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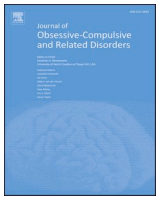

Treatment of co-occurring obsessive-compulsive and psychotic symptoms: A systematic review

Jennifer L. Buchholz [a,*], Heidi J. Ojalehto [a], Brenna B. Maddox [a], Samantha N. Hellberg [a,1], Katherine Wislocki [g], Jonathan S. Abramowitz [a], Charlie A. Davidson [d,e], Jordan E. Cattie [f], Emily M. Becker-Haimes [b,c]

[a] University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
[b] Department of Psychiatry University of Pennsylvania Perelman School of Medicine, USA
[c] Hall Mercer Community Mental Health, University of Pennsylvania Health System, USA
[d] Department of Psychology, Emory University, Atlanta, GA, USA
[e] Atlanta Center for Cognitive Therapy, Atlanta, GA, USA
[f] Emory Department of Psychiatry and Behavioral Sciences, Atlanta, GA, USA
[g] University of California Irvine, Irvine, CA, USA

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ABSTRACT

Up to 30% of individuals with psychosis also experience significant obsessive-compulsive symptoms (OCS). This common, yet understudied, co-occurrence is associated with a more severe clinical presentation and course than psychosis alone, making identifying effective treatments for this comorbidity profile critical. This systematic review synthesized the literature published between 1976 and 2020 on psychopharmacological, psychosocial, and other treatment approaches for co-occurring OCS and psychosis. Our review identified 42 studies examining psychopharmacological (n = 32), psychosocial (n = 7), and neuromodulation (n = 3) treatments. Results suggested that there is early support for the effectiveness of tricyclic antidepressants, selective serotonin reuptake inhibitors, and cognitive behavioral therapies as augmentative agents to traditional antipsychotic medications to reduce OCS within the context of psychosis. Considerable limitations across identified studies preclude reliable conclusions about the effectiveness of treatment for co-occurring OCS and psychosis. Gaps in the identified literature point to important areas for future research.

Obsessive-compulsive disorder (OCD) and psychosis frequently co-occur; approximately 12% of patients with schizophrenia or first episode psychosis also meet criteria for OCD (Achim et al., 2011; Mawn et al., 2020; Swets et al., 2014), vastly higher than the 1–3% OCD prevalence rate in the general population (Ruscio, Stein, Chiu, & Kessler, 2010). Further, up to 30% of patients with schizophrenia report significant obsessive-compulsive symptoms (OCS) that may not meet full diagnostic criteria for OCD (Hagen, Hansen, Joa, & Larsen, 2013; Poyurovsky et al., 2007; Swets et al., 2014) but are associated with substantial distress and impaired functioning (Adam, Meinlschmidt, Gloster, & Lieb, 2012). As such, we use the term OCS to refer to the presence of obsessive-compulsive symptoms, regardless of formal diagnostic status. The risk of psychosis is also greater among patients with OCS; a recent longitudinal study demonstrated that individuals first diagnosed with OCD had a three-fold higher risk of receiving a later diagnosis of schizophrenia and a five-fold higher risk of receiving a later diagnosis of schizoaffective disorder compared to individuals without OCD (Cederlöf et al., 2015).

Several theories exist to explain this common co-occurrence. Initially, clinicians proposed that OCS represented a variant or a prodrome of psychotic illness (Westphal, 1878). Later, Insel & Akiskal, 1986 proposed that OCS occur along a spectrum of insight, with patients at the far end of the spectrum displaying "obsessive-compulsive psychosis," whereby OCS shift into psychotic states when obsessions are no longer recognized as ego-dystonic and constitute delusions. In accordance with this and similar observations (e.g., Kozak & Foa, 1994), the DSM-IV (American Psychiatric Association, 1994) included a "with poor insight" specifier to denote when individuals with OCD fail to recognize

* Corresponding author.
E-mail address: jbuchholz@unc.edu (J.L. Buchholz).

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the irrationality of their OCS. This was further updated in the DSM-5 (American Psychiatric Association, 2013) criteria for OCD, which include specifiers for "good or fair insight" (the individual recognizes that OCS are definitely or probably not true), "poor insight" (the individual thinks OCS are probably true), and "absent insight/delusional beliefs" (the individual is completely convinced that OCS are realistic).

However, OCS and psychosis also co-occur as *independent* entities that may present with varying levels of phenomenological overlap (Porto, Bermanzohn, Pollack, Morrissey, & Siris, 1997). There is evidence for shared etiological pathways, including metacognitive processes (Moritz, Peters, Larøi, & Lincoln, 2010), genetic risk (Cederlöf et al., 2015), and neurobiological underpinnings (Hwang, Kim, Yum, & Opler, 2009; Poyurovsky et al., 2012). OCS can emerge prior to, concurrent with, or after the first onset of psychosis (Hwang et al., 2009), which suggests a distinct clinical course of OCS among patients with psychosis. Further, the utility of insight as a distinguishing factor between obsessions and delusions has been challenged. Many patients with OCS without psychosis show poor insight (Catapano, Sperandeo, Perris, Lanzaro, & Maj, 2001), and dimensions of delusions can occur with varying degrees of insight (Spitzer, 1990). OCS have also been observed to develop in response to treatment with antipsychotic medication, termed "antipsychotic-induced OCS", with numerous studies reporting the onset or exacerbation of OCS following treatment with a second-generation antipsychotic (Fonseka, Richter, & Müller, 2014; Schirmbeck & Zink, 2012).

While debate continues as to whether OCS are best considered a prodromal sign of psychosis (Cederlöf et al., 2015; Fontenelle et al., 2011) or distinct presentations, there is consistent evidence regarding the impact of this comorbidity on the severity and course of illness. Notably, the presence of OCS among patients with schizophrenia is associated with more severe global, positive, and negative symptoms (Cunill, Castells, & Simeon, 2009), more severe depressive symptoms, greater levels of hopelessness, greater risk of suicidal ideation and attempts (de Haan, Hoogenboom, Beuk, van Amelsvoort, & Linszen, 2005; Lysaker, Whitney, & Davis, 2006; Lysaker & Whitney, 2009), poorer global functioning, lower subjective well-being and quality of life (de Haan et al., 2012), worse social functioning (de Haan et al., 2012), and greater use of maladaptive coping strategies (Lysaker et al., 2006). Patients with comorbid OCS and psychosis also show poorer treatment response and worse long-term outcomes than patients without OCS (Fenton & McGlashan, 1986; Jaeger et al., 2008), and greater neuropsychological impairments (e.g., executive functioning, cognitive flexibility, visuo-spatial memory) compared to patients without OCS (Schirmbeck & Zink, 2012). Taken together, this work suggests that patients with co-occurring OCS and psychosis are at high risk for a poorer illness course and prognosis. Effective treatment strategies for this specific comorbidity profile are critical.

Despite the high co-occurrence of OCS and psychosis and associated impairment, there is limited information on how to treat OCS within the context of psychosis. Treatment recommendations for each differ. Treatment for OCS typically involves exposure and response prevention (ERP) and/or selective serotonin reuptake inhibitors (SSRIs; Olatunji, Davis, Powers, & Smits, 2013; Soomro, Altman, Rajagopal, & Browne, 2008) while recommended treatments for psychosis include antipsychotics, social skills training, family interventions (Combs & Mueser, 2017, p. 188), and, more recently, cognitive-behavioral therapy (CBT; Turner, van derGaag, Karyotaki, & Cuijpers, 2014). Importantly, OCS treatment trials often exclude patients with psychotic disorders (Eddy, Dutra, Bradley, & Westen, 2004; Odlaug et al., 2014), raising concerns about the efficacy of ERP and SSRIs in the context of psychosis. Despite limited data, practice guidelines advanced by the American Psychiatric Association for the treatment of co-occurring psychosis and OCS recommend the use of SSRIs in combination with antipsychotics (Koran, Hanna, Hollander, Nestadt, & Simpson, 2007). However, little is known regarding the extent to which SSRIs or ERP are effective for OCS in the context of co-occurring psychosis.

To date, there have been several reviews evaluating the evidence for treatment approaches to OCS in the context of psychosis. Regarding psychopharmacological interventions, these reviews–all now nearly 20 years out of date–supported the use of SSRIs and clomipramine hydrochloride for the treatment of OCS in schizophrenia (Chang & Berman, 1999). Of note, the data included in this review primarily comprised case reports and one controlled, double-blind study with a small sample size ($N = 6$). The authors also noted that patients in some of the case studies reported worsening of psychotic symptoms following the introduction of an SSRI or clomipramine and cautioned that adequate doses of neuroleptic agents should first be established. In a later review, Randhawa (2005) reported that treatment with clozapine, risperidone, olanzapine, and quetiapine were generally associated with worsening OCS in some case studies, but that one single-blind study demonstrated that treatment with risperidone improved OCS among patients with schizophrenia (Veznedaroglu, Ercan, Kayahan, Varan, & Bayraktar, 2003). The authors suggested that the effectiveness of pharmacotherapy for OCS in the context of psychosis may depend on the chronology of symptom development and that SSRIs may be required for patients whose OCS precede psychosis symptoms.

Two more narrative reviews, now both over a decade old, indicated mixed evidence for the safety and effectiveness of co-administration of antipsychotics with SSRIs for OCS and psychosis. Specifically, Poyurovsky, Weizman, and Weizman (2004) concluded that some studies have demonstrated favorable response to adjunctive SSRIs but that a substantial portion of patients with schizophrenia and co-occurring OCS demonstrate non-response. The authors similarly noted inconsistent findings for the use of typical antipsychotic agents for patients with schizophrenia and OCS, documenting potential exacerbation or new onset OCS after trialing antipsychotics (Poyurovsky et al., 2004). More recently, Hwang et al. (2009) also noted inconsistent evidence regarding the efficacy of various pharmacological approaches and suggested that this variation may be attributed to the presence of multiple etiological pathways for OCS in patients with schizophrenia. They concluded that clozapine monotherapy appears to be effective for the treatment of OCS for some, and yet may induce de novo OCS in other patients with schizophrenia. In such cases, the authors reported that there is evidence for the use of antipsychotics, such as amisulpride and haloperidol, as well as adjunctive SSRI therapy for the management of antipsychotic-induced OCS. A more recent review examined evidence for the treatment of clozapine-induced OCS specifically and concluded that aripiprazole as an add-on may be a promising treatment approach (du Montcel, Pelissolo, Schürhoff, & Pignon, 2019).

