#### Appendix 18: Clinical evidence statements

Psychological interventions evidence statements

Psychological vs. control (OCD)
Psychological vs. Psychological
Psychological vs. control (BDD)
Psychological vs. Psychological (BDD)

Pharmacological interventions evidence statements

SSRIs (adults)
Clomipramine (adults)
Tricyclic antidepressants
SNRIs
Monoamino-oxidase inhibitors
Anxiolytics
Other Pharmacological interventions
Augmentation strategies
SSRIs (children and adolescents)
Clomipramine (children and adolescents)
Pharmacological interventions (BDD)

Psychological vs. pharmacological interventions evidence statements

Combination therapy evidence statements

Other medical interventions evidence statements

## Psychological interventions evidence statements

<u>Psychological vs. control (OCD); Psychological vs. Psychological (OCD); Psychological vs. control (BDD); Psychological vs. Psychological (BDD)</u>

### Psychological vs. control (OCD)

Description	Statement level <sup>1</sup>	Statement
01 Behaviour therapy v Control		
01 Leaving the study early		
02 Clinician-guided BT vs. Systematic relaxation	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clinician-guided BT and systematic relaxation on the likelihood of leaving the study early ( $N = 1$ ; $n = 125$ ; $RR = 4.47$ ; 95% CI, 0.51 to 38.92). I
03 Computer-guided BT vs. Systematic relaxation	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between computer-guided BT and systematic relaxation on the likelihood of leaving the study early ( $N = 1$ ; $n = 123$ ; $RR = 2.32$ ; $95\%$ CI, $0.22$ to $24.88$ ). I
02 Non-responders (CGI)		
01 Clinician-guided BT vs. Systematic relaxation	s1x	There is evidence suggesting a difference favouring clinician-guided BT over systematic relaxation on the likelihood of response, defined as 'much improved' or 'very much improved' on the CGI (N = 1; n = $125$ ; RR = $0.51$ ; $95\%$ CI, $0.38$ to $0.69$ ). I
02 Computer-guided BT vs. Systematic relaxation	s2x	There is limited evidence suggesting a difference favouring computer-guided BT over systematic relaxation on the likelihood of response, defined as 'much improved' or 'very much improved' on the CGI ( $N = 1$ ; $n = 123$ ; $RR = 0.73$ ; 95% CI, 0.59 to 0.91). I
03 Y-BOCS	1	
01 ERP vs. Anxiety management	s1x	There is limited evidence suggesting a difference favouring ERP over anxiety management on reducing obsessive-compulsive symptoms as measured on the Y-BOCS ( $N = 1$ ; $n = 18$ ; SMD = -2.89; 95% CI, -4.3 to -1.48). I
03 Clinician-guided BT vs. Systematic relaxation	s1x	There is evidence suggesting a difference favouring clinician-guided BT over systematic relaxation on reducing obsessive-compulsive symptoms as measured on the Y-BOCS (N = 1; n = 121; SMD = -1.1; 95% CI, -1.49 to -0.72). I
04 Computer-guided BT vs. Systematic relaxation	s2x	There is limited evidence suggesting a difference favouring computer-guided BT over systematic relaxation on reducing obsessive-compulsive symptoms as measured on the Y-BOCS (N = 1; n = 121; SMD = -0.68; 95% CI, -1.05 to -0.31). I
04 Padua Inventory	s2x	There is limited evidence suggesting a difference favouring BT over control on reducing obsessive-compulsive symptoms as measured on the Padua Inventory ( $N = 2$ ; $n = 53$ ; $SMD = -0.63$ ; $95\%$ CI, $-1.19$ to $-0.07$ ). I
05 Depression: BDI or HAM-D		

<sup>&</sup>lt;sup>1</sup> See Chapter 2, Flowchart 2: Guideline Statement Decision Tree, for decisions on the clinical importance of the effect size

01 ERP vs. Anxiety management		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP and anxiety management on reducing depressive symptoms as measured on the BDI or HAMD ( $N = 1$ ; $n = 18$ ; $SMD = -0.55$ ; $95\%$ CI, $-1.5$ to $0.39$ ). I
02 ERP vs. Wait-list	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP and wait-list on reducing depressive symptoms as measured on the BDI or HAMD ( $N = 1$ ; $n = 35$ ; $SMD = -0.26$ ; $95\%$ CI, $-0.93$ to $0.41$ ). I
03 Clinician-guided BT vs. Systematic relaxation		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clinician-guided BT and systematic relaxation on reducing depressive symptoms as measured on the BDI or HAMD (N = 1; n = 121; SMD = $-0.28$ ; 95% CI, $-0.64$ to $0.08$ ). I
04 Computer-guided BT vs. Systematic relaxation		There is evidence suggesting there is unlikely to be a clinically important difference between computer-guided BT and systematic relaxation on reducing depressive symptoms as measured on the BDI or HAMD ( $N = 1$ ; $n = 121$ ; $SMD = -0.04$ ; $95\%$ CI, $-0.4$ to $0.31$ ). I
06 Maudsley Obsessive-Compuls	ive Inventor	у
01 ERP vs. Anxiety management		There is limited evidence suggesting a difference favouring ERP over anxiety management on reducing obsessive-compulsive symptoms as measured on the MOCI ( $N = 1$ ; $n = 18$ ; SMD = -1.8; 95% CI, -2.94 to -0.66). I
07 STAI: Trait		
01 ERP vs. Anxiety management		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP and anxiety management on reducing trait anxiety as measured on the STAI trait scale ( $N = 1$ ; $n = 18$ ; $SMD = -0.52$ ; $95\%$ CI, $-1.46$ to $0.42$ ). I
08 STAI: State		
01 ERP vs. Anxiety management		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP and anxiety management on reducing state anxiety as measured on the STAI state scale ( $N = 1$ ; $n = 18$ ; $SMD = -0.64$ ; $95\%$ CI, $-1.59$ to $0.32$ ). I
12 Work and Social Adjustment S	Scale	
01 Clinician-guided BT vs. Systematic relaxation	s2x	There is limited evidence suggesting a difference favouring clinician-guided BT over systematic relaxation on improving work and social adjustment as measured on the WSAS ( $N = 1$ ; $n = 121$ ; $SMD = -0.6$ ; 95% CI, $-0.96$ to $-0.23$ ). I
02 Computer-guided BT vs. Systematic relaxation	s2x	There is limited evidence suggesting a difference favouring computer-guided BT over systematic relaxation on improving work and social adjustment as measured on the WSAS ( $N = 1$ ; $n = 121$ ; SMD = -0.4; 95% CI, -0.76 to -0.04). I
13 Interference	<u> </u>	, , , , , , , , , , , , , , , , , , , ,
01 ERP vs. Anxiety Management		There is limited evidence suggesting a difference favouring ERP over anxiety management on reducing the impact of OCD on life and activities as measured on an interference rating scale ( $N = 1$ ; $n = 18$ ; SMD = -3.16; 95% CI, -4.64 to -1.67). I
03 Cognitive-Behavioural Therap	y v Control	,
01 Y-BOCS		
·		

01 CBT v Wait-list control	s2x	Thous is limited avidence suggesting a difference forcewing
(patients with obsessions only)	SZX	There is limited evidence suggesting a difference favouring cognitive-behavioural therapy over wait-list on reducing obsessive-compulsive symptoms as measured on the Y-BOCS in patients with
		obsessions only (N = 1; n = 29; SMD = -1.18; 95% CI, -1.98 to -0.38). I
03 CBT vs. Wait-list control: Group format	s1x	There is evidence suggesting a difference favouring cognitive-behavioural group therapy over wait-list on reducing obsessive-compulsive symptoms as measured on the Y-BOCS (N = 1; n = 47; SMD = $-1.18$ ; 95% CI, $-1.81$ to $-0.56$ ). I
02 Padua Inventory		
01 CBT vs. Wait-list control	s2x	There is limited evidence suggesting a difference favouring CBT over wait-list on reducing obsessive-compulsive symptoms as measured on the Padua Inventory (N = 1; n = 29; SMD = -0.83; 95% CI, -1.59 to -0.07). I
03 Current Functioning Assessm	nent	
01 CBT vs. Wait-list control	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and wait-list on reducing interference in life activities as measured on the CFA (N = $1$ ; n = $29$ ; SMD = $-0.46$ ; $95\%$ CI, $-1.2$ to $0.28$ ). I
04 Beck Anxiety Inventory		
01 CBT vs. Wait-list control	s2x	There is limited evidence suggesting a difference favouring CBT over wait-list on reducing anxiety symptoms as measured on the BAI ( $N = 1$ ; $n = 29$ ; SMD = -0.87; 95% CI, -1.64 to -0.1). I
05 Beck Depression Inventory		
01 CBT vs. Wait-list control	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and wait-list on reducing depressive symptoms as measured on the BDI ( $N = 1$ ; $n = 29$ ; SMD = 0.06; 95% CI, -0.67 to 0.79). I
06 NIMH-OC	s2x	There is limited evidence suggesting a difference favouring CBT over wait-list on reducing obsessive-compulsive symptoms as measured on the NIMH-OC (N = 1; n = $47$ ; SMD = $-1.24$ ; 95% CI, $-1.86$ to $-0.61$ ). I
10 Overvalued Ideas Scale		
01 CBGT vs. Wait-list control	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBGT and wait-list on reducing obsessive-compulsive symptoms as measured on the OVIS (N = 1; n = $47$ ; SMD = $-0.09$ ; 95% CI, $-0.66$ to $0.48$ ). I
11 WHOQOL-BREF: physical		
01 CBGT vs. Wait-list control	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBGT and wait-list on improving physical quality of life as measured on the WHOQOL-BREF physical scale (N = 1; n = 47; SMD = -0.57; 95% CI, -1.16 to 0.01). I
12 WHOQOL-BREF: psycholog	ical	
01 CBGT vs. Wait-list control	s2x	There is limited evidence suggesting a difference favouring CBGT over wait-list on improving psychological quality of life as measured on the WHOQOL-BREF psychological scale (N = 1; n = 47; SMD = -0.59; 95% CI, -1.18 to -0.01). I
13 WHOQOL-BREF: social		
01 CBGT vs. Wait-list control	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBGT and wait-list on improving social quality of life as measured on the WHOQOL-BREF social scale ( $N = 1$ ; $n = 47$ ; $SMD = -0.2$ ; $95\%$ CI, $-0.78$ to $0.37$ ). I

