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Systematic review and meta-analysis of metacognitive abilities in individuals with schizophrenia spectrum disorders

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ABSTRACT

Metacognitive deficits are well documented in schizophrenia spectrum disorders as a decreased capacity to adjust confidence to performance in a cognitive task. Because metacognitive ability directly depends on task performance, metacognitive deficits might be driven by lower task performance among patients. To test this hypothesis, we conducted a Bayesian meta-analysis of 42 studies comparing metacognitive abilities in 1425 individuals with schizophrenia compared to 1256 matched controls. We found a global metacognitive deficit in schizophrenia ($g = -0.57$, 95 % CrI [−0.72, −0.43]), which was driven by studies which did not control task performance ($g = -0.63$, 95 % CrI [−0.78, −0.49]), and inconclusive among controlled-studies ($g = -0.23$, 95 % CrI [−0.60, 0.16], $BF_{01} = 2.2$). No correlation was found between metacognitive deficit and clinical features. We provide evidence that the metacognitive deficit in schizophrenia is inflated due to non-equated task performance. Thus, efforts should be made to develop experimental protocols accounting for lower task performance in schizophrenia.

1. Introduction

Metacognition is the ability to monitor and control our own mental processes. Metacognitive deficits are thought to play an important role in schizophrenia spectrum disorders (hereafter: schizophrenia) (Hasson-Ohayon et al., 2018). These deficits are inferred both from subjective structured interviews (Semerari et al., 2003) and objective neuropsychological tasks (Koren et al., 2006), and have been linked to core features of schizophrenia including positive and negative symptoms (McLeod et al., 2014), lack of insight into illness (David et al., 2012), disorganisation (Vohs et al., 2014), functioning (Davies and Greenwood, 2020), and quality of life (Arnon-Ribenfeld et al., 2017).

Despite numerous studies, no meta-analysis has yet been conducted to examine metacognition in schizophrenia. Here we sought to conduct a systematic review and meta-analysis of neuropsychological measures of metacognitive performance in schizophrenia compared to matched healthy controls. From an experimental perspective, the gold standard to quantify metacognition is to assess how participants perform an experimental task (first-order task) and reflect on their own accuracy via confidence ratings (second-order task). Several studies employing this design have reported lower metacognitive performance in schizophrenia compared to healthy controls across different cognitive domains such as vision (Dietrichkeit et al., 2020; Jia et al., 2020; Moritz et al., 2014), audition (Gaweda and Moritz, 2019), emotion perception (Kother et al., 2012; Moritz et al., 2012; Pinkham et al., 2018), and memory (Berna et al., 2019; Mayer and Park, 2012; Moritz and Woodward, 2006a). However, these results are mitigated by recent studies that failed to reveal such metacognitive deficits (Faivre et al., 2019; Powers et al., 2017; Wright et al., 2020). Noticeably these studies controlled for potential group differences in first-order performance, either at the design level through adaptive staircase procedures (Levitt, 1971), or at the metric level through indices of metacognitive performance which are independent of first-order performance (Maniscalco and Lau, 2012). This is especially important in schizophrenia where cognitive impairments are well documented (Gopal and Variend, 2005; Heinrichs and Zakzanis, 1998) and associated with metacognitive deficits (Davies and

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Greenwood, 2020). This known issue in the field of metacognition (Galvin et al., 2003; Maniscalco and Lau, 2012) can be stated as follows: because it is easier to finely adjust confidence ratings following an easy task than a difficult one, any comparison of metacognitive performance between two conditions that differ in terms of task difficulty is non-specific: should a difference in metacognitive performance be observed, it is impossible to tell if it stems from first-order (i.e., task difficulty), or second-order origins (i.e., metacognitive processes per se). As first-order performance is typically lower in patients vs. controls, a putative metacognitive deficit may be merely inherited from a deficit at the first-order level, and thus not specific to second-order processing. To determine whether schizophrenia involves specific deficits in metacognitive abilities, we conducted a systematic review followed by a Bayesian meta-analysis on a sample of 42 studies. Our main hypothesis was that metacognitive deficits would be smaller in studies controlling for first-order performance. Following a pre-registered plan, we conducted additional subgroup analyses and meta-regressions to explore if metacognitive deficits vary across cognitive domains, the severity of schizophrenia symptoms, and antipsychotic dosage. We had preregistered two additional directional hypotheses regarding the influence of diagnosis (first-episode vs. chronic schizophrenia) and symptomatology (depression, insight) on metacognitive abilities, but these analyses were not conducted due to the scarcity of the available data.

2. Methods

This meta-analysis followed the PRISMA recommendations (Moher et al., 2009). The protocol was registered on PROSPERO (CRD42020188614) on May 26th 2020, before data extraction.

2.1. Inclusion criteria

Inclusion criteria followed the PICO framework.

