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Neurophysiological and Neuroimaging Phenotypes in Schizophrenia Spectrum Disorders

Systematic review and meta-analysis of the visual mismatch negativity in schizophrenia

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Abstract

Mismatch negativity (MMN) is an event-related potential component automatically elicited by events that violate predictions based on prior events. To elicit this component, researchers use stimulus repetition to induce predictions, and the MMN is obtained by subtracting the brain response to rare or unpredicted stimuli from that of frequent stimuli. Under the Predictive Processing framework, one increasingly popular interpretation of the mismatch response postulates that MMN represents a prediction error. In this context, the reduced MMN amplitude to auditory stimuli has been considered a potential biomarker of Schizophrenia, representing a reduced prediction error and the inability to update the mental model of the world based on the sensory signals. It is unclear, however, whether this amplitude reduction is specific for auditory events or if the visual MMN reveals a similar pattern in schizophrenia spectrum disorder. This review and meta-analysis aimed to summarise the available literature on the vMMN in schizophrenia. A systematic literature search resulted in 10 eligible studies that resulted in a combined effect size of $g = -.63$, CI [−.86, −.41], reflecting lower vMMN amplitudes in patients. These results are in line with the findings in the auditory domain. This component offers certain advantages, such as less susceptibility to overlap with components generated by attentional demands. Future studies should use vMMN to explore abnormalities in the Predictive Processing framework in different stages and groups of the SSD and increase the knowledge in the search for biomarkers in schizophrenia.

KEYWORDS
EEG, meta-analysis, MMN, schizophrenia, visual

Abbreviations: aMMN, auditory mismatch negativity; CPZ, Chlorpromazine; EEG, electroencephalography; MMN, mismatch negativity; PANSS, Positive and Negative Syndrome Scale; PPF, Predictive Processing framework; SPQ, Schizotypal Personality Questionnaire; SSD, schizophrenia spectrum disorders; vMMN, visual mismatch negativity.

1 | INTRODUCTION

Schizophrenia spectrum disorders (SSD) are a complex group of mental disorders defined by the manifestation of positive and negative symptoms, such as hallucinations or diminished emotional expression (DSM–5; American Psychiatric Association, 2013). In light of the challenging task of determining treatment effectiveness and predicting long-term outcomes, recent research has prioritised investigating the heterogeneity and complexity of symptoms in SSD (Baldwin et al., 2022; Chand et al., 2020; Green et al., 2020). However, the pathophysiological mechanisms of SSD remain largely unknown, and diagnosis is symptom constellation only.

The Predictive Processing framework (PPF) has appeared as an innovative approach to conceptualising the heterogeneity of symptoms in SSD (Corlett et al., 2009; Fletcher & Frith, 2009; McCleery et al., 2018). The PPF considers the brain as an inference engine that works according to Bayesian probability updating (Aitchison & Lengyel, 2017; Friston, 2010). Based on prior experiences, the brain actively develops an explanatory model of the world that is used to anticipate sensory inputs rather than react to them as a mere feature-detecting system (Lee et al., 2021). If the model is accurate, it correctly predicts the input signal; if not, a prediction error occurs (Clark, 2013; Friston, 2005, 2010). To improve the explanatory model, the goal is to minimise the prediction error by updating the prediction based on the new information (Friston, 2009, 2010). This mechanism is weighted by precision and reliability, so when it receives unreliable or noisy input, more weight is put on predictions, allowing for quicker interpretation of noisy stimuli (Yon et al., 2020). This framework fits with the brain's anatomy and physiology: as predictions propagate from higher-order areas, they modulate the firing of neurons in anticipation of sensory inputs, generating a hierarchical model of top-down and bottom-up information processing (Barrett & Simmons, 2015; Friston, 2005; Kok & de Lange, 2014).

The PPF suggests that a reduced weight on sensory information and overreliance on one's own prediction may explain the positive symptoms of SSD, such as delusions, hallucinations and false beliefs (McCleery et al., 2018). Within the schizophrenia spectrum, schizotypy, comprising a set of personality traits or symptoms, shares similarities with schizophrenia but is observable in the general population (Andersen, 2022). The assumption is that positive manifestations of SSD, including the positive schizotypy, can be explained in clinical or subclinical populations by over-precise, top-down priors that are not updated when confronted with information that should be trusted. This input is proposed to be perceived as noise, leading to a reduction in prediction error and a failure to update the predictive model. (Andersen, 2022; Fletcher & Frith, 2009; McCleery et al., 2018; van Schalkwyk et al., 2017; Wacongne, 2016).