14 WHOQOL-BREF: environmer	ntal	
01 CBGT vs. Wait-list control	s2x	There is limited evidence suggesting a difference favouring CBGT over wait-list on improving environmental quality of life as measured on the WHOQOL-BREF environmental scale (N = 1; n = 47; SMD = -1.05; 95% CI, -1.66 to -0.44). I
15 Non-responders (35% Y-BOCS	5)	
01 CBGT vs. Wait-list control	s2x	There is limited evidence suggesting a difference favouring CBGT over wait-list on the likelihood of response, defined as a 35% or more reduction on the Y-BOCS (N = 1; n = 47; RR = $0.32$ ; 95% CI, $0.17$ to $0.59$ ). I
16 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between cognitive-behavioural therapy and wait-list on the likelihood of leaving the study early ( $N = 2$ ; $n = 76$ ; $RR = 0.77$ ; 95% CI, 0.24 to 2.49). I
10 Children: Individual CBFT vs.	Wait-list co	ontrol
01 CY-BOCS	s2x	There is limited evidence suggesting a difference favouring individual CBFT over wait-list on reducing obsessive-compulsive symptoms as measured on the CY-BOCS (N = 1; n = 46; SMD = -2.73; 95% CI, -3.55 to -1.91). I
02 Delete in favour of CY-BOCS	s2x	There is limited evidence suggesting a difference favouring individual CBFT over wait-list on reducing obsessive-compulsive symptoms as measured on the NIMH-GOCS (N = 1; n = 48; SMD = -2.51; 95% CI, -3.28 to -1.74). I
03 Multidimensional Anxiety Scale for Children	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between individual CBFT and wait-list on reducing anxiety symptoms as measured on the MASC ( $N = 1$ ; $n = 34$ ; SMD = 0.06; 95% CI, -0.62 to 0.73). I
04 Children's Depression Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between individual CBFT and wait-list on reducing depressive symptoms as measured on the CDI ( $N = 1$ ; $n = 34$ ; $SMD = -0.26$ ; $95\%$ CI, $-0.94$ to $0.42$ ). I
05 McMaster Family Assessment Device - Mother's rating	s2x	There is limited evidence suggesting a difference favouring individual CBFT over wait-list on improving family functioning as measured on the MFAD mother's rating scale (N = 1; n = 32; SMD = -0.93; 95% CI, -1.67 to -0.19). I
06 McMaster Family Assessment Device – Father's rating	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between individual CBFT and wait-list on improving family functioning as measured on the MFAD father's rating scale ( $N = 1$ ; $n = 19$ ; SMD = -0.66; 95% CI, -1.59 to 0.27). I
11 Children: Group CBFT vs. Wa	it-list contro	ol .
01 CY-BOCS	s2x	There is limited evidence suggesting a difference favouring group CBFT over wait-list on reducing obsessive-compulsive symptoms as measured on the CY-BOCS (N = 1; n = 53; SMD = -2.54; 95% CI, -3.28 to -1.81). I
Delete in favour of CY-BOCS	s2x	There is limited evidence suggesting a difference favouring group CBFT over wait-list on reducing obsessive-compulsive symptoms as measured on the NIMH-GOCS (N = 1; n = 53; SMD = $-2.68$ ; 95% CI, $-3.44$ to $-1.93$ ). I
03 Multidimensional Anxiety Scale for Children	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between group CBFT and wait-list on reducing anxiety symptoms as measured on the MASC $(N = 1; n = 38; SMD = -0.59; 95\% CI, -1.26 to 0.07)$ . I

04 Children's Depression	s2x	There is limited evidence suggesting a difference favouring group
Inventory		CBFT over wait-list on reducing depressive symptoms as measured
		on the CDI (N = 1; n = 38; SMD = -0.78; 95% CI, -1.46 to -0.11). I
05 McMaster Family Assessment		There is limited evidence suggesting a difference favouring group
Device – Mother's rating		CBFT over wait-list on improving family functioning as measured
		on the MFAD mother's rating scale (N = 1; n = 40; SMD = -0.78; 95% $\mid$
		CI, -1.45 to -0.11). I
06 McMaster Family Assessment		The evidence is inconclusive and so it is not possible to determine if
Device – Father's rating		there is a clinically important difference between group CBFT and
		wait-list on improving family functioning as measured on the
		MFAD father's rating scale (N = 1; n = 23; SMD = -0.52; 95% CI, -1.36
		to 0.32). I

# Psychological vs. Psychological

Description	Statement level	Evidence statement
01 Behaviour therapy v Cognitiv	e therapy	
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on the likelihood of leaving the study early at the end of treatment ( $N = 4$ ; $n = 305$ ; $RR = 0.97$ ; 95% CI, 0.63 to 1.47). I
02 Not recovered (Y-BOCS) post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on the likelihood of recovering, defined as a reliable change on the Y-BOCS and a Y-BOCS score less than 13 ( $N = 2$ ; $n = 164$ ; $RR = 0.91$ ; 95% CI, 0.76 to 1.1). I
03 Not recovered (Y-BOCS) at 12 weeks follow-up	s2x	There is limited evidence suggesting a difference favouring group behaviour therapy over group cognitive therapy on the likelihood of recovering at 12 months follow-up, defined as a reliable change on the Y-BOCS and a Y-BOCS score less than 13 (N = 1; n = 93; RR = $0.74$ ; 95% CI, $0.6$ to $0.92$ ). I
04 Not reliable change (Y-BOCS) post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on the likelihood of a reliable change in obsessive-compulsive symptoms, defined as a Y-BOCS change of greater than 5 points ( $N = 2$ ; $n = 106$ ; $RR = 1.12$ ; 95% CI, 0.71 to 1.76). I
05 Non-responder (Y-BOCS 25%)		
01 Post-treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on the likelihood of response at the end of treatment, defined as a 25% or greater reduction on the Y-BOCS (N = 1; $n = 65$ ; $RR = 1.29$ ; 95% CI, 0.63 to 2.64). I
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on the likelihood of response at 26 weeks follow-up, defined as 25% or greater reduction on the Y-BOCS ( $N = 1$ ; $n = 65$ ; $RR = 1.26$ ; 95% CI, 0.65 to 2.45). I

03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on the likelihood of response at 52 weeks
		follow-up, defined as 25% or greater reduction on the Y-BOCS (N = 1; n = 65; RR = 0.97; 95% CI, 0.53 to 1.76). I
08 Y-BOCS post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms at the end of treatment as measured by the Y-BOCS ( $N = 4$ ; $n = 261$ ; SMD = 0.16; 95% CI, -0.09 to 0.4). I
09 Y-BOCS at 12 weeks follow- up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms at 12 months follow-up as measured by the Y-BOCS ( $N = 2$ ; $n = 144$ ; SMD = 0.07; 95% CI, -0.26 to 0.4). I
10 Y-BOCS at 26 weeks follow- up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms at 26 weeks follow-up as measured by the Y-BOCS ( $N = 1$ ; $n = 53$ ; SMD = -0.15; 95% CI, -0.69 to 0.39). I
11 Y-BOCS at 52 weeks follow- up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms at 52 weeks follow-up as measured by the Y-BOCS ( $N = 1$ ; $n = 48$ ; SMD = -0.43; 95% CI, -1.01 to 0.14). I
12 Y-BOCS at 2 years follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms at 2 years follow-up as measured by the Y-BOCS (N = $2$ ; n = $144$ ; SMD = $0.2$ ; 95% CI, $-0.13$ to $0.53$ ). I
13 Beck Depression Inventory post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing depressive symptoms at the end of treatment, as measured by the Beck Depression Inventory (N = 4; n = 260; SMD = 0.16; 95% CI, -0.08 to 0.41). I
14 Beck Depression Inventory at 12 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing depressive symptoms at 12 weeks follow-up, as measured by the Beck Depression Inventory (N = 2; n = 144; SMD = -0.1; 95% CI, -0.43 to 0.23). I
15 Beck Depression Inventory at 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing depressive symptoms at 26 weeks follow-up, as measured by the Beck Depression Inventory (N = 1; $n = 53$ ; SMD = 0.34; 95% CI, -0.2 to 0.89). I
16 Beck Depression Inventory at 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing depressive symptoms at 1 year follow-up, as measured by the Beck Depression Inventory (N = 1; n = 48; SMD = $0.28$ ; 95% CI, $-0.28$ to $0.85$ ). I
17 Beck Depression Inventory at 2 years follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing depressive symptoms at 2 years follow-up, as measured by the Beck Depression Inventory ( $N = 2$ ; $n = 144$ ; SMD = -0.11; 95% CI, -0.44 to 0.22). I

18 Beck Anxiety Inventory	1	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms post-treatment, as measured by the NIMH-OC ( $N = 1$ ; $n = 60$ ; SMD 0.15; 95% CI, -0.36 to 0.66). I
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms at 26 weeks follow-up, as measured by the NIMH-OC ( $N = 1$ ; $n = 53$ SMD = -0.08; 95% CI, -0.62 to 0.46). I
03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms at 1 year follow-up, as measured by the NIMH-OC ( $N = 1$ ; $n = 48$ ; SMD = -0.18; 95% CI, -0.75 to 0.39). I
19 Padua Inventory post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms as measured by the Padua Inventory (N = 3; n = 201; SMD = 0.05; 95% CI, -0.23 to 0.32). I
20 Padua Inventory at 12 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms at 12 weeks follow-up as measured by the Padua Inventory ( $N = 2$ ; $n = 144$ ; SMD = $-0.02$ ; $n = 95\%$ CI, $n = 0.35$ to $n = 0.31$ ). I
21 Padua Inventory at 2 years follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive-compulsive symptoms at 2 years follow-up as measured by the Padua Inventory ( $N = 2$ ; $n = 144$ ; SMD = -0.01; 95% CI, -0.34 to 0.32). I
22 Beck Anxiety Inventory post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing anxiety symptoms at the end of treatment as measured by the Beck Anxiety Inventory ( $N = 2$ ; $n = 144$ ; SMD = -0.15; 95% CI, -0.47 to 0.18). I
23 Beck Anxiety Inventory at 12 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing anxiety symptoms at 12 weeks follow-up as measured by the Beck Anxiety Inventory (N = 2; n = 144; SMD = -0.1; 95% CI, -0.42 to 0.23). I
24 Beck Anxiety Inventory at 2 years follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing anxiety symptoms at 2 years follow-up as measured by the Beck Anxiety Inventory ( $N = 2$ ; $n = 144$ ; SMD = -0.13; 95% CI, -0.46 to 0.2). I
08 Symptom Checklist-90	•	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing psychological distress as measured by the Symptom Checklist (N = 1; n = 57; SMD = 0.22; 95% CI, -0.3 to 0.74). I

