; Näätänen, 1990; Patel and Azzam, 2005); while P3a is a subcomponent of P3 and reflects the passive maintenance of the working memory trace, P3b reflects the active maintenance of the trace. Usually, P3 is elicited by novelty (e.g., the deviant stimulus in oddball paradigm) (Courchesne et al., 1975; Katayama and Polich, 1998). Another component, examined by one of the included studies, is N400 (Grisoni et al., 2019), which is elicited by a wide range of stimuli (e.g., drawings, photos, words, mathematical symbols) and is thought to play a role in their comprehension. Finally, one study examined P50 as a proxy of sensory gating, that is, the pre-attentive filtering of irrelevant stimuli (Wan et al., 2008). In addition to ERPs, some studies have focused on the theta wave, specifically involved in memory formation and attentional processes determining the individual predictive ability (Sauseng et al., 2010; Schacter, 1977).

2.5. *Appraisal of quality*

The quality of the included studies was assessed independently by two authors using the Newcastle Ottawa quality assessment scale (NOS) for case-control studies (Stang, 2010). The NOS is a widely used tool for assessing the quality of non-randomized studies in systematic reviews. It consists of three main categories: selection of study groups, comparability of groups, and ascertainment of exposure or outcome. Each category includes specific criteria related to study design and methodology. The NOS assigns stars to each study based on these criteria, providing a visual assessment of study quality. Studies with higher star ratings have higher methodological quality and a lower risk of bias. As for study selection, discrepancies were solved after consultation with a third reviewer.

3. Results

3.1. *Characteristics of the included studies*

Our search yielded a total of 4196 records. After removing duplicates, we screened the titles/abstracts of 2807 articles and the full text of 163 articles. Finally, we included 72 articles evaluating predictive coding tasks in the following neuropsychiatric disorders:

1. Schizophrenia spectrum disorders (n = 33 studies): specifically, schizophrenia (SCZ) only (n = 21), SCZ and/or schizoaffective disorder (n = 5), first-episode psychosis (n = 1), unspecified psychosis (n = 6).
2. Neurodevelopmental disorders (n = 33): specifically developmental language disorder (n = 1), attention-deficit/hyperactivity disorder (ADHD; n = 2), autism spectrum disorder (ASD; n = 30).
3. Mood disorders (n = 5): specifically bipolar disorder (BD; n = 3), depression (n = 1), and unspecified (n = 1).
4. Neurocognitive disorders (n = 4): including Alzheimer's disease (n = 1) and Parkinson's disease (n = 3).
5. Post-traumatic stress disorder (PTSD; n = 1).
6. Substance use disorders (n = 1).

Four articles included more than one diagnostic group compared to a single group of healthy controls (HC), specifically: schizophrenia (SCZ) and BD (Whitton et al., 2021); SCZ and unspecified mood disorder (Donaldson et al., 2020); SCZ, Alzheimer's disease, and BD (Baldeweg and Hirsch, 2015); ASD and ADHD (Gonzalez-Gadea et al., 2015). The PRISMA flow-chart of the study selection process is presented in Fig. 1.

Forty-five studies were conducted in Europe or the United Kingdom, 20 in the United States, 3 in Australia, 2 in Asia, and 2 in South America. As per the population age, seven studies were conducted in children (3–12 years old), 6 studies in adolescents (13–19 years old), 56 in adults (20–59 years old), and 2 in elderly people (60 + years old). In addition, one study (Pando-Naude et al., 2024) was conducted with mixed groups of adults and elderly people. Psychiatric comorbidities were reported in 58 studies for participants with neuropsychiatric disorders. In 38 of the included studies, patients were taking a psychopharmacological therapy, in one study medication use was an exclusion criterion, while 33 studies did not report whether participants were medicated.

3.2. *Tasks used for measuring predictive coding*

3.2.1. *Summary of findings*

3.2.1.1. Schizophrenia spectrum disorders. Characteristics of the studies including participants with SSD are presented in Table 1. Individuals with SCZ showed a consistent pattern of impaired non-social predictive coding, resulting in longer response times to unpredictable stimuli, jumping-to-conclusions bias, and greater inflexibility compared to HC. Even though they did not exhibit abnormal formation or use of priors, they presented difficulty in updating prior perceptions and adapting to environmental changes (Bansal et al., 2022; Limongi et al., 2018; Standke et al., 2021).

Despite non-social predictive coding impairments, individuals with SCZ could still leverage communicative gestures to predict the behavior of others, resulting in preserved social predictive coding (Okruszek et al., 2018, 2019).

Clinically, both negative and positive symptoms were found to negatively correlate with flexibility and the ability to maintain the working memory trace (Leptourgos et al., 2022; McCleery et al., 2018). Hallucinations also correlated with a greater perceptual bias toward expected stimuli (Cassidy et al., 2018). Predictive coding impairments in sensory gating and probabilistic reward learning improve in SSD when smoking (Whitton et al., 2021). The administration of ketamine in HC induced impairments similar to those observed in SCZ patients (Kort et al., 2017).

Electrophysiological studies showed a consistent pattern of reduced P3 in response to predictable stimuli (Fogelson et al., 2014), less N1 suppression (Ford et al., 2014), and higher P50 ratio (Whitton et al., 2021) in SCZ compared to HC. Additionally, they showed diminished MMN responses (i.e., reduced event-related EEG potentials) to both local irregularities and global regularities (Sauer et al., 2017). Age at onset of psychosis was positively correlated with MMN impairment; specifically, individuals with greater MMN amplitude impairment tended to experience psychosis at a younger age (Vogel et al., 2018). SCZ also showed reduced theta-band power for predictable stimuli, regardless of their level of predictability (Gomez-Pilar et al., 2018). Reduced MMN in SCZ was restored to near typical levels when an emotional stimulus context was added (Vogel et al., 2018). Prediction errors were also seen in people with clinical high-risk for psychosis and first-episode psychosis, in which MMN deficits remain stable over five years and predict the severity of symptoms (Hauke et al., 2023).

Functional neuroimaging (fMRI, PET) studies showed reduced activation during the prediction task in SSD compared to HC in several areas related to the flexible updating of priors, i.e., the dopaminergic midbrain/thalamus, striatum, and caudate nucleus), hippocampus, amygdala, pregenual anterior cingulate cortex and superior/middle/

Table 2
Characteristics of the studies evaluating predictive coding in autism spectrum disorder (ASD).