- *Population*: individuals with schizophrenia or related disorders (schizoaffective, schizophreniform), as defined by standard diagnostic criteria (DSM-III, DSM-III-R, DSM-IV, DSM-IV-R, DSM-IV-TR, DSM 5, ICD-10).
- *Intervention*: a computerized or manual experimental task with self-reported retrospective confidence judgments as behavioral measures on a confidence scale with more than one trial.
- *Comparison*: healthy controls.
- *Outcome*: meta-performance defined as the strength of the relationship between first-order performance (accuracy on a neuropsychological task in perception, memory, executive functions, social cognition, and agency) and retrospective confidence judgments in the first-order performance, repeated for each trial. Meta-performance indices included: meta-d', M-Ratio, AUROC2, logistic regression, confidence gap, knowledge corruption index, gamma correlation (for details on these measures, see Fleming and Lau, 2014).

2.2. Search strategy

We retrieved English written preprints and peer-reviewed articles in three databases – Pubmed, Web of Science, Scopus – with the following query applied to the title, abstract and keywords:

(schizophrenia OR schizophrenic OR schizo-affective OR schizoaffective) AND (confident OR confidence OR metacognition OR metacognitive OR "error awareness" OR "error monitoring").

2.3. Screening and data extraction

The search was performed on April 24th 2020, and no new search before analysis was performed. This query could not identify one article previously known by a co-author (Powers et al., 2017) as it contained non-matching key-words and reported metacognitive performance in supplementary materials. It was manually included in the list of publications. Two authors (MR and PS) screened studies for inclusion in parallel, using Cadima (https://www.cadima.info; see supplementary information (SI) for details). For each study group, MR and PS extracted the following primary outcomes:

- whether the study controlled for first-order performance between groups (TRUE or FALSE)
- metacognitive performance indices (see above)
- first-order accuracy (% correct, d')

Depending on the data available, either the mean and standard deviation, or raw statistics (t and F values) were extracted (SI). The following secondary outcomes were extracted:

- cognitive domain
- clinical characteristics including Positive and Negative Syndrome Scale scores (PANSS total, positive, and negative) and antipsychotic dosage (chlorpromazine equivalent).
- age (mean and standard deviation)
- sample size

2.4. Statistical analyses

All analyses were conducted in R. We used the brms package (Bürkner, 2017) based on the Stan framework (Carpenter et al., 2017) to fit Bayesian meta-analytic multilevel models.

Before testing our main hypothesis regarding the influence of equating first-order performance on metacognitive abilities, we fitted a global model M1 with fixed and random effects as follows:

$$M1: G_i \mid \sigma_i \sim \text{Intercept} + (\text{Intercept} \mid \text{study})$$

Where G_i denotes the Hedge's g effect size of study i, σ_i denotes the standard error of the effect size from study i, thereby accounting for different sample sizes across studies (SI). M1 estimated the overall effect-size of a difference in metacognitive performance between groups (the grand intercept of the model) while accounting for the between-study variability (random intercept per study; see SI for prior definition). To test the existence of a metacognitive deficit in schizophrenia (H1), we compared the estimations of M1 to the estimations of an alternative model M0 assuming that metacognitive deficit was inexistent (i.e., fixing the intercept at 0; H0).

$$M0: G_i \mid \sigma_i \sim 0 + (\text{Intercept} \mid \text{study})$$

Hypothesis testing:

Results were interpreted based on the relative evidence toward H0 (absence of a metacognitive deficit in schizophrenia) or H1 (presence of a metacognitive deficit in schizophrenia) given by the Bayes factor (BF), and the summary statistics of the posterior distribution (mean and 95 % credible interval, CrI). The BF is the ratio of the marginal likelihoods of each hypothesis. We note BF_{10} the ratio of evidence in favour of H1 and BF_{01} the ratio of evidence in favour of H0. We used the interpretation of BFs given by Wagenmakers et al. (2018), which translates continuous BF values into a categorical scheme. Thus, we considered the relative strength of evidence in favor of hypothesis H1 over H0 (resp. H0 over H1), to be anecdotal if $BF_{10} \in [1, 3]$ (resp. $[\frac{1}{3}, 1]$), moderate if $\in [3, 10]$ (resp. $[\frac{1}{10}, \frac{1}{3}]$), strong if $\in [10, 30]$ (resp. $[\frac{1}{30}, \frac{1}{10}]$), very strong if $\in [30, 100]$ (resp. $[\frac{1}{100}, \frac{1}{30}]$) and extremely strong if > 100 (resp. < 0.01).

For subgroup analyses, we retrieved the summary statistics (mean and 95 % CrI) of the difference between the two posterior distributions obtained in each group. Then we assessed in each case under which hypothesis (H0: absence of deficit or H1: existence of a deficit) the data was the most plausible.

