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Table 1. Studies including TMS-EMG outcomes

			Patient characteristics								Summary of findings	
Study	**Design**	**Number of SCZ patients (N of males)**	**Age (years) (mean±SD)**	**Illness duration (years) (mean±SD)**	**Clinical characteritics (mean±SD)**	**Medications**	**Task**	**TMS protocol**	**Stimulation site**	**Measures of cortical excitability**	**Main findings**	**Correlations**
Chroni et al. (2002)	cross-sectional	14 with SCZ 14 with major depression (MD) 14 with mania	39 (SCZ) 48 (MD) 42 (mania)	11.1 ± 9.8 (SCZ) 11.8 ± 9.2 (MD) 11.4 ± 9.1 (mania)	Clinical Global Impression Scale (CGI)	Antipsychotics (n=14), benzodiazepines (n=4), mood stabilizers (n=2), anticholinergics (n=11); remained on stable treatment regimens for at least 15 days before testing.	Participants were asked to perform an exercise involving the APB muscle of the right hand	At baseline, 5 stimuli trains (intensity = 115% of the participant's RMT, frequency = 0.3 Hz) were delivered with each train separated by 30 sec. Participants then asked to perform the "exercise" for 30 sec, followed immediately by a stimuli train, and this was repeated for another 4 times.	left M1	RMT defined as the lowest intensity capable of producing a MEP⩾ 100 μV in 3/5 consecutive trials; MEP facilitation (%) = mean MEP following exercise / baseline mean MEP (MEP amplitude was measured peak-to-peak)	SCZ patients had significantly lower RMT than HCs. Mean MEPs showed a significant increase after exercise in the HCs but not in any of the 3 patient groups. The difference in mean post-exercise MEP facilitation was significant between HCs and each one of the patient groups, but was not significant between the 3 patient groups themselves.	post-exercise MEP facilitation in the 42 patients (all patient groups included) did not correlate with disease or medication duration.

Kaster et al. (2015)	Longitudinal, open-label study (a new antipsychotic was selected by the patient in consultation with their psychiatrist). TMS measurements were performed at baseline, and 6 weeks and 6 months after the new antipsychotic. HCs were only assessed at baseline.	16 with medication resistance (11 M)	33.3 ± 10.9	9.4 ± 7.4	PANSS	Medications at baseline: antipshychotics (n=14), antidepressants (n=6), benzodiazepines (n=5), mood stabilizers (n=2). Data was not available for 1 patient. All 16 patients were switched to clozapine after baseline measurements	N/A	CSP: muscle actively contract at 20% of maximum voluntary contraction, stimulation intensity = 140% RMT, ISI = 5 sec. SICI and ICF: CS = 80% RMT, TS was adjusted to produce mean peak-to-peak MEP amplitude of 1 mV, ISI = 2, 4 (SICI), 10, 15 and 20 (ICF) ms	left M1	RMT; CSP duration; SICI and ICF: for both SICI and ICF the trials were averaged across the ISIs (e.g. for SICI it's 2 and 4 ms trials).	11 patients remained in the study after 6 weeks and 6 remained after 6 months. In patients, mean RMT at baseline, 6 weeks, and 6 months were not significantly different. At baseline CSP duration between patients and HCs were not significantly different; in patients, CSP was significantly longer after 6 weeks of treatment with clozapine, whereas no significant difference was found from 6 weeks to 6 months. No significant difference in SICI and ICF between HCs, patients at baseline, patients at 6 weeks and at 6 months.	Response to clozapine was defined as 20% reduction in PANSS from baseline, (total score) 23% response rate by 6 weeks. No significant correlation between change in CSP (baseline to 6 weeks) with change in PANSS scores as measured by total, positive, or negative scale. No significant difference in CSP change between clozapine responders and non-responders at 6 weeks. No significant correlation between CSP change with clozapine dose at 6 weeks.
Fitzgerald et al. (2002a)	cross-sectional	22 (17 M)	28.8 ± 7.9	N/A	PANSS; Montgomery-Asberg Depression Rating Scale	All patients were receiving treatment with a single antipsychotic:	N/A	CSP: stimulation was given at 10, 20, 30 and 40%	left M1	RMT; AMT (measured during a sustained contraction, 5–10% of	No significant difference in RMT or AMT between patients and HCs. CSP duration was	Data for patients and HCs were pooled and found a

		(MADRS); Simpson-Angus (SA) rating scale	olanzapine (n=14); risperidone (n=8). No concurrent use of lithium, mood stabilisers, or other antipsychotics. Use of long-acting or short-acting benzodiazepines within 18h of testing was not permitted	above the AMT (sustained contraction of 5% maximum). SICI and ICF: CS = AMT minus 5%, TS was set to produce a moderate MEP response (0.5–1.0 mV), 10 trials for each condition (i.e. single TS and at ISIs of 1, 2, 3, 4, 10, 15 and 20 ms), ITI = 5 sec	maximum); CSP: peak-to-peak MEP size and latency, and CSP latency and duration (calculated as the time of offset of the EMG activity suppression minus the time of onset); SICI & ICF = conditioned MEP amplitude / unconditioned MEP amplitude	significantly shorter in patients at 10, 30 and 40% above the AMT, no significant difference in CSP latency between groups; and no significant difference in MEP size or latency. SICI: When the 4 ISIs for SICI were pooled, there was a significant reduction in the degree of inhibition in the patient group (patients 40.2%, HCs 27.8%), but when each ISI was analysed separately, the patients had less SICI at each ISI but the differences were not significant. No significant between-group difference in ICF in the pooled data or at any of the 3 ISI levels.	significant positive correlation between the mean SICI (average of the 4 ISIs) and the CSP duration at the 30 and 40% intensity levels (30% and 40%). Also, a significant positive correlation between ICF and the CSP duration at 10 and 20% intensity was found. No significant correlation between the clinical ratings and medication dose and the TMS measures. No significant correlation between duration of treatment and RMT, AMT, CSP duration and latency, SICI and ICF, but significant positive

												correlation found between treatment duration and CSP-MEP size at 3 of the intensity levels (20%; 30%; 40%)
Liu et al. (2009)	cross-sectional	78 (69.2% M)	36.35 ± 11.35 (all); 31.29 ± 8.83 (unmedicated); 37.88 ± 11.47 (Olanzapine/Quetiapine); 36.20 ± 13.63 (Risperidone/Typical); 35.11 ± 11.56 (clozapine)	N/A	PANSS; Abnormal Involuntary Movement Scale (AIMS); Simpson-Angus Scale (SAS); Barnes Akathisia Scale (BAS)	unmedicated (n=7); clozapine (n=19); olanzapine/quetiapine (n=20/12), risperidone/typical antipsychotics (n=12/8); anticholinergics (n=10), benzodiazepines (n=11), the distributions of use among the subgroups did not significantly differ	N/A	CSP: participant pinched the dynamometer at 20% of maximal contraction force, stimulation intensity = 140% RMT, ISI = 5 sec. SICI and ICF: CS = 80% RMT, TS was suprathreshold, ISI = 2, 4 (SICI), 10, 15 and 20 (ICF) ms	left M1	RMT; unconditioned MEP size (a measure of motor excitability); absolute CSP duration = time from the MEP onset to the return of any voluntary muscular activity, SICI and ICF = conditioned MEP / unconditioned MEP	No significant difference in RMT or unconditioned MEP size between all patients (n=78) and HCs. Patients receiving clozapine had reduced SICI compared to HCs and patients taking other antipsychotics (n=52), but it did not differ between patients taking other antipsychotics and HCs. Among the 4 medication groups (unmedicated, Olanzapine/Quetiapine, Risperidone/Typical, clozapine): no significant difference in RMT, SICI or ICF among the groups, whereas the olanzapine/quietapine group showed higher unconditioned MEP size compared to the risperidone/typicals	In all 78 patients (as one group): PANSS total score correlated positively with unconditioned MEP size and negatively with CSP duration; positive symptoms severity correlated positively with unconditioned MEP size and SICI; negative symptoms severity correlated positively with unconditioned MEP size and negatively with CSP duration; general psychopathology score correlated

						group; as for CSP duration, patients taking clozapine > HCs > olanzapine/quetiapine group = risperidone/typical group > unmedicated patients.	positively with unconditioned MEP size and negatively with CSP duration; extrapyramidal and involuntary movements, as assessed by AIMS, SAS, and BAS, were not associated with RMT, SICI, or ICF. When the patients were split into medicated and unmedicated groups, significant positive correlation between PANSS total score and SICI in the unmedicated patients, but in the mediated patients no significant correlation between SICI and PANSS total or any subscale score. No significant correlation between SICI

												and CSP duration in HCs, all patients or any medication subgroups.
Lindberg et al. (2016)	cross-sectional	28 with SCZ (24 M); 21 healthy siblings (9 M)	32 ± 6.7 (SCZ); 31.5 ± 9.6 (siblings)	14.2 ± 6.9	PANSS; Simpson-Angus scale (SAS); Neurological Soft Signs scale (NSS); MMSE	22 patients on stable (> 3 months) atypical antipsychotics (6 were taking clozapine) and 7 were non-medicated for at least 6 months. Patients on mood stabilizers, antidepressants or benzodiazepines were excluded.	Stop signal task: assessed the ability to inhibit a prepared action, 225 go trials (the gauge hit the target and participants had to lift the finger), and 105 stop trials (the gauge stopped before hitting the target and participants had to inhibit the finger lift response). The go trials had 2 conditions: "go early" and "go late"	CSP: stimulation given at 120% and 140% AMT while maintaining 10% muscle contraction. SICI was used to measure task-related changes in motor excitability (the TS alone trials) and inhibition (paired-pulse trials) during the "go early", "go late" and "stop" conditions of the stop signal task. TS intensity was initially set to induce a MEP of about 1.5	left M1	RMT (defined at rest as the lowest stimulus intensity that evoked MEPs of 100 μV in at least 5 of 10 trials); AMT (10% of maximal voluntary contraction); unconditioned MEP amplitude; CSP duration (defined as the time from MEP onset to the re-occurrence of continuous EMG activity); SICI = [1 minus (mean conditioned MEP amplitude / mean unconditioned MEP amplitude)] × 100.	No significant differences in RMT and AMT were found among patients, siblings and HCs. Unconditioned MEP size was significantly higher in Go late than Go early and Stop conditions in all 3 groups, but no significant difference among groups for each condition. CSP duration at 140% AMT was longer than at 120% AMT, but no significant difference across the 3 groups at either intensity. SICI was significantly lower in the Go late condition than the other 2 conditions, and significantly lower in patients than the other 2 groups in the Stop condition only. Inhibition success rate (for Stop trials)	N/A

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					mV in the FDI at rest, then was adjusted to give reproducible MEPs on repeated Go trials and to a level where the participant was not disturbed by the stimulation. CS intensity was initially set to 90% AMT and was decreased to give 50% inhibition of the unconditioned MEP. Once the TS and CS were determined, the intensities were held constant for the duration of the experiment. ISI for paired-pulses = 3 ms	changed with stop time: shorter the stop time, bigger the difference in percentage correct inhibition across groups, and siblings had significantly higher successful inhibition rate across all stop times. No significant difference in stop-signal reaction time among groups.