First author	Year	Country	N ASD (% male)	Mean age ± SD / range	Comorbidities	Mean IQ ± SD (measure)	Medication	N controls (% male)	Mean age ± SD / range	Mean IQ ± SD (measure)	Predictive coding task
Allenmark	2021	Germany	22 (59.09 %)	30.4 ± 13.5	NR	105.9 ± 10.8 (WST)	NR	22 (59.09 %)	29.7 ± 12.9	105.9 ± 11.7 (WST)	Additional-singleton visual-search task
Arthur	2021	United Kingdom	30 (NR)	21.40 ± 5.09	NR	NR	NR	60 (NR)	21.78 ± 4.14	NR	Ad-hoc
Arthur et al.	2023	United Kingdom	29 (65.51 %)	21.28 ± 3.63	None	NR	NR	29 (34.48 %)	21.31 ± 3.3	NR	Ad-hoc
Brodski-Guerniero	2018	Germany	19 (100 %)	14–27	None	109.6 ± 18.6 (CFT)	Risperidone (2), psychostimulants (3), SSRI (2), Risperidone + Psychostimulant + SSRI (1)	19 (100 %)	14–27	109.4 ± 16.4 (CFT)	Ad-hoc
Gómez	2014	Germany	10 (NR)	30.3 ± 9.6	None	NR	NR	14 (NR)	29.7 ± 6.9	NR	Perceptual closure task
Gonzalez-Gadea	2015	Argentina	24 (95.83 %)	10.38 ± 1.97	None	39.63 ± 9.83 (RPM)	Risperidon (5)	19 (78.94 %)	11.63 ± 2.43	40.16 ± 8.20 (RPM)	Oddball-like*
Goris et al.	2018	Belgium	18 (72.22 %)	Adults	None	> 80 (WAIS-III)	None	24 (66.66 %)	Adults	> 80 (WAIS-III)	Ad-hoc
Goris et al.	2022	Belgium	27 (70.37 %)	35.63 ± 7.54	None	112.07 ± 14.44 (WAIS-III)	NR	27 (70.37 %)	35.63 ± 7.55	118 ± 12.26 (WAIS-III)	Oddball
Greene	2019	United States	25 (88 %)	14.78 ± 1.62	None	101.80 ± 18.02 (KBIT)	None	18 (94.44 %)	14.81 ± 2.08	104.83 ± 14.98 (KBIT)	Oddball-like
Grisoni	2019	Germany	20 (55 %)	38 ± 10.3	None	119.5 ± 8.4 (LPS−3)	NR	22 (36.36 %)	31.9 ± 11.1	116.8 ± 9.5 (LPS−3)	Distraction-oddball
Hudson	2021	United Kingdom	23 (78.26 %)	40.7 ± 8.6	None	106.5 ± 19.1 (WAIS)	NR	23 (69.56 %)	40.1 ± 12.5	112.5 ± 14 (WAIS)	Ad-hoc
Knight et al.	2020	United States	21 (85.71 %)	15	None	100 (Full Scale IQ)	NR	19 (36.84 %)	14	117 (Full Scale IQ)	Oddball-like
Lacroix	2021	France	33 (48.48 %)	32.56 ± 8.23	NR	124 ± 16.1 (WAIS)	NR	35 (42.86 %)	32.21 ± 7.51	114.09 ± 14.67 (WAIS)	Emotional Stroop after priming
Lacroix et al.	2022	France	109 (51.38 %) transgender/ non-binary	31.9 ± 7.6	Yes (comorbidities NR)	NR	NR	200 (34.5 %) transgender/ non-binary	32.6 ± 7	NR	Emotional shifting task, task switching task
Manning et al.	2017	United Kingdom	34 (85.29%)	9.11 ± 2	None	105.44 ± 14.94 (WASI-II)	NR	32 children (68.75%), 19 adults (36.84%)	9.2 ± 1.1 (children), 24.2 ± 3.9 (adults)	104.84 ± 14.11 (WASI-II, children), NR (adults)	Ad-hoc
Pesthy et al.	2023	Hungary, United Kingdom	22 (63.64 %)	27.32 ± 7.32	ADHD (5), OCD (3), GAD (2), BD (1), depression (1), SCZ (1)	NR	NR	20 (90 %)	25.4 ± 6.23	NR	Alternating serial reaction time
Prescott et al.	2022	United States	34 (70.58 %)	3.66 ± 0.33	None	75 ± 20 (MSEL Only	NR	34 (38.23 %)	2.16 ± 0.41	NR	Ad-hoc

(*continued on next page*)

Table 2 (*continued*)

First author	Year	Country	N ASD (% male)	Mean age ± SD / range	Comorbidities	Mean IQ ± SD (measure)	Medication	N controls (% male)	Mean age ± SD / range	Mean IQ ± SD (measure)	Predictive coding task
						Nonverbal Ratio)					
Randeniya et al.	2022	Australia	23 (47.83 %)	24.35 ± 6.08	ADHD (1), depression and anxiety (1)	NR	NR	23 (47.83 %)	24.04 ± 6.06	NR	Oddball
Sapey-Triomphe	2021	United Kingdom	26 (50 %)	27 ± 8.6	Anxiety (2), depression (3), PTDS (1), ADHD (3), OCD (1), agoraphobia (1), dyslexia (1)	> 75 (WAIS)	Yes (medication NR)	31 (51.61 %)	25 ± 4.7	NR	Visual discrimination task
Sapey-Triomphe et al.	2022	Belgium	25 (52 %)	27.2 ± 8.6	7 Yes (comorbidities NR)	> 70 (WAIS IV)	9 medicated	29 (44.82 %)	23.5 ± 3.5	NR	Motion detection task
Sapey-Triomphe	2023	Germany, Belgium	26 (50 %)	32.2 ± 9.5	ADHD (4), dyslexia (2), Tourette's syndrome (1)	112.1 ± 16.5 (WAIS-IV)	NR	26 (50 %)	30.9 ± 8.3	113.9 ± 12.3 (WAIS-IV)	Motion detection task
Seymour	2019	United Kingdom	18 (77.77 %)	16.67 ± 3.2	NR	43.84 ± 7.93 (Raven's Matrices)	NR	18 (83.33 %)	16.89 ± 2.8	48.71 ± 5.78 (Raven's Matrices)	Ad-hoc
Tan et al.	2023	China	22 (90.91 %)	6.73 ± 1.75	NR	96.27 ± 13.48 (PPVT) 108.73 ± 7.43 (CRT)	NR	19 (73.65 %)	7 ± 1.45	NR	Action anticipation task
Tewolde et al.	2018	United Kingdom	30 (16.66 %)	11.16 ± 2.23	None	105.93 ± 16.18 (WASI-II)	NR	30 (46.66 %)	10.48 ± 2.18	107.93 ± 16.03 (WASI-II)	Two exploration tasks
Thillay	2016	France	12 (83.33 %)	21.4 ± 10	NR	101 ± 5 (WAIS)	NR	12 (83.33 %)	21.7 ± 11	NR	Ad-hoc
Van De Cruys	2021	Belgium	24 (66.66 %)	12 ± 1.3	None	106 ± 17.23 (WISC-III)	NR	25 (52 %)	11.85 ± 1.5	107 ± 14.09 (WISC-III)	Visual search task
Van Laarhoven	2019	The Netherlands	30 (73.33 %)	18.55 ± 2.13	Severe comorbidities excluded	103 ± 16.47 (WAIS-IV)	NR	30 (80 %)	18.83 ± 1.32	111.97 ± 11.49 (WAIS-IV)	Ad-hoc
Van Laarhoven et al.	2020	The Netherlands	29 (72.41 %)	18.64 ± 2.11	All receiving clinical treatment (medication NR)	103.03 ± 16.76 (WAIS-IV)	NR	29 (79.31 %)	18.93 ± 1.22	112.07 ± 11.68 (WAIS-IV)	Ad-hoc
Vogel	2022	Germany	24 (62.5 %)	42.46 ± 8.79	None	> 80 (NR)	NR	24 (62.5 %)	42.33 ± 8.83	NR	Temoporal binding task
Von Der Lühe et al.	2016	Germany	16 (75 %)	41.56 ± 9.15	NR	116.88 ± 15.59 (WST)	NR	16 (62.5 %)	36.19 ± 12.11	115.31 ± 8.43 (WST)	Ad-hoc