In this context, the dependence of mismatch negativity (MMN) on the individuals' expectations and stimuli probabilities suggests the amplitude of this component could represent a prediction error in the PPF (Wacongne, 2016). This component is elicited mainly by oddball tasks that include an unpredicted or deviant visual or auditory stimulus and a standard or frequent one. Thus, this component accurately represents expectation violation and computations between expected and unexpected stimuli (Chennu et al., 2013; Male & O'Shea, 2023). MMN occurs at an early phase of stimuli perception between 100 and 250 ms after the onset of the rare stimulus in the frontal regions for auditory stimuli (Näätänen et al., 2007) and parieto-occipital for visual stimuli (Kimura et al., 2010; Stefanics et al., 2014). There is evidence that visual mismatch negativity (vMMN) can also be elicited by unexpected changes, similar to auditory mismatch negativity (aMMN), with simple variations such as orientation (Kimura et al., 2010), motion (Kremláček et al., 2006) or colour (Czigler et al., 2004) or more complex stimuli (Astikainen & Hietanen, 2009; Male & O'Shea, 2023).

Reduced aMMN amplitude is a robust and frequently replicated finding in SSD (Avissar et al., 2018; Erickson et al., 2016; Umbricht & Krljes, 2005). Meta-analyses on the aMMN in SSD show that the effect size of amplitude reduction is not affected by moderators such as age, sex ratio or duration of illness (Erickson et al., 2016; Umbricht & Krljes, 2005). Although a direct relation between amplitude reduction and duration of illness was not found in a review by Erickson and colleagues (2016), the authors described a higher degree of aMMN amplitude reduction in chronic schizophrenia compared with groups with first-episode psychosis, suggesting that the progressive deterioration observed in patients does not follow a linear trajectory (Erickson et al., 2016; Haigh et al., 2017). Experimental variability has also been amply studied. For example, Avissar et al. (2018) considered the use of simple deviants, stimuli that differ in basic properties such as duration, pitch and intensity, versus more complex deviants, such as environmental sounds or omission paradigms, and found that both paradigms elicited smaller amplitudes in schizophrenia, although a larger effect was found for simple stimuli. Other relevant experimental considerations are the deviant type—stimuli differing in duration reflected lower amplitudes than frequency deviants—and attentional demands—unattended tones revealed larger amplitude reductions than attended tones—which also seem to modulate effect

sizes between patients and control groups (Erickson et al., 2016; Umbricht & Krljes, 2005).

Considering that visual abnormalities have been documented in schizophrenia, such as visual hallucinations (Waters et al., 2014) or deficits in recognition of faces and facial expressions (Butler et al., 2012; Yoon et al., 2013), one can assume that the vMMN may also be disrupted, similarly to the aMMN (Avissar et al., 2018; Erickson et al., 2016; Umbricht & Krljes, 2005). At a cognitive level, and similarly to its auditory counterpart, vMMN may serve as a potential indicator for evaluating sensory memory and pre-attentive visual information processing (Maekawa et al., 2011).

In alignment with the PPF, the vMMN would also be considered an index of prediction error and, in parallel with the auditory domain, reveal an amplitude reduction in the SSD. Only two reviews have explored the literature regarding the vMMN in schizophrenia. Maekawa et al. (2011) found and described the results of three studies, whereas Kremlacek et al. (2016) included only one more. In this last meta-analysis, the authors reported reduced MMN amplitudes in SSD with a large Cohen's *d* effect size of .86.

In this sense, considering the lack of reviews in the vMMN exclusively in SSD as opposed to its counterpart in the auditory processing, this meta-analysis aims to expand the knowledge of mismatch alterations in schizophrenia in the visual domain. The most recent review by Kremlacek et al. (2016) concludes that vMMN in SSD points to impaired predictive processes in vision and considers that vMMN should be explored as a research tool or potential biomarker. In light of what has been described in the literature with aMMN in schizophrenia and the most recent review in vMMN (Kremláček et al., 2016), we expect that significantly reduced vMMN amplitudes will be found in SSD patients compared with healthy controls.

2 | METHOD

2.1 | Search strategy and inclusion criteria

The study protocol was publicly registered in PROSPERO (CRD42023412672) on 10 April 2023. Records were identified by systematically searching the PubMed (Medline) and Web of Science Core Collection databases using the search expression ('Schizophrenia' OR 'psychosis' OR 'psychotic disorder') AND ('Mismatch Negativity' OR 'MMN'). The search was conducted on 15 March 2023. Additional records were also retrieved by scanning the reference lists of literature reviews and included studies.

