Supplementary Table S1 Data about articles included in the guidelines organized through the PICO system

Reference	Population	Instrument/intervention vs comparator	Study type	Primary efficacy outcome measure	Secondary outcomes	Level of evidence
Bandelow B et al.[1]	73 studies (n=11,066) assessing SAD	Pharmacological, psychological, and combined treatments vs placebo	Meta-analysis	Pre-post scales results to measure anxiety symptoms for psychotherapy and medications compared to placebo	Pre-post scale results used to measure SAD symptoms (i.e. HAMA, GAD, and LSAS) for psychotherapy and medications compared to placebo.	2 The following psychological therapies had a large effect size (i.e. the confidence intervals of two treatments did not overlap): individual and group exposure CBT, IPT, mindfulness, non-face-to-face therapies, and psychodynamics, The following drug therapies had a large effect size: moclobemide, phenelzine, pregabalin, venlafaxine, escitalopram, fluvoxamine, paroxetine, and sertraline. Good effect sizes found for mindfulness, exercise, and CBT + drug combinations (although the tested medications were not reported).
Baldwin DS et al.[2]	3 RCTs (n=1,596)	Escitalopram vs. placebo	Meta-analysis	Changes in the LSAS score at week 12 according to different doses of escitalopram	Estimated difference with placebo in CGI-S score at week 12 and treatment response	1 The overall difference in treatment effect favored all doses of escitalopram vs placebo.
Barkowski S et al.[3]	36 RCTs (n=2,171)	Group psychotherapy vs. waitlist	Meta-analysis	Improvement in specific symptoms	Improvement in general psychopathology	1 The studies mainly used group CBT, so quantitative analyses were performed for group CBT. Medium to large positive effects emerged for waitlist-controlled trials for specific symptomatology: g = 0.84, 95% CI [0.72; 0.97] and general psychopathology: g = 0.62, 95% CI [0.36; 0.89]. Group psychotherapy was also superior to common factor control conditions in alleviating SAD symptoms, but not in improving general psychopathology.
Bernik et al.[4]	n=146	Sertraline + psychotherapy vs. psychotherapy alone; group cognitive-	RCT	Remission rates assessed with the CGI-I and response rates assessed with the SADS and the	Final CGI-I, SADS, and M-MSSE score and other scales assessing	2 Overall, sertraline + psychotherapy was superior to psychotherapy alone. Sertraline potentiated group CBT by enhancing acquisition of social skills.

		behavioral therapy vs. psychodynamic therapy; sertraline + group cognitive-behavioral therapy or sertraline + group psychodynamic therapy vs. placebo + group cognitive-behavioral therapy or placebo + group psychodynamic therapy		Multidimensional Scale of Social Expression-Motor Part	depressive and anxiety symptoms	
Canton J e tal.[5]	41 studies on generalized social anxiety	Medications and psychotherapy vs. placebo	Meta-analysis	Symptom reduction, odds ratio	-	1
		Paroxetine vs. placebo		OR=3.43 (CI 2.51-4.69)		1 In general, SSRIs became distinct from placebo by weeks 4-6 in a number of responses or other outcome measures, but SSRI-placebo differences tended to increase until 12 weeks of treatment. The SSRIs had a robust effect on preventing relapse of social phobia (pooled OR 0.25, 95% confidence interval [CI] 0.18-0.35).
		Sertraline vs. placebo		OR=2.48 (CI 1.82-3.37)		1
		Escitalopram vs. placebo		OR=2.05 (CI 1.47-2.86)		1
		Fluvoxamine vs. placebo		OR=1.98 (1.07-3.67)		1
		Fluoxetine vs. placebo		OR=2.73 (CI 1.67-4.48)		1
		Venlafaxine vs. placebo		OR=2.42 (CI 1.92-3.06)		1 Response onset across trials was evident at 4-6 weeks, although maximum separation from placebo continued until 12 weeks.