Boroojerdi et al. (1999)	cross-sectional	10 (9 M)	37.2 ± 10.8	7.8 ± 6.1	PANSS	olanzapine or clozapine (n=7), flupenthixol or haloperidol (n=3)	NA	Ipsilateral silent period: for MEP latency assessment, the coil was placed on the contralateral M1, stimulation intensity = 50% above RMT with the muscle at rest; for measurement of TCI the coil was placed on the ipsilateral M1, stimuli applied at 0.2 Hz with the same intensity. Participants were asked to maintain maximal activation of their ipsilateral FDI muscle before and during the stimulation (relax muscle for	bilateral M1	RMT (defined at rest as the lowest stimulus intensity that evoked MEPs of 100 μV in at least 5 of 10 trials); MEP latency in the contralateral FDI (a); onset latency of the inhibition of the FDI voluntary activity by ipsilateral stimulation (b); transcallosal conduction time = b minus a; duration of transcallosal inhibition	Data were pooled for the left and right sides (detected no significant side differences). No significant between group difference in RMT and MEP latency. Transcallosal conduction time was significantly delayed in patients and duration of TCI was significantly prolonged in patients.	N/A

								2-3 sec after stimulation)				
Du et al. (2019)	cross-sectional	24 (17 M)	36.51 ± 13.51	14.59 ± 14.75	Brief Psychiatric Rating scale (BPRS)	4 patients were not taking antipsychotics; atypical antipsychotics (n=19); typical antipsychotics (n=2); 1 patient was on both typical & atypical antipsychotics. No patient was taking benzodiazepines	N/A	SICI: ISI = 1 and 3 ms, CS = 80% RMT, TS = 120% RMT, 24 TS alone and 24 paired pulses (CS-TS)	left M1	RMT; unconditioned MEP amplitude (peak-to-peak); SICI = conditioned MEP / unconditioned MEP	No significant difference in RMT and unconditioned MEP amplitude between patients and HCs. SICI was significantly reduced in patients; no significant difference in SICI between smokers and non-smokers in patients or HCs.	N/A
Bajbouj et al. (2004)	cross-sectional	16 (12 M)	31.3 ± 10.5	57.8 ± 91.3 months	Brief Psychiatric Rating Scale (BPRS); PANSS; Global Assessment Scale (GAS); Extrapyramidal Motoric Symptom scale (EPS): 3.8 ± 5.7	5 patients were not taking antipsychotics or benzodiazepines; 11 were medicated: clozapine (n=2), olanzapine (n=2) , haloperidol (n=3), pimozid (n=1), amisulpride (n=2), fluphenazine (n=1), none took anticonvulsants, mood stabilisers or benzodiazepines	N/A	Post-excitatory inhibition and Ipsilateral transcallosal inhibition: stimulation intensity = 80% of maximum stimulator output, stimulation was given with maximally sustained contraction of bilateral FDI muscle	bilateral M1	RMT; duration of post-excitatory inhibition (from the onset of the EMG response to the end of the silent period, where the averaged tonic EMG activity again reaches the amplitude of the mean activity before the stimulus) in the contralateral FDI muscle; onset latency (the point where the averaged sustained EMG activity in the ipsilateral hand fell under the mean EMG amplitude before	Data from left and right hands were pooled since observed no significant side-to-side difference in patients or HCs. Durations of post-excitatory inhibition and of TCI were significantly longer in patients than HCs, whereas RMT and latency of TCI were not different between groups	Negative correlation between chlorpromazine equivalent and duration of post-excitatory inhibition was found. No sig correlation between the clinical scales and illness duration with the TMS measures

											the stimulus) and duration of transcallosal inhibition (measured from the onset latency until the EMG activity reaches the baseline level again) in ipsilateral muscle		
Fitzgerald et al. (2002b)	cross-sectional	20 olanzapine (17 M); 20 risperidone (15 M)	28.1 ± 9.9l (olan); 28.2 ± 8.4 (risp)	olan 4.7 ± 6.2; risp 5.3 ± 7.1	PANSS; Montgomery-Asberg Depression Rating Scale (MADRS); Abnormal Involuntary Movement Scale (AIMS); Assessment of Functioning (GAF)	Patients had been treated with their current dose for at least 14 days: olan mean dose 12.25 ± 6.1 mg, risp mean dose 4.1 ± 1.7 mg. Patients were not using lithium, mood stabilisers or other antipsychotics, or used benzodiazepines within 18h or testing	N/A	MEP size and CSP: sustained contraction of 5% of maximum force, stimulation intensity = 10%, 20%, 30% and 40% of RMT, 10 stimuli for each intensity. SICI & ICF: TS was set to produce MEPs of 0.5–1.0 mV, CS = AMT - 5%, (TS alone and at ISI of 1, 2, 3, 4, 10, 15 and 20 ms), ITI = 5s. Single-pulse TCI:	bilateral M1	RMT; AMT; peak-to-peak MEP size and latency; CSP duration (time that voluntary EMG activity reappeared minus the time of SP onset); SICI & ICF = conditioned MEP / unconditioned MEP. Onset and offset of single-pulse TCI (defined as the time points where the EMG trace fell persistently below and where it returned persistently to the baseline); single-pulse TCI duration (time of offset of TCI minus the onset). Dual-pulse TCI: resting TCI = %	The risp group showed higher RMT compared to the olan group. HCs had significantly longer CSP duration (higher the intensity, bigger the difference) than olan and risp groups but no significant difference between the 2 patient groups. Significant difference in single-pulse TCI duration between olan and HCs, and between olan and risp (HCs < olan > risp). Consistent SICI was seen at 1, 2, 3 and 4 ms and consistent ICF at 10 and 15 ms, no significant difference among the groups. For dual-pulse TCI, both medication groups	N/A

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							maximally sustained voluntary contraction on ipsilateral side of stimulation, applied at 155% of RMT. Paired-pulse TCI: 2 conditions - single TS to left M1, and a paired-pulse with the CS to the right M1 preceding the TS by 10 ms; the TS was set to produce a consistent MEPs of 0.5~1.5 mV in the contralateral APB muscle, CS = 125% RMT, the procedure was done twice, once at rest and once with the subject maintaining a 5%	reduction in the size of the MEP (peak-to-peak) in the TS alone condition vs paired-pulse condition; tonic TCI = MEP size of TS alone vs paired-condition, also duration of post-MEP silent period of TS alone vs paired-condition	showed significantly less reduction in MEP size in the paired condition at rest than HCs, but no difference between the medication groups. Also, the risp group showed significantly less reduction in the SP duration than olan group and HCs during tonic muscle contraction but no difference between olan and HCs

Ribolsi et al. (2011)	cross-sectional	16 medicated (15 M); 9 unmedicated for at least 1 month (8 M)	medicated 41.6 ± 9.4; unmedicated 40.5 ± 9.4	medicated: 19.2 ± 9.3; unmedicated 11 ± 8.8	Brief Psychiatric Rating Scale (BPRS); PANSS; Global Assessment Functioning (GAF)	medicated patients were taking typical and atypical antipsychotics: aripiprazole (n = 3); haloperidol (n = 5); clozapine (n = 4); amisulpride (n = 4); chlorpromazine (n = 1); risperidone (n = 3); quetiapine (n = 2). Other medications: antidepressants (n=3), benzodiazepines (n=14), mood stabilizer (n=1). None had extrapyramidal symptoms.	N/A	sustained contraction of the right APB TS intensity was adjusted to evoke a MEP of approx. 1 mV peak-to-peak in the relaxed left FDI, CS intensity = 110% RMT (inhibitory) or 80% AMT (facilitatory), ISI between CS and TS = 6, 8 and 15 ms, thus 4 conditions - TS alone and paired pulse with the 3 different ISIs, for each CS intensity, 20 pulses for TS alone and 10 trials for conditioned MEPs at each ISI	TS applied to the right M1; CS applied over left dorsal premotor cortex (defined as about 3cm anterior to M1)	RMT; AMT; peak-to-peak unconditioned MEP size; conditioned MEP size = % of unconditioned MEP size	No significant difference in RMT, AMT and unconditioned MEP size among medicated patients, unmedicated patients and HCs was found. Conditioned MEP size: medicated patients showed significantly less facilitation for CS = 80% AMT at ISI = 8 ms relative to HCs, for CS = 110% RMT; no significant difference among the 3 groups at any ISI.	Significant negative correlation between conditioned MEP size (at 80% AMT and 8 ms ISI) in patients and PANSS negative, but no correlation with PANSS positive, GAF, illness duration, (in medicated patients) chlorpromazine or diazepam. Regression analysis showed that conditioned MEP size was only predicted by PANSS negative score (standardized b = -.61)

Yildiz et al. (2015)	longitudinal: patients were followed up after 8 weeks to test the effect of a new atypical antipsychotic, TMS and PANSS administered on the 4th day (visit 1) of the new drug therapy, on the 5th day administered cognitive tests, 8 weeks later TMS, PANSS and cognitive tests were repeated	13 (5 M)	37.69 ± 9	12.31 ± 6.68	PANSS	Patients were switched to a new atypical antipsychotic (due to symptoms exacerbation) by the start of the study, during the 8 weeks 6 were on clozapine, 3 olanzapine, 3 risperidone and 1 quetiapine. Prior to the study, 3 were medication naïve and the others were on antipsychotics	N/A	CSP: intensity = 120% RMT, 10 trials. SICI & ICF: CS = sub-RMT, TS = supra-RMT, ISI = 1, 2, 3, 4, 5, 6 (for SICI) and 7, 8, 9, 10, 12, 14 (for ICF) ms. Single-pulse transcallosal inhibition (TCI): intensity = 155% RMT to the right ipsilateral M1, 10 trials	left M1	RMT; CSP duration = time between the onset and the termination of the EMG suppression; SICI & ICF = (conditioned MEP amplitude / unconditioned MEP amplitude) x 100; TCI: duration of the ipsilateral silent period = time between the onset and the end of EMG suppression	11 patients underwent repeat measurements at the end of the 8th week. Significant decrease in total, positive and general psychopathology scores after 8 weeks compared to baseline. No significant difference in RMT between HCs, and patients visit 1 & 2. CSP was significantly longer in the patients after 8 weeks relative to the baseline measurements in the controls. ICF was weaker in patients relative to HCs at the end of the 8th week for ISIs of 7, 8, 9, 10 and 12 ms; ICF significantly decreased at visit 2 compared to visit 1 for ISI of 14 ms. SICI did not differ between HCs and patients at visit 1 & 2 for any ISI. TCI was significantly longer in patients at visit 1 compared to HCs.	At visit 2, decreased SICI at 3 ms correlated with increasing chlorpromazine (higher the dose, larger the SICI). At visit 1, PANSS total and the general psychopathology scores were correlated with increased SICI (ISI 3 ms) (i.e. higher the PANSS scores, bigger the inhibition). SICI (ISI 3 ms) was also positively correlated with Visual Reproduction 1 and 2 test scores, the animal and name subtests of the Category Fluency Test, performance on the R-AVLT test with respect to the R-AVLT 1-5 cumulative learning test scores, R-

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AVLT 6 , R-AVLT 7 delayed recall scores, R-AVLT correct recognition scores, and the R-AVLT discrimination scores; and negatively correlated with R-AVLT wrong recognition scores, and Stroop Test scores on the word-colour subtest time and mistakes. Between visits 1 & 2, decrease in PANSS general psychopathology was positively correlated with decrease in ICF (ISI 7 ms) and change in SICI (ISI 3 ms) was correlated with changes in auditory verbal memory performance (positive correlation) as

												indicated by R-AVLT 7, and the Stroop Test word-colour subtest (negative correlation) .
Ustohal et al. (2017)	longitudinal: effect of risperidone was assessed at 4-week follow-up	13 hospitalized patients with first-episode SCZ	25.92 ± 4.81	N/A	PANSS	Patients were drug-naïve at baseline, no cytochrome P450 inhibitor or inductor, or benzodiazepines in the month before or during the study.	N/A	CSP: intensity = 150% RMT, measured over the moderately-activated right ADM muscle, CSP was assessed before risperidone treatment was initiated, and again 4 weeks later	left M1	RMT; CSP duration = interval between the end of MEP and the return of voluntary EMG activity	CSP data were unavailable from 1 patient due to technical difficulties. Risperidone significant increased CSP duration after 4 weeks of treatment. Did not find a significant difference between smokers and non-smokers. RMT at baseline (45% of maximal output) and after 4 weeks (46.1% of maximal output) did not differ.	No significant correlation between change in CSP duration and change in any PANSS scores
Bagewadi et al. (2019)	cross-sectional	45 (61.5% M)	28.6 ± 4.5	81.43 ± 57.35 months	Scale for the Assessment of Negative Symptoms (SANS); Scale for the Assessment of Positive Symptoms (SAPS)	the majority of patients were on atypical antipsychotics	Block 1 - rest; Block 2 - neutral action observation; Block 3 - context-based action observation	Single pulses were delivered with the intensity (SI-1mV) required to elicit ⩾ 1 mV MEPs. SICI: CS = 80% RMT, TS = SI-1mV, ISI = 3 ms. ICF:	left M1	RMT; SI-1mV and MEP size evoked by SI-1mV; SICI & ICF = [conditioned MEP/non-conditioned MEP] × 100; putative mirror neuron system (MNS) activity = (MCR during action observation - MCR during rest) x 100 / MCR	At block 1, RMT, SI-1mV and MEPs elicited by SI-1mV were similar between patients and HCs; patients had significantly lower ICF but similar SICI. Putative MNS activity was significantly greater during block 3 (block 3 minus block 1) than block 2 (block 2 minus	Social cognition was assessed using Social Cognition Rating Tools and Recognize Emotions in Neuropsychiatric Disorders. An average of individual test performance indices from these tests was