Records were included if they met the following criteria: (1) empirical studies with quantitative data written in English; (2) inclusion of at least one SSD (schizotypal disorder, brief psychotic disorder, delusional disorder, schizophreniform disorder, schizoaffective disorder, schizophrenia, psychotic disorder or first episode psychosis) or high schizotypy and one control group without a diagnosis of a psychiatric disorder or with low schizotypy traits; (3) inclusion of an electroencephalography recording during a visual task that reported amplitudes or latencies of the vMMN waveform in both groups or correlation analysis for schizotypy traits. Authors were asked to provide this information upon request whenever this information was lacking in the main text.

2.2 | Study selection and data coding

The total set of records was loaded to Rayyan (Ouzzani et al., 2016) to check for and remove duplicates. Three members of the research team (PM, FC and JD) were involved in determining eligibility criteria and approved search terms. PM screened the non-duplicate records by title and abstract to remove studies that were clearly out of topic. FC and JD screened the full texts of all retained studies to determine their eligibility for this review. Conflicts between researchers were solved by consensus in a meeting with the research team. The agreement between researchers and the number of records at each step, including the number and reasons for exclusions, are reported in the results section.

For data extraction, a spreadsheet was developed to extract the required information from the included studies, namely, (1) study information—title, authors and publication year; (2) variables related to the sample—sample size, the percentage of females, age mean and standard deviation, diagnostic criteria, mean years since diagnostic, age of onset, Chlorpromazine (CPZ) equivalent dosage mean, Positive and Negative Syndrome Scale (PANSS) positive, negative and total score; (3) MMN component amplitude and latency means and standard deviations, or *t* test and *p* value between groups, or correlation values, or *p* value and direction of the effect; (4) conditions related to the task—task name, task type (active or passive), stimuli type (e.g., face and gratings), deviant type (e.g., emotion and orientation), the process involved (affective—e.g., emotion recognition task; or cognitive—e.g., motion discrimination).

For effect size calculation in between-group analyses, means and standard deviation for each group were coded whenever possible. Alternatively, Cohen's *d*, *t* and *p* values were considered if these statistics permitted estimating the effect size of interest. For correlational

analyses, only zero-order Pearson correlations were retrieved. Effect sizes were computed as Hedges' *g* (Hedges, 1981) and in a way that a negative effect represents reduced amplitude in schizophrenia/high Schizotypy. In anterior and central derivations, when results clearly showed reversed polarities of the vMMN (likely the opposite end of the dipolar vMMN sources (e.g., Farkas et al., 2015), we treated increased positivities as an increased mismatch response, and as such, we considered a higher positive value in the control group as a negative effect. Recent reports have suggested the occipital cortex as the neural source of vMMN (Susac et al., 2014); however, some studies have also suggested the possibility of frontal generators (Kimura et al., 2010; Stefanics & Czigler, 2012). Central positivities have also been reported in studies with faces (Csukly et al., 2013; Stefanics et al., 2012).

Missing information for effect size calculation and other queries regarding included studies (e.g., unclear information and missing demographic information) were requested by e-mail to the authors. Studies were excluded if no information was available for effect size calculation, and the authors did not reply to the request. A total of 6 authors were contacted for missing information for one or more records. Only one author answered back with the requested information.

2.3 | Analytical strategy and meta-analytic methods

Data analysis was conducted using the Comprehensive Meta-Analysis software version 4 (Borenstein et al., 2022). The effect sizes for vMNN amplitude and latency were estimated as Hedges' *g* (Hedges, 1981), as this metric provides a correction factor that reduces the effect size overestimation bias (which may be especially problematic in studies with small samples). The software computed a composite score for each study using the mean of outcome/data entry to combine different data entries for each study.

Random effect models were used for the amplitude and latency meta-analysis. Heterogeneity of effect sizes and publication bias were also evaluated based on overall effect sizes for each analysis. The variability between studies, that is, the differences in effect sizes that are caused by factors other than chance (sampling error), were tested on overall effect sizes using the *Q* statistic (Cochran, 1954) and described via the I^2 statistic (Higgins et al., 2011). Heterogeneity was classified into high, medium or low, represented as $I^2 \geq 50\%$, $50\% > I^2 \geq 25\%$ or $25\% > I^2$. Subgroup analyses were used for categorical moderators, namely, (a) task type (active or passive), (b) stimuli type (face or non-face) and (c) electrode cluster (anterior, central or posterior). Meta-regressions were conducted for the continuous variables such as the percentage of females, age, PANSS total, positive and negative scores, CPZ equivalent dosage and mean years since diagnosis. Publication bias was assessed using Eggers's test of intercept bias (Egger et al., 1997). Hedges' *g* values, 95% confidence intervals and *p* values were reported for each analysis. Forest plots were created to provide a graphical overview of the data.