Two prior reviews specifically examined the benefit of psychosocial interventions for co-occurring OCS and psychosis. Schirmbeck and Zink (2013) performed a systematic review examining CBT for the treatment of OCS in schizophrenia and found CBT to be effective in reducing OCS in 80% of reported cases. Further, the authors deemed CBT for the treatment of OCS in patients with schizophrenia to be safe and well-tolerated, noting retention rates comparable to those seen in primary OCS patients and adverse effects in less than 10% of patients. This review was limited by the small number of total patients ($N = 30$), sole reliance on case reports and series, and inconsistent use of quantitative outcome measurement. Most recently, Tundo and Necci (2016) conducted a systematic review of the literature focusing on CBT treatment of OCS co-occurring with schizophrenia or schizoaffective disorder. Their Results supported the use of CBT for the treatment of antipsychotic-induced OCS and OCD not induced by antipsychotics and highlighted the lack of literature in this space.

Outside of psychopharmacological and psychosocial approaches, neuromodulation techniques to address OCS that occur in the context of psychosis are emerging. This research builds on promising findings regarding the effectiveness of such approaches for both OCD and psychosis. For example, a review of transcranial direct current stimulation (tDCS; Szymkowicz, McLaren, Suryadevara, & Woods, 2016) found some support for tDCS in alleviating symptoms of schizophrenia and

OCD, and there is some preliminary support for tDCS in the treatment for both OCS (Brunelin et al., 2018) and positive symptoms of schizophrenia (Kennedy, Lee, & Frangou, 2018; Kim et al., 2019). Similarly, therapeutic effects of repetitive transcranial magnetic stimulation (rTMS) have been demonstrated for OCD (Berlim, Neufeld, & Van den Eynde, 2013; Rehn, Eslick, & Brakoulias, 2018) and schizophrenia (Hovington, McGirr, Lepage, & Berlim, 2013). Some research has also pointed to the effectiveness of deep brain stimulation (DBS) for OCD (Gabriëls, Cosyns, Nuttin, Demeulemeester, & Gybels, 2003; Naesström, Blomstedt, & Bodlund, 2016), and Gault et al., 2018 noted several potential advantages to DBS for treatment-resistant schizophrenia. That said, results remain mixed. For example, a recent systematic review of brain stimulation for OCS (Rapinesi et al., 2019) found promising but inconsistent results for TMS and DBS, with less evidence supporting tDCS. Few studies, however, have examined neuromodulation for co-occurring OCS and psychosis, and such results have not yet been synthesized.

To date, there is no comprehensive, systematic review that includes psychopharmacological, psychosocial, and neuromodulation treatments for OCS and co-occurring psychosis. Furthermore, there has been no review of psychopharmacological interventions in this population in over a decade. Given the prevalence of this comorbidity profile and its associated poor prognosis, it is critical to evaluate the effectiveness of different treatment approaches for this population. Recent years have seen an expansion of research within this area. Accordingly, this systematic review aims to provide a clear and comprehensive overview of available evidence on the effectiveness of psychopharmacological, psychosocial, and other treatment approaches for co-occurring OCS and psychosis. Of note, this review broadly integrates research from several different fields (i.e., clinical psychology, psychiatry, OCD, and psychosis) with the aim of bridging related areas of work to provide a comprehensive picture of the current state of evidence. To our knowledge, this is the first systematic review to examine a range of interventions for co-occurring OCS and psychosis.

1. Method

A systematic literature search identified all relevant articles published in the last 45 years. An initial search for articles published between January 1st, 1976 and February 4th, 2020 was conducted in PsycINFO and PubMed using the following search strings, developed in collaboration with a medical librarian: ("obsessive-compulsive disorder" or "OCD" or "obsessive" or "compulsive" or "obsession" or "compulsion") and ("schizophrenia" or "schizophrenic" or "schizoaffective" or "schizotypal" or "psychosis" or "psychotic" or "prodrome" or "prodromal" or "hallucination" or "delusion" or "paranoid" or "paranoia"). The search returned 7133 unique records, which were divided among authors for abstract screening for inclusion, supported by Rayyan software (Ouzzani, Hammady, Fedorowicz, & Elmagarmid, 2016). Regular meetings were held to discuss uncertainties about an abstract's relevance with decisions made by consensus. 234 records were identified as meeting the following initial criteria: human participants, published and peer reviewed, English language, and reported on the treatment of comorbid OCS and psychosis.

Full-text reviews were conducted of the 234 records identified as meeting inclusion criteria after abstract screening. Studies were included in the review if: (a) participant(s) had both psychosis and OCS at treatment start, (b) an intervention was provided, and (c) at least one quantitative treatment outcome measure was utilized. Inclusion criteria were intended to allow for the broadest possible sample of studies that examined treatment of co-occurring psychosis and OCS. Studies were excluded if they reported on new onset OCS following administration of an antipsychotic only, as differences in the etiology of OCS onset likely impact treatment recommendations. Full text reviews were conducted by study team members with uncertainties flagged for review. The first and senior author independently reviewed uncertainties and then met to resolve discrepancies, obtaining 100% consensus on the ultimate inclusion of 42 records. Included studies were classified by intervention type: psychopharmacological, psychosocial, or neuromodulation and were reviewed by at least one member of the research team who extracted relevant information for synthesis. Fig. 1 illustrates the PRISMA study flow diagram.

2. Results

2.1. Overview of the literature

A total of 42 studies met inclusion criteria. Identified studies examined psychopharmacological ($n = 32$), psychosocial ($n = 7$), and neuromodulation ($n = 3$) treatments.

2.2. Psychopharmacological interventions

Summary of the Literature. Of 32 identified studies, our review returned 15 with heterogenous methodology that examined the impact of a single psychopharmacological agent as an augmented treatment intervention to standard medication regimens (e.g., stable antipsychotic medication), including one randomized controlled trial (RCT), one randomized crossover study, one single blind trial, 11 open label trials, and one naturalistic follow-up. Medications examined included tricyclic antidepressants (TCAs; clomipramine $n = 3$), SSRIs (fluoxetine $n = 2$, fluvoxamine $n = 3$, escitalopram $n = 2$), antipsychotics (aripiprazole $n = 1$, risperidone $n = 1$, ziprasidone $n = 1$), and an anticonvulsant (lamotrigine $n = 1$). One additional study (Schirmbeck and Zink, 2013) constituted a naturalistic prospective study examining two different medication combinations (clozapine and olanzapine vs. amisulpride and aripiprazole). All studies but one used the Yale-Brown Obsessive--Compulsive Scale (Y-BOCS; Goodman et al., 1989), the gold-standard assessment of the presence and severity of obsessions and compulsions, to monitor change in OCS over the study period. The Y-BOCS is a semi-structured interview that consists of a symptom checklist and severity scale that assesses time spent, interference, and distress associated with obsessions and compulsions. Higher scores reflect more severe OCS and the measure has demonstrated sensitivity to change with treatment (van Oppen, Emmelkamp, van Balkom, & van Dyck, 1995). Table 1 presents an overview of included studies, their design, and their main outcomes.

An additional 17 case studies or case series were identified that reported on the effects of a range of medications, including clozapine ($n = 5$), clomipramine ($n = 2$), fluoxetine ($n = 2$), fluvoxamine ($n = 1$) olanzapine ($n = 3$), citalopram ($n = 1$), paliperidone ($n = 1$), risperidone ($n = 1$) and milnacipran ($n = 1$). These studies varied widely in methodology, addressed a range of ages (M age = 29.5, Range = 16–55), and included predominantly males (66.6%). Nearly all ($n = 14$) reported on outcomes of a series of medication trials within a single individual. As such, we deemed them unlikely to lead to generalizable findings and excluded them from synthesis. See Supplemental Files for identified references of case studies.

Results for Tricyclic Antidepressants. Three independent studies examined the impact of clomipramine as an augmentative agent for OCS, all of which suggested the potential benefit for clomipramine to reduce OCS in individuals with psychosis. Yaryura-Tobias et al. (1976) conducted an eight-week open pilot trial in 10 individuals with a diagnosis of schizophrenia and co-occurring "severe" OCS. They tested the impact of up to 300 mg of clomipramine and found modest reductions in anxiety caused by rituals on the Obsessive-Compulsive Index (OCI; Foa et al., 1998). Zohar et al. (1993) conducted an open label trial using an "off-on-off" design to test the impact of 250–300 mg/day of clomipramine on OCS in a sample of five individuals diagnosed with schizophrenia or schizoaffective disorder. All participants showed reductions in OCS as measured by the Y-BOCS when clomipramine was added to their medication regimen; there was also evidence for return of OCS in four participants after discontinuation of the clomipramine. Berman

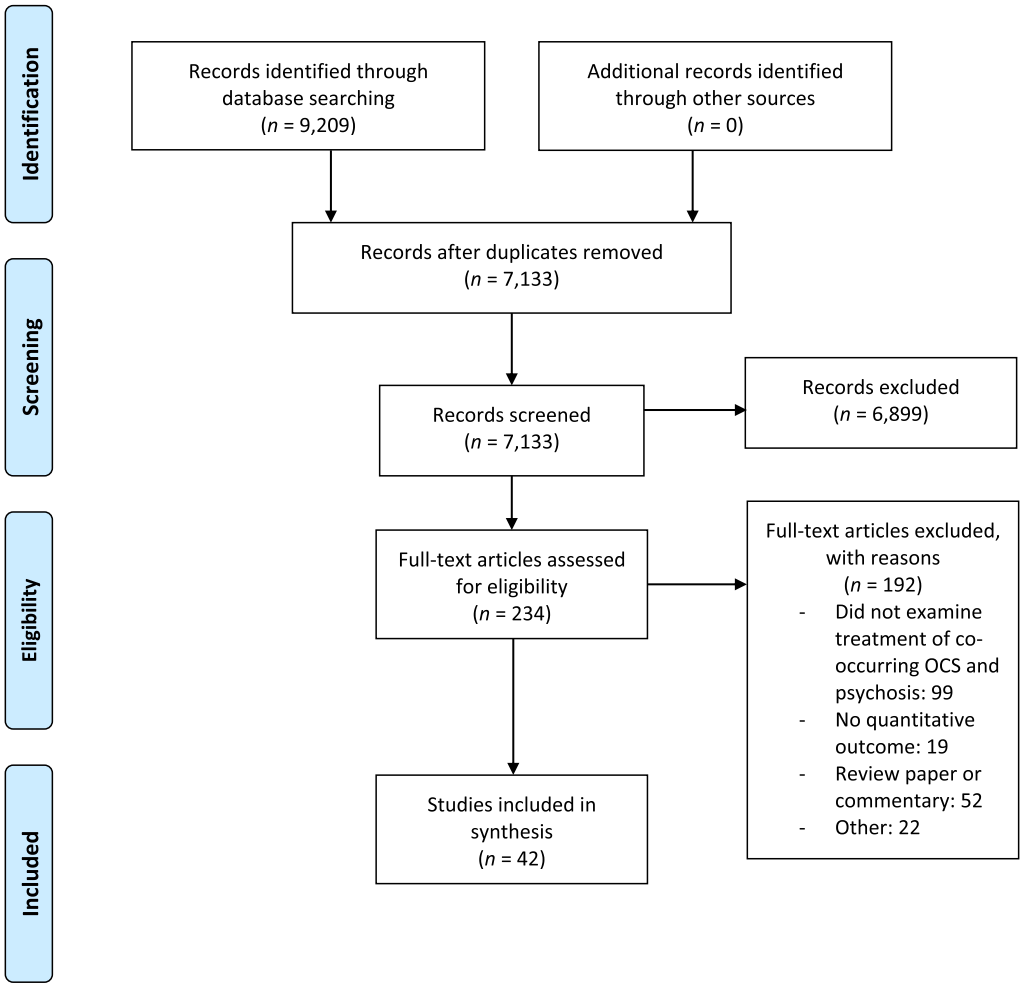

Fig. 1. PRISMA study flow diagram.

et al. (1995) conducted a 13-week randomized crossover trial with six adults diagnosed with schizophrenia who had at least two obsessions or compulsions, as indexed by the Y-BOCS. Participants were randomized first to either placebo or 100–250 mg/day of clomipramine for six weeks that was added to their stable dose of antipsychotic medications, then underwent a one-week drug washout during the seventh week, and then crossed over to the other condition for weeks 8–13. Results indicated that clomipramine led to more improvement in Y-BOCS ratings than did placebo; similar trends were seen for positive and negative symptoms of schizophrenia.