Legend: ADHD: attention deficit-hyperactivity disorder; BD: bipolar disorder; CFT: Culture Fair Intelligence Test; CRT: the cognitive reflection test; GAD: generalized anxiety disorder; IQ: intelligence quotient; KBIT: Kaufman Brief Intelligence Test; LPS-3: Leistungsprüfsystem; MSEL: Mullen Scales of Early Learning NR: not reported; OCD: obsessive-compulsive disorder; PPVT: Peabody Picture Vocabulary Test; PTSD: post-traumatic stress disorder; RPM: Raven's Progressive Matrices Test; SCZ: schizophrenia; SSRI: selective serotonin reuptake inhibitors; WAIS: Wechsler Adult Intelligence Scale; WASI: Wechsler Abbreviated Scale of Intelligence; WISC: Wechsler Intelligence Scale for Children; WST: Vocabulary test (Wortschatztest).

*This study included also groups of participants with other diagnosis, see Tables 3 and 4.

Table 3
Characteristics of the studies evaluating predictive coding in attention deficit-hyperactivity disorder (ADHD), developmental language disorder (DLD), mood disorders, post-traumatic stress disorder, substance use disorder, and neurocognitive disorders.

First Author	Year	Country	Diagnosis	N Patients (Male%)	Mean age ± SD/ Range	Comorbidities	Mean IQ ± SD (measure)	Medication	N Controls (% male)	Mean age ± SD	Mean IQ ± SD (measure)	Predictive Coding task
Baldeweg	2015	UK	BD	25 (48 %)	38.1 ± 10.3	NR	101.4 ± 9.28 (NART)	Atypical antipsychotics; mood stabilizers; antidepressants; antiparkinsonian agents	49 (51.02 %)	36.4 ± 11.5	112.4 ± 13.9 (NART)	Oddball-like*
Baldeweg	2015	UK	AD	15 (60 %)	71.2 ± 11.9	NR	108 ± 6.8 (NART)	NR	49 (51.02 %)	36.4 ± 11.5	112.4 ± 13.9 (NART)	Oddball-like*
Behroozmand	2018	United States	PD	15 (66.66 %)	67 (61–78)	None	28 ± 2 (MMSE)	Antiparkinsonian agents	15 (53.33 %)	65 ± 62–73	NR	Ad hoc
Donaldson	2020	United States	MD with PSD	75 (44 %)	48.4 ± 9	NR	NR	NR	248 (55.6 %)	50.5 ± 8.8	NR	Oddball*
Donaldson	2020	United States	PSD	25 (76 %)	49 ± 10.9	NR	NR	NR	248 (55.6 %)	50.5 ± 8.8	NR	Oddball*
Dzafic	2021	Australia	No PSD	22 (63.63 %)	36.05 ± 9.55	SD (9), PED (9), DD (17), AD (6)	107.62 ± 8.44 (WAIS-R)	Antipsychotics	22 (54.54 %)	34.09 ± 7.96	107.78 ± 5.83 (WAIS-R)	Oddball*
Eisenberg	2023	United States	PTSD	63 (19.04 %)	34.6 (20–49)	None	NR	NR	63 (17.46 %)	33.9 (18–50)	NR	Ad hoc
Gonzalez-Gadea	2015	Argentina	ADHD	15 (73 %)	11.73 ± 2.43	None	39.7 ± 8.93 (RPM)	Methylphenidate (11)	19 (78.94 %)	11.63 ± 2.43	40.16 ± 8.20 (RPM)	Oddball-like*
Haarsma et al.	2020	United Kingdom	FEP	30 (80 %)	24.8 ± NR	NR	NR	NR	32 (53.13 %)	22.6 ± NR	NR	Oddball-like
Haarsma et al.	2020	United Kingdom	RMS	29 (72.41 %)	21.5 ± NR	NR	NR	NR	32 (53.13 %)	22.6 ± NR	NR	Oddball-like
Harlé	2016	United States	MDI	62 (79 %)	38 ± 10.4	NR	109.1 ± 8.7 (WTAR)	NR	34 (70 %)	36.1 ± 11.1	111.6 ± 9.7 (WTAR)	Stop-Signal Task
Hauke	2023	United States	ESZ	19 (78.94 %)	23.91 ± 6.17	NR	NR	Antipsychotics: atypical only (13), atypical + typical (3); No antipsychotic (2); Unknown (1)	44 (61.36 %)	19.97 ± 5.5	NR	Oddball
Hauke	2023	United States	HP	38 (60.52 %)	17.4 ± 3.5	NR	NR	Antipsychotics: atypical only (10), atypical + typical (1); No antipsychotic (27)	44 (61.36 %)	19.97 ± 5.5	NR	Oddball
Hestvik	2022	United States	DLD	13 (61.53 %)	17.4 ± 3.5	NR	NR	NR	17 (58.82)	10.4 ± 1	NR	Ad hoc
Kort	2017	United States	KP	31 (61.29 %)	27 ± 4.3	None	NR	Antipsychotics	33 (75.75 %)	34.21 ± 8.99	NR	Speak/listen experimental paradigm*
Larsen et al.	2020	Australia	SDD	20 (75 %)	34.3 ± 7.33	NR	101.79 ± 7.21 (WAIS-R)	Antipsychotics	22 (54.54 %)	34.09 ± 7.96	102.29 ± 7.6 (WAIS-R)	Oddball
Larsen et al.	2020	Australia	No PSD	20 (65 %)	36.1 ± 9.32	NR	103.47 ± 9.16 (WAIS-R)	NR	22 (54.54 %)	34.09 ± 7.96	102.29 ± 7.6 (WAIS-R)	Oddball
Ramos-Grille	2018	Spain	PAD, GAD, AG, AD, PED	34 (29.4 %)	49 ± 12	MDD (58.8 %)DS (41.2 %)	NR	BZD (58.8 %); SSRI (50 %); SNRI (29.4 %);NDRI (2.9 %);SARI (8.8 %);NRI (5.9 %);NaSSA(2.9 %); TeCA (8.8 %);TCA (2.9 %);agomelatine (5.9 %); vortioxetine (5.9 %)	34 (29.4 %)	46 ± 13	NR	Social perception task
Richards	2020	Scotland (UK)	ADHD	17 (47.05 %)	34.12 ± 11.12	NR	122.53 ± 7.31 (WASI)	Stimulant medication (9); anti-depressants (5)	30 (63.33)	34.52 ± 11.12	118.38 ± 7.21 (WASI)	Motion detection task