Although originally not planned in the protocol, after examination of the included studies, we also found it important to report individual correlations and analysis performed in some studies for vMMN and self-report scores that assessed severity and clinical deficits, as these variables have also been explored in aMMN (Erickson et al., 2016; Umbricht & Krljes, 2005). We only performed moderation analysis for self-reports that included at least 3 studies, which was exclusive for PANSS scores. However, we included a qualitative description of other self-report findings in the severity and self-report scores section.

3 | RESULTS

3.1 | Search results

A detailed flow diagram of study selection was developed using the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Figure 1, Page et al., 2021). A total of 1316 records were retrieved after the electronic search, and 507 were identified as duplicates and removed. All 809 were screened by title and abstract, 81 were removed as out of topic and 82 were identified as reviews and removed to check for secondary references. The remaining records were full text screened for eligibility ($n = 646$). Eleven studies met the eligibility criteria and were included for data extraction. A manual search was performed to account for possible missing studies. This search was conducted through the reference list of included studies, the Google Scholar database and the publications of the included authors mentioned in our review. Additionally, we specifically searched for studies citing Urban et al. (2008), the first identified study that included vMMN in schizophrenia. This manual search resulted in the addition of two records. Thus, a total of 13 records were included in the review. We used Cohen's kappa to compare the agreement between the researchers regarding the decision to include or exclude the eligible studies, revealing a substantial agreement ($\kappa = .78$). The main characteristics of each report included in this review and meta-analysis

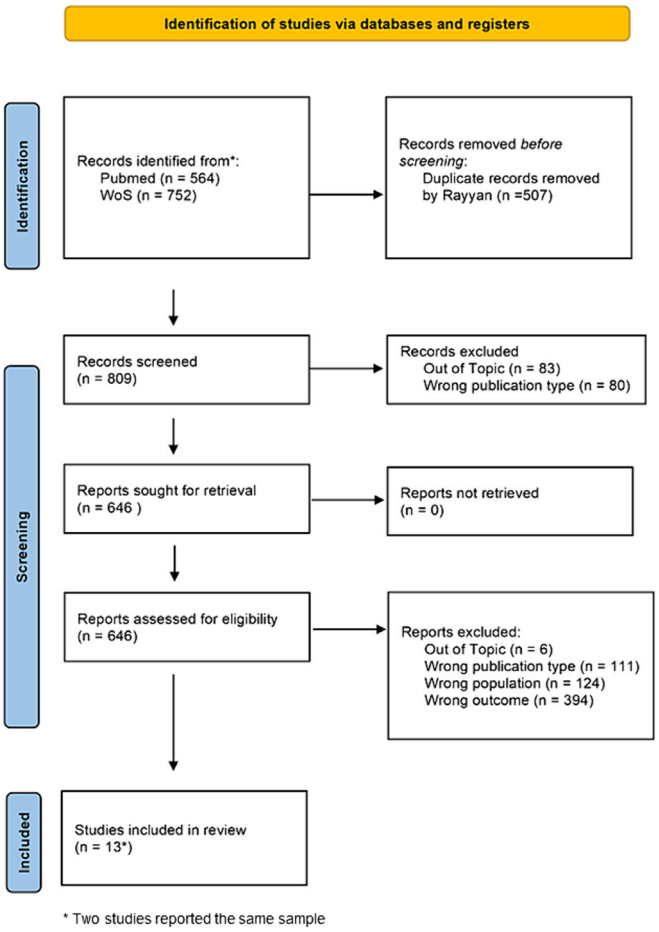

FIGURE 1 PRISMA 2020 flow diagram for new systematic reviews.

can be found in Table 1. From the 13 included reports, only 10 were incorporated in the overall amplitude analysis. One study (Maekawa et al., 2009) reported only latencies and was included only in the latency meta-analysis. Another study was a poster abstract (Libiger et al., 2010) that reported the same sample as Urban et al. (2008). Finally, only one study (Ford et al., 2022) explored the schizotypal traits in non-clinical populations. Unfortunately, this study reported only Spearman correlations and could not be included in the amplitude analysis. Nonetheless, we decided to include this report qualitatively in the severity subtopic, considering the importance of its analysis.