Irreversible MAOIs vs. placebo	OR=7.22 (CI 2.90-17.97)	3 Monoamine oxidase inhibitors were used because social phobia and atypical depression share the symptom of increased interpersonal sensitivity, and atypical depression is preferentially responsive to monoamine oxidase inhibitors. All four studies with this drug showed a significantly greater treatment response than placebo; however the pooled OR was heavily influenced by the results of one study. Exclusion of this study in a sensitivity analysis reduced the pooled OR from 7.22 to 4.58. There have also been positive open-label studies with tranylcypromine.
RIMAs vs. placebo	OR=2.96 (95% CI 1.78-4.91)	2 High heterogeneity (I^2 = 69%) was noted in the analysis of this drug class. Excluding three studies on reversible selective inhibitors of monoamine oxidase A (two brofaromine, one moclobemide) reduced the heterogeneity to 0%, but also reduced the pooled OR from 2.96 to 1.88.
Gabapentin and pregabalin vs. placebo	OR=3.11 (95% CI 1.92-5.04)	1 Gabapentin and pregabalin are both ligands at the alpha-2 delta site on voltage-gated calcium channels. Functionally, both drugs reduce the release of a range of excitatory neurotransmitters through binding to this site. There are three positive RCTs of alpha-2 delta ligands. The onset of anxiolytic effects is relatively rapid, occurring within the first week of treatment. The anxiolytic dose-response has only been formally assessed for pregabalin, and efficacy is only evident at the maximum dose (600 mg/day), but not at lower doses. This is in contrast with the effect of pregabalin in, e.g., GAD, where the anxiolytic dose-response is seen at much lower doses (150 mg/day). There are no long-term treatment or relapse prevention data for alpha-2 delta ligands.
SSRIs and MAOIs vs. psychotherapy	OR=1.86 (95% CI 0.94-3.69) in favor of drugs	2 There were no significant differences in effectiveness between SSRIs and psychological treatments (CBT types). The meta-analysis of four monoamine oxidase

						inhibitor trials suggests that these drugs may be superior to psychological treatments, but this result was not statistically significant. It is also important to consider how these treatments compare over the longer term. Three studies have published follow-up data on outcomes after a treatment-free period. In all three trials, psychological treatment led to greater maintenance of treatment gains or protection against relapse relative to the drug treatments.
		Medication vs. combined medication psychological treatment trials		OR=0.30 (95% 0.09-0.97) in favor of combined therapy.		2 For response rates in the SSRI and monoamine oxidase inhibitor studies, there were nonsignificant trends in favor of combined medication-psychological treatments over medication alone. For the single benzodiazepine study, there was a statistically significant advantage in favor of combined treatment (Figure 7). It should be noted that all studies were relatively small in size and, thus, may not have been adequately powered. For the pooled response rate in the SSRI studies, there was a nonsignificant trend in favor of combined medication-psychological treatments over psychological treatment alone. For the pooled response rate in the monoamine oxidase inhibitor studies, there was a significant trend in favor of combined medication psychological treatments over psychological treatment alone. It should be noted that all studies were relatively small in size and thus may not have been adequately powered.
Caponnetto et al.[6]	Patients diagnosed with social anxiety disorder but no other mental illness.	Immersive Virtual Reality Therapy using a head-mounted display and a digitally recreated virtual environment. Comparison: “symptoms before VRET with immersive virtual reality technologies” and “post-treatment” symptoms.	Systematic Review.	Post-treatment symptoms, regardless of whether social skills were acquired or whether greater adaptation to the social environment occurred.	None	3 Virtual reality therapies proved to be a valid alternative for acquiring the necessary social skills to improve SAD symptoms. Although there was not a significant difference between VRET and iVET, the low cost and flexibility of VRET opens new possibilities for achieving greater psychophysical well-being.

Carl E et al.[7]	30 studies (n=1,057) 8 studies tested. VRET for SAD	VRET vs Placebo	Meta-analysis	The efficacy of VRET compared to psychological placebo or waitlist conditions and *in vivo* exposure	-	3 The pooled effect size for 7 studies comparing VRET to psychological placebo or waitlist conditions for SAD and performance anxiety showed a large effect size for VRET (g = 0.97, SE = 0.18, 95% CI: 0.62 to 1.31). The pooled effect size for 6 studies comparing VRET to iVET for SAD and performance anxiety a small nonsignificant effect in favor of iVET (g = 0.06, SE = 0.22, 95% CI: -0.36 to 0.49).
Carperter JK et al.[8]	Patients aged18- 65 years who and met DSM-III-R, DSM-IV, or DSM-5 diagnostic criteria for acute stress disorder, GAD, OCD, PTSD, SAD or specific phobia as determined by a psychometrically sound and structured diagnostic instrument.	CBT vs. placebo	Meta-analysis	Effect sizes and response rates	Sample and study characteristics, including sample size, demographics, placebo type, CBT treatment type (exposure, cognitive or both; group vs. individual), number of sessions, type of analysis (completer vs. intention-to-treat), and year of publication.	1 Small to moderate effect sizes were found for SAD. The treatment response OR was 3.51 for CBT compared to placebo.
Cuijpers P et al.[9]	Patients with anxiety disorder, including SAD	CBT vs. placebo and others	Meta-analysis	Effect size indicating the post-test difference between groups was calculated using Hedges' g	Other effects related to scores and homogeneity.	2 More research is needed to improve treatment effects, especially for GAD and SAD, because even if the effects of treatments are generally positive, a considerable number of patients still do not benefit. There was great heterogeneity between the studies.