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Table 3 (*continued*)

First Author	Year	Country	Diagnosis	N Patients (Male%)	Mean age ± SD/ Range	Comorbidities	Mean IQ ± SD (measure)	Medication	N Controls (% male)	Mean age ± SD	Mean IQ ± SD (measure)	Predictive Coding task
Siciliano et al.	2023	Italy	BD	18 (33.33 %)	39 ± 7.99	None	27.64 ± 5.92 (RPM)	Antiepileptics (15); antipsychotics (9); lithium (8); anxiolytics (2); antidepressants (1); plypharmacy (11)	24 (37.5 %)	33.67 ± 11.06	30.08 ± 2.02 (RPM)	Sequence test. Faux Pas test
Trempler	2020	Germany	PD	21 (71.43 %)	58.81 ± 9.89	None	NR	Dopaminergic medication	21 (71.43 %)	60.05 ± 10.05	NR	Ad hoc

Legend: AD: Anxiety Disorder; ADHD: Attention-deficit/hyperactivity disorder; AG: Agoraphobia; ASD: Autism Spectrum Disorders; BD: bipolar disorder; BZD: Benzodiazepines; DLD: Developmental Language Disorder; DS: Dysthymia; ESZ: early-illness schizophrenia; FEP: first episode psychosis; GAD: Generalized anxiety Disorder HP: high risk for psychosis; MD: Mood disorder; MDI: methamphetamine dependent disorder; MMSE: Mini-Mental State Examination; NART: The National Adult Reading Test; NaSSA: Noradrenergic and specific serotonergic antidepressants; NDRI: Norepinephrine-Dopamine Reuptak Iinhibitors; NRI: Norepinephrine Reuptake Inhibitor; PAD: panic disorder; PD: Parkinson's disorder; PED: Personality Disorder; PTSD: Post Traumatic Stress Disorder; RMS: at-risk mental state patients; RPM: Raven's Progressive Matrices; SAD: schizoaffective disorder; SCZ: Schizophrenia; SCZH: Schizophrenia with hallucinations; SCZNH: Schizophrenia without hallucinations; SD: Substance use Disorders; KP: Ketamine Study Participants SSD: Schizophrenia spectrum disorder; RPM: Raven's Progressive Matrices Test; SARI: Serotonin Agonist and Reuptake Inhibitors; SNRI: Serotonin and Norepinephrine Reuptake Inhibitors; SSRI: Selective Serotonin Reuptake Inhibitors TCA: Tricyclic antidepressants; TeCA: Tetracyclic antidepressants; WAIS-R: Wechsler Adult Intelligence Scale; WASI: Wechsler Abbreviated Scale of Intelligence; WMSR: Wechsler Memory Scale; WRAT: Wide Range Achievement Test-3; WTAR: Wechsler Test of Adult Reading;
*This study included also groups of participants with other diagnosis, see Tables 1 and 2.

inferior frontal gyri (Standke et al., 2021). As for cognitive stability, that is the ability to focus on a task (Geddert and Egner, 2022), a network consisting of the inferior frontal gyrus pars orbitalis, the posterior long insular gyrus, and the pallidum, putamen, and caudate was significantly more activated during predictive coding tasks in the HC group, but not in the patient group (Standke et al., 2021). SCZ patients also showed reduced functional activity in the temporo-parietal junction and superior frontal gyrus compared to HC. The activity of the latter during predictive coding tasks was negatively correlated with psychotic symptoms (Standke et al., 2021). Compared to HC, patients with SCZ showed stronger functional connectivity between orbitofrontal and visual cortex, and between the right and left superior temporal gyrus. Conversely, reduced connectivity between the left and right hemispheres was observed for the inferior/superior frontal gyrus and primary auditory cortex during prediction tasks. Reduced connectivity between several brain areas was found in tasks involving movements, such as precuneus and medial prefrontal cortex during predictable motion; left frontal pole, superior frontal gyrus, and paracingulate gyrus during random motion; right cerebellum, left superior frontal gyrus, and paracingulate gyrus during arbitrary motion (Dzafic et al., 2021; Scheliga et al., 2022; Schmack et al., 2017).

Finally, structural MRI revealed that SCZ patients showed reduced gray matter in the dorsolateral anterior cingulate cortex (i.e., involved in uncertainty adaptation and decision-making), and striatum (i.e., involved in temporal precision and statistical regularities) (Cassidy et al., 2018).