01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if
		there is a clinically important difference between behaviour therapy and cognitive therapy on reducing anxiety as measured by the Anxiety/Discomfort scale ( $N = 1$ ; $n = 57$ ; SMD = 0.48; 95% CI, -0.04 to 1.01). I
10 Irrational Belief Inventory	<u> </u>	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing irrational beliefs as measured by the Irrational Belief Inventory ( $N = 1$ ; $n = 55$ ; SMD = 0.49; 95% CI, -0.05 to 1.03). I
11 Behavioural Avoidance Te	est: Avoidanc	e
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing avoidance of fearful situations post treatment as measured by the Behavioural Avoidance test - avoidance subscale ( $N = 1$ ; $n = 60$ ; $SMD = 0.34$ ; $95\%$ CI, $-0.17$ to $0.85$ ).
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing avoidance of fearful situations at 26 weeks follow-up as measured by the Behavioural Avoidance test avoidance subscale ( $N = 1$ ; $n = 53$ ; $SMD = 0.2$ ; $95\%$ CI, $-0.34$ to $0.74$ ).
03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing avoidance of fearful situations at 52 weeks follow-up as measured by the Behavioural Avoidance test - avoidance subscale ( $N = 1$ ; $n = 48$ ; $SMD = 0.14$ ; $95\%$ CI, -0.43 to 0.71). I
12 Behavioural Avoidance Te	est: Discomfo	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing discomfort with fearful situations at the end of treatment as measured by the Behavioural Avoidance test - discomfort subscale ( $N = 1$ ; $n = 60$ ; SMD = 0.07; 95% CI, -0.43 to 0.58). I
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing discomfort with fearful situations at 26 weeks follow-up as measured by the Behavioural Avoidance test - discomfort subscale ( $N = 1$ ; $n = 53$ ; SMD = -0.05; 95% CI, -0.59 to 0.49). I
03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing discomfort with fearful situations at 52 weeks follow-up as measured by the Behavioural Avoidance test - discomfort subscale ( $N = 1$ ; $n = 48$ ; $SMD = -0.14$ ; $95\%$ CI, $-0.71$ to $0.42$ ). I
13 Obsessive Thoughts Checl	klist: total	•
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive thoughts at end of treatment as measured by the Obsessive Thoughts Checklist ( $N = 1$ ;

		n = 60; SMD = 0.23; 95% CI, -0.28 to 0.74). I
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive thoughts at 26 weeks follow-up as measured by the Obsessive Thoughts Checklist ( $N = 1$ ; $n = 53$ ; SMD = 0.2; 95% CI, -0.34 to 0.74). I
03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing obsessive thoughts at 52 weeks follow-up as measured by the Obsessive Thoughts Checklist ( $N = 1$ ; $n = 48$ ; SMD = 0.29; 95% CI, -0.28 to 0.86). I
14 ITIQ - intrusive thoughts		
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing intrusive thoughts at end of treatment as measured by the Intrusive Thoughts and their Interpretation Questionnaire - intrusive thoughts subscale (N = 1; n = 60; SMD = 0.29; 95% CI, -0.22 to 0.8). I
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing intrusive thoughts at 26 weeks follow-up as measured by the Intrusive Thoughts and their Interpretation Questionnaire - intrusive thoughts subscale (N = 1; n = 53; SMD = 0.05; 95% CI, -0.49 to 0.59). I
03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing intrusive thoughts at 52 weeks follow-up as measured by the Intrusive Thoughts and their Interpretation Questionnaire - intrusive thoughts subscale ( $N = 1$ ; $n = 48$ ; SMD = 0.26; 95% CI, -0.31 to 0.83). I
15 ITIQ - interpretation/intru	sion	,
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on improving interpretation of intrusive thoughts at end of treatment as measured by the Intrusive Thoughts and their Interpretation Questionnaire - interpretation/intrusive thoughts subscale ( $N = 1$ ; $n = 60$ ; SMD = 0.02; 95% CI, -0.48 to 0.53).
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on improving interpretation of intrusive thoughts at 26 weeks follow-up as measured by the Intrusive Thoughts and their Interpretation Questionnaire - interpretation/intrusive thoughts subscale (N = 1; n = 53; SMD = - 0.37; 95% CI, -0.91 to 0.18). I
03 At 52 weeks follow-up  16 ITIQ - responsibility	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on improving interpretation of intrusive thoughts at 52 weeks follow-up as measured by the Intrusive Thoughts and their Interpretation Questionnaire - interpretation/intrusive thoughts subscale ( $N = 1$ ; $n = 48$ ; $SMD = -0.12$ ; $95\%$ CI, $-0.69$ to $0.44$ ). I

01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if
or rost treatment	54	there is a clinically important difference between behaviour therapy and cognitive therapy on reducing interpretation of responsibility at end of treatment as measured by the Intrusive Thoughts and their Interpretation Questionnaire - responsibility subscale ( $N = 1$ ; $n = 60$ ;
		SMD = 0.11; 95% CI, -0.4 to 0.61). I
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing interpretation of responsibility at 26 weeks follow-up as measured by the Intrusive Thoughts and their Interpretation Questionnaire - responsibility subscale (N = 1; n = 53; SMD = -0.37; 95% CI, -0.91 to 0.18). I
03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing interpretation of responsibility at 52 weeks follow-up as measured by the Intrusive Thoughts and their Interpretation Questionnaire - responsibility subscale (N = 1; n = 48; SMD = -0.02; 95% CI, -0.59 to 0.55). I
17 ITIQ - guilt	•	·
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing interpretation of guilt at end of treatment as measured by the Intrusive Thoughts and their Interpretation Questionnaire - guilt subscale (N = 1; n = 60; SMD = -0.09; 95% CI, -0.6 to 0.42). I
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing interpretation of guilt at 26 weeks follow-up as measured by the Intrusive Thoughts and their Interpretation Questionnaire - guilt subscale (N = 1; n = 53; SMD = -0.27; 95% CI, -0.81 to 0.27). I
03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing interpretation of guilt at 52 weeks follow-up as measured by the Intrusive Thoughts and their Interpretation Questionnaire - guilt subscale (N = 1; n = 48; SMD = -0.09; 95% CI, -0.66 to 0.47). I
18 ITIQ - inferiority	I	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing interpretation of inferiority at end of treatment as measured by the Intrusive Thoughts and their Interpretation Questionnaire - inferiority subscale ( $N = 1$ ; $n = 60$ ; SMD = 0.06; 95% CI, -0.44 to 0.57). I
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing interpretation of inferiority at 26 weeks follow-up as measured by the Intrusive Thoughts and their Interpretation Questionnaire - inferiority subscale (N = 1; n = 53; SMD = -0.33; 95% CI, -0.87 to 0.21). I
03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing interpretation of inferiority at 52

		weeks follow-up as measured by the Intrusive Thoughts and their Interpretation Questionnaire - inferiority subscale ( $N = 1$ ; $n = 48$ ; SMD = -0.2; 95% CI, -0.76 to 0.37). I
19 Salkovs.kis Responsibility	Scale	ριτίο -0.2, 70 /0 Ci, -0.70 to 0.07 j. i
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing responsibility for negative events at end of treatment as measured by the Salkovs.kis Responsibility scale ( $N = 1$ ; $n = 60$ ; $SMD = 0.18$ ; $95\%$ CI, $-0.32$ to $0.69$ ). I
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing responsibility for negative events at 26 weeks follow-up as measured by the Salkovs.kis Responsibility scale ( $N = 1$ ; $n = 53$ ; $SMD = 0.13$ ; $95\%$ CI, $-0.41$ to $0.67$ ). I
03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on reducing responsibility for negative events at 52 weeks follow-up as measured by the Salkovs.kis Responsibility scale ( $N = 1$ ; $n = 48$ ; $SMD = -0.09$ ; $95\%$ CI, $-0.66$ to $0.47$ ). I
20 Quality of life	1	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on improving quality of life at the end of treatment ( $N = 1$ ; $n = 60$ ; $SMD = 0.33$ ; $95\%$ CI, $-0.18$ to $0.84$ ). I
02 At 26 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on improving quality of life at 26 weeks follow-up ( $N = 1$ ; $n = 53$ ; $SMD = 0.04$ ; $95\%$ CI, $-0.5$ to $0.58$ ). I
03 At 52 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on improving quality of life at 52 weeks follow-up ( $N = 1$ ; $n = 48$ ; $SMD = 0.15$ ; $95\%$ CI, $-0.42$ to $0.71$ ). I
06 Behaviour therapy v Ratio	nal Emotive	
01 Non-responders (Anxiety)		- 7
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on the likelihood of response at the end of treatment, defined as "much improved" on the Anxiety/Discomfort scale ( $N = 1$ ; $n = 18$ ; $RR = 0.78$ ; 95% CI, 0.55 to 1.1). I
02 At 1-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on the likelihood of response at one month follow-up, defined as "much improved" on the Anxiety/Discomfort scale (N = 1; n = 18; RR = 1; 95% CI, 0.61 to 1.64). I
02 Maudsley Obsessive-Com	pulsive Inve	,
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing obsessive-compulsive symptoms at the end of treatment as measured by the Maudsley Obsessive-Compulsive Inventory (N = 2; n = 39; SMD = 0.25; 95% CI, -0.38 to 0.88). I

02 At 1-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if
oz At 1-month follow-up	54	there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing obsessive-compulsive
		symptoms at one month follow-up as measured by the Maudsley Obsessive-Compulsive Inventory ( $N = 1$ ; $n = 18$ ; $SMD = 0.3$ ; $95\%$ CI,
		-0.63 to 1.23). I
03 Anxiety Discomfort Scale		
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing anxiety and discomfort at the end of treatment as measured by the Anxiety/Discomfort scale $(N = 1; n = 18; SMD = 0.22; 95\% CI, -0.7 to 1.15)$ . I
02 At 1-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing anxiety and discomfort at one month follow-up as measured by the Anxiety/Discomfort scale ( $N = 1$ ; $n = 18$ ; SMD = -0.18; 95% CI, -1.11 to 0.75). I
04 Irrational Belief test		
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing irrational beliefs at the end of treatment as measured by the Irrational Belief Test ( $N = 2$ ; $n = 39$ ; SMD = 0.27; 95% CI, -0.37 to 0.9). I
02 At 1-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing irrational beliefs at one month follow-up as measured by the Irrational Belief Test ( $N = 1$ ; $n = 18$ ; SMD = 0.45; 95% CI, -0.49 to 1.39). I
05 Self-rating Depression scal	e	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing depression at the end of treatment as measured by the Self-rating Depression scale ( $N = 2$ ; $n = 39$ ; SMD = 0.4; 95% CI, -0.23 to 1.04). I
02 At 1-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing depression at one month follow-up as measured by the Self-rating Depression scale (N = 1; n = 18; SMD = 0.51; 95% CI, -0.44 to 1.45). I
06 Social Anxiety scale		-
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing anxiety at the end of treatment as measured by the Social Anxiety scale ( $N = 1$ ; $n = 18$ ; $SMD = 0.01$ ; 95% CI, -0.91 to 0.94). I
02 At 1-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing anxiety at one month follow-up as measured by the Social Anxiety scale (N = 1; n = 18; SMD = 0; 95% CI, -0.92 to 0.92). I
07 Hostility and Direction of 1	Hostility Qu	estionnaire: Intrapunitivity