Horigome et al.[10]	14 out of 22 studies assessing SAD. 9 included studies did not have group control.	VRET	Meta-analysis	Change in SAD measured mainly through change in LSAS score	RR of treatment discontinuation	2 The efficacy of VRET for SAD was significant and continued over long-term follow-up: Hedges' g for effect size at post-intervention, -0.86 (-1.04 to -0.68); 3 months post-intervention, -1.03 (-1.35 to -0.72); 6 months post-intervention, -1.14 (-1.39 to -0.89); and 12 months post-intervention, -0.74 (-1.05 to -0.43). Compared to iVET, the efficacy of VRET was similar at post-intervention but became inferior at later follow-up points. There was no significant difference in participant dropout rates between VRET and iVET.
Kampman et al.[11]	37 studies (n=2,991)	Technology-assisted interventions (ICBT, VRET, CBM) vs. placebo	Meta-analysis	Self-reported symptoms during post-assessment and follow up	Depressive symptoms	2 Patients undergoing ICBT and VRET showed significantly lower SAD symptoms at post assessment than passive control conditions.
Li et al.[12]	13 RCTs (n=2,585)	Paroxetine vs. placebo	SR	Changes in the LSAS total score and related subscales, CGI-S score, SADS score, and Sheehan Disability Scale for work, social, and family items.	Tolerability	1 Mean changes in total LSAS scores and subscale scores were all significantly greater in the active group. Response and remission rates were both significantly higher in the active group. Dropouts due to AEs were higher than placebo.
Liu et al.[13]	11 eligible RCTs and 5 single-arm trials	Mindfulness alone and associated vs other interventions	Meta-analysis	The primary outcome was the severity of clinical SAD symptoms	Secondary outcomes were depressive symptoms, mindfulness, quality of life and self-compassion.	2 In the between-group analysis of the 11 RCTs, Hedges' g = 0.00, while the within-group analysis showed a large pre-post effect size (g =1.20). MBIs were superior to the no-treatment comparator (g =0.89), equivalent to specific active treatment (g = -0.19), and less effective than evidence-based treatment (i.e., cognitive behavioral therapies) (g= -0.29). MBIs significantly alleviated depressive symptoms and improved mindfulness, quality of life, and self-compassion. Meta-regression analysis showed a dose-response relationship between the

						alleviation of SAD symptoms and MBI duration (β =0.659). Follow-up analysis showed that the effects of MBIs on SAD persisted for 12 months (g =0.231). An analysis of the 5 single-arm trials found that MBIs had a medium effect on alleviating SAD symptoms (g =0.48).
Liu H et al.[14]	34 studies (n=2,550)	CBM vs. placebo	Meta-analysis	Evaluated the degree to which certain variants of CBM improve SAD symptoms and cognitive behaviors.	Potential moderators of the effectiveness of CBM on SAD.	2 CBM had small but significant effects of on the primary symptoms of SAD, cognitive bias toward threat, and reactivity in stressful situations, but non-significant effects on secondary symptoms.
Liu X et al.[15]	5 RCTs (n=1,045)	Fluvoxamine vs. placebo	Meta-analysis	Mean change in LSAS and CGI-S; the tolerability outcome was discontinuation rate due to AEs	Response rate according to the CGI-I scale and tolerability	1 Mean changes in total LSAS and CGI-S scores were significantly greater in the active group. The response rate was higher in the fluvoxamine group. The discontinuation rate due to AEs was higher in patients who received fluvoxamine.
Mayo-Wilson et al.[16]	101 RCTs (n=13,164)	Psychological placebo and pharmacological vs. placebo/waitlist	Systematic Review and Meta-analysis	Assessed the severity of SAD symptoms, mainly through LSAS score.	-	1 Greater effects on outcomes were found for MAOIs, benzodiazepines, SSRIs, SNRIs, and anticonvulsants. Effects for psychological interventions were found for individual CBT, group CBT, exposure and social skills therapy, self-help with support, self-help without support and psychodynamic psychotherapy.
	6 studies (n=490)	Maclobemide vs. waitlist		-0·74 [-1·03 to -0·44]		1
	5 studies (n=125)	Phenelzine vs. waitlist		-1·28 [-1·57 to -0·98]		1