To test our main hypothesis, we assessed the influence of equating

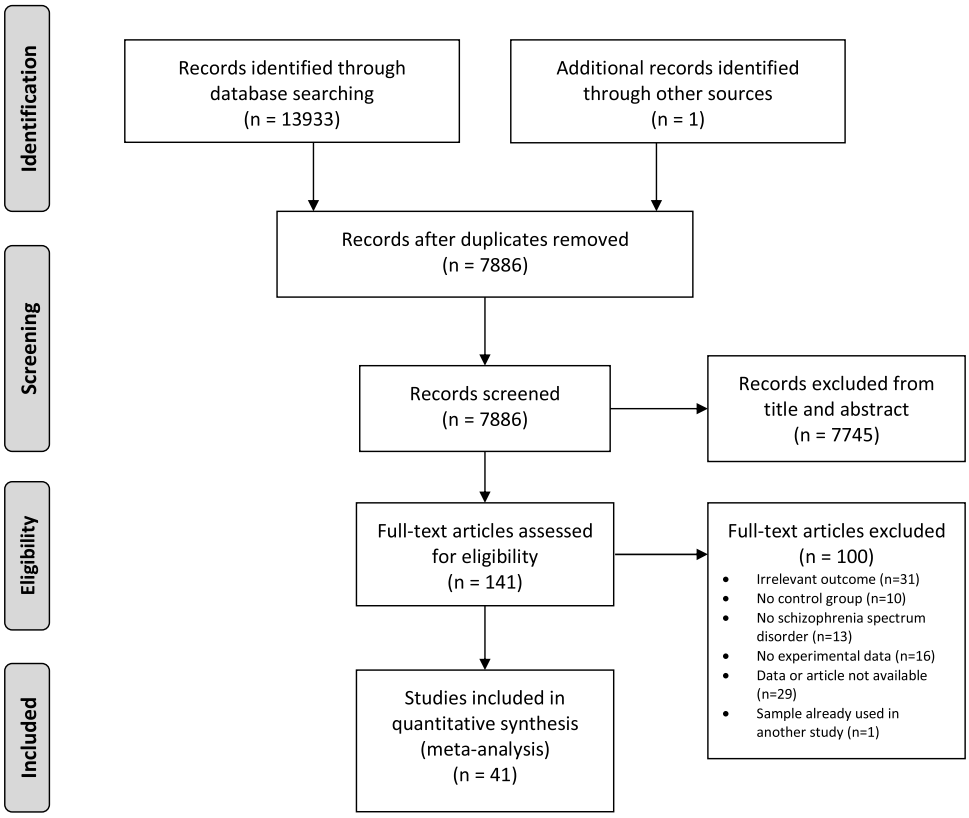

Fig. 1. Flow diagram of the selection process.

first-order performance with a model M2, identical to M1 including performance-matching as an additional binary predictor as follows:

$$M2: G_i \mid \sigma_i \sim Intercept + control\_type1 + (Intercept \mid study)$$

Where control_type1 is a binary predictor, TRUE for controlled-studies, FALSE otherwise.

All moderator analyses were first motivated by heterogeneity assessments. Three measures of heterogeneity were computed: the Q-statistic (Card and Little, 2016), the Q-between statistic (Borenstein et al., 2010), and the I^2 index for the percentage of the total variation due to between-studies variability (Higgins and Thompson, 2002). I^2 values between 0 and 0.25 suggest small magnitudes of heterogeneity, 0.25 to 0.50 medium magnitudes, and $>$ 0.50 large magnitudes. Exploratory subgroup analyses and meta-regressions were performed in case of significant Q-between and I^2 above 25 % (Huedo-Medina et al., 2006). Namely, we assessed the metacognitive deficit amplitude across cognitive domains by fitting a model identical to M1 with the between-study variable "cognitive domains" (perception, memory, others) as an additional categorical covariate. We also explored the correlation between metacognitive performance among patients and continuous variables by adding standardized (z-scores) PANSS scores and chlorpromazine equivalent as meta-regressors to M1.

2.5. Quality assessment

To quantify the risk of bias in individual studies, we assessed whether our selection contained extreme effect size values via a leave-one-out sensitivity analysis (SI). We also assessed the risk of bias according to the Newcastle-Ottawa Scale (NOS) adapted for case-control studies (SI). Publication bias was assessed using a funnel plot of observed outcomes against corresponding standard errors (Sterne and Harbord, 2004). The distribution of p-values was analyzed using the R package dmetar (Harrer et al., 2019) to examine whether some of the studies were subject to p-hacking (p-curve: Simonsohn et al., 2014).

3. Results

3.1. Study characteristics

Our search retrieved 13933 records, 7886 after duplicates removal. 7745 records were excluded after title and abstract screening (Fig. 1). Another 100 articles were excluded based on full-text screening, resulting in a selection of 41 articles.

One article was excluded because of a strongly deviant outcome identified via a leave-one-out analysis performed on the metacognitive deficit effect-size (SI). Among the 40 remaining articles, two were split into two independent studies as they involved different populations (young versus old: Gaweda (2015); hallucination-prone versus non-hallucination-prone: Powers et al. (2017). The final selection consisted of 42 studies, with a total population of 2681 participants (1425 patients) (Table 1).

Our selection included 10 perception (auditory and visual), 27 memory, 4 social cognition, and 1 agency studies. Because of their low number, social cognition and agency studies were regrouped into a generic category termed "others".