3.2.2. *Neurodevelopmental disorders*

As detailed in Table 2, individuals with neurodevelopmental disorders showed heterogeneous patterns in predictive processing compared to HC across different developmental levels. Children, adolescents, and adults with ASD exhibited impaired predictive abilities, characterized by reduced gaze visits and delays in responding to target stimuli compared to HC (Allenmark et al., 2021; Greene et al., 2019). However, individuals with ASD showed faster responses to certain stimuli (i.e., detecting the odd stimuli in patterns of similar stimuli) (Van de Cruys et al., 2021) and intact sensorimotor control. In addition, they could learn priors similarly to HC, although they showed less flexibility in adjusting precision in volatile contexts (i.e., an environment that is constantly changing) (Arthur et al., 2021). These findings related to predictive abilities suggest the presence of a more heterogeneous array of performances in ASD compared to HC.

Concerning tasks with social cues, individuals with ASD showed impaired delayed temporal binding for social events (Vogel et al., 2022) but intact social perception, that is the ability to identify and utilize social cues to make judgments about social roles, rules, relationships, context, or other characteristics of other people (Hudson et al., 2021). Specifically, people with ASD were able to make predictions about other people's goals only when the goal was explicit, whereas they showed prediction deficits when the goal was implicit (Hudson et al., 2021). Females outperformed males on interpersonal prediction tasks (Lacroix et al., 2021).

From a clinical point of view, a negative correlation was found between predictive abilities and the severity of a specific cluster of symptoms (i.e., restricted, repetitive, and stereotyped behaviors and interests) (Brodski-Guerniero et al., 2018).

EEG studies reported that individuals with ASD have alterations in anticipatory brain activity, that is the brain's preparation to respond to future events (Grisoni et al., 2019). Compared to HC, individuals with ASD showed reduced P3 peak and reduced bilateral superior frontal cortex activation in response to unexpected events and increased late activation of the left dorsolateral prefrontal cortex in response to expected events (Gonzalez-Gadea et al., 2015). In addition, expected events did not attenuate the N1 (van Laarhoven et al., 2019) and N4 (Grisoni et al., 2019) components (which predict self-initiated sounds and the presence of semantic congruencies respectively) in these individuals. Moreover, individuals with ASD showed reduced feedback connectivity from V4 to V1 (visual cortex), and decreased synchronization between alpha and gamma oscillations in V1 (Seymour et al., 2019). Despite MMN reductions, individuals with ASD exhibited increased contingent negative variation (i.e., slow cortical waves reflecting anticipatory attention and preparation), increased P3 amplitude (i.e., predictive sequence encoding), faster target-P3 (i.e., faster event-related processing), and shorter N2 latency (i.e., faster information processing) for the detection of predictable patterns (Thillay et al., 2016). Moreover, ASD showed no difference in MMN activity when detecting the odd stimulus in a pattern of regular stimuli compared to HC, and their anticipatory brain activity is less elicited by action and semantic sounds and more by pure tones compared to HC (Grisoni et al., 2019). In addition, ASD showed reduced active information storage (i.e., information that is actively maintained in the brain, ready to be used (Wibral et al., 2014; Zipser et al., 1993) in the hippocampus compared to HC (Gómez et al., 2014).

Functional neuroimaging showed that adults with ASD displayed altered predictive activity in different according to the level of abstraction of the predictions. High-level predictions (i.e., more abstract predictions) led to greater activations of the left orbitofrontal cortex, bilateral posterior cingulate cortex, and right posterior temporal sulcus in ASD compared to HC; mid-level predictions (i.e., predictions based on sensory information within a context) were associated with higher activation of the retrosplenial cortex, left putamen, and anterior cingulate cortex in ASD compared to HC; low-level predictions (i.e., sensory-motor predictions) correlated only with the activation of occipital cortex and postcentral gyrus (Sapey-Triomphe et al., 2023). Regardless of signal complexity, adolescents with ASD showed reduced activity in the posterior cingulate cortex, supramarginal gyrus, and precuneus compared to HC during predictive coding tasks (Brodski-Guerniero et al., 2018). Conversely, children with ASD showed reduced activity in the bilateral superior frontal cortex in response to unexpected events, and increased late activation of the dorsolateral prefrontal cortex in response to expected events (Gonzalez-Gadea et al., 2015).

Contrariwise to ASD, individuals with ADHD showed an increased responsivity to task-irrelevant stimuli (Gonzalez-Gadea et al., 2015) but maintained intact statistical learning on decision tasks similar to HC (Richards et al., 2020). ADHD showed reduced attention-related P3 in the superior frontal cortex, coupled with increased late frontal activation to unexpected stimuli (Gonzalez-Gadea et al., 2015). Differently from HC, children with DLD did not generate predictions to fill the gap in sentences (Hestvik et al., 2022). Studies involving participants with ADHD and DLD are presented in Table 3.

3.2.3. *Other disorders*

3.2.3.1. Mood disorders. Individuals with mood disorders showed impaired socio-emotional prediction abilities. More specifically, patients with depression showed increased trial-by-trial prediction errors specifically for negative (i.e., sadness and fear) but not positive (i.e., happiness) evoked emotions, while patients with BD showed impaired theory of mind and sequencing abilities (Ramos-Grille et al., 2022). In contrast, non-social predictive coding appeared preserved in individuals with BD (Baldeweg and Hirsch, 2015). In BD, smoking was associated with impaired sensory gating and reward-based learning performance, which appeared to be alleviated by the use of dopamine D2 receptor antagonists (i.e., i.e., antipsychotic dosage measured as chlorpromazine equivalents (Whitton et al., 2021). Characteristics of the studies including participants with mood disorders are presented in Table 3.

3.2.3.2. Post-traumatic stress disorder (PTSD). As presented in Table 3, patients with PTSD showed impaired predictive ability, especially after listening to their traumatic event (Eisenberg et al., 2023).

3.2.3.3. Substance use disorders. In a recent paper on people with substance use disorder, abstinent methamphetamine-dependent individuals showed impaired inhibitory control and reduced strategic adaptation compared with HC. Activity in orbitofrontal, parietal, and subcortical (caudate and thalamus) areas in response to unpredictable stimuli was also reduced in the clinical population (Harlé et al., 2016). The characteristics of the study are illustrated in Table 3.

3.2.3.4. Neurocognitive disorders. As for neurocognitive disorders, subjects with Alzheimer's disease exhibited similar results to HC in the predictive task (Baldeweg and Hirsch, 2015), whereas people with Parkinson's disease showed several impairments such as slower reaction time in response to predictable stimuli (Behroozmand and Johari, 2019), reduced probabilistic learning, and failure to adapt to high-probability conditions (Trempler et al., 2020). When asked to rate their pleasure when listening to different rhythms, they displayed a flatter response (Pando-Naude et al., 2024). The latter effect strongly correlated with the severity of symptoms (Pando-Naude et al., 2024). In HC, the activity of the right substantia nigra and caudate correlated positively with surprises, whereas this finding was not confirmed in off-medication patients with Parkinson's disease (Trempler et al., 2020). Even the introduction of dopaminergic medications does not restore learning from predictive errors (Trempler et al., 2020). Characteristics of the studies including participants with neurocognitive disorders are presented in Table 3.