1 study (n=12)	Alprazolam vs. waitlist	-0·85 [-1·40 to -0·30]	2 The largest effects were for MAOIs (class effect SMD -1·01, 95% CrI -1·56 to -0·45) and benzodiazepines (-0·96, -1·56 to -0·36), but the evidence for these effects was limited compared to the evidence for SSRIs and SNRIs (-0·91, -1·23 to -0·60)
4 studies (n=100)	Clonazepam vs. waitlist	-1·07 [-1·44 to -0·70]	2 The largest effects were for MAOIs (class effect SMD -1·01, 95% CrI -1·56 to -0·45) and benzodiazepines (-0·96, -1·56 to -0·36), but the evidence for these effects was limited compared with evidence for SSRIs and SNRIs (-0·91, -1·23 to -0·60)
1 study (n=34)	Gabapentin vs. waitlist	-0·89 [-1·42 to -0·37]	Inconclusive
1 study (n=9)	Levetiracetam vs. waitlist	-0·83 [-1·50 to -0·18]	Inconclusive
3 studies (n=199)	Pregabalin vs. waitlist	-0·72 [-1·07 to -0·37]	Inconclusive
2 studies (n=18)	Citalopram vs. waitlist	-0·83 [-1·28 to -0·39]	1
2 studies (n=675)	Escitalopram vs. waitlist	-0·88 [-1·20 to -0·56]	1
3 studies (n=107)	Fluoxetine vs. waitlist	-0·87 [-1·16 to -0·57]	1
5 studies (n=500)	Fluvoxamine vs. waitlist	-0·94 [-1·25 to -0·63]	1
12 studies (n=1,449)	Paroxetine vs. waitlist	-0.99 [-1.26;-0.73]	1
3 studies (n=535)	Sertraline vs. waitlist	-0·92 [-1·23 to -0·61]	1
5 studies (n=759)	Venlafaxine vs. waitlist	-0·96 [-1·25 to -0·67]	1
1 study (n=30)	Mirtazapine vs. waitlist	-0·81 [-1·45 to -0·16]	1 Mirtazapine, a noradrenergic and specific serotonergic antidepressant, was the only pharmacological

						intervention in a class by itself; its effect was not greater than waitlist (class effect SMD -0·80, 95% CrI -1·64 to 0·01), but only 30 people received the intervention.
Nordahl et al.[17]	n=102	Paroxetine vs. cognitive therapy vs. paroxetine + cognitive therapy and placebo	RCT	Symptoms measured with the Fear of Negative Evaluation Questionnaire	Anxiety symptoms assessed with the LSAS and BAI	2 Recovery rates were higher in the cognitive therapy group than the Sgroup. Cognitive therapy was also superior to paroxetine alone and pill placebo in 12-months of follow up.
Williams T et al.[18]	67 RCTs	Pharmacotherapy vs. placebo and head to head comparisons	Meta-analysis	Efficacy and acceptability measured with the LSAS and dropout rate	Response and tolerability measured by CGI-I and dropouts due to AEs	2 There was a response to paroxetine, brofaromine, bromazepam, clonazepam, escitalopram, fluvoxamine phenelzine, and sertraline. Olanzapine had the greatest result for treatment response efficacy, while buspirone had the worst. Brofaromine, escitalopram, fluvoxamine, paroxetine, pregabalin, sertraline, and venlafaxine performed worse than placebo regarding dropouts due to AEs.
		Olanzapine		MD -37.8 (-87.24;11.64)		Inconclusive.
		Bromazepam		MD -31.60 (-66.64;3.44)		2 Superior response to treatment was also observed for paroxetine, brofaromine, bromazepam, clonazepam, escitalopram, fluvoxamine, phenelzine, and sertraline.
		Clonazepam		MD -23.70 (-58.87;11.67)		2 Superior response to treatment was also observed for paroxetine, brofaromine, bromazepam, clonazepam, escitalopram, fluvoxamine, phenelzine, and sertraline.
		Sertraline		MD -17.45 (-43.76;8.88)		2 Superior response to treatment was also observed for paroxetine, brofaromine, bromazepam, clonazepam, escitalopram, fluvoxamine, phenelzine, and sertraline.
		Paroxetine		MD -15.89 (-29.94;-1.84)		2 Superior response to treatment was also observed for paroxetine, brofaromine, bromazepam, clonazepam,