3.2. Global metacognitive deficit in schizophrenia

The meta-analytic model M1 revealed lower metacognitive performance in the schizophrenia vs. control groups with an effect size $g = -0.57$, 95 % CrI $[-0.72, -0.43]$ (Fig. 2). Comparison against the null hypothesis (i.e., absence of a metacognitive deficit in schizophrenia modelled by M0) resulted in a Bayes factor favoring the alternative hypothesis $BF_{10} = 36.56 \times 10^6$, indicating extremely strong evidence in favor of a metacognitive deficit in schizophrenia. Of note, this pattern of results was robust to prior variations (SI).

Table 1
Study characteristics. KCI: knowledge corruption index; AUROC2: area under the type 2 receiver operating characteristic curve.

Study	Sample size		Age		Matched performance	Cognitive domain	Metacognitive index	NOS
	SCZ	HC	SCZ	HC				
Dietrichkeit et al. (2020)	39	20	34.72 ± 8.68	30.55 ± 8.54	no	perception	KCI	4.0
Jia et al. (2020)	38	38	22.6 ± 8.3	23 ± 4.6	yes	perception	AUROC2	5.0
Jones (2020)	215	151	41.72 ± 11.64	41.95 ± 12.42	no	others	confidence gap	5.0
Wright et al. (2020)	50	68	27.17 ± 1.3	25.7 ± 6.6	yes	perception	M-ratio	6.0
Berna et al. (2019)	10	10	36.3 ± 7.5	36.2 ± 8.4	yes	memory	meta-d' - d'	3.5
Faivre et al. (2019)	21	20	38.8 ± 8.77	42.6 ± 3.35	yes	perception	M-ratio	7.0
Gaweda et al. (2019)	33	33	35.82 ± 11.22	41.33 ± 14.8	no	perception	false perception	5.0
Davies et al. (2018)	31	18	26.16 ± 5.69	24.06 ± 4.87	yes	perception	M-ratio	7.0
Gawęda et al. (2018)	25	33	20.36 ± 2.16	20.27 ± 2.11	no	memory	KCI	5.7
Mayer et al. (2018)	24	24	40.67 ± 11.65	38.88 ± 9.66	no	memory	false memories	5.5
Pinkham et al. (2018)	31	32	35.65 ± 7.52	35.41 ± 7.07	no	others	AUROC2	3.5
Charles et al. (2017)	13	13	28.8 ± 5.9	28.8 ± 4.7	no	perception	meta-d'	5.0
Powers et al. (2017)	15	15	39.4 ± 13.47	46.07 ± 12.96	yes	perception	M-ratio	5.5
Powers et al. (2017)	14	15	38.29 ± 14.4	40.53 ± 13.04	yes	perception	M-ratio	5.5
Balzan et al. (2016)	25	50	39.96 ± 10.04	42.8 ± 15.46	no	memory	confidence in errors	4.0
Eifler et al. (2015)	29	25	37.22 ± 9.68	38.12 ± 10.72	no	memory	confidence gap	4.0
Eisenacher et al. (2015)	21	38	26.52 ± 5.57	25.08 ± 6.55	no	memory	confidence gap	3.5
Gaweda (2015)	13	17	22.08 ± 1.93	23.59 ± 1.87	no	memory	KCI	4.0
Gaweda (2015)	10	10	53.9 ± 3.21	57.4 ± 3.72	no	memory	KCI	4.0
Akdogan et al. (2014)	23	23	38 ± 8	37.5 ± 7.2	no	memory	gamma correlation	3.3
Mayer et al. (2014)	31	28	40.23 ± 9.1	37.89 ± 8.35	no	memory	false memories	4.0
Moritz et al. (2014)	55	45	38.22 ± 8.61	37.24 ± 13.93	no	perception	KCI	4.5
Gaweda et al. (2013)	54	34	35.17 ± 10.43	33.21 ± 11.33	no	memory	KCI	4.5
Peters et al. (2013)	27	24	37.96 ± 12.86	34.21 ± 11.33	no	memory	KCI	4.5
Gaweda et al. (2012)	32	32	32.81 ± 8.36	31.78 ± 11.67	no	memory	KCI	4.7
Kother et al. (2012)	76	30	34.26 ± 11.41	32.97 ± 10.88	no	others	KCI	4.0
Mayer et al. (2012)	28	29	38.32 ± 9.29	37.28 ± 8.41	no	memory	false memories	5.0
Metcalfe et al. (2012)	22	20	42.3 ± 11.1	38.1 ± 11.3	no	others	correlation perf-confidence	5.0
Moritz et al. (2012)	23	29	35.17 ± 11.12	34.24 ± 16.14	no	others	KCI	4.5
Peters et al. (2012)	47	47	35.72 ± 11.63	36.87 ± 11.89	no	memory	KCI	5.0
Bhatt et al. (2010)	25	20	47 ± 8.65	44.5 ± 8.81	no	memory	KCI	2.0
Kim et al. (2010)	12	13	40.2 ± 10.23	40.4 ± 9.34	no	memory	false memories	4.5
Moritz et al. (2008)	68	25	33.94 ± 10.45	32.04 ± 10.23	no	memory	confidence gap	4.0
Kircher et al. (2007)	27	19	32.8 ± 11.4	33.4 ± 13.4	no	memory	correlation perf-confidence	5.0
Peters et al. (2007)	23	20	36.3 ± 13.13	35.2 ± 9.71	no	memory	confidence gap	6.0
Moritz et al. (2006a)	31	61	33.77 ± 9.9	31.05 ± 8.75	no	memory	confidence gap	4.5
Moritz et al. (2006b)	30	15	24.73 ± 8.73	24.8 ± 8.99	no	memory	confidence gap	3.5
Moritz et al. (2006c)	35	34	36.29 ± 11.34	34.29 ± 11.38	no	memory	KCI	4.0
Moritz et al. (2005)	30	17	37.3 ± 10.16	37.67 ± 12.47	no	memory	KCI	4.5
Moritz et al. (2004)	20	20	33.2 ± 9.28	29.2 ± 12.51	no	memory	KCI	4.5
Moritz et al. (2003)	30	22	31.08 ± 8.3	27 ± 10.7	no	memory	confidence gap	4.0
Bacon et al. (2001)	19	19	31.7 ± 8.4	30.7 ± 8.2	no	memory	confidence gap	3.5