01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if
		there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing intrapunitivity at the end of treatment as measured by the Hostility and Direction of Hostility Questionnaire - intrapunitivity subscale ( $N = 1$ ; $n = 18$ ; SMD = -0.03; 95% CI, -0.95 to 0.9). I
02 At 1-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing intrapunitivity at one month follow-up as measured by the Hostility and Direction of Hostility Questionnaire - intrapunitivity subscale ( $N = 1$ ; $n = 18$ ; $SMD = 0.1$ ; 95% CI, -0.82 to 1.03). I
08 Hostility and Direction of H	Iostility Que	stionnaire: Extrapunitivity
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing extrapunitivity at the end of treatment as measured by the Hostility and Direction of Hostility Questionnaire - extrapunitivity subscale (N = 1; n = 18; SMD = 0.07; 95% CI, -0.85 to 1). I
02 At 1-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing extrapunitivity at one month follow-up as measured by the Hostility and Direction of Hostility Questionnaire - extrapunitivity subscale ( $N = 1$ ; $n = 18$ ; $SMD = -0.12$ ; $95\%$ CI, $-1.05$ to $0.8$ ). I
09 Dutch Obsessive-Compulsi	ve Question	naire
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing obsessive-compulsive symptoms as measured by the Dutch Obsessive-Compulsive Questionnaire] ( $N = 1$ ; $n = 21$ ; $n $
10 Anxiety Disability scale: ma	in OC symp	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational emotive therapy on reducing anxiety and discomfort as measured by the Anixety Discomfort scale for main obsessive-compulsive symptoms (N = 1; n = 21; SMD = $0.08$ ; 95% CI, $-0.78$ to $0.93$ ). I
07 BT v RET + exposure in viv	o (post RET	alone)
01 Maudsley Obsessive-Comp	ulsive Inven	atory
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and RET + exposure in vivo after initial treatment of RET alone on reducing obsessive-compulsive symptoms at the end of treatment as measured by the Maudsley Obsessive-Compulsive Inventory (N = 1; $n = 21$ ; SMD = 0.31; 95% CI, -0.55 to 1.17). I
02 At follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and RET + exposure in vivo after initial treatment of RET alone on reducing obsessive-compulsive symptoms at 8 weeks follow-up as measured by the Maudsley Obsessive-Compulsive Inventory ( $N = 1$ ; $n = 21$ ; SMD = 0.38; 95% CI, -0.48 to 1.25). I

01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and RET + exposure in vivo after initial treatment of RET alone on reducing obsessive-compulsive symptoms at the end of treatment as measured by the Dutch Obsessive-Compulsive Questionnaire (N = 1; n = 21; SMD = 0.31; 95% CI, -0.55 to 1.17). I
02 At follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and RET + exposure in vivo after initial treatment of RET alone on reducing obsessive-compulsive symptoms at 8 weeks follow-up as measured by the Dutch Obsessive-Compulsive Questionnaire (N = 1; n = 21; SMD = 0.27; 95% CI, -0.59 to 1.14). I
03 Anxiety Discomfort scale: n	nain OC syı	<u> </u>
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and RET + exposure in vivo after initial treatment of RET alone on reducing anxiety and discomfort as measured by the Anixety Discomfort scale for main obsessive-compulsive symptoms (N = 1; n = 21; SMD = -0.48; 95% CI, -1.35 to 0.39). I
04 Irrational Belief test		- 21, 51/1D0.40, 95 /6 Cl, -1.55 to 0.57). 1
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and RET + exposure in vivo after initial treatment of RET alone on reducing irrational beliefs at the end of treatment as measured by the Irrational Beliefs Test ( $N = 1$ ; $n = 21$ ; SMD = 0.58; 95% CI, -0.3 to 1.46). I
02 At follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and RET + exposure in vivo after initial treatment of RET alone on reducing irrational beliefs at 8 weeks follow-up as measured by the Irrational Beliefs Test (N = 1; n = 21; SMD = 0.66; 95% CI, -0.23 to 1.54). I
05 Self-rating Depression scale		
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and RET + exposure in vivo after initial treatment of RET alone on reducing depression at the end of treatment as measured by the Selfrating Depression scale ( $N = 1$ ; $n = 21$ ; SMD = 0.52; 95% CI, -0.35 to 1.4). I
02 At follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and RET + exposure in vivo after initial treatment of RET alone on reducing depression at 8 weeks follow-up as measured by the Self-rating Depression scale ( $N = 1$ ; $n = 21$ ; SMD = 0.02; 95% CI, -0.84 to 0.88). I
08 BT v CBT		
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on the likelihood of leaving the study early ( $N = 1$ ; $n = 35$ ; $RR = 6.74$ ; $95\%$ CI, $0.94$ to $48.29$ ). I
02 Non-remission (Y-BOCS)		2. 2. 2. 2. 2. 2. 2. 2. 2. 2. 2. 2. 2. 2
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01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on the likelihood of remission, defined as a score less than 16 on the Y-BOCS at the end of treatment ( $N = 1$ ; $n = 35$ ; $RR = 0.76$ ; $95\%$ CI, $0.41$ to $1.39$ ). I
02 At 3 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on the likelihood of remission, defined as a score less than 16 on the Y-BOCS at 3 months follow-up ( $N = 1$ ; $n = 35$ ; $RR = 0.76$ ; $95\%$ CI, $0.41$ to $1.39$ ). I
03 Y-BOCS		
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on reducing obsessive-compulsive symptoms at the end of treatment as measured by the Y-BOCS ( $N = 1$ ; $n = 35$ ; SMD = -0.08; 95% CI, -0.75 to 0.58). I
02 At 3 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on reducing obsessive-compulsive symptoms at 3 months follow-up as measured by the Y-BOCS ( $N = 1$ ; $n = 35$ ; SMD = 0.4; 95% CI, -0.27 to 1.07). I
03 At 6 months follow-up	s2y	There is limited evidence suggesting a difference favouring cognitive behavioural therapy over behaviour therapy on reducing obsessive-compulsive symptoms at 6 months follow-up as measured by the Y-BOCS ( $N = 1$ ; $n = 35$ ; SMD = 0.07; 95% CI, -0.59 to 0.74). I
04 At 12 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on reducing obsessive-compulsive symptoms at 12 months follow-up as measured by the Y-BOCS ( $N = 1$ ; $n = 35$ ; SMD = 0.07; 95% CI, -0.59 to 0.74). I
08 BDI	<b>.</b>	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on reducing depressive symptoms at the end of treatment, as measured by the Beck Depression Inventory ( $N = 1$ ; $n = 34$ ; $SMD = -0.1$ ; $95\%$ CI, $-0.77$ to $0.57$ ). I
02 At 3 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on reducing depressive symptoms at 3 months follow-up, as measured by the Beck Depression Inventory (N = 1; n = 34; SMD = 0.18; 95% CI, -0.5 to 0.85). I
03 At 6 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on reducing depressive symptoms at 6 months follow-up, as measured by the Beck Depression Inventory ( $N = 1$ ; $n = 34$ ; $SMD = 0.03$ ; $95\%$ CI, -0.64 to 0.71). I
04 At 12 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive behavioural therapy on reducing depressive symptoms at 12 months follow-up, as measured by the Beck

		Depression Inventory (N = 1; n = 34; SMD = -0.21; 95% CI, -0.89 to 0.46). I
09 Yogic meditation v Relaxation	n response	e + mindfulness meditation
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between Kundalini yoga and relaxation response plus mindfulness meditation on the likelihood of leaving the study early (N = 1; n = 22; RR = 1.39; 95% CI, 0.44 to 4.43). I
02 Y-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between Kundalini yoga and relaxation response plus mindfulness meditation on obsessive-compulsive symptoms as measured by Y-BOCS (N = 1; n = 22; SMD = -0.55; 95% CI, -1.41 to 0.31). I
03 Profile of Moods scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between Kundalini yoga and relaxation response plus mindfulness meditation on mood as measured by the Profile of Moods scale ( $N = 1$ ; $n = 14$ ; $SMD = -1.11$ ; 95% CI, -2.27 to 0.04). I
04 Perceived Stress scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between Kundalini yoga and relaxation response plus mindfulness meditation on level of stress as measured by the Perceived Stress Scale ( $N = 1$ ; $n = 14$ ; SMD = -0.67; 95% CI, -1.75 to 0.42). I
05 Purpose in Life scale	s2x	There is limited evidence suggesting a difference favouring Kundalini yoga over relaxation response plus mindfulness meditation on purpose in life as measured by the Purpose in Life scale ( $N = 1$ ; $n = 14$ ; $SMD = -1.2$ ; $95\%$ CI, $-2.38$ to $-0.03$ ). I
09 Self-exposure v Partner-assis	ted exposi	
01 Anxiety/discomfort - Main compulsion (assessor rated)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between self-exposure and partner-assisted exposure on reducing anxiety-discomfort due to main compulsions as measured by an independent assessor on an 9-point scale ( $N = 1$ ; $n = 12$ ; SMD = 0.37; 95% CI, -0.77 to 1.52). I
02 Anxiety/discomfort - Other compulsions (assessor rated)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between self-exposure and partner-assisted exposure on reducing anxiety-discomfort due to other compulsions as measured by an independent assessor on an 9-point scale ( $N = 1$ ; $n = 12$ ; $SMD = 0.6$ ; $95\%$ CI, $-0.56$ to $1.77$ ). I
03 Maudsley Obsessive- compulsive inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between self-exposure and partner-assisted exposure on reducing obsessive-compulsive symptoms as measured by the Maudsley Obsessive-compulsive inventory ( $N = 1$ ; $n = 12$ ; $SMD = 0$ ; $95\%$ CI, $-1.13$ to $1.13$ ). I
04 Social and Marital Adjustment: Marital	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between self-exposure and partner-assisted exposure on improving social and marital adjustment, as measured by the Social and Marital Adjustment Questionnaire - Marital subscale (N = 1; n = 12; SMD = -0.65; 95% CI, -1.83 to 0.53). I
05 Social and Marital Adjustment: Sexual	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between self-exposure and partner-assisted exposure on improving social and marital

		adjustment, as measured by the Social and Marital Adjustment Questionnaire - Sexual subscale (N = 1; n = 12; SMD = $0.83$ ; 95% CI, - $0.37$ to $2.03$ ). I
06 Social and Marital Adjustment: Social	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between self-exposure and partner-assisted exposure on improving social and marital adjustment, as measured by the Social and Marital Adjustment Questionnaire - Social subscale (N = 1; n = 12; SMD = 0.49; 95% CI, - 0.67 to 1.64). I
07 Anxious mood (assessor-rated)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between self-exposure and partner-assisted exposure on reducing anxious mood, as measured by an independent assessor on the Anxious Mood and Depression Scales ( $N = 1$ ; $n = 12$ ; $SMD = -0.62$ ; $95\%$ CI, $-1.79$ to $0.55$ ). I
08 Self-rating Depression scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between self-exposure and partner-assisted exposure on reducing depression, as measured by the Self-rating Depression scale (N = 1; n = 12; SMD = 0.61; 95% CI, - 0.56 to 1.78). I
10 Imaginal + live ERP v Live EI	RP	
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on the likelihood of leaving the study early ( $N = 1$ ; $n = 56$ ; $RR = 1$ ; 95% CI, 0.33 to 3.08). I
02 Relapse (multiple outcomes)		
01 At 20 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on the likelihood of relapse at the 20-week follow-up, defined as loss $50\%$ of the improvement on the CGI observed at the end of treatment (N = 1; n = $56$ ; RR = $1.6$ ; $95\%$ CI, $0.6$ to $4.29$ ). I
02 At 32 weeks follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on the likelihood of relapse at the 32-week follow-up, defined as loss 50% of the improvement on the CGI observed at the end of treatment ( $N = 1$ ; $n = 56$ ; $RR = 0.5$ ; 95% CI, 0.1 to 2.51). I
03 Y-BOCS obsessions		
01 At 9 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing obsessions, as measured by the Y-BOCS obsessions subscale ( $N = 1$ ; $n = 46$ ; $SMD = -0.11$ ; $95\%$ CI, $-0.69$ to $0.47$ ). I
02 At 20 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing obsessions at the 20-week follow-up, as measured by the Y-BOCS obsessions subscale ( $N = 1$ ; $n = 46$ ; $SMD = -0.17$ ; 95% CI, -0.74 to 0.41). I