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								CS and TS same as SICI, ISI = 10 ms. Participants received 14 single pulses, 14 SICI and 14 ICF, ITI = 5 sec while observing each of the 3 blocks; in the 3rd block, stimuli were delivered in a time-locked manner to coincide with the last 70 sec of the video that depicted goal-directed actions of the FDI muscle		during rest, it measures the % change of cortical reactivity from block 1 to either block 2 or block 3, MCR stands for motor cortical reactivity and refers to MEP, SICI & ICF	block 1) in both groups for MEP size, SICI & ICF; however, the increase in MNS activity during block 3 was bigger in HCs than patients in SI-1mV-MEP size and SICI but not ICF.	used to calculate the social cognition composite score. The social cognition composite score (n = 31) had a sig positive correlation with MNS-activity (ICF) during block 3.
Tang et al. (2014)	cross-sectional	17 inpatients (9 M)	31.71 ± 9.00	7.58 ± 4.72	PANSS	All patients were taking antipsychotics, 14 on monotherapy, and 3 were receiving 2 antipsychotics	N/A	CSP duration: 20% sustained contraction of the right APB muscle, intensity = 120% RMT,	left M1	RMT; 1 mV-MEP size; CSP duration = time from MEP onset to the recovery of voluntary EMG activity; SICI & ICF = conditioned MEP amplitude /	No significant difference in RMT between patients, people at ultra-high risk of psychosis (UHR) and HCs was found. MEP was smaller in patients than HCs. Patients showed a reduced	A negative correlation was found between PANSS positive score and 1 mV-MEP size and CSP duration. Also, PANSS

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								10 trials. SICI & ICF: CS = 80% RMT, TS was set to produce an average MEP of 1 mV in 5/10 trials, ISI = 3 (SICI) and 10 (ICF) ms, 10 trials each		unconditioned MEP amplitude	SICI than HCs, whereas no significant difference in ICF was found among groups. CSP duration was longer in patients and UHR than in HCs, but no difference between patients and UHR was found.	negative was sig correlated with 1mV MEP (positive correlation) and ICF (negative correlation)
Daskalakis et al. (2008a)	cross-sectional	6 unmedicated (4 M); 10 clozapine treated (7 M)	32.3 ± 9.8 (unmedicated); 30.2 ± 6.5 (clozapine-treated), mean duration of treatment was 2.5 ± 2.4 yrs	N/A	PANSS	6 patients were unmedicated for 1 month or more, 10 were taking clozapine	N/A	CSP: sustained moderate contraction of the right APB, intensity = 110% and 140% RMT, 10 trials at each intensity. SICI & ICF: CS = 80% RMT, TS was adjusted to produce an average MEP of 0.5–1.5 mV peak-to-peak amplitude in the contralateral APB, ISI = 2, 4	left M1	RMT; absolute CSP duration = time from the MEP onset to the return of any voluntary EMG activity; SICI & ICF = mean conditioned MEP amplitude (peak-to-peak) / mean unconditioned MEP amplitude	No significant difference in RMT between unmedicated and clozapine-treated patients and HCs was found. In all 3 groups stimulation at 140% RMT produced longer CSP duration than 110% RMT; for each intensity, clozapine treated patients had significantly longer CSP than unmedicated patients and HCs. No significant difference among groups was found in unconditioned MEP size, SICI and ICF.	In all patients, a significant positive correlation was found between PANSS positive scores and SICI (averaged across ISIs of 2 and 4 ms), whereas no significant correlations were found between this parameter and PANSS negative or global scores. Also, ICF (averaged across ISIs of 10, 15, 20 ms) was positively correlated with PANSS

								(inhibitory), 10, 15 and 20 (facilitatory) ms				positive score, but not with other symptoms dimensions.
Takahashi et al. (2013)	cross-sectional	20 (9 M)	27.4 ± 6.5	19.8 ± 12.5 months (duration of illness less than 3 yrs)	PANSS	unmedicated patients (n=3); 2nd generation antipsychotics (n=8); 2nd generation antipsychotics and benzodiazepines (n=7); benzodiazepines (n=2)	N/A	SICI & ICF: CS = 80% RMT, TS = 130% RMT, ISI = 2, 3 (SICI), 10, 15 (ICF) ms, ITI = 10s, 10 trials each for TS alone and the 4 ISIs	left M1	RMT; SICI & ICF = mean conditioned MEP / mean unconditioned MEP	No significant difference in RMT between patients and HCs. SICI & ICF: there was a significant effect of ISI (all 4 ISIs entered into an ANOVA) on MEP ratio (conditioned MEP/unconditioned MEP) in both groups, but no significant difference in MEP ratio between ISIs of 2 and 3 ms, and no difference in MEP ratio between ISIs of 10 and 15 ms; patients had sig less SICI (i.e. higher MEP ratio) than HCs; no significant difference found in ICF between groups.	Daily dose of antipsychotics and benzodiazepines did not correlate with any of the PANSS scores. In patients, SICI showed a significant negative correlation with the digit sequencing task (working memory) score, i.e. higher the task score, the more inhibition; SICI was not correlated with age, illness duration, daily dose of antipsychotics or benzodiazepines, or any PANSS scores. The TMS outcomes did not correlate with cognitive test

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											performance on list learning (verbal memory), token motor task (motor speed), category fluency and letter fluency (verbal fluency), symbol coding (attention and speed of information processing), and Tower of London (executive function)	
Daskalakis et al. (2008b)	cross-sectional	14 medicated (10 M); 6 unmedicated (4 M)	32.57 ± 11.71 (medicated); 32.67 ± 9.67 (unmedicated)	N/A	PANSS; Motor abnormalities assessed by Abnormal Involuntary Movement Scale (AIMS), Simpson-Angus Scale (SAS), Barnes Akathisia Scale (BAS)	6 patients were antipsychotics free for 1 month or longer, 14 were on a single antipsychotic	Use-dependent plasticity paradigm to assess time-limited reorganization of motor circuits: (1) measure the spontaneous direction of TMS-induced thumb movements; (2) train participants to produce brisk thumb movements opposite (180 degrees) to this	stimulation intensity = the lowest intensity necessary to produce consistent thumb movements in 1 axis (i.e. direction as in abduction/adduction or flexion/extension), stimuli were delivered at a frequency of 0.1 Hz for 10 minutes (i.e. 60 stimuli)	left M1	RMT; direction and acceleration of TMS-induced thumb movement	No significant difference in RMT among medicated and unmedicated patients and HCs. No significant difference across the 3 groups in TMS-induced acceleration (the "briskness") at baseline. Significant difference between unmedicated patients and	No association between training direction or accelerations and post-training orientation across all participants

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							baseline direction for 30 minutes at frequency of 1 Hz; and (3) measure the direction of TMS-induced thumb movement after training during the course of 30 min				HCs and between medicated patients and HCs was found in post-training thumb direction (measured by angular displacement), but not between medicated and unmedicated patients. Post-training acceleration: no significant difference among groups on TMS-induced movement amplitudes was found following training	
Daskalakis et al. (2002)	cross-sectional	15 unmedicated (14 med-naïve, 1 med-free for longer than 1 year, 8 M), 13 assessed in the TCI paradigm; 15 medicated (10 M)	unmedicated 33.1 ± 9.3; medicated 32.4 ± 9.0	unmedicated 8.5 ± 7.2; medicated 3.9 ± 5.8	PANSS; motor abnormalities assessed by Abnormal Involuntary Movement Scale (AIMS), Simpson-Angus Scale (SAS), and Barnes Akathesia Scale (BAS). No evidence of motor abnormalities in patients	medicated patients: olanzapine (n=11), risperidone (n=1), quetiapine (n=1), quetiapine + loxapine (n=1), methotrimeprazine + perphenazine (n=1)	CSP: sustained moderate contraction of right FDI, intensity = 10%, 20%, 30%, and 40% above AMT, 15 trials for each intensity. SICI & ICF: CS = 80% RMT, TS was adjusted to produce an average MEP of 0.5 to 1.5 mV peak-to-peak amplitude in the	left M1, for TCI it's left and right M1	RMT; CSP duration (time from the MEP onset to the return of any voluntary EMG activity) and MEP size; SICI & ICF = mean conditioned MEP size / mean unconditioned MEP size; TCI = mean conditioned	Unmedicated patients showed significantly lower RMT than medicated patients and HCs, but no difference between medicated patients and HCs. SICI: significant difference between unmedicated and HCs; when	In all 30 patients, SICI (averaged across ISI = 2 and 4 ms) was correlated with PANSS total (Pearson r = 0.50, spearman rank ρ = 0.53), positive (r = 0.46, ρ = 0.53) and global (r = 0.53, ρ = 0.56) scores but not with PANSS negative. No

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			contralateral FDI, ISI = 2, 4, 10, 15, and 20 ms, 12 trials for each condition (TS alone and 5 CS-TS pairs), ITI = 5 sec. Dual-pulse transcallosal inhibition: suprathreshold CS to the right M1, suprathreshold TS to the left M1, both set to produce MEPs of 0.5 to 1.5 mV peak-to-peak amplitude in the contralateral FDI, ISI = 2, 6, 10, 15, and 20 ms, ITI = 5 sec, 12 trials for each condition (TS alone and 5 CS-TS)	MEP amplitude / mean unconditioned MEP amplitude	averaged across both inhibitory ISIs (2 and 4 ms) unmedicated patients showed 31.2% less inhibition than HCs, and medicated patients showed 15.64% less inhibition than HCs. ICF: no group differences. CSP duration: significant differences between unmedicated and HCs (at 40% above the AMT) and between medicated and unmedicated (at 30% and 40% above the AMT) but not between medicated and HCs; when averaged across all intensities, unmedicated were 15.26 ms less than HCs who were 5.38 ms less than medicated; no significant	correlation between other TMS measures and PANSS scores

											group difference in CSP-MEP size. TCI: inhibition begins at ISI = 6 ms, significant difference between unmedicated and HCs but not between unmedicated and medicated or medicated and HCs; when averaged across all ISIs (2~20 ms), unmedicated showed 23.25 % less inhibition than HCs, medicated showed 9.92% less inhibition than HCs.	
Bridgman et al. (2016)	cross-sectional	11 (7 M)	38.5 ± 9.0	N/A	PANSS	patients were on a stable dose of antipsychotic medications for at least 1 month	Working memory performance was assessed by verbal N-back task (N = 1 and 3), 30 min after TMS assessments on the same day	Stimulation intensity = the intensity (SI-1mV) that elicits an average MEP amplitude of approximately 1 mV peak-to-peak in the relaxed APB muscle. LICI: CS = TS = SI-1mV, ISI = 100, 150 and 200 ms. SICI & ICF: CS = 80% RMT, TS = SI-1mV, ISI	left M1	RMT; LICI; SICI & ICF = 1 minus (mean conditioned MEP amplitude / mean unconditioned MEP amplitude); CSP duration = from time of MEP onset to the return of voluntary	Patients had significantly higher RMT than HCs. No difference in LICI was seen between groups; there was more LICI in the 100 ms ISI compared to both the 150 ms and the 200 ms ISIs regardless of diagnosis. There was	When patients' and HCs' data were pooled, 3-back task accuracy was associated with more SICI in the 4 ms ISI, but not in the 2 ms ISI. When separated by diagnosis, this trend was "approaching significance" only in the patients, (p =