3.2 | Meta-analysis on vMMN amplitude

The analysis comprised 10 studies and a total of 202 patients and 205 controls. Overall, a medium to large effect size effect was found ($g = -.63$, 95% CI [−.86, −.41], $p < .001$, Figure 2), revealing reduced vMMN amplitudes for the SSD group. The heterogeneity test identified a negligible variance across studies ($Q(9) = 11.08$, $p = .271$, $I^2 = 19\%$). There was no evidence of publication bias in this analysis ($b = -1.44$, $p = .588$).

3.2.1 | Effect size by design and task

Moderation analysis by region revealed that amplitudes in the SSD group were found to be smaller only in the posterior regions ($k = 10$, $g = -.54$, 95% CI [−.94, −.16], $p = .014$). In contrast, no difference was found in either the anterior ($k = 3$, $g = -.43$, 95% CI [−1.06, .21], $p = .189$) or central electrode clusters ($k = 3$, $g = -.91$, 95% CI [−1.86, .05], $p = .062$, Figure 3). Moderation analysis by stimuli (faces vs. non-faces) also revealed a qualitative difference in effect size, with a larger effect for non-faces ($k = 6$, $g = -.83$, 95% CI [−1.09, −.57], $p < .001$) than for faces ($k = 5$, $g = -.45$, 95% CI [−.83, −.08], $p = .018$, Figure S2).

The remaining categorical moderation analyses (task type, emotion) were also significant ($p < .004$) but revealed very similar medium effect sizes across moderators (Table 2). Forest plots for other categorical moderation analyses can be found in Figures S1–S3).

3.2.2 | Effect size by sample characteristics

We performed a meta-regression for age ($k = 10$), the proportion of females ($k = 8$), mean years since diagnosis ($k = 7$) and CPZ equivalent mean dosage ($k = 5$). Whereas there appeared to be a qualitatively positive relationship between CPZ equivalent mean dosage and effect size, this analysis was not significant ($p = .115$, Figure S4). None of the other regressions revealed statistically significant results ($p > .171$, Table 3).

3.2.3 | Effect size by severity and self-report scores

We performed meta-regressions for mean PANSS positive, negative and total scores, which were not significant ($p > .171$, Table 3, Figures S6 and S7).

Other symptom severity assessment scores were used by different authors. For example, two reports used the Schedule for Deficit Syndrome (SDS) and found that only the patient group with a severity rating greater than one revealed amplitude differences compared with the control group (Libiger et al., 2010; Urban et al., 2008). Neuhaus et al. (2013) used the Brief Psychiatric Rating Scale (BPRS) and Global Assessment of Functioning (GAF)

TABLE 1 Characteristics of the included studies.

	Healthy controls		Schizophrenia								
Study	*n*	% fem	Age (SD)	Group	*n*	% fem	Age (SD)	Duration of illness (years)	Time window (ms)	Stimuli type	Deviant type
Urban et al. (2005)	10	--	--	First-ep. SCZ	11	--	--	7.1	145–260 120–400	Gratings	Motion direction
Urban et al. (2008)	24	21	27.9 (9.3)	Chronic SCZ	24	21	27.9 (9.3)	--	100–200 100–205	Gratings	Motion direction
Maekawa et al. (2009)	12	--	--	SCZ	12	--	--	--	--	Circular windmill patterns	Number of vanes
Wang et al. (2010)	13	54	41 (8)	Chronic SCZ	13	46	43 (8)	7.1	350–525	Gratings	Monitor side
Csukly et al. (2013)	24	46	33.2 (9.8)	Chronic SCZ	24	46	34.2 (10.3)	16.5	170–220	Faces	Fearful face
Neuhaus et al. (2013)	24	46	38.0 (7.3)	Chronic SCZ	22	46	40.7 (11.3)	11.7	90–200	Letters	X or O
Farkas et al. (2015)	28	44	37.7 (8.4)	Chronic SCZ	27	43	38.2 (10.6)	8.7	100–450	Gabor patches	Orientation
She et al. (2017)	23	48	32.6 (11.3)	Chronic SCZ	23	48	32.3 (11.1)	--	150–450	Schematic faces	Happy face
Yin et al. (2018)	25	52	30.9 (13.2)	Chronic SCZ	25	52	31.3 (12.2)	--	150–250	Schematic faces	Emotional face
Vogel et al. (2018)	18	17	30.7 (6.5)	Chronic SCZ	17	18	31.9 (7.5)	--	150–400	Faces and sequences	Fearful face
Priyesh et al. (2021)	16	50	28.4 (5.5)	Chronic SCZ	16	50	30.2 (5.5)	--	100–200	Faces	Fearful face
Ford et al. (2022)	--	52	24.9	High traits	61	--	--	--	150–300	Faces	Emotional face