3.3. Metacognitive deficit in studies controlling for first-order performance

Our main hypothesis stipulated that metacognitive deficits would be decreased in studies controlling for first-order performance. The following analysis was further justified by a heterogeneity analysis which produced a significant Q-statistic (124.1, df = 41, $p < .001$) and a high amount of heterogeneity (I^2 statistic 0.66, 95 % CI [0.54, 0.76]), suggesting this moderator analysis was appropriate. Because metacognitive performance is known to depend on first-order performance (Maniscalco and Lau, 2012), and because the latter differed between groups (g = −0.64, 95 % CrI [−0.77, −0.52], $BF_{10} = 2.06 \times 10^{10}$), we sought to assess whether metacognitive deficits could stem from cognitive impairments that are well documented in schizophrenia (Gopal and Variend, 2005; Heinrichs and Zakzanis, 1998). Distinguishing studies controlling for first-order performance (N = 7) from those which did not (N = 35) revealed a significant moderation effect (Q-between = 6.82, df = 1, p = 0.009). Thus, we assessed the influence of performance-matching with a model M2, identical to M1 including performance-matching as an additional binary predictor. The sub-group of non-controlled studies had an overall metacognitive deficit of magnitude g = -0.63, 95 % CrI [−0.78, −0.49], which was reduced to g = −0.23, 95 % CrI [-0.60, 0.16] in the sub-group of controlled studies (Fig. 3A). Accordingly, the evidence ratio supporting our directional hypothesis that controlling for first-order performance decreases the magnitude of the metacognitive deficit was very strong ($BF_{10} = 51$) (Fig. 3B). Comparison against the null hypothesis among controlled studies revealed inconclusive evidence in favor of a metacognitive deficit in schizophrenia ($BF_{01} = 2.2$). Finally, a positive correlation between cognitive and metacognitive deficits was found among non-controlled studies (SI). Sub-group analyses reduced heterogeneity which however remained significant (SI).

3.4. Metacognitive deficits across cognitive domains

Next, in line with our pre-registered analysis plan and a significant moderation effect of cognitive domains (Qbetween = 38.5, df = 2, $p < .001$), we assessed how metacognitive deficits varied across cognitive domains (i.e., perception, memory, others). A subgroup analysis revealed the largest metacognitive deficit among memory studies, compared to perception and others. Mean value of the metacognitive deficit in the memory domain (g = -0.74, 95 % CrI [-0.89, -0.58], $BF_{10} = 7.74 \times 10^{156}$) was twice higher than in the perception domain (g = -0.33, 95 % CrI [-0.63, -0.04], $BF_{10} = 2.16$), and three times higher than in other domains (g = -0.26, 95 % CrI [-0.62, 0.10], $BF_{10} = 0.40$; see Figs. 4 and SI). Sub-group analyses reduced heterogeneity which however remained significant (SI).