03 At 32 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if
, ,	<b>7</b> 1	there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing obsessions at the 32-week follow-up, as measured by the Y-BOCS obsessions subscale (N = 1; n = 41; SMD = -0.21; 95% CI, -0.82 to 0.4). I
04 Y-BOCS rituals		
01 At 9 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing rituals, as measured by the Y-BOCS rituals subscale ( $N = 1$ ; $n = 46$ ; $N = -0.18$ ; $N = -0.18$ ; $N = -0.76$ to $N = -0.4$ ). I
02 At 20 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing rituals at the 20-week follow-up, as measured by the Y-BOCS rituals subscale ( $N = 1$ ; $n = 41$ ; $SMD = -0.25$ ; 95% CI, -0.86 to 0.37). I
03 At 32 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing rituals at the 32-week follow-up, as measured by the Y-BOCS rituals subscale ( $N = 1$ ; $n = 41$ ; $SMD = -0.33$ ; $95\%$ CI, $-0.95$ to $0.28$ ). I
05 Compulsions checklist		
01 At 9 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing compulsions, as measured by the compulsion checklist ( $N = 1$ ; $n = 46$ ; $SMD = -0.12$ ; $95\%$ CI, $-0.7$ to $0.46$ ). I
02 At 20 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing compulsions at the 20-week follow-up, as measured by the compulsion checklist ( $N = 1$ ; $n = 41$ ; $SMD = -0.11$ ; $95\%$ CI, $-0.72$ to $0.51$ ). I
03 At 32 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing compulsions at the 32-week follow-up, as measured by the compulsion checklist ( $N = 1$ ; $n = 41$ ; SMD = -0.35; 95% CI, -0.97 to 0.27). I
06 Beck Depression Inventory		
01 At 9 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing depression, as measured by the Beck Depression Inventory ( $N = 1$ ; $n = 46$ ; $SMD = -0.3$ ; $95\%$ CI, $-0.88$ to $0.28$ ). I
02 At 20 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing depression at the 20-week follow-up, as

		measured by the Beck Depression Inventory (N = 1; n = 41; SMD = -0.45; 95% CI, -1.07 to 0.17). I
		, and the second
03 At 32 weeks (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live exposure plus response prevention and live exposure plus response prevention on reducing depression at the 32-week follow-up, as measured by the Beck Depression Inventory (N = 1; n = 41; SMD = $-0.31$ ; 95% CI, $-0.92$ to $0.31$ ). I
11 ERP + relapse prevention v E	RP + associ	ative therapy
01 Non-responders (Y-BOCS 509	%)	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus response prevention and relapse prevention and exposure plus response prevention and associative therapy on the likelihood of response, defined as a 50% or greater reduction on the Y-BOCS (N = $1$ ; n = $20$ ; RR = $1.33$ ; 95% CI, $0.4$ to $4.49$ ). I
02 At 6 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus response prevention and relapse prevention and exposure plus response prevention and associative therapy on the likelihood of response at 6 months follow-up, defined as a 50% or greater reduction on the Y-BOCS (N = 1; n = 20; RR = 0.57; 95% CI, 0.24 to 1.35). I
02 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus response prevention and relapse prevention and exposure plus response prevention and associative therapy on the likelihood of leaving the study early ( $N = 1$ ; $n = 20$ ; $RR = 5$ ; 95% CI, 0.27 to 92.63).
03 Y-BOCS		
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus response prevention and relapse prevention and exposure plus response prevention and associative therapy on reducing obsessive-compulsive symptoms as measured by the Y-BOCS ( $N = 1$ ; $n = 18$ ; SMD = -0.16; 95% CI, -1.09 to 0.77). I
02 At 6-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus response prevention and relapse prevention and exposure plus response prevention and associative therapy on reducing obsessive-compulsive symptoms at 6 months follow-up as measured by the Y-BOCS ( $N = 1$ ; $n = 18$ ; SMD = -0.63; 95% CI, -1.58 to 0.33). I
04 Obessive-compulsive sympto	ms (assesso	or-rated)
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus response prevention and relapse prevention and exposure plus response prevention and associative therapy on reducing obsessive-compulsive symptoms as rated by an independent assessor ( $N = 1$ ; $n = 18$ ; $SMD = -0.57$ ; $95\%$ CI, $-1.53$ to $0.38$ ). I
02 At 6-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus response prevention and relapse prevention and exposure plus response prevention and associative therapy on reducing obsessive-

		compulsive symptoms as rated by an independent assessor at 6 months follow-up (N = 1; n = 18; SMD = -1.08; 95% CI, -2.09 to -0.07). I
05 Hamilton Depression Scale	•	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus response prevention and relapse prevention and exposure plus response prevention and associative therapy on reducing depression as measured by the Hamilton Depression Rating Scale (N = 1; n = 18; SMD = 0.45; 95% CI, -0.5 to 1.39). I
02 At 6-month follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus response prevention and relapse prevention and exposure plus response prevention and associative therapy on reducing depression at 6 months follow-up as measured by the Hamilton Depression Rating Scale ( $N = 1$ ; $n = 18$ ; $SMD = -0.66$ ; $95\%$ CI, $-1.63$ to $0.3$ ). I
12 Anxiogenic exposure v Neu	tral thoughts	S
01 Non-responders (multiple outcomes "much improved")	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on the likelihood of response, defined as a change in the main targeted problem of 16 or more (N = 1; n = 16; RR = 6.25; 95% CI, 0.35 to 112.52). I
02 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on the likelihood of leaving the study early ( $N = 1$ ; $n = 16$ ; $RR = 0.43$ ; $95\%$ CI, $0.06$ to $3.28$ ). I
03 Compulsions checklist	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on reducing obsessive-compulsive symptoms as measured by the Compulsions checklist (N = 1; n = 12; SMD = 0.53; 95% CI, -0.63 to 1.7). I
04 Obsessions - time	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on reducing the amount of time spent in obsessions $(N = 1; n = 12; SMD = -0.5; 95\% CI, -1.66 to 0.66)$ . I
05 Obsessions - discomfort	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on reducing the amount of discomfort ( $N = 1$ ; $n = 12$ ; SMD = -0.05; 95% CI, -1.18 to 1.08). I
06 Main target	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on reducing the severity of the main target symptom ( $N = 1$ ; $n = 12$ ; $SMD = 0.35$ ; $95\%$ CI, $-0.79$ to $1.5$ ). I

07 Beck Depression Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on reducing depression as measured by the Beck Depression Inventory ( $N = 1$ ; $n = 12$ ; $SMD = 0.1$ ; $95\%$ CI, $-1.04$ to
		1.23). I
08 Work adjustment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on improving work adjustment ( $N = 1$ ; $n = 12$ ; SMD = 0.13; 95% CI, -1 to 1.27). I
09 Home adjustment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on improving home adjustment ( $N = 1$ ; $n = 12$ ; $SMD = -0.59$ ; 95% CI, -1.75 to 0.58). I
10 Social adjustment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on improving social adjustment ( $N = 1$ ; $n = 12$ ; $SMD = 0.25$ ; 95% CI, -0.89 to 1.39). I
11 Private adjustment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on improving private adjustment ( $N = 1$ ; $n = 12$ ; SMD = 0; 95% CI, -1.13 to 1.13). I
13 BTSTEPS + scheduled suppor	t v BTSTEPS	5 + requested support
01 Y-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between BTSTEPS plus scheduled support and BTSTEPS plus requested support on reducing obsessive-compulsive symptoms as measured by the Y-BOCS ( $N = 1$ ; $n = 36$ ; $SMD = -0.55$ ; $95\%$ CI, $-1.22$ to $0.12$ ). I
02 Target triggers discomfort	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between BTSTEPS plus scheduled support and BTSTEPS plus requested support on reducing discomfort on target symptoms (N = 1; n = 36; SMD = -0.28; 95% CI, -0.94 to 0.39). I
03 Work and Social Adjustment Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between BTSTEPS plus scheduled support and BTSTEPS plus requested support on improving work and social adjustment as measured by the Work and Social Adjustment Scale ( $N = 1$ ; $n = 36$ ; SMD = -0.36; 95% CI, -1.02 to 0.3). I
04 Leaving the study early	s1x	There is evidence suggesting a difference favouring BTSTEPS plus scheduled support over BTSTEPS plus requested support on the likelihood of leaving the study early (N = 1; n = 44; RR = 0.23; 95% CI, 0.08 to 0.7). I
14 Family-based BT v Patient-based	sed BT	1, 5:00 to 5:1 j. 2
01 MOCI		
01 Post-treatment	s2x	There is limited evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing obsessive-compulsive symptoms as measured by the Maudsley Obsessive-compulsive inventory (N = 1; n = 30; SMD = -0.89; 95% CI, -1.65 to -0.14). I

02 Follow-up at 6 months	s1x	There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing obsessive-compulsive symptoms at 6 months follow-up as measured by the Maudsley Obsessive-compulsive inventory (N = 1; n = 30; SMD = -1.44; 95% CI, -2.25 to -0.62). I			
02 Zung Self-rating Depressio	n Scale	,			
01 Post-treatment	s1x	There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing depression as measured by the Zung Self-rating Depression scale (N = 1; n = 30; SMD = -1.38; 95% CI, -2.19 to -0.58). I			
02 Follow-up at 6 months	s1x	There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing depression at 6 months follow-up as measured by the Zung Self-rating Depression scale (N = 1; n = 30; SMD = -1.81; 95% CI, -2.67 to -0.94). I			
03 Social adjustment: Occupat	ion				
01 Post-treatment	s2x	There is limited evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on improving social adjustment at work as measured by the Global Assessment of Severity (N = 1; n = 30; SMD = -0.91; 95% CI, -1.67 to -0.16). I			
02 Follow-up at 6 months	s1x	There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on improving social adjustment at work at 6 months follow-up as measured by the Global Assessment of Severity (N = 1; n = 30; SMD = -1.34; 95% CI, -2.15 to -0.54). I			
04 Social adjustment: family	1	<u> </u>			
01 Post-treatment	s2x	There is limited evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on improving family adjustment as measured by the Global Assessment of Severity (N = 1; n = 30; SMD = -0.78; 95% CI, -1.52 to -0.03). I			
02 Follow-up at 6 months	s2x	There is limited evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on improving family adjustment at 6 months follow-up as measured by the Global Assessment of Severity (N = 1; n = 30; SMD = -0.82; 95% CI, -1.57 to -0.07). I			
05 Social adjustment: househo	05 Social adjustment: household responsibilities				
01 Post-treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between family-based behaviour management and patient-based behaviour management on improving household responsibility as measured by the Global Assessment of Severity (N = 1; n = 30; SMD = $-0.56$ ; 95% CI, $-1.29$ to 0.18). I			
02 Follow-up at 6 months  06 Social adjustment: leisure-t	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between family-based behaviour management and patient-based behaviour management on improving household responsibility at 6 months follow-up as measured by the Global Assessment of Severity (N = 1; n = 30; SMD = -0.51; 95% CI, -1.24 to 0.22). I			