						escitalopram, fluvoxamine, phenelzine, and sertraline.
				Mirtazapine	MD -14.53 (-38.87;9.82)	Inconclusive.
				Gabapentin	MD -11.50 (-47.62; 24.62)	Inconclusive.
				Phenelzine	MD -8.65 (-28.65; 11.38)	2 Superior response to treatment was also observed for paroxetine, brofaromine, bromazepam, clonazepam, escitalopram, fluvoxamine, phenelzine, and sertraline.
				Moclobemide	MD -8.51 (-25.87; 8.86)	Inconclusive.
				Brofaromine	MD - 8.10 (-43.29; 27.09)	2 Superior response to treatment was also observed for paroxetine, brofaromine, bromazepam, clonazepam, escitalopram, fluvoxamine, phenelzine, and sertraline.
				Escitalopram	MD -8.05 (-41.81;25.71)	2 Superior response to treatment was also observed for paroxetine, brofaromine, bromazepam, clonazepam, escitalopram, fluvoxamine, phenelzine, and sertraline.
				Levetiracetam	MD -3.82 (-31.80;24.15)	Nonsignificant.
				Fluvoxamine	MD -2.12 (-21.88;17.64)	2 Superior response to treatment was also observed for paroxetine, brofaromine, bromazepam, clonazepam, escitalopram, fluvoxamine, phenelzine, and sertraline.
				Atomoxetine	MD 2.60 (-35.38; 40.58)	Nonsignificant.
				Vilazodone	MD 15.60 (-22.05;53.25)	Nonsignificant.
				Venlafaxine	MD 30.47 (7.76;53.18)	Inconclusive.
Williams T et al.[19]	66 RCTs (n=11,597)	Pharmacotherapy vs. placebo	Systematic Review	Reduction in SAD symptoms according to the LSAS, Reduced depressive symptoms, functional disability, and dropout rates	Regarding the secondary outcome of SAD symptom severity, SSRIs, the SNRI venlafaxine, MAOIs, RIMAs, benzodiazepines,	1 Very low-quality evidence of treatment response for SSRIs. There was also evidence of benefit from MAOIs, RIMAs, benzodiazepines, pregabalin, and gabapentin. SSRIs were the only medication to prove effective in reducing relapse based on moderate-quality evidence.

			the antipsychotic olanzapine, and the noradrenergic and specific serotonergic antidepressant atomoxetine were associated with reduced SAD symptoms, but most of the evidence was of very low quality.	
1 study (n=30; 15 active/15 placebo)	5HT1A partial agonists (Buspirone) vs. placebo	1.00 [0.07 to 14.55]		Nonsignificant
3 studies (n=532; 369 active/163 placebo)	Anticonvulsant GABAs (gabapentin and pregabalin) vs. placebo	1.60 [1.16 to 2.20]		Nonsignificant
2 studies (n=228; 118 active/110 placebo)	Levetiracetam vs. placebo	0.98 [0.70 to 1.37]		Nonsignificant
2 studies (n=132; 67 active/65 placebo)	Benzodiazepines vs. placebo	4.03 [2.45 to 6.65]		Nonsignificant
2 trials	Antipsychotics (olanzapine and quetiapine) Quetiapine	1.07 [0.27 to 4.23]		Nonsignificant
2 studies (n=97; 49 active/ 48 placebo)	Beta-blockers vs. placebo	1.09 [0.63 to 1.88]		Nonsignificant
4 studies	MAOI (phenelzine) vs.	2.36 [1.48, 3.75]		3