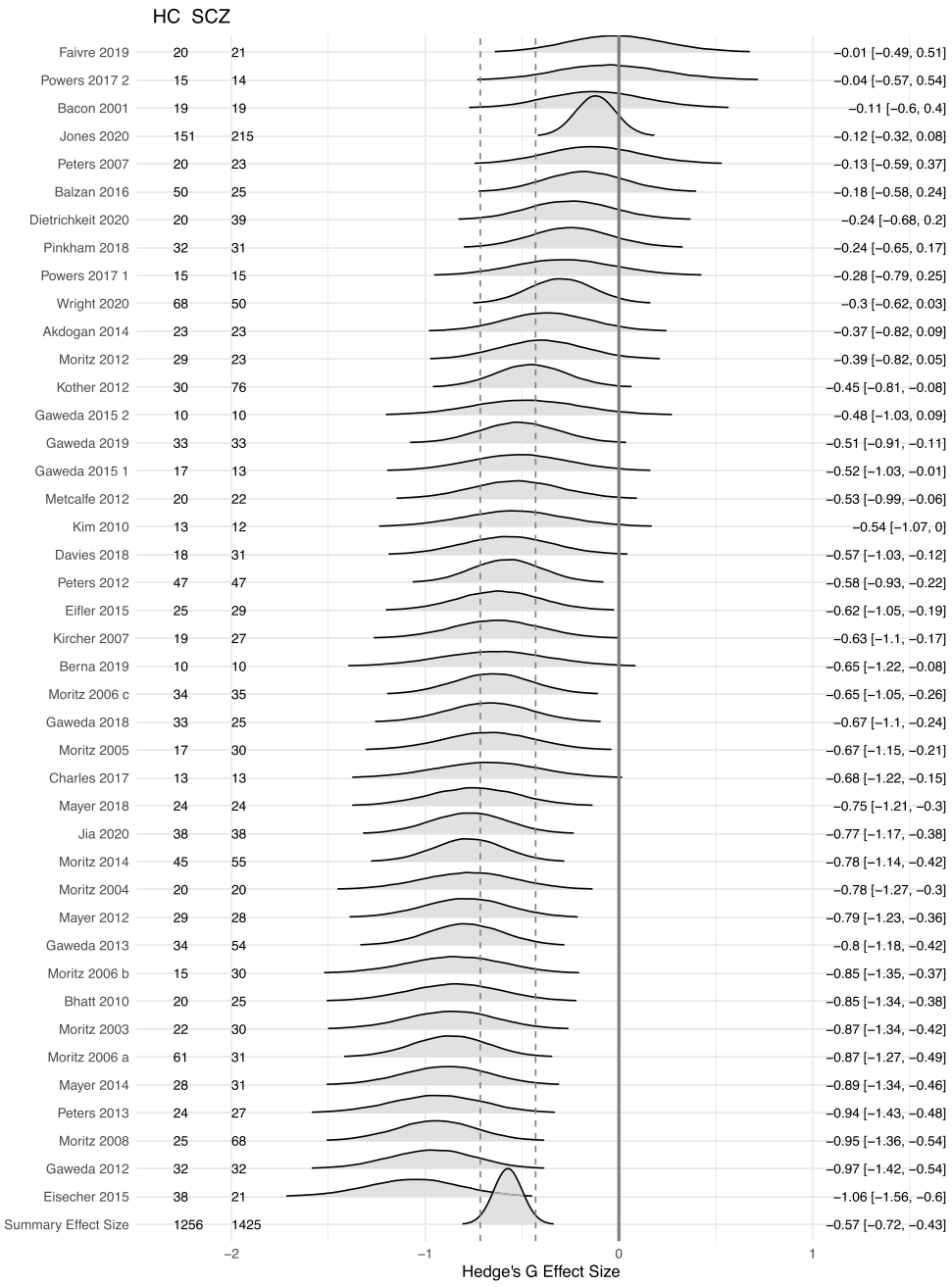

Fig. 2. Forest plot of the metacognitive deficit in schizophrenia. Left: Authors with publication year and sample sizes; Middle: posterior distribution of the effect size; Right: mean and 95 % CrI of the posterior distribution. The summary effect size is displayed on the last row: the solid vertical grey line is centred on zero (i.e., equivalent metacognitive performance between groups), and the dashed vertical lines depict the boundaries of the 95 % CrI.

3.5. Meta-regression analyses

Finally, we performed further meta-regressions to explore how metacognitive deficits co-varied with the severity of positive and negative symptoms (PANSS equivalent scores) and antipsychotic dosage (chlorpromazine equivalent), with a prior of mean 0 and SD = 1. We had pre-registered the hypothesis of a negative correlation between meta-performance and PANSS positive scores. However, meta-regression analyses provided inconclusive evidence regarding the influence of symptom severity on the metacognitive deficit: $BF_{10} = 0.88$ for PANSS total scores (N = 35), $BF_{10} = 0.91$ for PANSS positive scores (N = 32) and $BF_{10} = 0.75$ for PANSS negative scores (N = 33) (see SI, Fig. S6).

Similarly, we found no evidence for an association between metacognitive performance and pharmacological treatment (N = 20), with an evidence ratio ($BF_{10} = 0.99$) suggesting inconclusive data (see SI, Fig. S6).

3.6. Risk of bias in selected studies

A quality evaluation using the Newcastle-Ottawa Scale suggested that about half the studies had a relatively high risk of bias with scores < 5/9 (SI and (Luchini et al., 2017)). The shape of the funnel plot revealed no asymmetry (Egger's test: z = - 0.07, p = 0.94; Figs. 5A and SI), suggesting no clear publication bias. Plus, testing the right-skewness of the P-curve (Fig. 5B) with Stouffer's method revealed that both the half (p's < 0.025) and full p-curves (p's < 0.05) were right-skewed with p < .001,