01 Post-treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between family-based behaviour management and patient-based behaviour management on improving leisure-time activities as measured by the Global Assessment of Severity (N = 1; n = 30; SMD = -0.32; 95% CI, -1.05 to
		0.4). I
02 Follow-up at 6 months	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between family-based
		behaviour management and patient-based behaviour management
		on improving leisure-time activities at 6 months follow-up as
		measured by the Global Assessment of Severity ( $N = 1$ ; $n = 30$ ; SMD
		= -0.7; 95% CI, -1.45 to 0.04). I

# Psychological vs. control (BDD)

Description	Statement level	Statement
01 CBT v No treatment/wait-list	control	
01 Non-responders (DSM-BDD + BDDE)	s2x	There is limited evidence suggesting a difference favouring CBT over no treatment/wait-list control on the likelihood of response ( $N = 1$ ; $n = 54$ ; $RR = 0.2$ ; 95% CI, 0.09 to 0.44). I
02 Body Dysmorphic Disorder Examination	s1x	There is evidence suggesting a difference favouring CBT over no treatment/wait-list control on reducing BDD symptoms as measured by the BDDE ( $N = 2$ ; $n = 73$ ; $SMD = -2.35$ ; $95\%$ CI, $-2.96$ to $-1.73$ ). I
05 Rosenberg Self-Esteem Scale	s2x	There is limited evidence suggesting a difference favouring CBT over no treatment/wait-list control on increasing self-esteem as measured by the RSES ( $N = 1$ ; $n = 54$ ; SMD = -0.86; 95% CI, -1.42 to -0.3). I
06 Brief Symptom Inventory	s2x	There is limited evidence suggesting a difference favouring CBT over no treatment/wait-list control on reducing psychological distress as measured by the BSI (N = 1; n = $54$ ; SMD = $-0.79$ ; 95% CI, $-1.35$ to $-0.24$ ). I
07 Y-BOCS (BDD)	s2x	There is limited evidence suggesting a difference favouring CBT over no treatment/wait-list control on reducing BDD symptoms as measured by the Y-BOCS (BDD) ( $N = 1$ ; $n = 19$ ; SMD = -1.81; 95% CI, -2.92 to -0.7). I
08 MADRS	s2x	There is limited evidence suggesting a difference favouring CBT over no treatment/wait-list control on reducing depressive symptoms as measured by the MADRS (N = 1; n = 19; SMD = $-1.53$ ; 95% CI, $-2.58$ to $-0.47$ ). I
09 Hospital Anxiety	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and no treatment/wait-list control on reducing anxiety symptoms as measured by the HADI anxiety scale ( $N = 1$ ; $n = 19$ ; $SMD = -0.12$ ; $95\%$ CI, $-1.02$ to $0.78$ ). I
10 Hospital Depression	s2x	There is limited evidence suggesting a difference favouring CBT over wait-list control on reducing depressive symptoms as measured by the HADI depression scale (N = 1; n = 19; SMD = $-1.66$ ; 95% CI, $-2.73$ to $-0.58$ ). I
12 Social Phobia	s2x	There is limited evidence suggesting a difference favouring CBT over wait-list control on reducing social phobia as measured by the SPAI ( $N = 1$ ; $n = 19$ ; SMD = $-1.89$ ; 95% CI, $-3.02$ to $-0.77$ ). I

Psychological vs. Psychological (BDD)		
Description	Statement level	Statements and Statistics
01 BT v CT (mid-treatment)		
01 Non-responders (Y-BOCS non-reliable change)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive therapy at mid-treatment on the likelihood of response, defined as a reliable change on the Y-BOCS ( $N = 1$ ; $n = 10$ ; $RR = 1$ ; 95% CI, 0.08 to 11.93). I
02 Y-BOCS (BDD)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive therapy at mid-treatment on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = $10$ ; SMD = $0.91$ ; $95\%$ CI, $-0.43$ to $2.25$ ). I
03 BDD Examination	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive therapy at mid-treatment on reducing obsessive-compulsive symptoms as measured by the BDD examination ( $N = 1$ ; $n = 10$ ; $SMD = 1.17$ ; $95\%$ CI, $-0.23$ to $2.58$ ). I
04 Defects Related Beliefs Test	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive therapy at mid-treatment on reducing dysfunctional beliefs about appearance as measured by the Defects Related Beliefs test ( $N = 1$ ; $n = 10$ ; $SMD = 0.69$ ; $95\%$ CI, $-0.61$ to $1.99$ ). I
05 Beck Anxiety Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive therapy at mid-treatment on reducing anxiety as measured by the Beck Anxiety Inventory ( $N = 1$ ; $n = 10$ ; SMD = 0.65; 95% CI, -0.64 to 1.94). I
06 Beck Depression Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive therapy at mid-treatment on reducing depression as measured by the Beck Depression Inventory ( $N = 1$ ; $n = 10$ ; SMD = 0.23; 95% CI, -1.02 to 1.47). I
07 Body Satisfaction Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive therapy at mid-treatment on reducing dissatisfaction with body parts as measured by Body Satisfaction Scale ( $N = 1$ ; $n = 10$ ; SMD = 0.26; 95% CI, -0.99 to 1.51). I
08 Social Avoidance and Distress Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive therapy at mid-treatment on reducing social avoidance and affective discomfort as measured by the Social Avoidance and Distress scale ( $N = 1$ ; $n = 10$ ; $SMD = 0.15$ ; $95\%$ CI, $-1.09$ to $1.39$ ). I
09 Quality of Life Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive therapy at mid-treatment on improving quality of life as measured by the Quality of Life Inventory (N = 1; n = $10$ ; SMD = $-0.14$ ; 95% CI, $-1.38$ to $1.11$ ). I
02 BT v CBT (post-treatment)	14	
01 Non-responders (Y-BOCS non-reliable change)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive behavioural therapy at the end of treatment on

		the likelihood of response, defined as a reliable change on the Y-BOCS (N = 1; n = 10; RR = 3; 95% CI, 0.15 to 59.89). I
02 Y-BOCS (BDD)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive behavioural therapy at the end of treatment on reducing obsessive-compulsive symptoms as measured by the Y-BOCS ( $N = 1$ ; $n = 10$ ; $SMD = 0.48$ ; $95\%$ CI, $-0.79$ to $1.75$ ). I
03 BDD Examination	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive behavioural therapy at the end of treatment on reducing dysfunctional beliefs about appearance as measured by the Defects Related Beliefs test (N = 1; n = 10; SMD = 1.25; 95% CI, -0.17 to 2.68). I
04 Defects Related Beliefs Test	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive behavioural therapy at the end of treatment on reducing dysfunctional beliefs about appearance as measured by the Defects Related Beliefs test ( $N = 1$ ; $n = 10$ ; SMD = 0.76; 95% CI, -0.55 to 2.07). I
05 Beck Anxiety Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive behavioural therapy at the end of treatment on reducing anxiety as measured by the Beck Anxiety Inventory ( $N = 1$ ; $n = 10$ ; SMD = 0.51; 95% CI, -0.77 to 1.78). I
06 Beck Depression Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive behavioural therapy at the end of treatment on reducing depression as measured by the Beck Depression Inventory ( $N = 1$ ; $n = 10$ ; SMD = 0.12; 95% CI, -1.12 to 1.36). I
07 Body Satisfaction Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive behavioural therapy at the end of treatment on reducing dissatisfaction with body parts as measured by Body Satisfaction Scale ( $N = 1$ ; $n = 10$ ; SMD = 0.15; 95% CI, -1.1 to 1.39). I
08 Social Avoidance and Distress Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive behavioural therapy at the end of treatment on reducing social avoidance and affective discomfort as measured by the Social Avoidance and Distress scale (N = 1; n = 10; SMD = -0.07; 95% CI, -1.31 to 1.17). I
09 Quality of Life Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behavioural therapy and cognitive behavioural therapy at the end of treatment on improving quality of life as measured by the Quality of Life Inventory ( $N = 1$ ; $n = 10$ ; $SMD = -0.18$ ; $95\%$ CI, $-1.07$ to $1.42$ ). I

## Pharmacological interventions evidence statements

SSRIs (adults); Clomipramine (adults); TCAs; SNRIs; MAOIs; Anxiolytics; Other Pharmacological; Augmentation strategies; SSRIs (children/adolescents); Clomipramine (children/adolescents); Pharmacological (BDD)

### SSRIs (adults)

Description	Statement level	Statement and Statistics
01 SSRIs vs. placebo (acute phas	se)	
01 Adverse events	s2y	There is limited evidence suggesting a difference favouring placebo over SSRIs on the likelihood of reporting adverse events in adults with OCD (N = $10$ ; n = $1786$ ; RR = $1.16$ ; $95\%$ C.I., $1.1$ to $1.23$ ). I
02 Serious adverse events	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on the likelihood of reporting serious adverse events ( $N = 1$ ; $n = 189$ ; $RR = 0.99$ ; $95\%$ C.I., $0.2$ to $4.78$ ). I
03 Attempted suicide	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on the likelihood of attempted suicide ( $N = 1$ ; $n = 189$ ; $RR = 0.99$ ; 95% C.I., 0.06 to 15.59). I
02 Leaving study early	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and placebo on the likelihood of leaving the study early in adults with OCD ( $N = 16$ ; $n = 2623$ ; $RR = 0.98$ ; 95% C.I., 0.85 to 1.13). I
03 Leaving study early due to adverse events	s1y	There is evidence suggesting a difference favouring placebo over SSRIs on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 13; n = 3009; RR = $2.15$ ; 95% C.I., $1.62$ to $2.86$ ). I
04 Non-responders (OCD)	s1x	There is evidence suggesting a difference favouring SSRIs over placebo on the likelihood of treatment response, defined as a 25%+ or 35%+ reduction on the Y-BOCS or a score of 1 or 2 on the Clinical Global Impressions scale, in adults with OCD (N = 9; n = 2588; RR = 0.77; 95% C.I., 0.73 to 0.82). I
05 Non-responders (MDD)		
01 Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and placebo on the likelihood of treatment response, defined as achieving a $50\%$ or greater reduction on the Hamilton Rating Scale for Depression, in adults with OCD (N = 1; n = $46$ ; RR = $0.9$ ; $95\%$ C.I., $0.69$ to $1.18$ ). I
06 Non-remitters		
01 Fluvoxamine	s2x	There is limited evidence suggesting a difference favouring fluvoxamine over placebo on the likelihood of remission, defined as a score of 8 or less or the Y-BOCS, in adults with OCD (N = 1; n = $253$ ; RR = $0.9$ ; $95\%$ C.I., $0.82$ to $0.98$ ). I
07 Y-BOCS	s2x	There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = $10$ ; n = $1629$ ; SMD = $-0.42$ ; $95\%$ C.I., $-0.52$ to $-0.32$ ). I