	(n=235; 121 active/114 placebo)	placebo				Very low-quality evidence for MAOIs
	1 study (n=60; (30 active/30 control)	Mirtazapine vs. placebo		1.00 [0.28, 3.63]		Nonsignificant
	8 studies (n=1,270; 632 active/638 placebo)	RIMAs vs. Placebo		1.83 [1.32 to 2.55]		3 There was evidence that this medication was efficacious compared to placebo (k = 5, RR 1.32; 95% CI 1.14 to 1.52, N = 1063; P < 0.001). Furthermore, there was moderate-quality evidence that moclobemide had a long-term effect on treatment efficacy in participants with SAD compared to placebo (k = 1, RR 1.50; 95% CI 1.12 to 2.00, N = 90. Comparisons between medication classes revealed that the response to RIMAs was smaller than that of benzodiazepines (x^2 = 6.63, df = 1, P = 0.01; I^2 = 84.9%).
	4 studies (n=1,173; 630 active/543 placebo)	Venlafaxine vs. placebo		1.30 [0.85 to 1.99]		2 Treatment withdrawal was higher for venlafaxine than placebo (SSRIs: k = 24, RR 2.59; 95% CI 1.97 to 3.39, N = 5131, low-quality evidence; venlafaxine: k = 4, RR 3.23; 95% CI 2.15 to 4.86, N = 1213, moderate-quality evidence), but there were low absolute rates of withdrawal for both medications classes compared to placebo.
	24 studies (n=4,984; 2,767 active/2,217 placebo)	SSRIs vs. Placebo		1.65 [1.48 to 1.85]		1 SSRIs were the only medication that proved effective in reducing relapse based on moderate-quality evidence.
Yang L et al.[20]	17 RCTs (n=1,134)	Psychological interventions for children and adolescents	Meta-analysis	Change in anxiety symptoms measured by anxiety rating scales and acceptability (all-cause dropout)	Remission, quality of life/functional improvement and depressive symptoms	1 Psychological interventions were more effective than control (SMD -1.13; RR 8.99 and NNT=3.3). Psychological interventions were also superior to control in improving quality of life and reducing depressive symptoms.

AEs = adverse events; CBM = cognitive bias modification; CBT = cognitive behavioral therapy; CGI = Clinical Global Impression; CGI-I = Clinical Global Impression- improvement index; CGI-S = Clinical Global Impression- severity Index; GABA = gamma-aminobutyric acid; GAD = generalized anxiety disorder; HAMA = Hamilton Anxiety Scale; ICBT = Internet-delivered cognitive behavioral therapy; IPT = interpersonal therapy; iVET = *in vivo* exposure therapy; LSAS = Liebowitz Social Anxiety Scale; MAOI = monoamine oxidase inhibitor; MBI = mindfulness-based intervention; MD = mean difference; M-MSSE = Multidimensional Scale of Social Expression; OR = odds ratio; RIMA = selective inhibitors of monoamine oxidase A; RR = relative risk; SAD = social anxiety disorder; SADS = Social Avoidance and Distress Scale; SMD = standardized mean difference = SNRI = serotonin and noradrenaline reuptake inhibitor; SSRI = selective serotonin reuptake inhibitor; VRET = virtual reality exposure therapy.

http://doi.org/10.47626/1516-4446-2024-3878
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Supplementary Table S2 Risk of bias of systematic reviews with or without meta-analyses for pharmacological therapies

Study	Study eligibility criteria	Identification and selection of studies	Data collection and study appraisal	Synthesis and findings	Risk of bias in the review
Bandelow B et al. 2015	Low	Low	Low	Low	Low
Baldwin DS, et al. 2016	Low	Low	Low	Low	Low
Barkowski S et al., 2016	Low	Low	Low	Low	Low
Canton J et al., 2012	Low	Low	Low	Low	Low
Caponnetto et al. 2021	High	High	High	High	High
Carl E et al., 2018	High	High	High	High	High
Carperter JK et al., 2018	Low	Low	Low	Low	Low
Cuijpers P et al., 2016	Low	Low	Unclear	High	Unclear
Horigome et al., 2020	Low	Low	Low	Low	Low
Jakubovski et al. 2019	High	High	High	High	High
Kampmann IL et al 2016	Low	Low	Low	Low	Low
Li et al. 2020	Low	Low	Low	Low	Low
Liu X et al. 2021	Low	Low	Low	Low	Low

http://doi.org/10.47626/1516-4446-2024-3878
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Liu H et al. 2017	Low	Low	Low	Low	Low
Liu X et al. 2018	Low	Low	Low	Low	Low
Mayo-Wilson et al. 2014	Low	Low	Low	Low	Low
Nordahl et al. 2016	Low	Low	Low	Low	Low
Van Dis et al. 2019	Low	Low	Low	Low	Low
Scaini et al. 2016	Low	Low	Low	Low	Low
Williams et al. 2017	Low	Low	Low	Low	Low
Williams et al. 2020	Low	Low	Low	Low	Low
Yang L et al., 2019	Low	Low	Low	Low	Low

Supplementary Table S3 Risk of bias of randomized clinical trials on pharmacological therapies

Authors	Randomization process	Risk of bias arising from the timing of identification or recruitment of participants	Deviation from intended interventions	Missing outcome data	Measurement of the outcome	Selection of the reported results	Overall bias
Bernik et al., 2018	Low	Low		Low	Low	Low	Low
Nordahl et al., 2016	Low	Unclear	Low	High	Low	Low	Unclear

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