Limitations of TCA Studies. All three studies were limited by small sample sizes (range = 5–10), majority male samples (66.7–80% male), and highly variable attrition rates (0–40%). Furthermore, dosages of clomipramine administered varied across studies. Race and ethnicity of participants were not reported. There was also some concern for side effects of clomipramine preventing individuals from being able to titrate up to the therapeutic dose.

Results for SSRIs. Seven studies examined the impact of SSRIs as augmentative agents on OCS in the context of psychosis, with SSRIs added to a stable medication regimen for psychosis symptoms. All studies used the Y-BOCS as a primary outcome measure. In general, results indicated that SSRIs were well-tolerated and led to improvements in OCS, with the most support for the use of fluvoxamine to reduce OCS.

Of the studies that examined fluvoxamine, two (Poyurovsky et al., 1999; Reznik & Sirota, 2000) used open label trial designs and one (Reznik et al., 2008) used an RCT design. All three studies showed reductions in OCS in response to fluvoxamine. Specifically, Poyurovsky and colleagues (1999) examined the impact of up to 150 mg/day of fluvoxamine on OCS in a sample of 10 individuals with schizophrenia, all of whom had baseline Y-BOCS scores greater than or equal to seven. Seven participants completed the study and showed reduced OCS. In another open label trial, Reznik and Sirota (2000) enrolled 16 individuals with schizophrenia and refractory OCS (Y-BOCS $\geq$ 26) and tested the efficacy of 150–200 mg of fluvoxamine added to an established antipsychotic regimen on both OCS and positive and negative symptoms of schizophrenia. The addition of fluvoxamine was associated with a 33% reduction in OCS on average, and reductions in both positive and negative symptoms of schizophrenia. Finally, in a RCT of 30 individuals with schizophrenia and elevated OCS (Y-BOCS $\geq$ 26), Reznik and colleagues (2001) compared the efficacy of adding 150–200 mg/day of fluvoxamine to antipsychotics compared to antipsychotics alone on OCS. Results indicated that adding fluvoxamine led to greater reductions in OCS than antipsychotics alone.

Both studies examining fluoxetine (Agarwal & Agarwal, 2000; Kiran & Chaudhury, 2018) used open label trial designs with small sample sizes. Specifically, in a sample of seven participants with schizophrenia with co-occurring OCS (diagnosed through clinical interview), Agarwal and Agarwal (2000) demonstrated improvement in OCS for five of seven participants after 40–80 mg/day of fluoxetine. Kiran and Chaudhury (2018) examined whether the addition of fluoxetine on top of an antipsychotic (olanzapine) improved anxiety symptoms for 93 hospitalized individuals with schizophrenia, with a subsample analysis examining the specific impact on OCS for eight individuals. Their findings indicated little impact of olanzapine on OCS in this subsample, but reductions in

Table 1
Psychopharmacological interventions.

	Study Design	Study Length	Sample Size	Attrition	Dosage	Outcome Measure[a]		Participant Characteristics			Main Findings
						Baseline *M* (SD)	Post *M* (SD)	Age Range	Gender (% Male)	Race/ Ethn.	
TCA: Clomipramine (CMI)											
Yaryura-Tobias, Neziroglu, and Bergman (1976)	Open trial	8 wks	10	40%	25–300 mg; on top of other meds	Scores on the OCI not reported	Scores on the OCI not reported	18–65	Not reported	Not reported	CMI led to reductions in scores on an OCI subscale for "anxiety caused by rituals"
Zohar, Kaplan, and Benjamin (1993)	Open label trial; off-on-off design	Varied (3 wks–3 mon.)	5	0%	250–300 mg; on top of other meds	27.8 (7.36)	8.4 (4.93)	18–36	80%	Not reported	CMI led to reduction of OCS; discontinuing medication led to OCS resumption
Berman et al. (1995)	Randomized crossover study	13 wks (6 w/ placebo or CMI; wk 7 washout; wks 8–13 cross)	6	16.7%	100–250 mg	22.33 (4.55)	13.5 (7.48)	34–52	66.7%	Not reported	CMI outperformed placebo in the treatment of obsessions and compulsions. Two patients could not receive the full dose of CMI due to side effects
SSRI: Fluoxetine (Fluox)											
Agarwal and Agarwal (2000)	Open label trial	12 wks	7	0%	40–80 mg	30.3	14	18–35	85.7%	Not reported	Reduction in OCS and schizophrenic symptoms for 5 participants, 2 showed no change.
Kiran and Chaudhury (2018)	Open label trial	16 wks	93 total (8 with OCS + SCZ subsample only)	Not reported	Not reported (8 wks antipsychotic; fluox. added 8 wks).	32.88 (2.53)	23.13 (2.17)	18–55	85%	Not reported	Reduction in OCS after administration of fluox.
SSRI: Fluvoxamine (Fluv.)											
Poyurovsky et al., 1999	Open label trial	12 wks	10	30%	Up to 150 mg/day	21.0 (5.2)	13.3 (8.7)	36–60	29%	Not reported	Reduction in OCS as well as positive and negative schizophrenic symptoms
Reznik and Sirota (2000)	Open label trial	8 wks	16	0%	150–200 mg/day	26.5 (4.45)	17.78 (7.34)	19–67	69%	Not reported	Reduction in OCS and symptoms of schizophrenia
Reznik et al., 2001	RCT	8 wks	30		150–200 mg/day	Fluv.: 27.18 (4.54) No Fluv.: 26.06 (.47)	Fluv.: 19.18 (8.1) No Fluv.: 22.43 (5.0)	25–64	73%	Not reported	Greater reduction in OCS from fluvoxamine augmentation than those who were on antipsychotics only
SSRI: Escitalopram											
Stryjer et al., 2013	Open label trial	12 wks	15	13.3%	20 mg/day	28.9 (7.2)	23.3 (8.8)	21–61	66.7%	Not reported	Reduction in OCS as well as positive and negative schizophrenic symptoms
Rubin-Kahana et al., 2019	Open label trial	12 wks	15	13.3%	20–40 mg/day	29.4 (6.64)	18.67 (9.42)	22–54	47%	Not reported	Reduction in OCS as well as positive and negative schizophrenic symptoms
Antipsychotic: Aripiprazole											
Glick et al., 2008	Open label trial	6 wks	15	53.3%	10–30 mg/day	25.43 (4.61)	12.42 (6.19)	18–37	86%	71% white, 29% other	Modest improvement in OCS for some, no exacerbation, high attrition
Antipsychotic: Risperidone											
Veznedaroglu et al. (2003)	Single blind trial	8 wks	40	10%	$M = 3.5$ mg ($SD = 1.8$)	2.53 (6.43)	0.53 (1.00)	18–60	55.6%	Not reported	Reduction in OCS, no evidence of exacerbation
Antipsychotic: Ziprasidone											
		8 wks			80–200 mg/day			18–65			

(*continued on next page*)

Table 1 (*continued*)

	Study Design	Study Length	Sample Size	Attrition	Dosage	Outcome Measure[a]		Participant Characteristics			Main Findings
						Baseline *M* (SD)	Post *M* (SD)	Age Range	Gender (% Male)	Race/ Ethn.	
Juven-Wetzler, Fostick, Cwikel-Hamzany, Balaban, and Zohar (2014)	Open label, non-randomized		45 (29 subsample OCS + SCZ)	Not reported		23.97 (6.81)	Not reported		48 3%	Not reported	Reduction in OCS at least 25% for approximately half of sample, half experienced no effect or exacerbation
Antipsychotics: Clozapine/Olanzapine (CLZ/OLZ) vs Amisulpride/Aripiprazole (AMS/APZ)											
Schirmbeck et al., 2013	Naturalistic follow-up of two groups	12-months	75	Not reported		CLZ/OLZ: 11.1 (9.3) AMS/APZ: 2.2 (3.8)	CLZ/OLZ: 12.0 (9.9) AMS/ APZ:0.9 (1.8)			Not reported	All but 1 patent treated with CLZ or OLZ showed persisting or increasing OCS, patients treated with AMS or APZ showed decreases in compulsive symptoms
Anticonvulsant: Lamotrigine											
Poyurovsky, Glick, and Koran (2010)	Open label trial	8 wks	11	18%	Up to 200 mg	22.9 (6.1)	17.4 (3.6)	23–32	56%	Not reported	Reduction in OCS and depressive symptoms; 5 of 9 considered treatment responders

Note.
[a] Unless explicitly noted, the Y-BOCS was used as the primary outcome measure in all studies.

OCS after introduction of fluoxetine; however, the specific dosages provided to individuals with OCS were not reported.

Both studies that examined escitalopram used open label trial designs. Specifically, Stryjer and colleagues (2013) enrolled 15 patients diagnosed with schizophrenia with baseline Y-BOCS scores greater than 16 for a 12-week trial of up to 20 mg/day. Rubin-Kahana and colleagues (2019) enrolled 15 patients diagnosed with schizophrenia who had baseline Y-BOCS scores greater than 12 for a 13-week trial of up to 40 mg/day of escitalopram. Both trials found beneficial effects on OCS, as well as additional reductions in positive and negative schizophrenia symptoms. However, of note, the dosage studied by Rubin-Kahana and colleagues is now above current limitations of escitalopram due to concern for QT interval prolongation (i.e., electrical activity of the heart that places patients at risk for ventricular arrhythmias), limiting the potential clinical utility of findings.

Limitations of SSRI Studies. Studies again were limited by small sample sizes (range = 7–30), primary use of open label trial designs, and variable (0–30%) attrition rates. Race and ethnicity of participants were not reported for any study, lending concern for generalizability of findings. Finally, inclusion criteria for what constituted "co-occurring OCS" varied across studies or was not reported.