08 NIMH-OC	s2x	There is limited evidence expressing a difference foregoing CCDI-
US MINITI-OC	SZX	There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD ( $N = 7$ ; $n = 904$ ; SMD = -0.37; 95% C.I., -0.5 to -0.23). I
09 General Rating Scale - comp	ulsions	5/12 0.07/30% C.I., 0.0 to 0.20/. I
01 Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and placebo on compulsive symptoms as measured by the General Rating Scale compulsions subscale in adults with OCD (N = 1; n = 16; SMD = $-0.74$ ; 95% C.I., $-1.76$ to $0.29$ ). I
10 General Rating Scale - obsess	sions	
01 Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and placebo on obsessive symptoms as measured by the General Rating Scale obsessions subscale in adults with OCD (N = 1; n = 16; SMD = $0.50$ ; 95% C.I.,-1.50 to 0.50). I
11 Maudsley Obsessive Compu	lsive Inven	ntory
01 Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and placebo on obsessive-compulsive symptoms as measured by the Maudsley Obsessive Compulsive Inventory in adults with OCD ( $N = 1$ ; $n = 16$ ; SMD =-0.43; 95% C.I., -1.42 to 0.57). I
12 Obsessive-Compulsive Chec	klist	
01 Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and placebo on obsessive-compulsive symptoms as measured by the Obsessive-Compulsive Checklist in adults with OCD ( $N = 1$ ; $n = 16$ ; SMD =-0.43; 95% C.I.,-1.43 to 0.56). I
13 OCD Scale (CPRS)		
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on obsessive-compulsive symptoms as measured by the CPRS obsessive-compulsive subscale in adults with OCD (N = 1; n = 37; SMD = 0.30; 95% C.I., -0.35 to 0.95). I
14 Beck Depression Inventory		
01 Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and placebo on depressive symptoms as measured by the Beck Depression Inventory in adults with OCD (N = 1; n = 16; SMD =-0.33; 95% C.I.,-1.32 to 0.66). I
15 Hamilton Rating Scale for Depression	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and placebo on depression symptoms as measured by the Hamilton Rating Scale for Depression in adults with OCD (N = 2; n = 124; SMD = $-0.14$ ; 95% C.I., $-0.50$ to 0.21). I
16 Montgomery-Asberg Depression Rating Scale	s2x	There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing depressive symptoms as measured by the Montgomery-Asberg Depression Rating Scale in adults with OCD (N = 3; n = 608; SMD = -0.28; 95% C.I., -0.44 to -0.11). I
17 Hamilton Anxiety Scale	•	

04 El	T 4	
01 Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and placebo on anxiety symptoms as measured by the Hamilton Anxiety Rating Scale in adults with OCD (N = 1; n = 16; SMD =-0.75; 95% C.I.,-1.77 to 0.28). I
18 Clinical Global Impressions	,	
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on the severity of illness as measured by the Clinical Global Impressions scale in adults with OCD ( $N = 1$ ; $n = 37$ ; SMD = -0.10; 95% C.I., -0.74 to 0.55). I
19 Clinical Global Impressions:	severity	
01 Paroxetine	s2x	There is limited evidence suggesting a difference favouring paroxetine over placebo on reducing the severity of illness as measured by the CGI severity of illness subscale in adults with OCD ( $N = 1$ ; $n = 293$ ; $SMD = -0.36$ ; $95\%$ C.I., $-0.61$ to $-0.06$ ). I
20 Sheehan Disability Scale - fan	nily	
01 Citalopram	s2x	There is limited evidence suggesting a difference favouring citalopram over placebo on reducing impairment of family life as measured by the SDS family subscale in adults with OCD (N = 1; n = $203$ ; SMD = $-0.33$ ; 95% C.I., $-0.61$ to $-0.06$ ). I
21 Sheehan Disability Scale - soc	ial	
01 Citalopram	s2x	There is limited evidence suggesting a difference favouring citalopram over placebo on reducing impairment of social life as measured by the SDS social subscale in adults with OCD (N = 1; n = 203; SMD = -0.33; 95% C.I., -0.61 to -0.05). I
22 Sheehan Disability Scale - wo	rk	
01 Citalopram	s2x	There is limited evidence suggesting a difference favouring citalopram over placebo on reducing impairment of work as measured by the SDS work subscale in adults with OCD (N = 1; n = 203; SMD = -0.35; 95% C.I., -0.63 to -0.08). I
23 Symptom Checklist-90	•	
01 Paroxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on psychological distress as measured by the Symptom Checklist-90 in adults with OCD (N = 1; n = 246; SMD = -0.21; 95% C.I.,-0.48 to 0.06). I
Subanalysis: Y-BOCS in patients with comorbid depression	s s2	There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with comorbid depression (K = $4$ ; N = $489$ ; SMD = $-0.58$ ; $95\%$ C.I., $-0.76$ to $-0.4$ ). I
Subanalysis: Non-responders (OCD) (CGI or Y-BOCS) in patients with comorbid depression	s2	There is limited evidence suggesting a difference favouring SSRIs over placebo on the likelihood of treatment response, defined as a 25%+ or 35%+ reduction on the Y-BOCS and/or CGI "much improved" or "very much improved" in patients with comorbid depression (K = 3; N = 763; SMD = -0.73; 95% C.I., -0.66 to -0.79). I
02 SSRIs at different doses (acut	e phase)	
01 Adverse effects	ı	
01 Citalopram 40mg vs. Citalopram 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 40mg and 20mg of citalopram on the likelihood of reporting adverse effects in adults with OCD ( $N = 1$ ; $n = 200$ ; $RR = 0.94$ ; 95% C.I., 0.79 to 1.13). I

02 Citalopram 60mg vs. Citalopram 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 20mg of citalopram on the likelihood of reporting adverse effects in adults with OCD (N = 1; n = $202$ ; RR = $0.99$ ; 95% C.I., $0.84$ to $1.18$ ). I
03 Citalopram 60mg vs. Citalopram 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 40mg of citalopram on the likelihood of reporting adverse effects in adults with OCD (N = 1; n = 198; RR = 1.05; 95% C.I., 0.88 to 1.26). I
07 Sertraline rapid titration vs. slow titration	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between rapid dose sertraline and slow dose sertraline on the likelihood of reporting adverse effects in adults with OCD (N = 1; n = 32; RR = $1.04$ ; 95% C.I., 0.70 to 1.56). I
02 Leaving study early		•
01 Citalopram 40mg vs. Citalopram 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 40mg and 20mg of citalopram on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 200$ ; $RR = 0.98$ ; 95% C.I., 0.51 to 1.86)
02 Citalopram 60mg vs. Citalopram 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 20mg of citalopram on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 202$ ; $RR = 0.96$ ; 95% C.I., 0.50 to 1.83)
03 Citalopram 60mg vs. Citalopram 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 40mg of citalopram on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 198$ ; $RR = 0.98$ ; $95\%$ C.I., $0.51$ to $1.89$ )
04 Fluoxetine 40mg vs. Fluoxetine 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 40mg and 20mg of fluoxetine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 105$ ; $RR = 0.95$ ; 95% C.I., 0.49 to 1.81)
05 Fluoxetine 60mg vs. Fluoxetine 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 20mg of fluoxetine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 108$ ; $RR = 0.96$ ; 95% C.I., 0.51 to 1.82)
06 Fluoxetine 60mg vs. Fluoxetine 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 40mg of fluoxetine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 107$ ; $RR = 1.02$ ; 95% C.I., 0.53 to 1.96)
07 Paroxetine 40mg vs. 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 40mg and 20mg of paroxetine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 174$ ; $RR = 1.46$ ; 95% C.I., 0.79 to 2.70)
08 Paroxetine 60mg vs. 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 20mg of paroxetine on the likelihood of leaving the study early in adults with OCD (N = 1; n = 173; RR = 1.41; 95% C.I., 0.75 to 2.62)
09 Paroxetine 60mg vs. 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 40mg of paroxetine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 171$ ; $RR = 0.96$ ; 95% C.I., 0.55 to 1.67)

10 Sertraline 100mg vs. Sertraline 50mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 100mg and 50mg of sertraline on the likelihood of leaving the study early in adults with OCD (N = 1; n = 161; RR = 1.57; 95% C.I., 0.93 to 2.64)
11 Sertraline 200mg vs. Sertraline 50mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 200mg and 50mg of sertraline on the likelihood of leaving the study early in adults with OCD (N = 1; n = 160; RR = 1.24; 95% C.I., 0.71 to 2.16)
12 Sertraline 200mg vs. Sertraline 100mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 200mg and 100mg of sertraline on the likelihood of leaving the study early in adults with OCD (N = 1; n = 161; RR = 0.79; 95% C.I., 0.49 to 1.27)
13 Sertraline rapid titration vs. Sertraline slow titration	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between rapid dose sertraline and slow dose sertraline on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 32$ ; $RR = 1.32$ ; $95\%$ C.I., 0.25 to 6.88)
03 Leaving study early due to a	dverse eve	
01 Citalopram 40mg vs. Citalopram 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 40mg and 20mg of citalopram on the likelihood of leaving the study early due to adverse events in adults with OCD (N = 1; n = 200; RR = 1.56; 95% C.I., 0.45 to 5.36)
02 Citalopram 60mg vs. Citalopram 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 20mg of citalopram on the likelihood of leaving the study early due to adverse effects in adults with OCD ( $N = 1$ ; $n = 202$ ; $RR = 1.02$ ; $95\%$ C.I., 0.26 to 3.97)
03 Citalopram 60mg vs. Citalopram 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 40mg of citalopram on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 198; RR = 0.65; 95% C.I., 0.19 to 2.24)
04 Fluoxetine 40mg vs. Fluoxetine 20mg	s1x	There is evidence suggesting a difference favouring 40mg of fluoxetine over 20mg of fluoxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 2; n = 281; RR = $0.40$ ; 95% C.I., $0.23$ to $0.71$ )
05 Fluoxetine 60mg vs. Fluoxetine 20mg	s2x	There is limited evidence suggesting a difference favouring 60mg of fluoxetine over 20mg of fluoxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 2; n = 285; RR = 0.51; 95% C.I., 0.31 to 0.85)
06 Fluoxetine 60mg vs. Fluoxetine 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 40mg of fluoxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = $2$ ; n = $286$ ; RR = $1.27$ ; 95% C.I., 0.66 to $2.41$ )
07 Paroxetine 40mg vs. 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 40mg and 20mg of paroxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 174; RR = 0.91; 95% C.I., 0.37 to 2.25)