								= 2, 4 (SICI), 10, 15, 20 (ICF) ms. CSP: intensity = 140% RMT, sustained 20% maximum contraction of APB muscle		EMG activity that is half the size of the background EMG	significantly less SICI in the 2 ms and 4 ms ISI in patients compared to HCs; and there was significantly more SICI in the 2 ms ISI than the 4 ms ISI regardless of diagnosis. No difference between groups in CSP duration or ICF.	0.09) and not in the HCs (p = 0.14). A negative correlation was also found between RMT and 3-back performance across all subjects.
Fitzgerald et al. (2004)	TMS measures of cortical excitability were made before and after a period of rTMS, but SICI and ICF were only recorded before the rTMS train	medicated 16 (10 M); unmedicated 10 (8 M)	32.2 ± 8.8 (medicated); 32.6 ± 8.3 (unmedicated)	8.8 ± 10.4 (medicated); 6.4 ± 5.1 (unmedicated)	PANSS; Simpson-Angus rating scale (SA); Global Assessment of Functioning (GAF).	Unmedicated patients had not been treated with any oral antipsychotic for at least 3 months or depot for at least 12 months. Medicated patients were receiving a single antipsychotic for a minimum of 1 month: olanzapine (n=7); risperidone (n=4); quetiapine (n=5). No concurrent treatment with anticonvulsant or lithium, and excluded who were regularly taking a	N/A	AMT: sustained contraction of right APB muscle at 5% of maximum force. MEP size: intensity = 120% RMT, 10 trials. CSP: stimulation intensity = 120% AMT, sustained contraction at 5% of maximum, 10 trials. SICI & ICF: CS = 5% below the AMT, TS was adjusted to produce MEPs of 0.5~1.0 mV, ISI = 2 (SICI) and 15 (ICF) ms, 10 trials for each condition (TS alone and 2 CS-TS pairs), ITI = 5	left M1	RMT; AMT; MEP size (measured as the area under the curve); CSP duration (measured from the time of stimulation to the return of spontaneous EMG activity); SICI & ICF = 1 minus (mean conditioned MEP size / mean unconditioned MEP size). Change in RMT, MEP size and CSP duration were	No difference in RMT, AMT, MEP area or ICF between medicated and unmedicated patients and HCs at baseline, whereas both patient groups had a shorter CSP duration than the HCs, and reduced SICI was found in medicated patients than HCs but found no difference between unmedicated and any group. Comparing response to rTMS (post-	Significant positive correlation between change in RMT and baseline CSP duration (i.e. participants with a shorter CSP duration at baseline had less change in RMT following rTMS). No relationship found between the baseline or the change scores of the TMS outcome measures with the psychopatholog y scales for the

					benzodiazepine, who had taken any long acting benzodiazepines in the previous 3 days or a short acting benzodiazepine within 18 h of testing	sec. rTMS: a single 15-min train, intensity = 110% RMT, frequency 1 Hz	calculated by subtracting the pre-rTMS scores from the post-rTMS scores	minus pre-rTMS) across groups: significant difference was found in change in RMT between HCs and unmedicated and between HCs and medicated (HCs > medicated and unmedicated), no difference between the 2 patient groups. Also there was an increase in AMT level in the control and medication treated group but not the unmedicated patients. No difference in change in MEP size among groups. There was a significant decrease in CSP duration in the HCs and an increase (not significant) in CSP duration in both patient groups. The	patient groups (pooled)

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											results did not change after excluding left-handed.	
Strube et al. (2016)	The anodal-tDCS and PAS sessions were 4~8 days apart	20 (13 M)	31.5 ± 9.0	7.1 ± 5.9	PANSS; GAF; CGI	medication free (n=1), the others were taking stable ongoing antipsychotics for 1 week before testing (9 on monotherapy, 10 on a combination of 2 antipsychotics)	N/A	The stimulation intensity corresponded to an average MEP amplitude of 1 mV (S1 mV). 40 single stimuli were delivered at baseline; after PAS and tDCS, 20 single stimuli delivered at time-points 0, 5, 10, 20 and 30 min. SICI & ICF were obtained at baseline and 15 mins after the plasticity protocols, CS = 80% RMT, TS = S1 mV (intensities not adjusted after plasticity protocols), ISI = 2, 3 (SICI), 7, 9, 12 (ICF) ms. Anodal-tDCS: intensity = 1 mA, duration = 13 min,	left M1	RMT; S1 mV; SI-1 mV MEP size; SICI & ICF (absolute MEP values of each condition in mV)	Baseline of A-tDCS session: no significant group differences for RMT, S1 mV, SI-1 mV MEP, and ICF; significantly less SICI (i.e. higher absolute MEP values) in patients than HCs at ISI = 2 and 3 ms and averaged 2 & 3 ms. Baseline of PAS session: reduced SICI in patients at ISI = 2 and 3 ms and averaged 2 & 3 ms, and reduced ICF in patients at ISI = 9 ms and mean 9~12 ms value. LTP induction via tDCS (pre- vs post-tDCS): In HCs, significant increase in MEP size at 0 and 20 min and on average over all time points; In patients, all time points showed significant MEP size increase. LTP induction via PAS	The averaged MEP size of all time points following tDCS was correlated positively with PANSS positive, negatively with PANSS general psychopathology, total, and CGI scores, and positively with GAF, but these correlations would not survive corrections for multiple comparisons and thus have to be interpreted with caution. SCZ smokers showed a significant negative correlation between Fagerstrom values (for nicotine dependence)

								anodal electrode positioned on the left M1 (area for the right FDI muscle), cathodal electrode placed above the ipsilateral right orbit. PAS: 180 pairs of peripheral nerve (ulnar nerve) stimuli (PNS) followed by TMS stimuli with an ISI of 25 ms, PNS intensity = 300% of the individual perceptual threshold, resulting in an average electrical intensity of 8.3 ± 2.1 mA, TMS intensity = S1 mV		(pre- vs post-PAS): In HCs, significant MEP increase for all post-PAS time points compared to baseline and for the average of all time points; In patients, no increase in MEP size. SICI & ICF after tDCS: significant increase in absolute MEP size for TS alone, 3 ms SICI, and 7, 9 & 12 ms ICF in patients. SICI & ICF after PAS: significant increase in MEP size for TS alone, and 7 and 12 ms ICF in HCs.	and mean post-tDCS MEP size
Fitzgerald et al. (2003)	cross-sectional	9 medicated (6M); 9 unmedicated (6M)	medicated 27.7 ± 5.1; unmedicated 33.8 ± 8.2	medicated 4.11 ± 3.05; unmedicated 6.0 ± 4.81	PANSS; Montgomery–Åsberg Depression Rating Scale (MADRS); Simpson-Angus rating	9 patients had not been treated with any oral medication for at least 3 months or depot for at least 12 months; 9 were taking a single antipsychotic medication for at least	N/A	LICI: CS = TS = the intensity that produced an average MEP of 0.5~1.0 mV (about 20% above RMT),	left M1	RMT; LICI = mean conditioned MEP size / mean unconditioned MEP size; I-wave facilitation	No difference in RMT among medicated and unmedicated patients and HCs. A significant degree of LICI was seen in all	No significant correlation between I-wave facilitation and LICI, or between I-wave

					(parkinsonism); GAF. Symptom severity rating was done within 48 h of the testing procedure	1 month (4 on olanzapine, 2 risperidone, 2 quetiapine). Participants were not taking anticonvulsants or lithium, or long-acting benzodiazepines, and short-acting benzodiazepines were not permitted within 18 h of testing	ITI = 5 sec. I-wave facilitation: CS intensity was set to produce an average MEP of 1 mV, TS = 90% RMT, ISI = 1.2 (to assess I-wave facilitation during the 1st I-wave peak) and 2 (facilitation does not usually occur, a control condition) ms	= conditioned MEP size / unconditioned MEP size	groups, but no significant difference among the groups. Significant I-wave facilitation was seen in all groups and the degree of facilitation was greatest in the medicated group and least in the HCs, there was a significant difference between the HCs and medicated group; at ISI = 2 ms, no facilitation was seen and no difference among groups.	facilitation and psychopathology and parkinsonism scores (data pooled for the patient groups)
Du and Hong (2018)	The inter-session interval for patients was 24.60 ± 19.25 days, and for HCs was 29.41 ± 23.88 days	25 (18 M)	36.86 ± 13.65	16.40 ± 15.2	BPRS	medication free (n=5), typical antipsychotics (n=1), atypical antipsychotics (n=18), both types (n=1), anti-depressants (n=1). No change in medication or dose between the 2 testing sessions	N/A	SICI & ICF: CS = 80% RMT, TS = 120% RMT, ISI = 1, 3 (SICI), 6, 9, 12, 15, 18, 21, 30, 40, 80, 120, 200 and 500 (ICF) ms, ITI = 4~10 sec	left M1	RMT; SICI & ICF (ratio) = mean conditioned MEP / mean unconditioned MEP	No significant difference in RMT between sessions or groups. MEP amplitudes evoked by 120% RMT did not show significant difference between sessions in HCs and patients, suggesting a stable and comparable cortical excitability at M1 between sessions and groups. Significantly

											reduced SICI in patients at ISI = 1 and 3 ms (ratio merged from the 2 sessions). Patients and HCs did not differ in ICF at ISIs of 9, 12 and 15 ms (the 2 sessions are merged), and none of the other ISIs showed sig group differences.	
Mehta et al. (2014a)	cross-sectional	33 antipsychotic-naive (18 M); 21 medicated (9 M)	33.60 ± 9.74 (med-naïve); 9.19 ± 6.60 (medicated)	41.12 ± 44.20 (med-naïve); 50.42 ± 42.65 (medicated) months	PANSS	Patients in the medicated group were taking atypical antipsychotics (12 risperidone, 4 olanzapine, 3 risperidone + olanzapine, 1 olanzapine + amisulpride, and 1 aripiprazole) with median duration of treatment being 60 days	Participants viewed a static picture of a hand and lock while receiving TMS measurements	MEPs obtained with (a) stimulus intensity equal to 120% of RMT and (b) SI-1 mV. SICI: CS (80% of RMT) was given 3 ms before a TS (SI-1 mV) with the right hand at rest. LICI: A supra-threshold CS (SI-1 mV) was given 100 ms before a supra-threshold TS (SI-1 mV).	left M1	MEPs amplitude; RMT; SICI & LICI (conditioned MEP / non-conditioned MEP) × 100	Baseline cortical excitability during the rest state (i.e. viewing a static picture): No significant difference in RMT, SI-1 mV, LICI, MEPs amplitude with 120% RMT or SI-1 mV among the medication-naive patients, medicated patients and HCs. Antipsychotic-naïve patients had significantly less SICI compared to HCs, but no significant difference in SICI between the medicated patients and HCs.	Theory of mind (ToM) and social perception were assessed using the Social Cognition Rating Tools in Indian Setting (SOCRATIS). Emotion processing was assessed using the Tool for Recognition of Emotions in Neuropsychiatric Disorders (TRENDS). Each test provided an index of the respective test performance,

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which was equivalent to the score of an individual on the test divided by the maximum score possible. An average of z-scores of the 3 individual tests formed the SC composite score. SICI was inversely correlated with SC composite score in drug-naive patients. Among the individual SC dimensions, emotion recognition index had the strongest inverse correlation with SICI. Linear regression showed that group status (medicated vs drug-naive) significantly predicted SC composite score, and