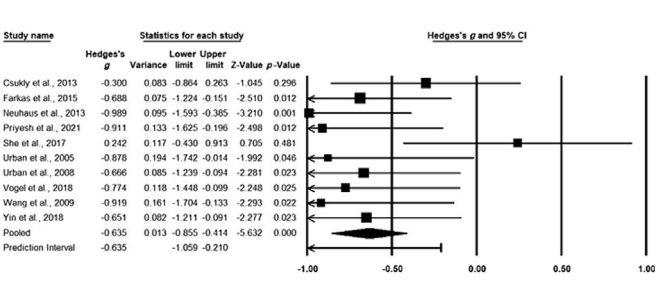

FIGURE 2 Forest plot for overall meta-analysis of visual mismatch negativity (vMMN) amplitude. Each study is identified by the study name, and Hedges' *g* values, 95% confidence intervals and *p* values are reported, as well as the overall effect. Negative effect sizes indicate reduced amplitudes in the schizophrenia spectrum disorder (SSD) group.

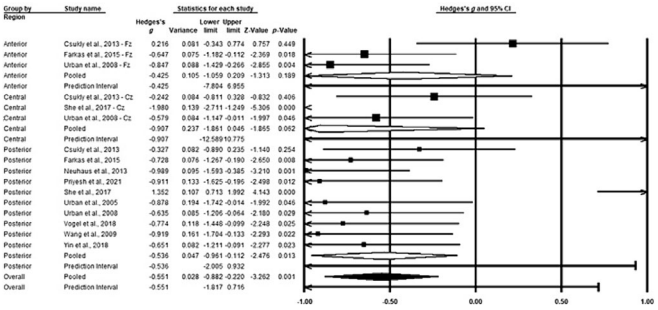

FIGURE 3 Forest plot for meta-analysis of Event-related potential (ERP) component amplitude grouped by anterior, central and posterior electrode clusters. Each study is identified by study name, and Hedges' *g* values, 95% confidence intervals, *p* values and the overall effect for each subgroup are reported.

TABLE 2 Moderator analysis statistics (Hedges' *g*, confidence interval—95% and *p* value).

		Heterogeneity statistics					Confidence interval—95%		
	Moderator	***Q***	***p* value**	***k***	**Category**	**Hedges' *g***	**Lower limit**	**Upper limit**	***p* value**
Amplitude	Stimuli	11.97	.287	5	Faces	−.45	−.83	−.08	.018
				6	Non-faces	−.83	−1.09	−.57	.000
	Task type	11.08	.271	7	Active	−.58	−.89	−.26	.001
				3	Passive	−.75	−1.24	−.38	.000
	Emotion	7.32	.396	4	Fear	−.59	−.99	−.19	.004
				4	Happy	−.48	−.79	−.16	.003
	Region	**6.31**	**.000**	3	Anterior	−.43	−1.06	.21	.189
				3	Central	−.91	−1.86	.05	.062
				10	**Posterior**	**−.54**	**−.94**	**−.16**	**.014**
Overall		**11.08**	**.271**	**10**		**−.63**	**−.86**	**−.41**	**.000**

TABLE 3 Meta-regression analysis statistics (coefficient, confidence interval—95% and *p* value).

			Confidence interval—95%		
Covariate	***k***	**Coefficient**	**Lower limit**	**Upper limit**	***p* value**
Age	9	−.01	−.06	.05	.880
Percentage of females	8	.01	−.02	.03	.607
Mean years since diagnosis	6	−.05	−.16	.06	.384
CPZ equivalent mean	6	.00	−.00	.00	.322
PANSS total	5	.00	−.02	.02	.903
PANSS positive	4	−.19	−.46	.08	.162
PANSS negative	4	−.06	−.17	.05	.258

Abbreviations: CPS, Chlorpromazine; PANSS, Positive and Negative Syndrome Scale.

scores and found no significant correlations with the MMN amplitudes.