Results for Antipsychotics. Four studies examined the effect of antipsychotic medications on reductions in OCS. Each study examined different medications, including aripiprazole (Glick et al., 2008; Schirmbeck & Zink, 2013), risperidone (Veznedaroglu et al., 2003), ziprasidone (Juven-Wetzler et al., 2014), and clozapine, olanzapine, and amisulpride (Schirmbeck & Zink, 2013). All studies used the Y-BOCS as a primary outcome measure and results were mixed. Glick and colleagues (2008) tested 10–30 mg/day of aripiprazole over six weeks in a sample of adults diagnosed with schizophrenia and OCS $\geq$ 16 on the Y-BOCS. This open label trial yielded overall reductions in OCS across the sample; however, four people discontinued the trial due to worsening symptoms (n = 2) or side effects (n = 2). Veznedaroglu et al. (2003) tested the impact of risperidone on OCS in a sample of 40 adults diagnosed with schizophrenia over eight weeks, also finding beneficial effects. However, the mean baseline Y-BOCS score was low (M = 2.53) and there was no minimum criterion on the Y-BOCS for study inclusion, making generalizations from these findings difficult. In contrast, Juven-Wetzler et al. (2014) found more mixed findings for ziprasidone in an eight-week trial of 45 adults with schizophrenia, 29 of whom had significant OCS in the subsample of interest for this review. Specifically, they found that about half the sample experienced significant reduction in OCS on the Y-BOCS; however, the remaining half of the sample experienced no effect or experienced a worsening effect of OCS over the trial period. Finally, Schirmbeck and colleagues (2013) conducted a longitudinal follow-up of 75 adults diagnosed with schizophrenia or schizoaffective disorder; participants were classified into two groups as a function of the medications they were prescribed. Group 1 (n = 41) received either of clozapine or olanzapine (CLZ/OLZ) and Group 2 (n = 34) received either amisulpride or aripiprazole (AMS/APZ). Of individuals treated with CLZ/OLZ who reported OCS at the beginning of the 12-month follow-up, all but one showed persisting or increasing OCS. In contrast, individuals treated with AMS/APZ showed decreases in compulsive symptoms over time. Of note, Y-BOCS scores were lower at baseline among individuals treated with AMS or APZ compared to those treated with CLZ or OLZ.

Limitations of Antipsychotic Studies. Primary limitations of antipsychotic studies include lack of replication of studies using the same pharmaceutical agent and high attrition rates (up to 53%). Only one trial (Glick et al., 2008) reported race and ethnicity of participants (who all identified as White), lending further concern for generalizability. Inclusion criteria for OCS severity also varied across studies.

Other Medications or Their Combination. One additional study examined an alternative psychopharmacological agent, lamotrigine. Poyurovsky et al. (2010) examined lamotrigine, an anticonvulsant that modulates glutamate, as an adjunctive agent on OCS in a sample of 11

adults diagnosed with schizophrenia or schizoaffective disorder and a Y-BOCS score ≥ 16 in an eight-week, open-label trial. Their findings indicated overall reduction in Y-BOCS scores across the sample, with 5 of 9 treatment completers (55.5%) being classified as treatment responders (i.e., had a 35% or more decrease in Y-BOCS scores).

2.3. Psychosocial interventions

Summary of the literature. Seven studies examined a psychosocial intervention to address OCS in the context of psychosis. All studies examined a form of CBT. Table 2 presents an overview of included studies, their design, and main outcomes.

Description of Studies. Of the studies that examined CBT for co-occurring psychosis, one was a naturalistic study of 21 participants (Tundo et al., 2014), one was an RCT comparing mindfulness therapy versus progressive muscle relaxation (PMR) in 90 participants with psychosis (Moritz et al., 2015), and five were characterized as case studies (Christodoulides, Callcott, & Dudley, 2011; Ekers et al., 2004; Gole, 2015; Hagen et al., 2014; Kobori, Sato, Katsukura, & Harada, 2008). Given the sparseness of psychosocial literature identified, case studies were included in the synthesis, although we place emphasis in our synthesis on the two non-case studies. The mean age of participants in the study led by Tundo et al. (2014) was 29 (range 18–37; 8 female), and the mean age in the study led by Moritz and colleagues was 38.11 in the mindfulness group and 37.46 in the PMR group.

Results for Psychosocial Interventions. All seven studies provided support for the effectiveness of CBT-based interventions for co-occurring psychosis and OCS. All studies explicitly reported improvements in OCS or related phenomena and/or OCD diagnostic status among participants with co-occurring psychosis, using the Y-BOCS, obsessive-compulsive subscale of the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983), Obsessive-Compulsive Inventory-Revised Abbreviated (OCI-R; Foa et al., 2002), and/or diagnostic criteria. Specifically, Tundo et al. (2014) followed 21 individuals age 18–65 years who met DSM-IV criteria for OCD and either schizophrenia or schizoaffective disorder as assessed by the Structured Clinical Interview for DSM-IV (SCID-I; First et al., 1997). Participants had OCS of at least moderate severity (Y-BOCS total score ≥16) and stabilized schizophrenia or schizoaffective disorder, as defined by a Positive and Negative Symptoms Scale (PANSS; Kay et al., 1987) total score ≤75. Patients received between one and 40 h of CBT ($M = 34.3$ for patients with schizoaffective disorder; $M = 31.1$ for patients with schizophrenia), with treatment comprising imaginal and in vivo exposure, ritual-prevention and/or delay, cognitive therapy, and additional interventions used to supplement exposure and ritual-prevention strategies. Patients were followed for one year after baseline; of note, several patients were still receiving CBT at that time. Findings indicated gradual but continuous and significant improvement in Y-BOCS and Clinical Global Impressions-Severity of Illness (CGI-S; Guy, 1976) scores from baseline to 6 months, and to a lesser extent from 6 months to 12 months after baseline.

Moritz et al. (2015) recruited 90 participants aged 18–65 years who had a diagnosis of schizophrenia/psychosis that was previously established by a psychiatrist, psychotherapist, or another physician or mental health expert. Although the presence of OCS was not a criterion for study inclusion, OCS (according to the OCI-R) was measured and included in analyses. Of note, the mean baseline OCI-R score was below the clinical cutoff for both groups (19.24 in the mindfulness group and 18.87 in the PMR group). Patients were randomly assigned to six weeks of either self-guided mindfulness or self-guided PMR. The mindfulness manual included an introduction to the concept of mindfulness and descriptions of several classic techniques and exercises. The PMR manual included background/rationale, exercises, and answers to potential questions that might arise. Audio files accompanied both interventions. The authors reported a treatment completion rate of 71% and adherence rates of 61.5% in the mindfulness group and 51.1% in PMR group. Importantly,

Table 2
Psychosocial interventions.

	Study Design	Study Length	Sample Size	Attrition	Outcome Measure[a]		Participant Characteristics			Main Findings
					Baseline *M* (SD)	Post *M* (SD)	Age Range	Gender (% Male)	Race/ Ethn.	
Tundo et al. (2014)	Naturalistic Study	Varied	21	24%	30.8 (6.7)	26.8 (8.0)	18–37	42%	Not reported	CBT led to reduction of OCS, with a lower likelihood of improvement among patients with lifetime alcohol/substance abuse disorder.
Moritz et al. (2015)	Randomized controlled trial	6 weeks	90	29%	PMR: 18.87 (7.13) on OCI-R; Mindfulness: 19.24 (8.16) on OCI-R	Not reported (shown graphically)	18–65	42%	Not reported	PMR and mindfulness both showed a decline in OC symptoms at a medium effect size
Christodoulides et al. (2011)	Case Study	17 weekly sessions	1	N/A	25 on CORE; 32 on IIT; 43 on IoU	7 on CORE; 0 on IIT; 9 on IoU	50	100%	Not reported	CBT led to reduction of OCS
Ekers, Carman, and Schlich (2004)	Case Study	20 h	1	N/A	31	17	31	100%	Not reported	ERP led to reduction of OCS
Gole (2015)	Case Study	18 weekly sessions	1	N/A	54 on IUS-12; 9 on BSI-OC	26 on IUS-12; 6 on BSI-OC	Not reported	100%	Not reported	Clinical Philosophy led to reductions in intolerance of uncertainty and OC symptoms
Hagen et al. (2014)	Case Study	3 weekly sessions	1	N/A	24	5	Late 20s	100%	White	CBT led to reduction of OCS
Kobori et al. (2008)	Case Study	19 weekly sessions	1	N/A	31	11	26	100%	Not reported	CBT led to reduction of OCS

Note.
[a] Unless explicitly noted, the Y-BOCS was used as the primary outcome measure in all studies.

adherence was defined as reading the full manual, which may not capture the nuances of treatment adherence (e.g., consistently engaging in the recommended exercises). Symptom change on the OCI-R was measured six weeks after the baseline assessment. The effect of time for both groups bordered on significance at a medium effect size, and there were no significant group differences or interaction effects. These findings suggest the potential utility of mindfulness and PMR for OCS among individuals with psychosis.

With respect to the case studies, all participants identified as male between the ages of 25 and 50 (except for Gole, 2015, where no age was reported). While all reported on CBT, the exact content of each intervention varied. Four explicitly included exposure with response prevention in their intervention (Ekers et al., 2004; Hagen et al., 2014; Kobori et al., 2008) while Gole (2015) described treatment as "Clinical Philosophy," a novel philosophy-oriented method within the broader framework of third wave CBT and existential analysis. The length of treatment varied from three to 19 sessions. The Y-BOCS was included as an outcome measure in three (Ekers et al., 2004; Hagen et al., 2014; Kobori et al., 2008). Among studies that did not include the Y-BOCS, Christodoulides et al. (2011) used Clinical Outcomes in Routine Evaluation (CORE; Evans et al., 2000), and the Interpretation of Intrusive Thoughts (IIT; Freeston et al., 1995); Gole (2015) indexed OCS via the obsessive-compulsive subscale of the BSI. The timing and frequency of outcome measurement also varied across studies. Four studies included a follow-up assessment after treatment was complete (Christodoulides et al., 2011; Ekers et al., 2004; Hagen et al., 2014; Kobori et al., 2008), which ranged from six months to two years post-treatment. Of note, Kobori et al. (2008) reported that the patient had not relapsed at the two-year follow-up but did not report whether a quantitative assessment measure was used at that time. Across all case studies, CBT was determined to be effective for five individuals with co-occurring OCS and psychosis.

Limitations of studies reviewed. Although the reviewed studies indicate the potential benefits of CBT for OCS with co-occurring psychosis, the small samples sizes (*N*s ranging from 1 to 90; modal $N = 1$) and variability in treatment length, therapeutic strategies, delivery method, and outcome variables are considerable limitations. Moreover, race was only reported in one study (Hagen et al., 2014), ethnicity was not reported in any study, and male identifying participants were overrepresented, which may limit the generalizability of these findings. The RCT conducted by Moritz et al. (2015) and the naturalistic study conducted by Tundo et al. (2014) included more rigorous methodology (i.e., Type 3 according to the Nathan and Gorman (2002) classification system), yet additional double-blind and placebo-controlled trials, as well as standardized treatment protocols, are needed to isolate the specific effects of psychosocial interventions.