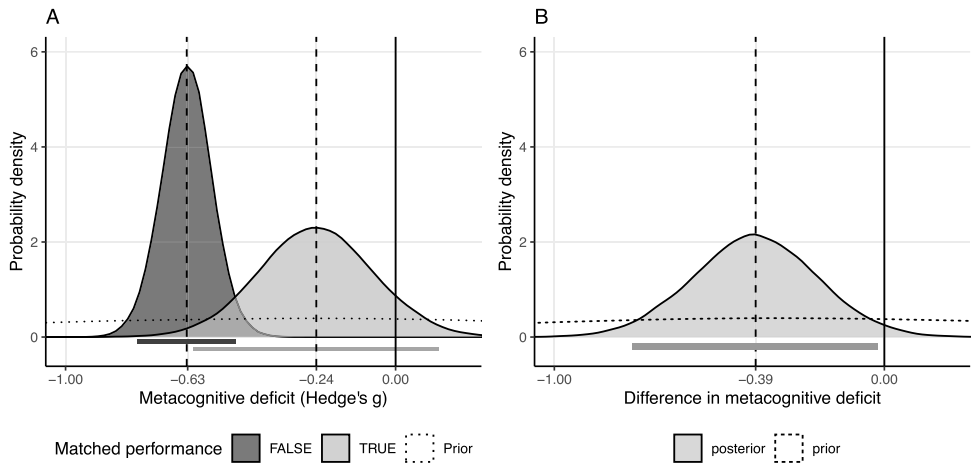

Fig. 3. A: Posterior distributions of the metacognitive deficit. Dark gray: non-controlled first-order performance (n = 35), Light gray: controlled first-order performance (n = 7). B: Posterior distribution of the difference in effect size between studies which did or did not control for first-order performance. In both panels, dotted lines represent the prior distributions, vertical dashed lines the mean posterior values, and the horizontal bars the 95 % CrI.

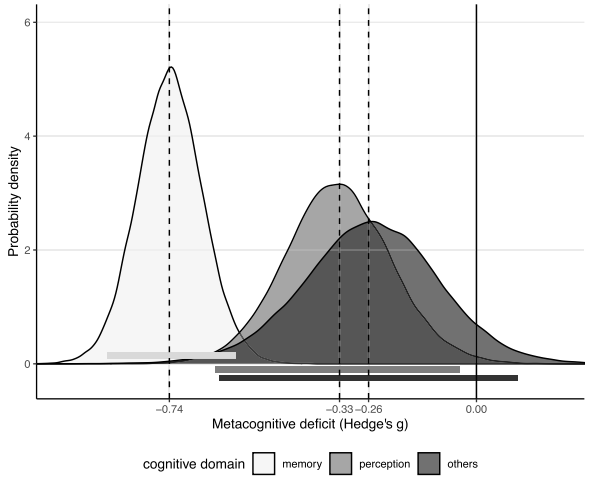

Fig. 4. Posterior distributions of the metacognitive deficit (Hedge's g effect size) according to each cognitive domain. The vertical dashed lines represent mean values and the horizontal bars the 95 % CrI.

suggesting that our study sample was not contaminated by p-hacking.

4. Discussion

The present meta-analysis based on 42 studies and 2681 individuals aimed at synthesizing the literature on the metacognitive abilities among individuals with schizophrenia. At first sight, our findings confirmed a deficit in metacognitive abilities in schizophrenia, but with high heterogeneity. The effect was of medium magnitude, which is smaller than the large effects reported in prior meta-analyses regarding cognitive impairments (Schaefer et al., 2013). The leave-one-out sensitivity analysis confirmed this effect was robust to outliers. We found several sources for heterogeneity that we describe hereafter.

4.1. Main result

Because patients' first-order cognitive deficits risked to artificially inflate metacognitive deficits (Galvin et al., 2003), our main hypothesis was that metacognitive deficits would be reduced in studies equating first-order performance between groups. Results indicated strong evidence in favor of our hypothesis, as metacognitive deficits were twice smaller in studies controlling for first-order performance, most of them

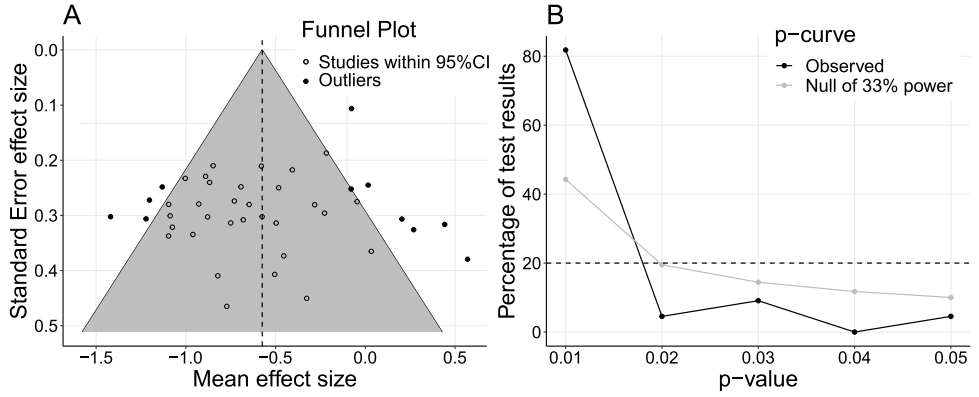

Fig. 5. A: Funnel plot centered on the overall effect size. The vertical dashed line represents the global metacognitive deficit. The gray area represents the 95 % CI of the overall effect size. Each dot represents a study, full dots represent outliers. B: Observed p-curve (black) and theoretical p-curve expected for low-powered (33 %) studies (gray). Horizontal dashed line: Expected uniform distribution for null effects.