												SICI was not a significant predictor.
Hasan et al. (2011)	cross-sectional; TMS measurements were performed in the same order within an experimental session before and after a 13 min train of tDCS. All post-tDCS measurements were conducted within 30 min after the tDCS-intervention.	9 with recent-onset SCZ; 13 with multi-episode SCZ.	29.33 ± 7.8 (RO-SCZ); 36.00 ± 8.0 (ME-SCZ)	RO-SCZ had a single psychotic episode (lasting at least for one month), no relapse and a duration of psychosis less than 2 years. ME-SCZ had had more than two psychotic episodes, at least one relapse and a duration of psychosis more than 2 years	PANSS; GAF; CGI. ME-SCZ showed a higher disease severity, reduced social functioning (GAF) and increased psychopatholog y as indicated by a higher PANSS total score compared RO-SCZ.	20 patients were treated with antipsychotics (14 in monotherapy, 6 with risperidone and 6 with quetiapine). In the RO-SCZ group, 1 patient received citalopram and 1 diazepam. In the ME-SCZ group, 1 received mirtazapine, 1 biperiden/ diazepam and 1 biperiden/Lorazepam.	N/A	MEPs were recorded before tDCS and 5 min after the stimulation. SICI and ICF: CS = 80% RMT, TS intensity = SI-1 mV, ISI = 3 (SICI) and 12 (ICF) ms. CSP duration: stimulus intensity = SI-1 mV (25~30% of maximal contraction). For SICI, ICF, and CSP measures, RMT and SI-1 mV were adjusted after tDCS. Anodal tDCS: The anodal electrode was placed over the representation al field of the right FDI as identified by TMS, and the cathodal electrode was	left M1	RMT; MEPs amplitude evoked by SI-1 mV; SICI & ICF = (mean amplitude of conditioned MEP / mean amplitude of unconditioned MEP); CSP duration	At baseline, patients presented significantly elevated RMT and reduced SICI compared to HCs (differences did not occur in the 3-group comparison). No difference in ICF, CSP duration or SI-1 mV MEP size among RO-SCZ, ME-SCZ and HCs at baseline. After tDCS, there was significant facilitation of 1 mV-MEP within all groups; HCs showed higher 1 mV-MEPs compared to ME-SCZ, but not compared to RO-SCZ; RO-SCZ showed a trendwise higher 1 mV-MEPs compared to ME-SCZ (p = 0.084). No change in SICI within patients (n=22) and HCs (pre- vs post-tDCS) and patients still had	No correlation was found between the main outcome parameters and antipsychotic dose, PANSS, CGI, GAF, number of psychotic episodes and duration of psychosis across the patients groups and for each patient group separately

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								located contralaterally above the right orbit, a continuous current flow of 13 min with an intensity of 1 mA was used to induce changes in motor cortical excitability				reduced SICI than HCs; compared to baseline, only RO-SCZ showed an enhanced SICI, and RO-SCZ had an enhanced SICI (more inhibition) compared to ME-SCZ after tDCS, but no difference between RO-SZ and HCs post-tDCS. All 3 groups showed change in CSP duration post-tDCS (didn't specify in which direction). No change in ICF post-tDCS in all 3 groups.
Koch et al. (2008)	cross-sectional	14 medicated patients (11 M); 6 unmedicated patients (5 M)	35.71 (medicated); 34.3 (unmedicated)	14.28 (medicated); 9.16 (unmedicated)	PANSS; GAF; SAS; BAS; AIMS	Medicated patients were taking typical or atypical antipsychotics (aripiprazole, n=3; clozapine, n=3; amisulpride, n=2; olanzapine, n=1; chlorpromazine, n=1; risperidone, n=4). 13 patients were taking benzodiazepines (lorazepam, n=6; alprazolam, n=2; lormetazepam, n=4; diazepam, n=1)	N/A	Cortico-cortical connectivity between the posterior parietal cortex (PPC) and the M1 was tested with a twin-coil procedure. CS was applied at different intensities over the right PPC before MEPs were obtained by	For right PPC, the coil was positioned over P4; right M1	MEP amplitude	No significant difference in RMT or SI-1 mV MEP among the medicated patients, unmedicated patients and HCs. The strength of parieto-motor connectivity differed between SCZ patients and the HCs, both medicated and unmedicated patients in	In patients (n=20), the mean amount of facilitation across ISIs induced by the CS at 90% RMT correlated with GAF score ($r = .46$; $p < .05$) and the PANSS negative score ($r = -.48$; $p < .05$), showing that patients with a

								right M1 stimulation with participants at rest. CS = 110% or 90% RMT (RMT was tested over the ipsilateral M1); TS intensity = SI-1 mV in the relaxed left FDI. ISIs between CS and TS were 2, 4, 6, 8, 10, and 15 ms.			
										comparison with HCs had less facilitation for CS intensity = 90% RMT at ISI = 2, 4 and 15 ms. In HC, CS applied over the ipsilateral PPC at 90% of RMT intensity was able to increase the excitability (i.e. increased the MEP amplitudes) of the hand area of the right M1, with peaks at ISIs of 4 and 15 msec but failed to induce any facilitatory parieto-motor interaction in medicated and unmedicated patients.	better global functioning and lower negative symptoms had less impaired connectivity. The same parameter positively correlated with illness duration. No other correlations were found. In the medicated group the facilitation across ISIs at 90% RMT did not correlate with the CPZ equivalent dose or with benzodiazepines (i.e. diazepam equivalents)
Reid et al. (2002)	cross-sectional; patients and HCs had TMS measurements at rest and after exercising the APB muscle	11 with SCZ (9 M); 10 with major depressive episode (4 M)	27.27 ± 6.25 (SCZ); 48.30 ±12.84 (MD)	N/A	N/A	In SCZ patients, 4 were taking risperidone, 3 olanzapine, 3 clozapine and 1 quetiapine	N/A	TMS was administered at 110% RMT. 16 baseline recordings were produced using trains of 4 with an ISI of 3 sec, and trains were separated by 30 sec.	left M1	MEP; facilitation (calculated in 2 forms: post-minus pre-exercise MEPs; % increase of post-exercise amplitudes over mean baseline amplitudes)	No difference in RMT among SCZ, MD and HCs. At baseline, HCs had significantly lower MEP amplitude than the other 2 groups. Facilitation (post-exercise MEP as % of increase compared to

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							Participants then asked to perform a hand exercise (first, oppose the thumb using a dynamometer as hard as possible to determine the maximal voluntary contraction; at least 10 minutes later the participant was asked to exercise for 30 seconds at 20% of their maximal voluntary contraction using the same dynamometer) followed by 16 post-exercise recordings (4 trains) at the same stimulus parameters	baseline): the 2 patient groups was significantly lower than the HCs but did not differ from each other. Facilitation (post-minus pre-exercise MEPs): the MD group was significantly lower than the HC and SCZ groups.

Soubasi et al. (2010)	cross-sectional	51 (33 M)	34.4 ± 8.5	9.0 ±7.7	Acute extrapyramidal symptoms and tardive dyskinesia were initially evaluated at study entry using the Simpson-Angus scale (SAS) and the Abnormal Involuntary Movement scale (AIMS), respectively. Patients who had a score ⩾ 1 on any item of either scale were excluded.	Antipsychotics remained unchanged for type and dose for at least 2 months. 39 were treated with a single atypical antipsychotic and 12 with a combination of a typical and an atypical drug (olanzapine, n=20; quetiapine, n=8; ziprasidone, n=11; olanzapine and haloperidol, n=4; quetiapine and haloperidol, n=2; ziprasidone and haloperidol, n=6). None was receiving anticonvulsants or benzodiazepines.	N/A	Stimulus intensity was initially set at 70–80% of maximum output and increased and decreased by 2% steps to ensure supramaximal stimulation. SI-max = the lowest stimulus intensity required to produce maximum MEP. After 3 min, the RMT was estimated. Train of responses to SI-max were then recorded from each muscle (the left and right APB) and the MEP with the highest amplitude, which was most often the one with the shortest latency as well, was selected for analysis. Frequency	bilateral M1	SI-max; with intensity = SI-max, MEP amplitude and latency (from stimulus artifact to onset of negative peak); RMT; SP1 (silent period obtained by SI-1); SP2 (silent period obtained by SI-2)	Left M1: RMT, SI-max and MEP latency (ms) were significantly higher/longer in patients than HCs; no significant difference in MEP amplitude between groups; and no significant side-to-side difference in SI-max, RMT, MEP latency and amplitude in patients or HCs. SP1 (ms), but not SP2 was significantly longer in patients. Right M1: RMT, SI-max and MEP latency were significantly higher/longer in patients than HCs; no group difference in MEP amplitude. SP1, but not SP2 was significantly longer in patients. However, when SP1 was expressed as a ratio over the corresponding stimulus intensity applied in each participant (i.e.	They explored the relationship between the 2 SPs to the corresponding 2 SIs in each individual: a positive correlation of individual SP2-SP1 difference to the corresponding SI2-SI1 difference, which was significant for HCs but not for patients, was found (data for the right and left hemispheres are pooled together)

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								was 0.2~0.3 Hz. Silent period was determined while the participant exerted isometric contraction at 80% of maximal voluntary contraction; stimulus intensity = 130% of RMT (SI-1) and 90% of maximal output (SI-2)			SP1/SI-1), no group difference was found. Patients on ziprasidone (n=24) demonstrated the highest SI-max for both hemispheres (difference not significant in right cortex), and the highest RMT for the left hemisphere; patients receiving olanzapine (n=17) demonstrated the lowest RMT for the left hemisphere, and those on quetiapine (n=10) showed intermediate values.	
Ahlgren-Rimpilainen et al. (2013)	cross-sectional	11 hospitalised patients (6 M)	42.6 ± 13.7	22.5 ± 12.63 (conventional antipsychotics users, n=6); 11.00 ± 8.94 (atypical antipsychotics users, n=5)	PANSS; AIMS; Barnes Akathisia Scale (BAS); SAS for extrapyramidal symptoms; Calgary depression scale for depression associated with SCZ	4 patients were on clozapine treatment and 1 was using zotepine (an AA agent like clozapine). 6 used combinations of CA (2 CA users had additionally risperidone, but because they showed clinically significant extrapyramidal signs, they were assessed to belong to the group of CA users). 1 AA user	N/A	Biphasic pulses with an intensity of 60 to 80% of max output were applied, the stimulation intensity constantly exceeded the motor threshold. In each series of stimuli, 5 stimuli were given with an	bilateral M1	Latency and duration of CSPs: because multiple CSPs were observed (i.e. a single stimulus elicited more than 1 SP) predominantly in patients, they measured the latency and duration of the first of the multiple CSPs, and to calculate	At the dominant hemisphere: patients and HCs did not differ in latency of CSP in ADM or TA; but patients had a significantly higher number of CSPs in ADM and TA; no significant differences in the first CSP duration or in the total duration of CSP in ADM or TA	In both hemispheres, TMS measures (latency & duration of the 1st SP, total duration, total number of CSPs) did not correlate with clinical scores, daily dose of antipsychotics, duration of