2.4. Neuromodulation

Summary of the literature. Three studies examined neuromodulation (Mendes-Filho, Belmonte-de-Abreu, Pedrini, Cachoeira, & Lobato, 2013; Plewnia et al., 2008; Verma, Kumar, Mahapatra, & Shah, 2018). Table 3 presents an overview of included studies, their design, and their main outcomes.

Description of studies. Three approaches to neuromodulation were examined across included studies: rTMS (Mendes-Filho, de Jesus, Belmonte-de-Abreu, Cachoeira, & Rodrigues Lobato, 2016), DBS (Plewnia et al., 2008), and tDCS (Verma et al., 2018). The study led by Mendes-Filho and colleagues was a double-blind pilot RCT, whereas Plewnia and colleagues and Verma and colleagues both reported on a single case. Mendes-Filho and colleagues examined the effectiveness of rTMS in a sample of 12 adults (2 female; mean age = 41) with a diagnosis of either schizophrenia ($n = 11$) or schizoaffective disorder ($n = 1$) and a score of 16 or higher on the Y-BOCS. rTMS was applied to the supplementary motor area of six participants, who were compared with six participants who received sham rTMS (i.e., stimulation that induces local sensations on the scalp similarly to the disturbances caused by the active stimulation). Y-BOCS scores, Brief Psychiatric Rating Scale (BPRS; Overall and Gorham, 1962) scores, and serum Brain Derived Neurotrophic Factor (BDNF) levels were assessed at pre-treatment, post-treatment, and 4 weeks after treatment.

Plewnia et al. (2008) reported on a 51-year-old female treated with unilateral DBS of the right nucleus accumbens. The participant was described as having intractable OCD and residual symptoms of schizophrenia. Medication (60 mg citalopram, 300 mg quetiapine) remained unchanged during DBS treatment. Symptoms were assessed with the Y-BOCS and Global Assessment of Functioning (GAF; Hall, 1995) at 6 months, 1 year, and 2 years after implantation.

Verma et al. (2018) examined the effects of cathode placement at supplementary motor cortex and anode at right occipital region as an add-on approach to pharmacotherapy to manage co-occurring OCD in a 32-year-old male with chronic schizophrenia. Symptoms were assessed with the Y-BOCS after ten consecutive days of twice-daily sessions, and again after ten more sessions.

Results for neuromodulation. Findings from the two case studies suggest some response to neuromodulation for co-occurring OCS and psychosis. Specifically, Plewnia et al. (2008) reported a 25% reduction in OCS and no increase in psychotic symptoms with DBS. The patient reportedly appraised the effect of stimulation as satisfying and never requested a cessation of treatment. Verma et al. (2018) observed a "clinically significant reduction" in OCS (e.g., Y-BOCS change from 31 to 17) after treatment with tDCS as an adjunct to pharmacotherapy (clozapine 500 mg; fluoxetine 80 mg/d cross tapered with fluvoxamine optimized to 200 mg/d). In the pilot RCT led by Mendes-Filho et al. (2016), no significant differences were found between active rTMS and

Table 3
Neuromodulation.

	Study Design	Study Length	Sample Size	Attrition	Outcome Measure[a]		Participant Characteristics			Main Findings
					Baseline *M* (SD)	Post *M* (SD)	Age Range	Gender (% Male)	Race/ Ethn.<	
Mendes-Filho et al. (2016)	Randomized controlled trial	20 sessions of 20 min each	12	0%	Active: 26.0 (6.2); Sham: 25.8 (5.7)	Not reported (shown graphically)	18–65	82%	Not reported	rTMS did not significantly change OC symptoms relative to sham
Plewnia et al. (2008)	Case Study	3 months of DBS	1	N/A	32	24	51	0%	Not reported	DBS led to reduction of OCS
Verma et al. (2018)	Case Study	20 sessions of 20 min each; 10 additional sessions	1	N/A	31	24; 17 after 10 additional sessions	32	100%	Not reported	tDCS led to reduction of OCS

Note.
[a] Unless explicitly noted, the Y-BOCS was used as the primary outcome measure in all studies.

sham groups on any measures. Both active and sham rTMS were well tolerated, and no significant adverse events were observed in either group.

Limitations of studies reviewed. Sample size was a notable limitation across neuromodulation studies. Although study design (i.e., double-blind pilot RCT) was a strength of the study led by Mendes-Filho and colleagues, the small sample size ($N = 12$) precludes definitive conclusions about the utility of rTMS for OCS co-occurring with schizophrenia. Although findings from Plewnia and colleagues and Verma and colleagues suggest that future research on DBS and tDCS is warranted, the case study design lacks methodological rigor. Race and ethnicity were not reported in any study.

3. Discussion

This systematic review is the first to comprehensively synthesize the existing literature on psychopharmacological, psychosocial, and neuromodulation interventions for co-occurring OCS and psychosis. Given the prevalence of this comorbidity profile, and its association with higher illness severity and lower overall functioning, an understanding of the evidence base for various treatment approaches is essential. The treatment outcomes summarized in this review have implications for future research directions and clinical recommendations.

This review notably expands on previous reviews that yielded emerging support for certain pharmacotherapies (Poyurovsky et al., 2004) and emerging support for CBT (Schirmbeck & Zink, 2013; Schirmbeck & Zink, 2013; Tundo & Necci, 2016). The pharmacological studies reviewed in the present study provided some preliminary evidence for effectiveness of both TCAs and SSRIs as augmentative agents on OCS in the context of psychosis, although as with prior reviews, the continued lack of rigorous, adequately powered clinical trials makes firm conclusions challenging. Consistent with earlier reviews, findings remain decidedly more mixed about the utility of antipsychotics for addressing OCS, particularly with their associated risk of OCS exacerbation. This risk is also likely higher than what is demonstrated in this review, given that we did not include studies that specifically reported on new-onset OCS following an antipsychotic trial. One study also provided preliminary evidence for the effectiveness of lamotrigine, warranting further research.

Consistent with previous reviews of CBT for co-occurring OCS and psychosis, our review found general support for its efficacy, although the definition of "CBT" varied widely and rigorous trial data remain absent. For example, five of the seven studies of CBT emphasized exposure-based techniques, suggesting that this gold-standard treatment for OCS can be delivered in the context of psychosis. Importantly, interventions were classified as "third wave" CBT in two studies—mindfulness in Moritz et al. (2015) and "Clinical Philosophy" in Gole (2015). Preliminary meta-analytic findings point to the potential utility of third-wave interventions that incorporate principles of mindfulness (e.g., acceptance and commitment therapy; ACT) for OCD (Bluett, Homan, Morrison, Levin, & Twohig, 2014), but significant limitations of the studies included in the empirical review preclude drawing conclusions about the relative effectiveness of ACT versus OCD (i.e., inclusion of only one case study, three small multiple baseline studies, and one RCT, only female participants in the RCT; with all studies conducted by a single research group). Mindfulness-based interventions have also been shown to be moderately effective in treating negative symptoms in schizophrenia (Khoury, Lecomte, Gaudiano, & Paquin, 2013). Given the potential benefits of integrating principles of mindfulness and acceptance with exposure-based treatment for OCS (e.g., Buchholz & Abramowitz, 2019; Twohig et al., 2018), this approach should be considered for future examination in the context of psychosis. Specifically, there is a need for treatment development efforts that integrate CBT for psychosis (see Rector & Beck, 2012) with ACT-informed ERP-based techniques. As such, treatments that are designed and empirically tested specifically for this clinical population will constitute a major development in the field and an important resource for clinicians.

Finally, our review also included three studies of neuromodulation for treating OCS in the context of psychosis; to our knowledge, this is the first systematic review to include studies of neuromodulation for this symptom presentation. Case studies provided some support for DBS and tDCS, yet an RCT of rTMS did not find differences between the active treatment and sham placebo. Thus, although some patients may appear to improve following neuromodulation, placebo effects likely account for observed changes in symptoms.

The current review highlighted several major limitations of the existing literature. First, studies reviewed varied widely in methodology. Consistent with prior reviews, most studies that met criteria for inclusion in the review were case studies or case series. Because these studies are based on extremely small samples and do not involve random assignment to a treatment or control groups, one cannot draw strong conclusions about the specificity of the treatment effect. That is, any improvement observed could be explained by factors such as the variable course of OCS and psychotic symptoms, the use of additional treatments, and the expectation of improvement (i.e., placebo effects). Thus, emphasis is best placed on the three RCTs and one randomized crossover study included in this review. There was also considerable heterogeneity regarding length and frequency of treatment, and timing of outcome measurement. This area is ripe for additional crossover trials with consistent and clearly delineated treatment protocols to isolate the unique effects of treatment augmentation (e.g., antipsychotic medication augmented with an SSRI).

Second, most studies did not report sociocultural variables such as race, ethnicity, cultural background, or socioeconomic status. This is a major concern, given the known racial diagnostic bias in psychotic disorders (Schwartz & Blankenship, 2014; Olbert, Nagendra, & Buck, 2018), systemic inequities in healthcare (Shavers & Shavers, 2006), and underrepresentation of marginalized racial and ethnic groups in clinical research (Fisher & Kalbaugh, 2011). Furthermore, extant data suggest that sociocultural factors impact the course of treatment for both OCD (Williams et al., 2015) and schizophrenia (Oluwoye et al., 2018); however, these data are limited in large part due to the underrepresentation of minoritized groups in clinical trials for OCS and psychosis (Wetterneck et al., 2012; Williams, Beckmann-Mendez, & Turkheimer, 2013; Williams, Powers, Yun, & Foa, 2010). Given that previous research has found that race affects OCS (Wu & Wyman, 2016) and schizophrenia (Nagendra et al., 2018) symptomology, and that racial discrimination has demonstrated negative mental health consequences (Berger & Sarnyai, 2015; Williams et al., 2017), treatments that have demonstrated efficacy and effectiveness in samples of primarily white participants may not show equivalent outcomes among minoritized groups. Thus, it is imperative that future studies examining this comorbidity profile employ recommended strategies (e.g., Williams et al., 2013) for recruiting diverse samples and report comprehensive demographic data.

Third, no studies included participants under the age of 18, despite epidemiological research suggesting that both OCS and psychosis typically develop during adolescence and young adulthood (McGrath et al., 2016; Ruscio et al., 2010). Given research suggesting that early intervention is associated with better clinical response for psychosis (Fusar-Poli et al., 2017), coupled with evidence that patients with co-occurring OCS and psychosis show poorer treatment response and worse long-term outcomes (Fenton & McGlashan, 1986; Jaeger et al., 2008), this is a major gap in the literature.