Finally, only one study reported amplitudes of MMN in a community sample with varying schizotypal traits. This report was not included in the meta-analysis, considering it only reported Spearman rank correlations. Ford et al. (2022) used the Schizotypal Personality Questionnaire (SPQ) total and subscales scores to analyse correlations with the MMN amplitudes. This study reported a negative association between SPQ total and Interpersonal Features subscale scores, which goes in the opposite direction of what has been described in clinical Schizophrenia, but this effect lost significance after correction for false discovery rate.

3.3 | Meta-analysis on vMMN latency

This analysis comprised four studies. Overall, we found no statistically significant differences in MMN latencies between controls and patients ($g = -.410$, 95% CI [−.921, .101], $p = .116$). The heterogeneity test indicated no significant variance across studies, despite the moderate I^2 value observed ($Q(3) = 6.815$, $p = .078$, $I^2 = 56\%$). There was no evidence of publication bias in this analysis ($b = 1.80$, $p = .162$).

4 | DISCUSSION

In light of what has been described in the literature for the aMMN in schizophrenia (Avissar et al., 2018; Erickson et al., 2016; Umbricht & Krljes, 2005) and in the available reviews on the vMMN (Kremláček et al., 2016; Maekawa et al., 2011), we expected to find decreased vMMN amplitudes in SSD patients (vs. healthy controls). Overall, this meta-analysis, including 10 studies, revealed reduced vMMN amplitudes in the SSD and, thus, possibly reflects cognitive dysfunction, such as abnormalities in the automatic detection of unpredicted changes in the visual environment (Stefanics et al., 2014). This result is consistent with what was reported by Kremláček et al.

in 2016 ($d = .86$) with four studies, although with a more moderate effect size ($g = .63$). Reduced amplitudes seem to be present in patients with schizophrenia compared with controls in experiments with visual and auditory stimuli. However, findings with aMMN have been much more present in the literature, and meta-analyses revealed larger effects of $g = .78$ (Avissar et al., 2018), $g = .95$ (Erickson et al., 2016) and $d = .99$ (Umbricht & Krljes, 2005). Our main results are closer to the effect for complex sensory deviants found by Avissar and colleagues in 2018 ($g = .59$), who also included complex abstract/pattern deviants (e.g., environmental sounds) (Fisher et al., 2014), abstract violations (e.g., descending frequency tones in ascending frequency sequences) (Gjini et al., 2010) or different vowels (Kasai et al., 2002) rather than the simple physical deviants (e.g., pure tone pitch deviant) (Javitt et al., 2000). Interestingly, in our study, larger effect sizes were found for non-complex stimuli. Non-face stimuli ($g = .83$, Table 2) (e.g., gratings, Gabor patches and circular windmill patterns) are indeed expected to be relatively less complex to process than emotional faces. As such, there seems to be no need to use complex stimuli or paradigms to detect reduced vMMN amplitudes in Schizophrenia, as has been described for aMMN (Avissar et al., 2018). Still, more studies are needed to support this hypothesis.

The moderation analysis for electrode clusters also uncovered that differences between groups were exclusive to the posterior region. This finding is aligned with the suggestion that the neural bases of the vMMN are located in the brain's nonprimary visual areas (Susac et al., 2014), in the same way that the auditory cortices are the main generators of the aMMN (Alho, 1995). The scalp distribution and source generators of the vMMN are still debatable. However, there seems to be a consensus regarding higher posterior negativity and an opposite effect, that is, a positivity, in anterior regions, which can also occur in central regions for face stimuli (Csukly et al., 2013; Stefanics et al., 2012; for a review, see Stefanics et al., 2014). Nonetheless, in this meta-analysis, only two studies reported a positivity effect. Farkas et al. (2015) used Gabor patches and found a negative peak in parieto-occipital regions and a reversed effect in prefrontal regions. Csukly et al. (2013) used emotional faces and found a positivity effect in prefrontal and central regions. Both studies were included in the subgroup analysis of the anterior region ($k = 3$), with only one study (Urban et al., 2008) reporting negative amplitudes at Fz for both groups. For central regions ($k = 3$), all but Csukly et al. (2013) described a negative effect (She et al., 2017; Urban et al., 2008).

Another finding of Erickson et al. (2016) was that a possible overlap between the N2b in active tasks due to attention could significantly impact the aMMN amplitude. We did not replicate this finding with the vMMN as we found no differences between studies with active and passive tasks. This may be due to the more posterior location of the vMMN neural generators. Although research has suggested that the prefrontal cortex might contribute to vMMN (Kimura et al., 2010), all studies found a posterior negativity except for She et al. (2017), which reported this only in the central region. This result suggests that the vMMN is less susceptible to overlap with components generated in the prefrontal cortex, such as N2b (Luck, 2014), and so not as influenced by the task characteristics, for instance active versus passive tasks.