3.2.4. *Quality of the included studies*

The overall quality of the included studies was rated as high ($\geq$7 points). Quality appraisal is presented in the Supplementary materials (Table S1).

4. Discussion

Over the last 20 years, there has been a growing interest in the predictive coding framework and its relevance for individuals with neuropsychiatric disorders. To the best of our knowledge, this is the first systematic review to synthesize findings from case-control studies investigating predictive coding paradigms across different diagnostic clusters following the TRANSD recommendation to improve transdiagnostic research in psychiatry (Fusar-Poli, 2019; Fusar-Poli et al., 2019). Seventy-two studies were included that investigated whether brain predictive abilities were different between individuals with and without a neuropsychiatric disorder.

Findings suggest that neuropsychiatric disorders are characterized by general impairments of performance across different tasks based on predictive coding. Some impairments appear disorder-specific, although certain commonalities between different conditions have been found. Hereafter, we will discuss the main findings for each diagnostic category.

4.1. *Schizophrenia spectrum disorders*

Robust evidence supports the hypothesis of abnormal brain predictive coding abilities in SCZ. People with SCZ show abnormal predictive errors during non-social predictive tasks compared to HC, and the magnitude of prediction errors was able to predict the severity of psychotic disorder five years later. Specifically, SCZ shows a neural pattern consisting of reduced theta waves, reduced ERP to target stimuli, and less repetition suppression compared to HC. These alterations are manifested behaviorally through premature responses and impaired flexibility.

Consistent with previous literature, alterations of brain predictive mechanisms (i.e., decreased precision in both prior beliefs and sensory perception) may contribute to the formation of delusions and hallucinations (Damiani et al., 2022, 2024; Sterzer et al., 2018). Specifically, in the early stages of the disorder, individuals with SCZ displayed reduced perceptual prior bias compared to HC, whereas they have strong cognitive priors in the later stages of psychosis. Psychotic-like states, and thus predictive coding impairments can also be induced by administering ketamine to healthy individuals (Damiani et al., 2020).

In contrast to non-social predictive coding, individuals with SCZ show preserved emotional predictive inference. These findings appear to be consistent with previous literature suggesting that emotional context facilitates the formation of predictive errors (Vogel et al., 2015; Watanabe et al., 2013). This facilitation was seen only in emotional contexts. When exposed to uncontextualized emotion (i.e., emotional oddball), SCZ patients showed aberrant results compared to HC (Csukly et al., 2013). Communicative gestures were used by SCZ individuals to anticipate another subject's behavior, showing almost typical performances. The literature supports this tendency, also showing that individuals with SCZ are more likely to perceive ambiguous communication signals as self-referred (White et al., 2016). Finally, it is well-known that people with psychotic disorders have high rates of nicotine dependence (George and Krystal, 2000). Nicotine smoking is associated with improved sensory gating (i.e., pre-attentive filtering of irrelevant stimuli) and probabilistic reward-based learning (i.e., learning by probabilistic reward) in individuals with SCZ and is therefore used to stabilize predictive errors (Whitton et al., 2021).

In fMRI studies, individuals with SCZ show reduced activity in the temporoparietal junction, precuneus, and medial prefrontal cortex during motion perception tasks. In morphometric MRI studies, decreased gray matter in the dorsolateral anterior cingulate cortex and striatum is suggestive of impairment in uncertainty adaptation and decision-making processes (Baiano et al., 2007; McCutcheon et al., 2019; Simpson et al., 2010). The striatum is thought to be a region of broad modulatory effects mediated by cholinergic interneurons, dopamine, and N-methyl-D-aspartate (NMDA) receptors (Chiara et al., 1994). The deficits activity of the striatum and frontal areas may contribute to impulsivity and inflexibility (Dalley et al., 2008; Sakagami and Pan, 2007; Trempler et al., 2017), while reduced activity in the anterior cingulate cortex, hippocampus, and amygdala support their role in generating predictive errors (Guo et al., 2018; Heckers, 2001; Laurens et al., 2003). These alterations contribute to predictive impairments as well as to negative and positive psychotic symptoms (Baaré et al., 1999; Gur et al., 2000; Nakamura et al., 2008; Onitsuka et al., 2004; Sun et al., 2009; Wible, 2012).

4.1.1. *Neurodevelopmental disorders*

Individuals with ASD show some similarities and some differences in predictive processing at all developmental stages compared to HC. They show impaired non-social predictive abilities characterized by reduced neural activity in several areas, including those involved in decision-making, working memory, and sensory perception; this is manifested behaviorally as impaired eye gaze and slower reaction time (Greene et al., 2019; Lacroix et al., 2021). This impairment is not generalizable to all predictive tasks; indeed, it is absent when individuals with ASD respond to certain stimuli. Specifically, when they are asked to detect a target stimulus in a pattern of similar stimuli or the motion of an object, individuals with ASD process the information faster and better (Van de Cruys et al., 2021). There is consensus on the evidence that individuals with ASD have reduced flexibility compared to typically developing individuals, resulting in improved precision (i.e., reliability) at the expense of accuracy (i.e., average closeness to physical reality). The latter finding is consistent with the predictive coding models of ASD (Pellicano and Burr, 2012; Van Boxtel and Lu, 2013), which postulate that individuals with ASD present impairments in high-level sensory perception (i.e., hypo-priors) rather than low-level sensory perception (Kern et al., 2007; Tavassoli et al., 2014).

There is evidence that individuals with ASD utilize the behavior of other people as a social-emotional cue to predict their actions or infer mental states only in the presence of an explicit intention. This may suggest that the preserved interpersonal predictive coding is nuanced in ASD. Impairments in the use of non-explicit prior information may lead to difficulties in anticipating the actions of others based on their goal or movement trajectory (Ganglmayer et al., 2020) or mental states (Palmer et al., 2015). Temporal binding deficits may also contribute to this impairment (Brock et al., 2002).

There is also a sex difference in performance, as females with ASD perform better in interpersonal predictive tasks, suggesting better social and cognitive profile in females with ASD without intellectual disability that potentially may play a role in social camouflaging (Cook et al., 2021; Dean et al., 2017).