A strength of this review is our comprehensive synthesis of psychopharmacological, psychosocial, and neuromodulation research, which is consistent with efforts to disrupt the traditional "siloed" approach to psychiatric and psychological research (Graham, Callaghan, & Richardson, 2014). Nonetheless, most studies reviewed emphasized only one therapeutic approach rather than the combined effects of medication and therapy, even when participants were treated with a

combined approach; future studies should make sure to report all therapies an individual is receiving to address OCS and psychosis.

Our conclusions should be interpreted considering this review's methodological limitations. First, our systematic literature search excluded "gray literature" (e.g., conference abstracts, unpublished trial data, dissertations/theses) as well as non-English language studies. Selection and publication bias may have therefore limited the comprehensiveness of this review and generalization of findings to non-English speaking regions.

3.1. Future directions

In addition to the methodological recommendations for future studies we discussed above (i.e., improving standardization of reporting for client demographics, therapies received, etc.), Results of this review suggest the following future directions:

1. To inform the personalization of treatments and advance the literature in this space, it is critical for researchers to examine the underlying treatment mechanisms (e.g., cognitive-behavioral change) that are implicated in the co-occurrence of OCS and psychosis.
2. The increasing proliferation of comprehensive first episode psychosis programs (FEP; e.g., coordinated specialty care; Bello et al., 2017; Powell, Hinger, Marshall-Lee, Miller-Roberts, & Phillips, 2021) presents unique opportunities to identify individuals with OCS in the context of psychosis who are at known risk for poorer clinical course. To date, these programs have not traditionally included routine screening for OCS or direct treatment of OCS. This is an area ripe for research regarding how best to integrate targeted OCS screening into FEP treatment. If such screening is indicated, future work also should aim to identify the best strategies for identifying individuals with OCS and psychosis and connecting them with tailored treatment.
3. OCS and psychosis treatment researchers continue to be relatively siloed. Moving forward, it is important that researchers in these fields collaborate more effectively in both the design, delivery, and dissemination of treatments for these at-risk individuals. Individuals specialized in the presentations of OCS and psychosis may be able to identify areas of overlap in the phenomenology, presentation, and effective treatment of each condition that can help in identifying efficient and effective intervention targets for individuals with comorbid psychosis and OCS. Routine assessment of both OCS and psychosis in clinical trials (e.g., using the Y-BOCS and PANSS) would increase researchers' ability to draw conclusions about such symptoms when they co-occur.
4. Our updated review indicates that, despite significant passage of time and increased interest in and understanding of this population, studies largely rely on case study or case series design. How can we more rapidly develop confidence in an evidence-base for effective treatments for this low-base rate, but highly burdensome, clinical presentation? Given overall low base rates of OCD and psychosis, recruitment for fully-powered clinical trials presents feasibility challenges. One option is to develop a clinical registry or consortium of sites willing to treat OCS in the context of psychosis who engage in harmonization of data progress monitoring and treatment practices to facilitate aggregation of findings.
5. As the treatment science advances, it may be particularly important for researchers to think about implementation challenges during treatment design (i.e., "design for dissemination"; Chambers, 2020). For example, ERP in particular suffers from low uptake even for OCD alone (Becker-Haimes et al., 2017; Reid et al., 2018), with clinician concerns about exacerbating symptoms a major reason for its underuse (Olatunji, Deacon, & Abramowitz, 2009). Efforts to increase use of ERP with individuals also experiencing psychotic symptoms may be a particular challenge. Strategies from the implementation science literature designed to dually enhance a treatment's engagement of target clinical mechanisms while maximizing acceptability and feasibility for stakeholders (e.g., intervention mapping; Durks et al., 2017) may be particularly salient in this context to facilitate access to the most effective treatments for this vulnerable clinical population.

4. Conclusions

Enhancing treatment models for co-occurring OCS and psychosis has high potential to offset significant psychiatric burden. Our review indicates some emerging evidence for treatments to reduce individuals' suffering, but firm conclusions are precluded by a lack of rigorous clinical trial data to suggest the superiority of one treatment (psychopharmacological or psychosocial) over placebo, with little data to guide treatment combination and sequencing approaches. Rigorous research, perhaps supported by a clinical registry or consortium of sites engaged in data harmonization as described above, with longitudinal follow-is needed to truly advance the scientific literature on how best to address the complex needs of individuals with OCS and psychosis.

Declaration of competing interest

The authors declare no conflict of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jocrd.2021.100704.

References

Achim, A. M., Maziade, M., Raymond, É., Olivier, D., Mérette, C., & Roy, M. A. (2011). How prevalent are anxiety disorders in schizophrenia? A meta-analysis and critical review on a significant association. *Schizophrenia Bulletin, 37*(4), 811–821.

Adam, Y., Meinlschmidt, G., Gloster, A. T., & Lieb, R. (2012). Obsessive–compulsive disorder in the community: 12-month prevalence, comorbidity and impairment. *Social Psychiatry and Psychiatric Epidemiology, 47*(3), 339–349.

Agarwal, V., & Agarwal, K. (2000). Treatment of obsessive compulsive symptoms in schizophrenia with fluoxetine. *Indian Journal of Psychiatry, 42*(3), 291.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Becker-Haimes, E., Okamura, K., Wolk, C., Rubin, R., Evans, A., & Beidas, R. (2017). Predictors of clinician use of exposure therapy in community mental health settings. *Journal of Anxiety Disorders, 49*, 88–94.

Bello, I., Lee, R., Malinovsky, I., Watkins, L., Nossel, I., Smith, T., & Dixon, L. B. (2017). OnTrackNY: The development of a coordinated specialty care program for individuals experiencing early psychosis. *Psychiatric Services, 68*(4), 318–320.

Berger, M., & Sarnyai, Z. (2015). "More than skin deep": Stress neurobiology and mental health consequences of racial discrimination. *Stress: The International Journal on the Biology of Stress, 18*(1), 1–10.

Berlim, M. T., Neufeld, N. H., & Van den Eynde, F. (2013). Repetitive transcranial magnetic stimulation (rTMS) for obsessive–compulsive disorder (OCD): An exploratory meta-analysis of randomized and sham-controlled trials. *Journal of Psychiatric Research, 47*(8), 999–1006.

Berman, I., Sapers, B., Chang, H., Losonczy, M., Schmildler, J., & Green, A. (1995). Treatment of obsessive-compulsive symptoms in schizophrenic patients with clomipramine. *Journal of Clinical Psychopharmacology, 15*(3), 206–210.

Bluett, E. J., Homan, K. J., Morrison, K. L., Levin, M. E., & Twohig, M. P. (2014). Acceptance and commitment therapy for anxiety and OCD spectrum disorders: An empirical review. *Journal of Anxiety Disorders, 28*(6), 612–624.

Brunelin, J., Mondino, M., Bation, R., Palm, U., Saoud, M., & Poulet, E. (2018). Transcranial direct current stimulation for obsessive-compulsive disorder: A systematic review. *Brain Sciences, 8*(2), 37.

Buchholz, J. L., & Abramowitz, J. (2019). Using an acceptance and commitment therapy approach when exposure and cognitive therapy become rituals in the treatment of obsessive-compulsive disorder. *Journal of Cognitive Psychotherapy, 33*(3), 256–268.

Catapano, F., Sperandeo, R., Perris, F., Lanzaro, M., & Maj, M. (2001). Insight and resistance in patients with obsessive-compulsive disorder. *Psychopathology, 34*(2), 62–68.

Cederlöf, M., Lichtenstein, P., Larsson, H., Boman, M., Rück, C., Landén, M., et al. (2015). Obsessive-compulsive disorder, psychosis, and bipolarity: A longitudinal cohort and multigenerational family study. *Schizophrenia Bulletin, 41*(5), 1076–1083.

Chambers, D. A. (2020). Sharpening our focus on designing for dissemination: Lessons from the SPRINT program and potential next steps for the field. *Translational behavioral medicine, 10*(6), 1416–1418.