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		had additionally daily lorazepam and 1 CA user and 1 AA user had daily lorazepam.	ISI of 1~5 sec. Responses were recorded using a pair of monopolar needle electrodes that were inserted into the abductor digiti minimi (ADM) muscles in the upper extremities and tibialis anterior (TA) muscles in the lower extremities at a distance of 3 cm from each other. For CSP, muscles were voluntarily maximally preactivated and the following parameters were recorded on the contralateral side: the latency, duration, and total number of silent periods of the activated muscle; the	the total duration of the CSP, durations of the first and later occurring CSPs were added together in respective stimulation site.	between the groups. Nondominant hemisphere: the groups did not differ in latency of CSP in ADM or TA; the patients had a higher number of CSPs in the nondominant ADM (non-significant after Bonferroni correction); no significant differences in the number of CSPs in TA or in the first CSP duration in ADM or TA; total duration of CSP was significantly longer in ADM in patients, but not in TA. No significant side-to-side differences within either group in any of the measures. CA vs AA vs HCs: CA seemed to have the shortest mean first and total CSP duration in the nondominant extremities compared to HCs	illness or age of the patients. In nondominant ADM, a positive correlation was obtained between the number of CSPs and PANSS

								presence of the SP was defined as a simultaneous decrease of amplitude of muscular activity below 0.05 mV/div in 5 consecutive measurements.				and AA; AA seemed to have the longest mean first and total CSP duration in the nondominant extremities, but also in the dominant TA, where also the mean number of CSPs was the highest of all
Basavaraju et al. (2015)	cross-sectional	18 with ego-boundary disturbance (EBD) (9 M); 32 without EBD (14 M)	33.11 ± 8.20 (with EBD); 29.97 ± 8.59 (without EBD)	63.55 ± 56.05 (with EBD); 35.84 ± 34.78 (without EBD) (months)	PANSS	12 were receiving risperidone, 4 olanzapine, 3 risperidone + olanzapine, 1 olanzapine + amisulpride and 1 aripiprazole. Median duration of treatment was 60 days. The rest were drug-naïve (9 in EBD group, 20 in without EBD group)	3 conditions: "rest" state, actual observation of an action, and virtual action observation	4 TMS paradigms: single-pulses at 120% RMT, MT1 (the minimum stimulation intensity evoking 1 mV peak-to-peak amplitude in the resting FDI), SICI and LICI. For SICI, CS (80% of RMT) was given 3 ms before a supra-threshold TS (MT1) with the right hand at rest. For LICI, a supra-threshold CS (MT1) is given 100 ms before a	left M1	RMT; MEPs for 120% RMT and MT1 (mV); SICI & LICI = (conditioned MEP/nonconditioned MEP) × 100. MNA (mirror neuron activity) = % change of motor excitability from resting to action observation states (average of virtual and actual observation) = (motor reactivity at action observation - motor reactivity at rest) x 100 / motor reactivity at rest	Significantly greater MNA in patients without EBD than in patients with EBD for the MT1 and 120% RMT stimulus paradigms, indicating less mirror neuron activity in patients with symptoms of EBD

Hasan et al. (2012)	cross-sectional	18 with first episode SCZ (14 M); 18 at risk of psychosis (14 M);	25.3 ± 6.3 (1st-episode SCZ); 24.11 ± 5.3 (at risk)	N/A	In SCZ patients, PANSS, GAF and CGI	All at-risk individuals were antipsychotic-naïve, but 7 received an antidepressant and 1 zopiclone. All, except 3, SCZ patients were taking atypical antipsychotics in monotherapy, but at the time of the TMS measurements, no patients had had a continuous treatment lasting longer than 6 weeks.	N/A	supra-threshold TS (MT1) SICI and ICF: CS intensity = 80% RMT, TS intensity = SI-1 mV; ISI = 3 (SICI), 7 and 15 (ICF) ms. For CSP, data recorded from the FDI muscle under voluntary contraction with 25% to 30% maximum force while stimulating M1 with 120% RMT	left M1	MEP size evoked by SI-1 mV; RMT; SICI; ICF; CSP duration	SI-1 mV-MEPs differed significantly across groups, the at-risk patients had smaller 1 mV-MEPs than 1st-episode patients and HCs, but no difference between HCs and 1st-episode patients. SICI differed significantly across groups, the at-risk and 1st-episode patients had less inhibition than HCs, but no difference between the at-risk and 1st-episode groups. CSP duration differed significantly across groups, 1st-episode patients had higher CSP duration than the prodromal and HC group, but no difference between the latter 2. No significant difference in ICF among groups.	In the 1st-episode patients, no correlations between SICI and ICF with PANSS scores, or between SICI, ICF, CSP duration with CPZ equivalent dose; CSP duration positively correlated with PANSS total and GAF scores. In the prodromal group, RMT negatively correlated with the positive symptom scores from the PANSS and Structured Interview for Prodromal Symptoms (SIPS), and CSP duration negatively correlated with PANSS

												negative, general psychopathology and total scores. However, the detected correlations did not survive correction for multiple comparisons.
Mehta et al. (2014b)	cross-sectional	Same as Mehta et al. (2014a)	Same as Mehta et al. (2014a)	Same as Mehta et al. (2014a)	PANSS	Same as Mehta et al. (2014a)	3 conditions: "rest" state, actual action observation, and virtual action observation	Same as Mehta et al. (2014a)	left M1	MEPs amplitude; SICI and LICI (conditioned MEP / non-conditioned MEP) × 100. For single-pulse paradigms, the difference in MEPs between rest and action observation states (averaged across virtual and actual action observation conditions) formed the measure of putative MNA; for paired-pulse paradigms, the difference in cortical inhibition (SICI and LICI) between rest and	In HCs, MEPs amplitude was significantly higher during action observation than rest state for 120% RMT and SI-1mV, and SICI was reduced during action observation. In contrast, antipsychotic-naive patients showed no significant difference between rest and action-observation states for 120% RMT, SI-1mV and SICI. In medicated patients, MEPs amplitude (for SI-1 mV) was increased and SICI was reduced	In the med-naive group, MNA measured using 120% RMT, SI-1mV and SICI were positively correlated with the ToM index. In the pooled patients group, MNA measured using SICI, SI-1mV and 120% RMT were positively correlated with the ToM index, and MNA measured using SICI was also

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						action-observation states formed the measure of putative MNA	during action observation. LICI did not showed modulation by action observation in any of these groups. Further, med-naive patients showed less MNA compared to HCs and medicated patients for all measures except LICI; and medicated patients had higher MNA during action observation for SI-1mV-MEP and SICI. No difference in MNA for LICI among the 3 groups.	positively correlated with the emotion recognition index.

Abbreviations: SCZ = schizophrenia or schizoaffective disorder; HCs = healthy controls; ISI = interstimulus interval; ITI = intertrial interval; CS = conditioning stimulus; TS = test stimulus; M1= primary motor cortex

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Table 2. Studies including TMS-EEG outcomes

			Patient characteristics									Summary of findings	
Study	**Design**	**Number of patients (N of males)**	**Age (years) (mean±SD)**	**Illness duration (years) (mean±SD)**	**Clinical characteristics (mean±SD)**	**Medications**	**Task**	**TMS protocol**	**Stimulation site**	**Masking sound or sham TMS**	**Measures of cortical excitability**	**Between-group (or time point) comparisons**	**Correlations**
Ferrarelli et al. (2008)	cross-sectional	16 (13 M)	33.5±8	11.1±6.4	N/A	14 were on 2nd-generation antipsychotics; 2 were unmedicated	N/A	The brain was stimulated using single-pulse stimuli at an intensity that generated an intracranial electric field of 120 V/m (suprathreshold), ISI = 0.5~0.7 Hz.	right premotor cortex, the coordinates of the stimulation site was not provided	Played masking noise	RMT; global mean field power (GMFP); event-related spectral perturbation (ERSP) and intertrial coherence (ITC) in the gamma band (30~50Hz)	No significant difference in RMT between patients and HCs. GMFP was decreased in patients between 12 and 100 ms post-stimulus relative to HCs, and the biggest decrease occurred at 22 and 55ms in several fronto-central electrodes. ERSP was significantly reduced in patients between 12~100 ms post-TMS in 4 fronto-central channels (including Cz and FC2) close to the TMS stimulation.	ERSP and ITC was not correlated with duration of illness or medication dose

												ITC was significantly reduced in patients within the first 100 ms in 5 fronto-central electrodes (same fronto-central region that showed GMFP and ERSP gamma reduction, including Cz and FC2).	
Canali et al. (2015)	cross-sectional	12 (9 M) with SCZ; 12 (2 M) with bipolar disorder (BPD); 12 (4 M) with major depression (MD)	38 ± 9 (SCZ); 36 ± 7 (BPD); 46 ± 8 (MD)	13 ± 6 (SCZ); 11 ± 9 (BPD); 18 ± 10 (MD)	PANSS	6 BPD were taking lithium salts. All MD were on antidepressant treatment, 8 of them also on benzodiazepines and 3 of them also on mood stabilizers. SCZ patients were taking antipsychotics (5 typical, 7 atypical)	N/A	Intensity of TMS-induced electric field was always > 90 V/m for each participant, delivered at a frequency randomly jittered between 1.5 and 1.8 sec (equivalent to about 0.5~0.6 Hz)	left premotor area (Brodmann area 6), the coordinates of stimulation site not provided	Played masking noise	ERSP in 8~50 Hz (1 Hz bin resolution), measured at the channel closest to the stimulation site. Natural frequency was defined as the frequency bin with the largest cumulated ERSP over time	TMS significantly activated the beta/gamma band response (range 21–50 Hz) in HCs between 20~300 ms post-TMS. The frequencies were significantly reduced in patients with bipolar disorder, major depression and SCZ (range 11–27 Hz). Frontal natural frequency	No significant correlations between natural frequencies and PANSS scores, or between natural frequencies and medication doses.

												was significantly reduced in the patient groups compared with HCs but did not differ among the patient groups themselves.	
Farzan et al. (2010)	cross-sectional	14 (10 M) with SCZ; 14 (9 M) with BPD	37.5 ± 10.4 (SCZ); 32.6 ± 13.4 (BPD)	9.8 ± 7.3 (SCZ); 7.7 ± 9.3 (BPD)	PANSS	2 SCZ patients were unmedicated (1 medication-naïve and 1 medication-free for 6 months) and 12 were on medication (clozapine, n = 5; risperidone, n = 3; haloperidol, n = 2; quetiapine, n = 1; perphenazine, n = 1)	N/A	LICI: CS = TS = intensity adjusted to produce mean peak-to-peak MEP of 1 mV, this corresponded to 65.8 ± 17.1% of stimulator output in patients with SCZ, 72.1 ± 15.2% in BPD, and 62.8 ± 11.8% in HCs; ISI = 100 ms, ITI = 5 sec	left M1 (optimal site for APB activation) and DLPFC (Talairach co-ordinates = -50, 30, 36)	Sham stimulation was administered using the same parameters as the active stimulation over the DLPFC and motor cortex (it preserved the auditory stimulation produced by TMS clicks) to control for the effect of auditory evoked potentials	Inhibition = [1 - area under rectified curve (conditioned) / area under rectified curve (unconditioned)] X 100. EEG data were decomposed into 5 frequency bands: delta (1–3.5 Hz), θ (4–7 Hz), α (8–12 Hz), β (12.5–28 Hz) and γ (30–50 Hz) and for each frequency band inhibition was obtained through the equation above, time of interest = 50~150 ms post-TS	Motor cortex (LICI at C3): inhibition in the γ band did not significantly differ among the 3 groups. DLPFC (LICI at AF3): SCZ patients had significantly lower inhibition in the γ band compared to BPD patients and HCs. No difference among groups for other frequency bands in motor cortex and DLPFC. No significant difference in the response to TS alone among groups for any frequency	No significant correlation between CIγ and medication dose (converted chlorpromazine equivalents) in SCZ and BPD patients treated with antipsychotics (n = 19)