Finally, there is a negative correlation between predictable information and the severity of symptoms (i.e., restricted, repetitive, and stereotyped behaviors and interests) in individuals with ASD (Brodski-Guerniero et al., 2018). Furthermore, there is reduced activity in the cerebellum of individuals with ASD compared to HC, highlighting the role of the cerebellum in both predictive inference (Popa and Ebner, 2019; Sokolov et al., 2017) and autism symptoms (Pierce and Courchesne, 2001). Further studies should focus on understanding the weight of each trait severity in predicting coding impairment in order to better understand which trait to focus rehabilitation on.

The scattered findings reported in studies about predictive coding and ASD may be related to a wide variety of factors, such as the differences in tasks, the presence of co-occurring conditions, variations in medication use, potential cultural influences that shape responses and behaviors, as well as the intrinsic heterogeneity of ASD itself, which may manifest uniquely in each individual (Lombardo et al., 2019). All the studies included in the present review involved individuals without intellectual disability, thus excluding a large proportion of the ASD population (La Malfa et al., 2004; Matson and Shoemaker, 2009).

Individuals with ADHD show increased sensitivity to task-irrelevant stimuli, but still have preserved statistical learning abilities similar to neurotypical individuals. Diminished neural responses in children with ADHD are consistent with previous literature reporting impairment in attentional predictive error (Barry et al., 2003; Senderecka et al., 2012) and increased switching costs to unexpected events (Gumenyuk et al., 2005). Differences in results between children and adults are consistent with the fact that there may be remission of symptoms in adults with ADHD (Barkley et al., 2002; Ramtekkar et al., 2010).

Finally, children with DLD reduced linguistic predictive performance compared to HC and reduced predictive error (i.e., represented by pre-emptive eye movements and reduced lateralized MMN), which correlates with vocabulary size (Friederici, 2006; Mani and Huettig, 2012). Indeed, children with DLD struggle to learn patterns in language, especially those related to syntax (i.e., the way words are put together to make sentences) and morphology (i.e., the structure of words), which may lead to an impairment in predicting upcoming parts of sentences, such as different word types (e.g., nouns or verbs), inflectional morphs (e.g., sing, ed), or complex sentence structures (e.g., passive voice) (Jones and Westermann, 2021). Similar results have been found in previous literature in children with dyslexia (Beach et al., 2022; Zhao et al., 2019), supporting the hypothesis that predictive coding may be impaired in various neurodevelopmental disorders. Further research should focus on confirming this hypothesis in order to identify children with a neurodevelopmental disorder early and give them a tailored intervention (Haker et al., 2016).

4.1.2. *Mood disorders*

Studies found that depressed individuals displayed reduced predictive performance during emotional tasks with a significant negative bias. These results are consistent with the notion that individuals with depression present distorted feedback and feedforward predictive mechanisms. Specifically, they tend to neglect or underestimate positive information which disconfirms their negative expectations (i.e., inflexible negative interpretation of events), resulting in negative predictions and biased learning, self-reinforcing a negative feedback loop (Kube et al., 2020; Paulus et al., 2019).

The reduced performance in social predictive tasks shown by individuals with BD is consistent with previous literature on the theory of mind deficits in individuals with BD (Bora et al., 2016; Olivito et al., 2022). Conversely, the non-social prediction ability in BD is similar to HC, consistent with previous literature reporting non-significant differences in cognitive performance between BD and HC (Vöhringer et al., 2013).

The cerebellum was found to play an abnormal role in the pathophysiology of mood disorders (Schutter, 2016; Olivito et al., 2022), psychiatric disorders (Escelsior et al., 2019), and predictive impairments (Friston and Herreros, 2016). Individuals with BD who experienced predominant manic episodes showed more extensive cerebro-cerebellar changes than those who had predominant depressive episodes compared to HC (Argyropoulos et al., 2021). In addition, structural and functional studies have shown increased cerebellar volume (Depping et al., 2018) and decreased synchronization of neural activity (Liu et al., 2010) in patients with major depression compared to HC. Thus, abnormalities in the cerebellum – a common substrate in mood disorders given its role in emotion regulation (Schutter and Van Honk, 2005) – may contribute to impairments in social prediction (Minichino et al., 2014).

Alterations in predictive performance may also be related to nicotine use, whose prevalence is much higher in patients with BD than in the general population (Thomson et al., 2015). This habit is related to impaired sensory gating and reward-based learning in individuals with BD, and it is attenuated by the use of dopamine D2 receptor antagonists (i.e., chlorpromazine) (Whitton et al., 2021).

Finally, mood disorders are typically managed with medications acting on the monoaminergic and glutamatergic systems, both playing a critical role in mood, behavioral, and cognitive regulation, including predictive performance (Gilbert et al., 2022; Goulty et al., 2023; Pal, 2021; Sanacora et al., 2008). Future research should test whether BD and depressed patients have similar or different predictive performance during mood episodes and the euthymic phases and whether their performance changes over time while on medication.

4.1.3. *Neurocognitive disorders*

Individuals with Alzheimer's disease show similar predictive errors to HC. These results seem to contradict previous literature supporting the fact that individuals with Alzheimer's disease have impaired predictive processes (Papadaniil et al., 2016; Tsolaki et al., 2017). This discrepancy may be due to differences in the sample of subjects, their medications, and their level of cognitive impairment. Further research should clarify whether or not there are predictive impairments in individuals with Alzheimer's disease.

On the other hand, individuals with Parkinson's disease showed impaired predictive coding compared to HC (Minks et al., 2014; Pekkonen et al., 1995). In addition, when it was attempted to restore the dopaminergic system – which is crucial for predictive abilities (Galea et al., 2012) – through the use of dopaminergic medications, it was found that the performance did not improve. This observation is in contrast with previous research showing improvements in patients taking medications acting on the dopaminergic system (Tomassini et al., 2019; Wolpe et al., 2015). One possible explanation for this difference may be related to dysfunctions in neurotransmitter systems that interact with dopamine to modulate cognitive flexibility under uncertainty (Aly and Turk-Browne, 2018).

Moreover, at the EEG, MMN duration, but not frequency, is negatively correlated with aging, resulting in impaired predictive errors (Pekkonen et al., 1995). The literature also suggests that abnormal MMN in patients with neurocognitive disorders (i.e., Alzheimer's disease and vascular dementia) may be predictive of the course of the disorder itself (Jiang et al., 2017), and thus may be used for preventive intervention.