Chang, H. H., & Berman, I. (1999). *Treatment issues for patients with schizophrenia who have obsessive-compulsive symptoms*.
Christodoulides, T., Callcott, P., & Dudley, R. (2011). Conceptualizing and targeting beliefs in the treatment of a man with obsessive compulsive disorder and overvalued ideation. *Cognitive Behaviour Therapist, 4*(2), 68–77.
Combs, D. R., & Mueser, K. T. (2017). *Schizophrenia and severe mental illness. Treatments for Psychological Problems and Syndromes*.
Cunill, R., Castells, X., & Simeon, D. (2009). Relationships between obsessive-compulsive symptomatology and severity of psychosis in schizophrenia: A systematic review and meta-analysis. *Journal of Clinical Psychiatry, 70*(1), 70–82.
Derogatis, L. R., & Melisaratos, N. (1983). The brief symptom inventory: An introductory report. *Psychological Medicine, 13*, 596–605.
Durks, D., Fernandez-Llimos, F., Hossain, L. N., Franco-Trigo, L., Benrimoj, S. I., & Sabater- Hernández, D. (2017). Use of intervention mapping to enhance health care professional practice: A systematic review. *Health Education & Behavior, 44*(4), 524–535.
Eddy, K. T., Dutra, L., Bradley, R., & Westen, D. (2004). A multidimensional meta-analysis of psychotherapy and pharmacotherapy for obsessive-compulsive disorder. *Clinical Psychology Review, 24*(8), 1011–1030.
Ekers, D., Carman, S., & Schlich, T. (2004). Successful outcome of exposure and response prevention in the treatment of obsessive compulsive disorder in a patient with schizophrenia. *Behavioural and Cognitive Psychotherapy, 32*(3), 375–378. https://doi.org/10.1017/S135246580400147X
Evans, J. M.-C., Frank, M., Barkham, M., Audin, K., Connell, J., & Graeme McGrath, C. (2000). Core: Clinical outcomes in routine evaluation. *Journal of Mental Health, 9*(3), 247–255.
Fenton, W. S., & McGlashan, T. H. (1986). The prognostic significance of obsessive-compulsive symptoms in schizophrenia. *American Journal of Psychiatry, 143*(4), 437–441. https://doi.org/10.1176/ajp.143.4.437
First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1997). *Structured clinical interview for DSM-IV Axis I disorders (SCID I)*. New York: Biometric Research Department.
Fisher, J. A., & Kalbaugh, C. A. (2011). Challenging assumptions about minority participation in US clinical research. *American Journal of Public Health, 101*(12), 2217–2222.
Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., Hajcak, G., et al. (2002). The obsessive-compulsive inventory: Development and validation of a short version. *Psychological Assessment, 14*(4), 485.
Foa, E. B., Kozak, M. J., Salkovskis, P. M., Coles, M. E., & Amir, N. (1998). The validation of a new obsessive–compulsive disorder scale: The Obsessive–Compulsive Inventory. *Psychological Assessment, 10*(3), 206.
Fonseka, T. M., Richter, M. A., & Müller, D. J. (2014). Second generation antipsychotic-induced obsessive-compulsive symptoms in schizophrenia: A review of the experimental literature. *Current Psychiatry Reports, 16*(11), 510. https://doi.org/10.1007/s11920-014-0510-8
Fontenelle, L. F., Lin, A., Pantelis, C., Wood, S. J., Nelson, B., & Yung, A. R. (2011). A longitudinal study of obsessive-compulsive disorder in individuals at ultra-high risk for psychosis. *Journal of Psychiatric Research, 45*(9), 1140–1145. https://doi.org/10.1016/j.jpsychires.2011.03.005
Freeston, M. H., Ladouceur, R., Provencher, M., & Blais, F. (1995). Strategies used with intrusive thoughts: Context, appraisal, mood, and efficacy. *Journal of Anxiety Disorders, 9*(3), 201–215.
Fusar-Poli, P., McGorry, P. D., & Kane, J. M. (2017). Improving outcomes of first-episode psychosis: An overview. *World Psychiatry, 16*(3), 251–265.
Gabriëls, L., Cosyns, P., Nuttin, B., Demeulemeester, H., & Gybels, J. (2003). Deep brain stimulation for treatment-refractory obsessive-compulsive disorder: Psychopathological and neuropsychological outcome in three cases. *Acta Psychiatrica Scandinavica, 107*(4), 275–282.
Gault, J. M., Davis, R., Cascella, N. G., Saks, E. R., Corripio-Collado, I., Anderson, W. S., … Abosch, A. (2018). Approaches to neuromodulation for schizophrenia. *Journal of Neurology, Neurosurgery & Psychiatry, 89*(7), 777–787.
Gole, M. (2015). Clinical philosophy in the treatment of paranoid schizophrenia and obsessive–compulsive disorder. *Archives of Psychiatry and Psychotherapy, 17*(4), 53–60. https://doi.org/10.12740/APP/60949
Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., … Charney, D. S. (1989). The Yale-Brown obsessive compulsive scale: I. Development, use, and reliability. *Archives of General Psychiatry, 46*(11), 1006–1011.
Graham, B. M., Callaghan, B. L., & Richardson, R. (2014). Bridging the gap: Lessons we have learnt from the merging of psychology and psychiatry for the optimisation of treatments for emotional disorders. *Behaviour Research and Therapy, 62*, 3–16.
Guy, W. B. R. R. (1976). Clinical global impression. *Assessment manual for Psychopharmacology*, 217–222.
de Haan, L., Hoogenboom, B., Beuk, N., van Amelsvoort, T., & Linszen, D. (2005). Obsessive- compulsive symptoms and positive, negative, and depressive symptoms in patients with recent-onset schizophrenic disorders. *Canadian Journal of Psychiatry, 50*, 519–524.
de Haan, L., Sterk, B., & van der Valk, R. (2012). Presence of obsessive compulsive symptoms in first-episode schizophrenia or related disorders is associated with subjective well-being and quality of life. *Early Intervention in Psychiatry*. https://doi.org/10.1111/j.1751-7893.2012.00377.x. Published online: 2 July 2012.
Hagen, K., Hansen, B., Joa, I., & Larsen, T. K. (2013). Prevalence and clinical characteristics of patients with obsessive-compulsive disorder in first-episode psychosis. *BMC Psychiatry, 13*(1), 1–6.
Hagen, K., Solem, S., Larsen, T. K., Joa, I., Kvale, G., & Hansen, B. (2014). Obsessive-compulsive disorder (OCD) in patients with first-episode psychosis (FEP): A prospective study. *Clinical Neuropsychiatry: Journal of Treatment Evaluation, 11*(6), 164–169.
Hall, R. C. (1995). Global assessment of functioning: A modified scale. *Psychosomatics, 36*(3), 267–275.
Hovington, C. L., McGirr, A., Lepage, M., & Berlim, M. T. (2013). Repetitive transcranial magnetic stimulation (rTMS) for treating major depression and schizophrenia: A systematic review of recent meta-analyses. *Annals of Medicine, 45*(4), 308–321.
Hwang, M. Y., Kim, S. W., Yum, S. Y., & Opler, L. A. (2009). Management of schizophrenia with obsessive-compulsive features. *Psychiatria Clinica, 32*(4), 835–851.
Insel, T. R., & Akiskal, H. S. (1986). Obsessive-compulsive disorder with psychotic features: A phenomenologic analysis. *American Journal of Psychiatry, 143*(12), 1527–1533.
Jaeger, M., Messer, T., Laux, G., Pfeiffer, H., Naber, D., Schmidt, L. G., et al. (2008). Standardized remission criteria in schizophrenia: Descriptive validity and comparability with previously used outcome measures. *Pharmacopsychiatry, 41*, 190–195.
Juven-Wetzler, A., Fostick, L., Cwikel-Hamzany, S., Balaban, E., & Zohar, J. (2014). Treatment with ziprasidone for schizophrenia patients with OCD. *European Neuropsychopharmacology, 24*(9), 1454–1462. https://doi.org/10.1016/j.euroneuro.2014.06.014
Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophrenia Bulletin, 13*(2), 261.
Kennedy, N. I., Lee, W. H., & Frangou, S. (2018). Efficacy of non-invasive brain stimulation on the symptom dimensions of schizophrenia: A meta-analysis of randomized controlled trials. *European Psychiatry, 49*, 69–77.
Khoury, B., Lecomte, T., Gaudiano, B. A., & Paquin, K. (2013). Mindfulness interventions for psychosis: A meta-analysis. *Schizophrenia Research, 150*(1), 176–184.
Kim, J., Iwata, Y., Plitman, E., Caravaggio, F., Chung, J. K., Shah, P., & Gerretsen, P. (2019). A meta-analysis of transcranial direct current stimulation for schizophrenia: "Is more better?". *Journal of Psychiatric Research, 110*, 117–126.
Kiran, C., & Chaudhury, C. (2018). Correlates and management of comorbid anxiety disorders in schizophrenia. *Industrial Psychiatry Journal, 27*(2), 271.
Kobori, O., Sato, H., Katsukura, R., & Harada, S. (2008). Cognitive behavioural therapy for obsessive compulsive symptoms affected by past psychotic experience of schizophrenia: A case report. *Behavioural and Cognitive Psychotherapy, 36*(3), 365–369. https://doi.org/10.1017/S1352465808004360
Koran, L. M., Hanna, G. L., Hollander, E., Nestadt, G., & Simpson, H. B. (2007). *Practice guideline for the treatment of patients with obsessive-compulsive disorder*.
Kozak, M. J., & Foa, E. B. (1994). Obsessions, overvalued ideas, and delusions in obsessive- compulsive disorder. *Behaviour Research and Therapy, 32*(3), 343–353.
Lysaker, P. H., & Whitney, K. A. (2009). Obsessive-compulsive symptoms in schizophrenia: Prevalence, correlates and treatment. *Expert Review of Neurotherapeutics*, 99–107.
Lysaker, P. H., Whitney, K. A., & Davis, L. W. (2006). Obsessive-compulsive and negative symptoms in schizophrenia: Associations with coping preference and hope. *Psychiatry Research*, 253–259.
Mawn, L., Campbell, T., Aynsworth, C., Beckwith, H., Luce, A., Barclay, N., … Freeston, M. H. (2020). Comorbidity of obsessive-compulsive and psychotic experiences: A systematic review and meta-analysis. *Journal of Obsessive-Compulsive and Related Disorders, 26*.
McGrath, J. J., Saha, S., Al-Hamzawi, A. O., Alonso, J., Andrade, L., Borges, G., & Kessler, R. C. (2016). Age of onset and lifetime projected risk of psychotic experiences: Cross-national data from the world mental health survey. *Schizophrenia Bulletin, 42*(4), 933–941.
Mendes-Filho, V. A., Belmonte-de-Abreu, P., Pedrini, M., Cachoeira, C. T., & Lobato, M. I. R. (2013). rTMS as an add-on treatment for resistant obsessive-compulsive symptoms in patients with schizophrenia: report of three cases. *Revista Brasileira de Psiquiatria, 35*(2), 210–211. https://doi.org/10.1590/1516-4446-2012-1035
Mendes-Filho, V. A., de Jesus, D. R., Belmonte-de-Abreu, P., Cachoeira, C. T., & Rodrigues Lobato, M. I. (2016). Effects of repetitive transcranial magnetic stimulation over supplementary motor area in patients with schizophrenia with obsessive-compulsive-symptoms: A pilot study. *Psychiatry Research, 242*, 34–38. https://doi.org/10.1016/j.psychres.2016.05.031
du Montcel, C. T., Pelissolo, A., Schürhoff, F., & Pignon, B. (2019). Obsessive-compulsive symptoms in schizophrenia: An up-to-date review of literature. *Current Psychiatry Reports, 21*(8), 1–8.
Moritz, S., Cludius, B., Hottenrott, B., Schneider, B. C., Saathoff, K., Kuelz, A. K., et al. (2015). Mindfulness and relaxation treatment reduce depressive symptoms in individuals with psychosis. *European Psychiatry : The Journal of the Association of European Psychiatrists, 30*(6), 709–714. https://doi.org/10.1016/j.eurpsy.2015.05.002
Moritz, S., Peters, M. J. V., Larøi, F., & Lincoln, T. M. (2010). Metacognitive beliefs in obsessive-compulsive patients: A comparison with healthy and schizophrenia participants. *Cognitive Neuropsychiatry, 15*(6), 531–548. https://doi.org/10.1080/13546801003783508
Naesström, M., Blomstedt, P., & Bodlund, O. (2016). A systematic review of psychiatric indications for deep brain stimulation, with focus on major depressive and obsessive-compulsive disorder. *Nordic Journal of Psychiatry, 70*(7). https://doi.org/10.3109/08039488.2016.1162846
Nagendra, A., Schooler, N. R., Kane, J. M., Robinson, D. G., Mueser, K. T., Estroff, S. E., … Penn, D. L. (2018). Demographic, psychosocial, clinical, and neurocognitive baseline characteristics of Black Americans in the RAISE-ETP study. *Schizophrenia Research, 193*, 64–68.