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												band. In all groups, level of suppression did not change after controlling for the effect of auditory evoked potentials in the DLPFC or M1.	
Frantseva et al. (2014)	cross-sectional	16 (12 M)	36.7 ± 10.4	9.7 ± 7.3	PANSS	14 patients were medicated (clozapine, n = 6; risperidone, n = 3; haloperidol, n = 2; quetiapine, n = 1; perphenazine, n = 1; olanzapine, n = 1), with no other psychotropic medications; 2 were unmedicated	N/A	Intensity of single-pulse stimuli set to produce mean MEP amplitude of 1 mV peak-to-peak at rest, ISI = 5 sec	left M1	To control for the effect of TMS click-induced auditory evoked potentials, single-pulse sham-stimulation was given to all participants at the same intensity as used for active stimulation but with the coil angled at 90° from the scalp resting on one wing of the coil	Average global voltage (estimated as a surface area under the rectified EEG traces across all electrodes for each participant); Time-frequency signal power in delta (1–3.5 Hz), theta (4–7 Hz), alpha (8–12 Hz), beta (12–28 Hz), and gamma (30–50 Hz) bands	No significant difference in RMT and stimulus intensity between patients and HCs. Patients had significantly higher average global voltage than HCs between 400~750 ms post stimulus, but no difference in 75~150 ms. Patients also showed higher absolute signal voltage than HCs on topographic plots at around 200	Positive PANSS score was positively correlated with the time-varying maximum gamma power (total power averaged across 60 electrodes for each participant for the time period of interest) between 400~700 ms. Negative PANSS score was positively correlated with maximum theta and delta power at 200 ms

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ms and between 400~750 ms, and subtracting sham-EEG signal from active TMS-EEG signal did not diminish the difference statistically. The topography plots suggest that patients experienced more prolonged and widespread activation in response to TMS. Patients showed significantly increased signal power between 400~800 ms in delta band in ipsilateral frontal and temporo-parietal leads and in bilateral occipital and parietal electrodes; and in beta-

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												gamma band in ipsilateral (C3, C5, CP3, CP5, P3, P5, and P7) and contralateral channels (F8, FT8, FC6, C6, CP6, and T8).	
Noda et al. (2017)	cross-sectional	12 (8 M)	41 ± 10	N/A	Patients were clinically stable determined by the PANSS score of ⩽ 70. 11/12 patients interviewed with PANSS	Patients were on a stable dose of antipsychotic medications for at least one month, and were not taking anticholinergic drugs, benzodiazepines, or glutamate modulators	N/A	SICI & ICF: CS = 80% RMT, TS = SI-1 mV, ISI = 2 (SICI) and 10 (ICF) ms	Left DLPFC, the target was individually determined based on the EEG cap navigated F5 electrode site method	not used	Modulation of TEPs amplitudes by SICI & ICF in the DLPFC ROI: the TEP components were P30, N45, P60, N100 and P180, change in TEP (absolute change in amplitude) = conditioned amplitude minus unconditioned amplitude. The left DLPFC ROI was defined as electrodes Fp1, AF3, AF7, F1, F3, F5, F7, FC1, FC3 and FC7. Modulation of frequency band powers by SICI & ICF in delta (1–3 Hz), theta (4–7 Hz), alpha (8–14 Hz), beta (14–30 Hz), and gamma (30–	In both patients and HCs, P60 amplitude was significantly reduced by SICI, but the reduction was smaller in patients. P60 and N100 were significantly changed (amplitudes became more positive) following ICF in HCs, but no TEP amplitudes were increased by ICF in patients. Topography plots of TEPs showed that in HCs, SICI reduced excitation	No correlation between CPZ equivalent dose and the clinical or cognitive measures; and no correlation between the CPZ equivalent dose and the modulation of TEP components induced by SICI and ICF. Change in N100 amplitude by ICF was positively correlated with PANSS total score; change in P60 amplitude by SICI was negatively correlated with the longest span of the Letter-Number Span Test in

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				50 Hz) bands: change in power (as a ratio) for each frequency band = conditioned power / unconditioned power. Time-frequency analysis for SICI & ICF: ERSP	over the frontal area on P60, whereas ICF increased excitation over the left frontal area on P60 (i.e. more excitatory modulation) and N100 (i.e. less inhibition); in patients, the topographical changes are poor. Patients showed significantly less inhibitory modulation (i.e. higher conditioned / unconditioned power ratio) than HCs on delta frequency band by SICI in left DLPFC. No significant difference in modulations on any frequency bands with ICF in left DLPFC between	patients. No significant correlation with the other cognitive tests scores (Wechsler Test of Adult Reading, Letter-Number Span Test, the Trail Making Test Parts A & B, and Hopkins Verbal Learning Test) in patients.

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												groups. Time-frequency plot showed that HCs had significantly more inhibitory modulations during SICI (conditioned minus unconditioned power was more negative in HCs) and more facilitatory modulations during ICF (conditioned minus unconditioned power was more positive in HCs) compared to patients	
Radhu et al. (2015)	cross-sectional	38 with SCZ (25 M); 27 (11M) with obsessive compulsive disorder (OCD)	35.71 (SCZ); 36.15 (OCD)	N/A	Brief Psychiatric Rating Scale (BPRS)	SCZ patients were taking a variety of antipsychotics, antidepressants, mood stabilizers and/or benzodiazepines, 11 were taking clozapine	N/A	LICI: ISI = 100 ms, ITI = 5 sec, the intensity of both CS and TS were set to elicit an average MEP of 1 mV peak-to-peak upon delivery of 20 pulses over the motor cortex (no significant group difference for the stimulus intensity)	left M1, left DLPFC (Talairach coordinates = -50, 30, 36)	not used	Event-related spectral perturbation (ERSP) (μV^2/Hz) was computed separately for single pulse and paired pulse conditions, LICI = single pulse minus paired-pulse. The DLPFC ROI includes	LICI of DLPFC analysed across all channels: (1) all groups showed significant within-group inhibition in most channels - lower frequencies tend to show extended	For LICI of DLPFC, negative correlation found between BPRS total score and the size of the largest significant cluster of inhibition, after removing 2 outliers. No correlation

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					electrodes FP1, FPZ, FP2, AF3, AF4, F7, F5, F3, F1, FZ, F2, F4, F6, F8, FT7, FC5, FC3, FC1, FCZ, FC2, FC4, FC6 and FT8; the M1 ROI includes T7, C5, C3, C1, CZ, C2, C4, C6, T8, TP7, CP5, CP3, CP1, CPZ, CP2, CP4, CP6 and TP8	inhibition up to ~400 ms post stimulus, whereas higher frequencies show inhibition over narrower or specific temporal regions; (2) overall inhibition (1~50 Hz) was larger in HCs than SCZ, and significant difference between SCZ and OCD; (3) LICI was significantly different between HCs and SCZ in theta, alpha, beta and gamma bands, and significantly different between SCZ and OCD in theta and alpha bands. In the DLPFC ROI, overall inhibition (1~50 Hz) was larger in	between the size of largest cluster of inhibition (LICI of DLPFC) and CPZ equivalents in SCZ patients treated with antipsychotics (n=38)

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											HCs than in SCZ as well as in all frequency bands, and significant difference between SCZ and OCD in overall inhibition, theta, alpha and beta bands. LICI of M1 analysed across all channels: (1) all groups showed within-group inhibition and no difference between any groups across all frequency bands; (2) time-frequency plots showed inhibition in most channels in all 3 groups. In the M1 ROI, no significant difference between any groups across all frequency bands.

Radhu et al. (2017)	cross-sectional	19 with SCZ (10 M); 30 first-degree relatives of patients with SCZ (13 M)	30.2 (SCZ); 53.8 (relatives)	N/A	Brief Psychiatric Rating Scale (BPRS)	Patients were taking a variety of antipsychotics, antidepressants, mood stabilisers and/or benzodiazepines, 9 were taking clozapine	N/A	LICI: ISI = 100 ms, ITI = 5 sec, the intensity of both CS and TS were set to elicit an average MEP of 1 mV peak-to-peak	left M1; left DLPFC (Talairach coordinates = -50, 30, 36)	not used	ERSP was computed independently for the single-pulse and paired-pulse conditions, inhibition = power of single pulse minus power of paired pulse. 9 electrodes were used for the analysis of inhibition (F1, Fz, F2, FC1, FCz, FC2, C1, Cz, C2) for DLPFC and M1 stimulation	LICI of DLPFC: for overall inhibition (2~50 Hz), HCs = unaffected first-degree relatives > SCZ patients; for gamma (30~50 Hz) inhibition, HCs > SCZ but no difference between HCs and relatives or between relatives and SCZ. LICI of M1: no significant difference between any groups in overall (2–50 Hz) or gamma (30–50 Hz) inhibition in the ROI	For LICI of DLPFC, no significant correlation between overall inhibition and CPZ equivalent, or between gamma inhibition and CPZ equivalent; no significant relationship between BPRS score and overall or gamma inhibition

Ferrarelli et al. (2019)	cross-sectional	16 (12 M) patients with first-episode psychosis	22.5 ± 5.2	N/A	Scale for the Assessment of the Positive and Negative Symptoms (SAP and SAN)	FEP patients had no more than 2 months of lifetime antipsychotic treatment: 9 were antipsychotic naïve, 7 had <1 month exposure to antipsychotic medications at the time of the study	N/A	intensity of single pulses = 110% RMT, stimuli delivered at 0.4 to 0.6 Hz	left M1 (targeted a motor region adjacent to the hand area to ensure that no hand movement was observed in or reported by any participant to avoid re-afferent somatosensory activity)	Played masking noise	RMT (measured in the right FDI muscle); GMFP; ERSP and ITC were averaged between 8~45 Hz and 20~300 ms; the power spectra were also expressed as the % of power in a given frequency, called the relative spectral power (RSP). Clustering analysis was performed for ERSP, ITC and RSP	No significant difference in RMT between FEP and HCs. GMFP did not differ between groups. ITC (p=0.0524) and ERSP (p=0.0502) were decreased in FEP at trend level significance at electrodes FCz and C1 for the frequency band 28~42 Hz and 26~40 Hz, respectively. FEP showed significantly decreased RSP than the HCs in the 27~33 Hz range (beta/low gamma) in a cluster of fronto-central electrodes overlying the M1	No correlation between TMS-evoked EEG parameters (ERSP, ITC) and CPZ equivalent dose in medicated FEP patients. No correlation between ERSP, ITC and clinical scores

Noda et al. (2018)	cross-sectional	12 (8 M)	41 ±10	N/A	PANSS	Patients were on a stable dose of antipsychotic medications for at least 1 month, and no specific anticholinergic drugs or benzodiazepines more than lorazepam equivalent dose of 2 mg	N/A	SAI was delivered at ISIs relative to the somatosensory evoked potential (SSEP) at N20 (SSEP evoked by MNS is a negative deflection measured from somatosensory areas at a latency of about 20 ms). For M1-SAI, ISI = N20 + 2 ms; for DLPFC-SAI, ISI = N20 + 4 ms at the F5 electrode site. ITI = 5 sec, inter-block interval = 5~10 min (block refers to M1 or DLPFC stimulation). The conditioning median nerve stimulation (MNS) intensity was adjusted to 3 times the sensory threshold individually. Intensity of TMS was set to induce 1 mV peak-to-peak MEP amplitude	Left M1 and DLPFC (administered at the F5 electrode site)	not used	MEPs; TEPs (P30, N45, P60, N100, and P180). ROI for left M1 (FC1, FC3, FC5, C1, C3, C5, CP1, CP3, CP5) and DLPFC (Fp1, AF3, AF7, F1, F3, F5, F7, FC1, FC3, FC7). The modulation of TEPs by MNS was calculated as follows: SAI = [amplitude of TEP induced by SAI condition] / [amplitude of TEP induced by single pulse TMS]	Effect of SAI on MEPs: no significant difference in the mean intensity to induce 1 mV peak-to-peak MEP amplitude, or the degree of attenuation by SAI between patients and HCs Effect of SAI on TEPs: There was positive modulation (i.e. increased amplitude) of P180 in the M1 in patients and HCs (however, not sure if modulation was significant in the HCs), and there was significant difference in the level of modulation of P180 between groups, with patients > HCs.	Negative correlation in patients between modulation of N100 at left DLFPC and executive function as measured with the ratio of Trail Making Test (TMT) part B to part A - bigger modulation of N100 correlated with worse performance. This correlation remained significant after Bonferroni correction, and remained significant after controlling for age as a covariate in this correlation.