concerning the perceptual domain. In this subset of studies, assessing the presence of a metacognitive deficit revealed inconclusive evidence. By contrast, a correlation between cognitive and metacognitive deficits was found among non-controlled studies, indicating that first-order performance is a critical moderator of metacognition which should be controlled for when assessing metacognitive deficits in schizophrenia.

4.2. Metacognitive deficits across cognitive domains

We also explored possible differences in metacognitive deficits across cognitive domains (perception, memory, others), and found the most prominent deficits among memory studies. As such, this result is not sufficient to confirm the presence of a specific meta-memory deficit in schizophrenia, as all the memory studies but one did not control for differences in first-order performance between groups. Given that the magnitude of the meta-memory deficit we found is lower than the one of episodic verbal memory (range between -1.53 and -1.11 SD) (Gopal and Variend, 2005; Heinrichs and Zakzanis, 1998; Schaefer et al., 2013), arbitrating between the existence of a specific meta-memory deficit or the side effect of a non-controlled first-order factor will require the development of more robust experimental protocols. Of note, this meta-analysis did not examine the literature based on judgments of learning or feeling of knowing, which may reveal different patterns of results (Souchay et al., 2006).

4.3. Unexplained heterogeneity

Despite moderation analyses, heterogeneity remained high even after clustering studies according to performance matching and cognitive domains. This heterogeneity may be explained by the different diagnoses included in our selection of studies. The category of first episode of psychosis may be particularly problematic, as it included variable diagnoses (mania with psychosis, bipolar disorder with psychosis, depression with psychosis, delusional disorder, substance-induced psychotic disorder, psychosis not otherwise specified, acute and transient psychotic disorder, brief psychotic disorder). Heterogeneity may also come from the use of idiosyncratic first-order tasks (e.g., memory performance was quantified using recognition, source memory and spatial delayed response tasks) and confidence scales (e.g., ordinal vs. continuous scales, full vs. half scales, etc.). Finally, one should consider that the same research group co-contributed a large number of selected studies, with metacognitive deficits of larger magnitudes than the one estimated by other authors (SI). With this in mind, it will be important to use more systematic paradigms among more diverse study samples in the future.

4.4. Perspectives

Additional analyses evaluating how metacognitive deficits varied as a function of clinical scores (PANSS total, positive, negative) and antipsychotic dosage (chlorpromazine equivalent) revealed inconclusive evidence for correlation in each case. As we had no access to individual data, correlations were based on summary statistics extracted from each experimental group, which is suboptimal. As with all meta-analyses, our findings are shaped and limited by selection and analytical methods, and the information made available to researchers in the studies selected for review. Thus, they may be contradicted by other relevant studies referenced in non-searched databases. The scarcity of data prevented us from running planned analyses regarding the link between metacognitive performance and clinical/cognitive insight. Establishing this link is of crucial importance to validate confidence calibration as a valid empirical construct for clinical practice, and to refine current strategies to improve insight in schizophrenia. We encourage authors to share anonymized individual data similar to what is done for healthy controls (Rahnev et al., 2020) on a dedicated repository (https://osf.io/cfm5d/). Our findings point to several areas for future research. First, few studies included in this meta-analysis measured mood, despite it being an important determinant of metacognition (Lin et al., 2019), with a bias toward underconfidence in depression (Hoven et al., 2019). No study included in this meta-analysis focused on the metacognition of executive function. Further studies are needed because meta-executive functions have been linked with attenuated psychosis syndrome (Koren et al., 2019). Further studies should also investigate whether metacognitive abilities are associated with insight, relapse and psychosocial functioning before using it in clinical settings.

5. Conclusion

This is the first meta-analysis to examine metacognitive deficits based on confidence judgments in schizophrenia. Our results show that this deficit is inflated due to non-equated first-order performance, and varies across cognitive domains. Importantly, metacognitive deficits may also be overestimated in other psychiatric and neurological conditions involving cognitive impairments. Efforts should be made to develop experimental protocols accounting for lower first-order performance in schizophrenia before including the accuracy of confidence judgments as a cognitive dimension in neuropsychological batteries for clinical applications.

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The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Author contributions

MR, PS, MP, PR and NF developed the study concept and contributed to the study design. Data selection and extraction were performed by MR and PS. MR, LN and NF analyzed data. MR and NF drafted the paper; all authors provided critical revisions and approved the final version of the paper for submission.

Data availability statement

Bibliographic data and analyses scripts are publicly available: https://gitlab.com/nfaivre/meta_analysis_scz_public.

Declaration of Competing Interest

The authors declare no competing interests.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.neubiorev.2021.03.017.

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