The overall reduction in MMN amplitude suggests a failure in higher-order processes rather than specific sensory alterations in the visual or auditory domain. Considering that the MMN is believed to represent prediction error or expectation violation in the PPF (Wacongne, 2016), our results align with this framework and provide more evidence for the reduced prediction error hypothesis in schizophrenia across stimuli modalities. The PPF suggests that the heterogeneity of manifestations of SSD, particularly positive symptoms, can be explained by over-precise top-down priors that fail to adjust when confronted with low-noise input that should be trusted. Consequently, these stimuli may be perceived as noise, resulting in a diminished prediction error and a failure to update the predictive model (Griffin & Fletcher, 2017; van Schalkwyk et al., 2017). In this context, a reduction in MMN amplitude could represent a reduction in prediction error in the PPF. Mechanistically, it remains unclear whether reduced prediction error in SSD is due to increased precision of priors, reduced precision of sensory input or both, as none of the included studies manipulated both expectations and the level of noise/reliability of the stimuli.

The amplitude reduction of the vMMN component could be a promising candidate biomarker, particularly for patients with visual symptomatology. Still, more studies are needed to replicate other relevant findings found in previous aMMN meta-analyses, such as smaller effect sizes for analysis by first-episode psychosis and bipolar disorder groups compared to chronic schizophrenia (Erickson et al., 2016) or correlations with the duration of illness (Umbricht & Krljes, 2005) and more robust results with simple stimuli and deviants (Avissar et al., 2018). Particularly, research focused on examining the development of the disease, from high-risk clinical populations to chronic illness, would bring important knowledge in understanding the nature of vMMN impairment in schizophrenia. Additionally, such studies can shed light on the potential of vMMN as a biomarker with clinical applications.

5 | LIMITATIONS AND FUTURE DIRECTIONS

A clear limitation that needs to be addressed in the field is the small number of studies available to be included in this meta-analysis. Unfortunately, not enough studies have explored vMMN to reach robust conclusions, although no heterogeneity was found in the overall findings. Also, the number of studies included in some moderation analyses might have been underpowered to find differences.

Nonetheless, the current results may open the use of visual stimuli in the search for a better understanding of mismatch responses in SSD. Visual stimuli offer important advantages compared with the auditory domain. For example, in the study of emotions, although it is possible to use auditory or other expressions, most of our non-verbal communication channels are through facial expressions (Oh Kruzic et al., 2020). Another potential advantage is that this component does not seem to overlap with components generated in the prefrontal cortex. Thus, similar results can be found for active and passive tasks, as opposed to what is described for aMMN (Erickson et al., 2016). From a clinical point of view, this holds potential utility, given that this component can be recorded without requiring the management of voluntary attention. This aspect proves beneficial for designing more engaging tasks, such as games, or tasks that allow simultaneous recording of other components of interest, like P3b, which reflects higher-order cognitive processes.

Future research should not only increase the knowledge of the MMN's visual domain, particularly in different stages and groups of the SSD, such as first-episode psychosis, but also explore other sensory modalities. Considering that this reduction has been found for the visual and auditory domains, one can consider that it might reflect a general dysfunction. In that sense, studies should also search for similar prediction error abnormalities in other exteroceptive modalities, such as somatosensory oddball tasks (Huang et al., 2010) and in interoception (Ardizzi et al., 2016).

6 | CONCLUSION

Overall, this meta-analysis revealed a reduction in the vMMN amplitude in schizophrenia, in line with findings in the auditory domain and the proposed abnormalities in the PPF for psychosis. In addition, this component offers certain advantages, such as being less susceptible to overlap with components generated in the prefrontal cortex due to voluntary attentional task demands, attributed to its posterior negativity instead of anterior, and its potential for investigating non-verbal communication and affective aspects. Future studies should use vMMN to explore abnormalities in the PPF in different stages and groups of the SSD and increase the knowledge in the search for biomarkers in schizophrenia.

AUTHOR CONTRIBUTIONS

PM: conceptualisation; data curation; formal analysis; investigation; writing – original draft preparation. FC: Formal analysis; investigation; writing – review & editing. JD: investigation; writing – review & editing. RP: Conceptualisation; writing – review & editing. CS: writing – review & editing. FFS: conceptualisation; writing – review & editing.

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CONFLICT OF INTEREST STATEMENT

The authors have declared no conflict of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created.

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SUPPORTING INFORMATION

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