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								in the right FDI muscle.			DLPFC-SAI: In the DLPFC, N100 became significantly more positive (i.e. amplitude reduced) in patients but the N100 amplitude increased in HCs, and there was significant group difference in modulation of N100, with patients < HCs
Lett et al. (2016)	cross-sectional	23 (a subgroup of the patients from Radhu et al., 2015)	N/A	N/A	N/A	N/A	N/A	LICI: TS = CS = intensity that elicited an average MEP of 1 mV peak-to-peak upon delivery of 20 pulses over the motor cortex, ISI = 100ms, ITI = 5 sec	left DLPFC (Talairach coordinates = -50, 30, 36)	not used	LICI was assessed by comparing single-pulse versus paired-pulse conditions. The number of significant voxels within the biggest cluster of inhibition was calculated for every participant, which was used to reflect the degree of LICI	23 patients and 33 HCs completed the TMS-EEG and genetic protocols: In the DLPFC, GAD1 T-allele (the "risk genotype") carriers predicted greater LICI cluster size in HCs and lower LICI cluster size in patients.

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								after DLPFC stimulation. LICI was assessed at frequencies ranging from 1~50 Hz using cluster-based analysis.	Another sample completed cognitive tests and genetic protocol: analysis using the general linear model showed that GAD1 genotype was a significant predictor of performance on letter-number span, digit span and Stroop ratio after covarying for age and IQ.

Abbreviations: SCZ = schizophrenia or schizoaffective disorder; HCs = healthy controls; ISI = interstimulus interval; ITI = intertrial interval; CS = conditioning stimulus; TS = test stimulus; M1= primary motor cortex

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RoBANS

1. The selection of participants Selection biases caused by the inadequate selection of participants	
Criteria for judgments of a 'Low risk' of bias	**Case-control study** • The case and control groups were selected from comparable population groups • The case group (diagnosis) was clearly defined, with validated diagnostic instrument (e.g. DSM, ICD). • It was clearly demonstrated that the control group is not the patient group (i.e. no history of diagnosis of psychotic disorder)
	Before-after study The study participants were consecutively recruited, and the data were collected prospectively.
Criteria for judgments of a 'High risk' of bias Any one of the following conditions:	**Case-control study** • The case and control groups are not the comparable population groups. • The patient definitions were generated by self-reported or merged data. • It was not clearly confirmed that the control group excluded patients.
	Before-after study • The participants was not recruited consecutively. • Retrospective data collection was performed.
Criteria for judgments of an 'Unclear risk' of bias	It is uncertain whether the selection of participants resulted in a ‘high risk’ or a ‘low risk’ of bias
2. Confounding variables Selection biases caused by the inadequate confirmation and consideration of confounding variables	
Criteria for judgments of a 'Low risk' of bias Any one of the following conditions:	**Non-randomized studies (except for before-after studies)** • The major confounding variables (e.g. age, sex or any additional factor) were adequately confirmed and considered during the design phase (e.g. through matching, participation restriction, or other methods). • The major confounding variables were adequately confirmed and adjusted for during the analysis phase (e.g. through stratification, propensity score approaches, statistical adjustments, or other methods)
	Before-after study

	A natural progression and learning effect (this effect occurs if past experience improves future execution skills) can be excluded during the consideration of diseases and interventions.
Criteria for judgments of a 'High risk' of bias Any one of the following conditions:	**Non-randomized study (except for before-after studies)** • The major confounding variables were not considered. • Although the existence of major confounding variables was confirmed, these variables were not adequately considered during the design and analysis phases.
	Before-after study Natural progression and a learning effect are relatively evident in the considerations of diseases and interventions.
Criteria for judgments of an 'Unclear risk' of bias	It is uncertain whether the confounding variables resulted in a 'high risk' or a 'low risk' of bias
3. Measurement of exposure (intervention) Performance biases caused by inadequate measurements of exposure	
Criteria for judgments of a 'Low risk' of bias	The experimenter was blinded during collection of exposure data
Criteria for judgments of a 'High risk' of bias	A clear case of performance bias
Criteria for judgments of an 'Unclear risk' of bias	It is uncertain whether the exposure measurement resulted in a 'high risk' or a 'low risk' of bias
4. Blinding of outcome assessments Detection biases caused by the inadequate blinding of outcome assessments	
Criteria for judgments of a 'Low risk' of bias Any one of the following conditions:	• The outcome assessments were blinded. • Although blinding was not present, its absence was judged to have no effect on the outcome measurements
Criteria for judgments of a 'High risk' of bias	Blinding was not performed or incomplete, and this lack of appropriate blinding appears likely to have affected the outcome measurements.
Criteria for judgments of an 'Unclear risk' of bias	It is uncertain whether the blinding of the outcome assessments resulted in a 'high risk' or a 'low risk' of bias
5. Incomplete outcome data Attrition biases caused by the inadequate handling of incomplete outcome data	
Criteria for judgments of a 'Low risk' of bias	**Non-randomized studies (except for before-after studies)**

Any one of the following conditions:	• There are no missing data. • The missing data did not affect the study outcomes. • The quantity of missing data was a product of similar developments in both the intervention (exposure) and the control groups, and the causes of these developments are similar.
	Before-after study Information about the number of participants before and after the study exists, and the baseline did not differ with respect to completed and failed study participants.
Criteria for judgments of a 'High risk' of bias Any one of the following conditions:	**Non-randomized studies (except for before-after studies)** The missing data could affect the study outcome. These effects may be attributed to the differences in the missing data between the intervention (exposure) group and the control group, or the effects may be caused by the absence of important measurements.
	Before-after study Differences exist with respect to the baseline for successful and failed participants
Criteria for judgments of an 'Unclear risk' of bias	It is uncertain whether the incomplete outcome data resulted in a ‘high risk’ or a ‘low risk’ of bias
6. Selective outcome reporting Reporting biases caused by the selective reporting of outcomes	
Criteria for judgments of a 'Low risk' of bias Any one of the following conditions:	• The experimental protocol is available, and the pre-defined primary/secondary outcomes were described as planned. • All of the expected outcomes were included in the study descriptions (in the absence of the experimental protocols).
Criteria for judgments of a 'High risk' of bias Any one of the following conditions:	• The pre-defined primary outcomes were not fully reported. The outcomes were not reported in accordance with the previously defined standards. • Primary outcomes that were not pre-specified in the study existed (except for outcomes with clear explanations, such as unexpected adverse effects). • The existence of incomplete reporting regarding the primary outcome of interest. • The absence of reports on important outcomes that would be expected to be reported for studies in related fields.

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Criteria for judgments of an 'Unclear risk' of bias	It is uncertain whether the selective outcome reporting resulted in a 'high risk' or a 'low risk' of bias

Search terms for each database

PsycInfo

1. transcranial magnetic stimulation/
2. "transcranial magnetic stimulation".ab.
3. "tms".ab.
4. exp schizophrenia/
5. schizophreni*.ab.
6. "schizoaffective disorder*".ab.
7. exp interneurons/
8. exp glutamic acid/
9. exp gamma aminobutyric acid/
10. exp neural inhibition/
11. exp pyramidal neurons/
12. excita*.ab.
13. inhibit*.ab.
14. GABA*.ab.
15. glutam*.ab.
16. "excitation-inhibition balance".ab.
17. "E-I balance".ab.
18. 1 or 2 or 3
19. 4 or 5 or 6
20. 7 or 8 or 9 or 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17
21. 18 and 19 and 20

Embase

1. transcranial magnetic stimulation/
2. ("transcranial magnetic stimulation" or "tms").ab.
3. exp schizophrenia/
4. (schizophreni* or "schizoaffective disorder*").ab.
5. exp interneuron/
6. exp glutamic acid/
7. exp 4 aminobutyric acid/
8. exp nerve cell inhibition/
9. exp pyramidal nerve cell/

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10. (excita* or inhibit* or GABA* or glutam* or "excitation-inhibition balance" or "E-I balance").ab.
11. 1 or 2
12. 3 or 4
13. 5 or 6 or 7 or 8 or 9 or 10
14. 11 and 12 and 13

Medline

1. transcranial magnetic stimulation/
2. "transcranial magnetic stimulation".ab.
3. "transcranial magnetic stimulation".ab.
4. exp schizophrenia/
5. schizophreni*.ab.
6. "schizoaffective disorder*".ab.
7. exp interneurons/
8. exp glutamic acid/
9. exp gamma aminobutyric acid/
10. exp neural inhibition/
11. exp pyramidal neurons/
12. excita*.ab.
13. inhibit*.ab.
14. GABA*.ab.
15. glutam*.ab.
16. "excitation-inhibition balance".ab.
17. "E-I balance".ab.
18. 1 or 2 or 3
19. 4 or 5 or 6
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Risk of bias assessment

Table 7. Assessment of bias

Study	Selection of participants	Confounding variables	Measurement of exposure	Blinding of outcome assessment	Incomplete outcome data	Selective reporting
Boroojerdi et al. (1999)	low	low	unclear	unclear	low	low
Chroni et al. (2002)	low	high	unclear	unclear	low	low
Daskalakis et al. (2002)	low	high	unclear	unclear	unclear	low
Fitzgerald et al. (2002a)	high	high	unclear	low	low	low
Fitzgerald et al. (2002b)	high	low	low	low	low	low
Reid et al. (2002)	low	high	unclear	unclear	low	low
Fitzgerald et al. (2003)	low	low	low	low	low	low
Takahashi et al. (2003)	high	low	unclear	unclear	low	low
Bajbouj et al. (2004)	high	low	unclear	unclear	low	low
Fitzgerald et al. (2004)	low	low	unclear	low	low	low
Daskalakis et al. (2008a)	low	high	unclear	unclear	low	low
Daskalakis et al. (2008b)	low	high	unclear	unclear	low	low
Ferrarelli et al. (2008)	high	high	unclear	unclear	low	low
Koch et al. (2008)	low	high	unclear	unclear	low	low
Liu et al. (2009)	low	low	unclear	unclear	low	low
Farzan et al. (2010)	high	low	unclear	unclear	low	low
Soubasi et al. (2010)	low	low	unclear	unclear	low	low
Hasan et al. (2011)	low	low	unclear	unclear	low	low
Ribolsi et al. (2011)	low	high	unclear	unclear	low	low
Hasan et al. (2012)	low	low	unclear	unclear	low	low
Ahlgren-Rimpilainen et al. (2013)	low	low	unclear	unclear	low	low
Frantseva et al. (2014)	low	low	unclear	unclear	low	low
Mehta et al. (2014a)	low	low	unclear	unclear	low	low
Mehta et al. (2014b)	low	low	unclear	unclear	low	low
Tang et al. (2014)	low	low	unclear	unclear	low	low
Yildiz et al. (2015)	low	low	low	unclear	unclear	low
Basavaraju et al. (2015)	low	low	unclear	unclear	low	low
Canali et al. (2015)	high	high	unclear	unclear	low	low
Kaster et al. (2015)	low	low	low	low	unclear	low
Radhu et al. (2015)	low	high	unclear	unclear	low	low
Bridgman et al. (2016)	low	high	unclear	unclear	low	low
Lett et al. (2016)	low	low	unclear	low	low	low

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Lindberg et al. (2016)	low	high	unclear	unclear	unclear	low
Strube et al. (2016)	low	low	unclear	unclear	low	low
Ustohal et al. (2017)	low	low	low	unclear	unclear	low
Noda et al. (2017)	low	high	unclear	unclear	low	low
Radhu et al. (2017)	low	high	unclear	unclear	low	low
Du and Hong (2018)	low	high	unclear	unclear	low	low
Ferrarelli et al. (2019)	high	high	unclear	unclear	low	low
Noda et al. (2018)	low	high	unclear	unclear	low	low
Bagewadi et al. (2019)	low	low	unclear	unclear	low	low
Du et al. (2019)	low	high	unclear	unclear	low	low