4.1.4. *Trauma- and stress-related disorders*

In individuals with PTSD, the severity of symptoms is negatively correlated with the accuracy of their predictions. These findings are consistent with studies suggesting that individuals with PTSD may exhibit altered MMN due to the specific symptoms of the disorder (e.g., hyperarousal, insomnia, and impaired concentration) (Ge et al., 2011; Menning et al., 2008; Morgan and Grillon, 1999). Cognitive theories suggest that PTSD may develop as a result of an extremely stressful trauma that leads to an overly negative appraisal of the trauma itself and biased memory recall (Ehlers and Clark, 2000). When exposed to positive events, healthy individuals typically make the previous negative events irrelevant (Garrett and Sharot, 2017; Sharot, 2011). This mechanism, also known as optimism bias, disappears when subjects perceive a threatening environment (Garrett et al., 2018). Similarly, individuals with PTSD perceive the external environment as threatening; this may result in a negative prediction bias (Kube et al., 2020; Linson and Friston, 2019; Wilkinson et al., 2017).

It is important to underline that traumatic events and early-life adverse experiences may similarly alter predictive coding across different psychiatric conditions. If it is true that individuals with mental disorders are more prone to traumatic experiences (Mauritz et al., 2013), traumas in childhood or adolescence increase the likelihood of developing a psychiatric disorder (Copeland et al., 2018). The predictive coding framework proposed that a shared feature in PTSD-related and schizophrenia-related hallucinations is the overweighting of prior beliefs over sensory stimuli. In both disorders, stress was identified as a common trigger leading the brain to prioritize speed of encoding over accuracy, increasing the chance of formulating precise but inaccurate prior beliefs (de Filippis et al., 2024; Lyndon and Corlett, 2020).

4.1.5. *Substance use and related disorders*

Recently abstinent methamphetamine addicts showed impaired predictive ability and greater inflexibility than HCs. Methamphetamine-dependent subjects show impaired cognitive abilities (i.e., attention, memory, and executive functions) after one month or less of abstinence (Monterosso et al., 2005; Simon et al., 2010). Similar results have been found for addiction to other substances (i.e., cocaine) (Pace-Schott et al., 2008). These impairments should improve over time and show at least a minimal recovery within one year (Medina et al., 2004; Salo et al., 2009). Future studies should measure changes in predictive performance during the drug abstinence period. In addition, they should evaluate predictive abilities in both behavioral addictions (e.g., gambling) and substance addictions (e.g., cocaine or tobacco addiction) during the abuse and withdrawal periods. Indeed, literature has suggested that both substance and behavioral addictions share common craving substrates which may lead to similar cognitive impairments (Kulkarni et al., 2023).

4.2. *Strengths, limitations, and perspectives for future research*

This systematic review provides a comprehensive and transdiagnostic approach to examining predictive coding framework across a range of neuropsychiatric disorders and comparing similarities and differences between them.

Our results should be interpreted in light of some limitations. Additionally, we only selected studies focused on predictive coding as primary framework of reference. Moreover, different studies had a limited sample size and may be underpowered, leading to more statistical biases. Given the heterogeneity of included studies, future research should focus on creating paradigms to standardize prediction across different disorders. In addition, factors potentially affecting the performance during the predictive coding tasks, such as the individual's sex/gender, age, age of onset, childhood traumas and adversities, active pharmacological/psychological treatment, or psychiatric comorbidities, should be considered as potential confounders. For instance, individuals without a psychiatric diagnosis tend to rely more on perceptual priors with increasing age (Chan et al., 2021). Concerning medications, a fascinating but underpowered study on schizophrenia showed lower

Table 4
Summary of the main findings of predictive coding in neuropsychiatric disorders.

Diagnostic category	Findings
Schizophrenia spectrum disorders	• Consistent impairments in non-social predictive coding • Generalized impairments in social predictive coding that are more similar to those of healthy controls when emotional contexts are involved • Association of predicting coding impairments with symptom severity
Neurodevelopmental disorders	• ASD: impaired non-social predictive abilities characterized by stronger cognitive priors and reduced flexibility; specific deficits in social cue processing that improve to near-typical levels when intentions are explicitly stated • ADHD: increased sensitivity to irrelevant stimuli, but preserved statistical learning abilities, with symptoms often decreasing over development • DLD: difficulty in predicting sentence gaps, reflecting broader linguistic prediction impairments
Mood disorders	• Consistent impairments in social, but not non-social, predictive coding
Neurocognitive disorders	• Alzheimer's disease: mixed evidence, possibly due to sample and methodological heterogeneity • Parkinson's disease: consistent predictive impairments
Post-traumatic stress disorder	• Negative predictive bias
Substance use disorder	• Impaired predictive performance during early abstinence

Legend: ASD: autism spectrum disorder; ADHD: attention deficit-hyperactivity disorder; DLD: developmental language disorder

prediction errors in patients receiving higher antipsychotic dosages (Horga et al., 2014). The lack of literature addressing confounders calls for studies specifically examining their role in the relationship between brain activity and predictive coding abilities.

Of note, there is currently a dearth of research on predictive coding abilities in patients with certain common mental disorders (e.g., obsessive-compulsive disorder, anxiety disorders, feeding and eating disorders). Future studies should also include these psychiatric conditions.

5. Conclusions

This systematic review has shown that the brain predictive coding activity may be impaired in neuropsychiatric disorders (see Table 4 for a summary of the main findings). However, current literature provides a broad and complete picture of predictive coding in SCZ and ASD only. People within the schizophrenia spectrum show a consistent pattern of impaired non-social predictive coding, while predictive coding deficits are more selective for social cues in individuals on the autism spectrum.

Although substantially more evidence on other diagnostic categories is needed for a more accurate inference, our findings should prompt further attention to the computational modelling of cognition in neuropsychiatric conditions using clinical, electrophysiological, and neuroimaging techniques.

The predictive coding framework offers a robust heuristic to interpret the brain's ability to detect and correct discrepancies between expected and actual sensory input, fine-tuning pre-existing knowledge and expectations used to interpret new information, and improving the accuracy and reliability of sensory information processing. Given the neurobiological basis of predictive coding impairments, further in-depth studies within the mental health domain call for the implementation of the predictive coding framework on different neuropsychiatric conditions and considering potential confounders (such as medications in use and sex/gender) to tailor personalized treatment strategies. These strategies may be crucial to develop cognitive-enhancing rehabilitation and pharmacological approaches.

Declaration of Competing Interest

All authors declare they have no conflicts of interest.

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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.2025.106020.

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