Nathan, P. E., & Gorman, J. M. (2002). Efficacy, effectiveness, and the clinical utility of psychotherapy research. In P. E. Nathan, & J. M. Gorman (Eds.), *A guide to treatments that work* (2nd ed., pp. 642–654). New York: Oxford University Press.
Odlaug, B. L., Weinhandl, E., Mancebo, M. C., Mortensen, E. L., Eisen, J. L., Rasmussen, S. A., & Grant, J. E. (2014). Excluding the typical patient: Thirty years of pharmacotherapy efficacy trials for obsessive-compulsive disorder. *Annals of Clinical Psychiatry: Official Journal of the American Academy of Clinical Psychiatrists, 26*(1), 39.
Olatunji, B. O., Davis, M. L., Powers, M. B., & Smits, J. A. (2013). Cognitive-behavioral therapy for obsessive-compulsive disorder: A meta-analysis of treatment outcome and moderators. *Journal of Psychiatric Research, 47*(1), 33–41.
Olatunji, B. O., Deacon, B. J., & Abramowitz, J. S. (2009). The cruelest cure? Ethical issues in the implementation of exposure-based treatments. *Cognitive and Behavioral Practice, 16*(2), 172–180.
Olbert, C. M., Nagendra, A., & Buck, B. (2018). Meta-analysis of Black vs. White racial disparity in schizophrenia diagnosis in the United States: Do structured assessments attenuate racial disparities? *Journal of Abnormal Psychology, 127*(1), 104.
Oluwoye, O., Stiles, B., Monroe-DeVita, M., Chwastiak, L., McClellan, J. M., Dyck, D., & McDonell, M. G. (2018). Racial-ethnic disparities in first-episode psychosis treatment outcomes from the RAISE-ETP study. *Psychiatric Services, 69*(11), 1138–1145.
van Oppen, P., Emmelkamp, P. M., van Balkom, A. J., & van Dyck, R. (1995). The sensitivity to change of measures for obsessive-compulsive disorder. *Journal of Anxiety Disorders, 9*(3), 241–248.
Ouzzani, M., Hammady, H., Fedorowicz, Z., & Elmagarmid, A. (2016). Rayyan-a web and mobile app for systematic reviews. *Systematic Reviews, 5*(1), 1–10. https://doi.org/10.1186/s13643-016-0384-4
Overall, J. E., & Gorham, D. R. (1962). The brief psychiatric rating scale. *Psychological Reports, 10*(3), 799–812.
Plewnia, C., Schober, F., Rilk, A., Buchkremer, G., Reimold, M., Wachter, T., et al. (2008). Sustained improvement of obsessive-compulsive disorder by deep brain stimulation in a woman with residual schizophrenia. *International Journal of Neuropsychopharmacology, 11*(8), 1181–1183. https://doi.org/10.1017/S1461145708009188
Porto, L., Bermanzohn, P. C., Pollack, S., Morrissey, R., & Siris, S. G. (1997). A profile of obsessive-compulsive symptoms in schizophrenia. *CNS Spectrums, 2*(3), 21–25.
Powell, A. L., Hinger, C., Marshall-Lee, E. D., Miller-Roberts, T., & Phillips, K. (2021). Implementing coordinated specialty care for first episode psychosis: A review of barriers and solutions. *Community Mental Health Journal, 57*, 268–276.
Poyurovsky, M., Faragian, S., Pashinian, A., Levi, A., Viosburd, A., Stryjer, R., & Weizman, A. (2007). Neurological soft signs in schizophrenia patients with obsessive-compulsive disorder. *Journal of Neuropsychiatry and Clinical Neurosciences, 19*(2), 145–150.
Poyurovsky, M., Glick, I., & Koran, L. (2010). Lamotrigine augmentation in schizophrenia and schizoaffective patients with obsessive-compulsive symptoms. *Journal of Psychopharmacology, 24*(6), 861–866. https://doi.org/10.1177/0269881108099215
Poyurovsky, M., Weizman, A., & Weizman, R. (2004). Obsessive-compulsive disorder in schizophrenia. *CNS Drugs, 18*(14), 989–1010.
Poyurovsky, M., Zohar, J., Glick, I., Koran, L. M., Weizman, R., Tandon, R., et al. (2012). Obsessive-compulsive symptoms in schizophrenia: Implications for future psychiatric classifications. *Comprehensive Psychiatry, 53*(5), 480–483.
Randhawa, R. S. (2005). A review of the pharmacotherapy of obsessive-compulsive disorder and schizophrenia: The case of Sam. *Cognitive and Behavioral Practice, 12*(4), 395–402.
Rapinesi, C., Kotzalidis, G. D., Ferracuti, S., Sani, G., Girardi, P., & Del Casale, A. (2019). Brain stimulation in obsessive-compulsive disorder (OCD): A systematic review. *Current Neuropharmacology, 17*(8), 787–807.
Rector, N. A., & Beck, A. T. (2012). Cognitive behavioral therapy for schizophrenia: An empirical review neil A. Rector, PhD and aaron T. Beck, MD (2001). Reprinted from the J nerv ment dis 189: 278–287. *The Journal of Nervous and Mental Disease, 200*(10), 832–839.
Rehn, S., Eslick, G. D., & Brakoulias, V. (2018). A meta-analysis of the effectiveness of different cortical targets used in repetitive transcranial magnetic stimulation (rTMS) for the treatment of obsessive-compulsive disorder (OCD). *Psychiatric Quarterly, 89*(3), 645–665.
Reid, A. M., Guzick, A. G., Fernandez, A. G., Deacon, B., McNamara, J. P., Geffken, G. R., … Striley, C. W. (2018). Exposure therapy for youth with anxiety: Utilization rates and predictors of implementation in a sample of practicing clinicians from across the United States. *Journal of Anxiety Disorders, 58*, 8–17.
Reznik, I., & Sirota, P. (2000). An open study of fluvoxamine augmentation of neuroleptics in schizophrenia with obsessive and compulsive symptoms. *Clinical Neuropharmacology, 23*(3), 157–160.
Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the national comorbidity survey replication. *Molecular Psychiatry, 15*(1), 53–63.
Schirmbeck, F., & Zink, M. (2012). Clozapine-induced obsessive-compulsive symptoms in schizophrenia: A critical review. *Current Neuropharmacology, 10*(1), 88–95.
Schirmbeck, F., & Zink, M. (2013). Obsessive-compulsive syndromes in schizophrenia: A case for polypharmacy? *Polypharmacy in Psychiatry Practice, II*, 233–261 (Springer, Dordrecht).
Schirmbeck, F., & Zink, M. (2013). Cognitive behavioural therapy for obsessive-compulsive symptoms in schizophrenia. *Cognitive Behaviour Therapist, 6*.
Schwartz, R. C., & Blankenship, D. M. (2014). Racial disparities in psychotic disorder diagnosis: A review of empirical literature. *World Journal of Psychiatry, 4*(4), 133.
Shavers, V. L., & Shavers, B. S. (2006). Racism and health inequity among Americans. *Journal of the National Medical Association, 98*(3), 386.
Soomro, G. M., Altman, D. G., Rajagopal, S., & Browne, M. O. (2008). Selective serotonin re- uptake inhibitors (SSRIs) versus placebo for obsessive compulsive disorder (OCD). *Cochrane Database of Systematic Reviews*, (1).
Spitzer, M. (1990). On defining delusions. *Comprehensive Psychiatry, 31*(5), 377–397.
Swets, M., Dekker, J., van Emmerik-van Oortmerssen, K., Smid, G. E., Smit, F., de Haan, L., et al. (2014). The obsessive compulsive spectrum in schizophrenia, a meta-analysis and meta-regression exploring prevalence rates. *Schizophrenia Research, 152*(2–3), 458–468.
Szymkowicz, S. M., McLaren, M. E., Suryadevara, U., & Woods, A. J. (2016). Transcranial direct current stimulation use in the treatment of neuropsychiatric disorders: A brief review. *Psychiatric Annals, 46*(11), 642–646.
Tundo, A., & Necci, R. (2016). Cognitive-behavioural therapy for obsessive-compulsive disorder co-occurring with psychosis: Systematic review of evidence. *World Journal of Psychiatry, 6*(4), 449.
Tundo, A., Salvati, L., Cieri, L., Daniele, M., Di Spigno, D., Necci, R., et al. (2014). Effectiveness and outcome predictors of cognitive-behavioural therapy for obsessive-compulsive disorder co-occurring with psychosis. *Journal of Psychopathology, 20*(2), 127–133.
Turner, D. T., van der Gaag, M., Karyotaki, E., & Cuijpers, P. (2014). Psychological interventions for psychosis: A meta-analysis of comparative outcome studies. *American Journal of Psychiatry, 171*(5), 523–538.
Twohig, M. P., Abramowitz, J. S., Smith, B. M., Fabricant, L. E., Jacoby, R. J., Morrison, K. L., & Ledermann, T. (2018). Adding acceptance and commitment therapy to exposure and response prevention for obsessive-compulsive disorder: A randomized controlled trial. *Behaviour Research and Therapy, 108*, 1–9.
Verma, R., Kumar, N., Mahapatra, A., & Shah, B. (2018). Effectiveness of tDCS augmentation for co-morbid obsessive compulsive disorder in chronic schizophrenia: A case report. *Asian journal of psychiatry, 38*, 9–11.
Veznedaroglu, B., Ercan, E. S., Kayahan, B., Varan, A., & Bayraktar, E. (2003). Reduced short- term obsessive–compulsive symptoms in schizophrenic patients treated with risperidone: A single-blind prospective study. *Human Psychopharmacology: Clinical and Experimental, 18*(8), 635–640.
Westphal, C. (1878). Uber die Verrucktheit. *Allgemeine Zeitschrft fur Psychiatrie, 34*, 252.
Wetterneck, C. T., Little, T. E., Rinehart, K. L., Cervantes, M. E., Hyde, E., & Williams, M. (2012). Latinos with obsessive-compulsive disorder: Mental healthcare utilization and inclusion in clinical trials. *Journal of obsessive-compulsive and related disorders, 1*(2), 85–97.
Williams, M. T., Beckmann-Mendez, D. A., & Turkheimer, E. (2013). Cultural barriers to African American participation in anxiety disorders research. *Journal of the National Medical Association, 105*(1), 33–41.
Williams, M., Powers, M., Yun, Y. G., & Foa, E. (2010). Minority participation in randomized controlled trials for obsessive–compulsive disorder. *Journal of Anxiety Disorders, 24*(2), 171.
Williams, M. T., Sawyer, B., Leonard, R. C., Ellsworth, M., Simms, J., & Riemann, B. C. (2015). Minority participation in a major residential and intensive outpatient program for obsessive-compulsive disorder. *Journal of Obsessive-Compulsive and Related Disorders, 5*, 67–75.
Williams, M. T., Taylor, R. J., Mouzon, D. M., Oshin, L. A., Himle, J. A., & Chatters, L. M. (2017). Discrimination and symptoms of obsessive–compulsive disorder among African Americans. *American Journal of Orthopsychiatry, 87*(6), 636.
Wu, K. D., & Wyman, S. V. (2016). Examination of racial differences in assessment of OCD symptoms and obsessive beliefs. *Journal of Obsessive-Compulsive and Related Disorders, 10*, 10–18.
Yaryura-Tobias, J., Neziroglu, F., & Bergman, L. (1976). Chlorimipramine, for obsessive-compulsive neurosis: An organic approach. *Current Therapeutic Research, 20*(4, Pt 2), 541–548.
Zohar, J., Kaplan, Z., & Benjamin, J. (1993). Clomipramine treatment of obsessive compulsive symptomatology in schizophrenic patients. *The Journal of Clinical Psychiatry, 54*(10), 385–388.