08 Paroxetine 60mg vs. 20mg	s4	The evidence is inconclusive and so it is not possible to determine if
		there is a clinically important difference between 60mg and 20mg of paroxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD ( $N = 1$ ; $n = 173$ ; $RR = 1.38$ ; 95%
09 Paroxetine 60mg vs. 40mg	s4	C.I., 0.61 to 3.11)  The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 40mg of paroxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 171; RR = 1.52; 95% C.I., 0.65 to 3.53)
10 Sertraline 100mg vs. Sertraline 50mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 100mg and 50mg of sertraline on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 161; RR = 1.81; 95% C.I., 0.70 to $4.66$ )
11 Sertraline 200mg vs. Sertraline 50mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 200mg and 50mg of sertraline on the likelihood of leaving the study early due to adverse effects in adults with OCD ( $N = 1$ ; $n = 160$ ; $RR = 1.00$ ; 95% C.I., 0.34 to 2.97)
12 Sertraline 200mg vs. Sertraline 100mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 200mg and 100mg of sertraline on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 161; RR = 0.55; 95% C.I., 0.21 to 1.42)
13 Sertraline rapid titration vs. Sertraline slow titration	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between rapid-dose sertraline and slow-dose sertraline on the likelihood of leaving the study early due to adverse effects in adults with OCD ( $N = 1$ ; $n = 32$ ; $RR = 0.88$ ; 95% C.I., 0.14 to 5.52)
04 Non-responders	1	/
01 Citalopram 40mg vs. Citalopram 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 40mg and 20mg of citalopram on the likelihood of treatment response, defined as a 25% or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = $200$ ; RR = $1.11$ ; 95% C.I., $0.82$ to $1.51$ )
02 Citalopram 60mg vs. Citalopram 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 20mg of citalopram on the likelihood of treatment response, defined as a 25% or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = $202$ ; RR = $0.81$ ; 95% C.I., $0.57$ to $1.15$ )
03 Citalopram 60mg vs. Citalopram 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 40mg of citalopram on the likelihood of treatment response, defined as achieving a 25% or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = 198; RR = 0.73; 95% C.I., 0.52 to 1.02)

04 Fluoxetine 40mg vs. Fluoxetine 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 40mg and 20mg of fluoxetine on the likelihood of treatment response in adults with OCD (N = $2$ ; n = $281$ ; RR = $0.96$ ; $95\%$ C.I., $0.83$ to $1.12$ )
05 Fluoxetine 60mg vs. Fluoxetine 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 40mg and 20mg of fluoxetine on the likelihood of treatment response in adults with OCD (N = 2; n = 285; RR = 0.93; 95% C.I., 0.79 to 1.08)
06 Fluoxetine 60mg vs. Fluoxetine 40mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 40mg of fluoxetine on the likelihood of treatment response in adults with OCD (N = 2; n = 286; RR = 0.96; 95% C.I., 0.82 to 1.13)
07 Paroxetine 40mg vs. Paroxetine 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 40mg and 20mg of paroxetine on the likelihood of treatment response in adults with OCD (N = 1; n = 174; RR = $0.84$ ; 95% C.I., $0.67$ to $1.05$ )
08 Paroxetine 60mg vs. Paroxetine 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 20mg of paroxetine on the likelihood of treatment response in adults with OCD (N = 1; n = 173; RR = 0.80; 95% C.I., 0.64 to 1.01)
09 Paroxetine 60mg vs. Paroxetine 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 40mg of paroxetine on the likelihood of treatment response in adults with OCD (N = 1; n = 171; RR = 0.95; 95% C.I., 0.74 to 1.23)
05 Y-BOCS		
01 Citalopram 40mg vs. Citalopram 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 40mg and 20mg of citalopram on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 1$ ; $n = 200$ ; $SMD = -0.07$ ; $95\%$ C.I., $-0.35$ to $0.21$ )
02 Citalopram 60mg vs. Citalopram 20mg	s2	There is limited evidence suggesting a difference favouring 60mg of citalopram over 20mg of citalopram on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 1$ ; $n = 202$ ; $SMD = -0.28$ ; $95\%$ C.I., $-0.56$ to $0.00$ )
03 Citalopram 60mg vs. Citalopram 40mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 40mg of citalopram on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 1$ ; $n = 198$ ; $SMD = -0.22$ ; $95\%$ C.I., $-0.49$ to $0.06$ )
04 Fluoxetine 40mg vs. Fluoxetine 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 40mg and 20mg of fluoxetine on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 2$ ; $n = 246$ ; $SMD = -0.03$ ; $95\%$ C.I., $-0.28$ to $0.22$ )
05 Fluoxetine 60mg vs. Fluoxetine 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 20mg of fluoxetine on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = 2; n = 249; SMD = -0.21; 95% C.I., -0.46 to 0.04)

06 Fluoxetine 60mg vs. Fluoxetine 40mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 40mg of fluoxetine on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 2$ ; $n = 241$ ; $SMD = -0.18$ ; $95\%$ C.I., $-0.43$ to $0.08$ )
07 Sertraline rapid titration vs. Sertraline slow titration	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between rapid-dose sertraline and slow-dose sertraline on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = 1; n = 27; SMD = -0.28; 95% C.I., -1.04 to 0.48)
06 NIMH-OC		
01 Citalopram 40mg vs. Citalopram 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 40mg and 20mg of citalopram on obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = 1; n = 200; SMD = $-0.04$ ; 95% C.I., $-0.31$ to 0.24)
02 Citalopram 60mg vs. Citalopram 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 20mg of citalopram on obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD ( $N = 1$ ; $n = 202$ ; $SMD = -0.12$ ; $95\%$ C.I., $-0.39$ to $0.16$ )
03 Citalopram 60mg vs. Citalopram 40mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 40mg of citalopram on obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = 1; n = 198; SMD = $-0.07$ ; 95% C.I., $-0.35$ to $0.20$ )
07 Hamilton Rating Scale for De	epression	
01 Fluoxetine 40mg vs. Fluoxetine 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 40mg and 20mg of fluoxetine on depressive symptoms as measured by the Hamilton Rating Scale for Depression in adults with OCD (N = 1; n = $104$ ; SMD = $-0.11$ ; 95% C.I., $-0.49$ to $0.28$ )
02 Fluoxetine 60mg vs. Fluoxetine 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 20mg of fluoxetine on depressive symptoms as measured by the Hamilton Rating Scale for Depression in adults with OCD ( $N = 1$ ; $n = 106$ ; SMD = -0.25; 95% C.I., -0.63 to 0.13)
03 Fluoxetine 60mg vs. Fluoxetine 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60mg and 40mg of fluoxetine on depressive symptoms as measured by the Hamilton Rating Scale for Depression in adults with OCD ( $N = 1$ ; $n = 106$ ; SMD = -0.13; 95% C.I., -0.51 to 0.25)
08 Montgomery-Asberg Depres	sion Rating	Scale
01 Citalopram 40mg vs. Citalopram 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 40mg and 20mg of citalopram on depressive symptoms as measured by the Montgomery-Asberg Depression Rating Scale in adults with OCD (N = 1; n = 200; SMD = $-0.09$ ; 95% C.I., $-0.36$ to $0.19$ )
02 Citalopram 60mg vs. Citalopram 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 20mg of citalopram on depressive symptoms as measured by the Montgomery-Asberg Depression Rating Scale in adults with OCD (N = 1; n = 202; SMD = -0.09; 95% C.I., -0.37 to 0.18)
03 Citalopram 60mg vs. Citalopram 40mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 40mg of citalopram on depressive symptoms as measured by the Montgomery-Asberg Depression Rating Scale in adults with OCD (N = 1; n = 198; SMD = -0.02; 95% C.I., -0.30 to 0.26)

04 Fluoxetine 40mg vs.	s4	The evidence is inconclusive and so it is not necesible to determine if
Fluoxetine 20mg	54	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 40mg and 20mg of
Tuoxetine zonig		fluoxetine on depressive symptoms as measured by the
		Montgomery-Asberg Depression Rating Scale in adults with OCD (N
		= 1; n = 104; SMD = -0.27; 95% C.I., -0.66 to 0.11)
05 Fluoxetine 60mg vs.	s4	The evidence is inconclusive and so it is not possible to determine if
Fluoxetine 20mg		there is a clinically important difference between 60mg and 20mg of
		fluoxetine on depressive symptoms as measured by the
		Montgomery-Asberg Depression Rating Scale in adults with OCD (N
		= 1; n = 106; SMD = -0.26; 95% C.I., -0.65 to 0.12)
06 Fluoxetine 60mg vs.	s3	There is evidence suggesting that there is unlikely to be a clinically
Fluoxetine 40mg		important difference between 60mg and 40mg of fluoxetine on
		depressive symptoms as measured by the Montgomery-Asberg
		Depression Rating Scale in adults with OCD (N = 1; n = 106; SMD =
09 Sheehan Disability Score	- family	0.00; 95% C.I., -0.38 to 0.38)
01 Citalopram 40mg vs.	s3	There is evidence suggesting that there is unlikely to be a clinically
Citalopram 20mg	83	There is evidence suggesting that there is unlikely to be a clinically important difference between 40mg and 20mg of citalopram on
Charopiani Zonig		impairment of family life as measured by the SDS family subscale in
		adults with OCD (N = 1; n = 200; SMD = -0.13; 95% C.I., -0.40 to 0.15)
02 Citalopram 60mg vs.	s3	There is evidence suggesting that there is unlikely to be a clinically
Citalopram 20mg	33	important difference between 60mg and 20mg of citalopram on
Chareprant 2011g		impairment of family life as measured by the SDS family subscale in
		adults with OCD (N = 1; n = 202; SMD = -0.21; 95% C.I., -0.48 to 0.07)
03 Citalopram 60mg vs.	s3	There is evidence suggesting that there is unlikely to be a clinically
Citalopram 40mg		important difference between 60mg and 40mg of citalopram on
		impairment of family life as measured by the SDS family subscale in
		adults with OCD (N = 1; n = 198; SMD = -0.08; 95% C.I., -0.36 to 0.19)
10 Sheehan Disability Score	- social	
01 Citalopram 40mg vs.	s3	There is evidence suggesting that there is unlikely to be a clinically
Citalopram 20mg		important difference between 40mg and 20mg of citalopram on
		impairment of social life as measured by the SDS social subscale in
		adults with OCD (N = 1; n = 200; SMD = 0.04; 95% C.I., -0.23 to 0.32)
02 Citalopram 60mg vs.	s3	There is evidence suggesting that there is unlikely to be a clinically
Citalopram 20mg		important difference between 60mg and 20mg of citalopram on
		impairment of social life as measured by the SDS social subscale in
		adults with OCD (N = 1; n = 202; SMD = -0.17; 95% C.I., -0.45 to 0.11)
03 Citalopram 60mg vs.	s3	There is evidence suggesting that there is unlikely to be a clinically
Citalopram 40mg		important difference between 60mg and 40mg of citalopram on
		impairment of social life as measured by the SDS social subscale in
11.01 1 D: 1.11: 0	1	adults with OCD (N = 1; n = 198; SMD = -0.21; 95% C.I., -0.49 to 0.07)
11 Sheehan Disability Score		
01 Citalopram 40mg vs.	s3	There is evidence suggesting that there is unlikely to be a clinically
Citalopram 20mg		important difference between 40mg and 20mg of citalopram on
		impairment of work as measured by the SDS work subscale in adults with OCD ( $N = 1$ ; $p = 200$ ; SMD = $0.08$ ; $95\%$ C L $0.36$ to $0.19$ )
02 Citalogram 60mg vo	62	with OCD (N = 1; n = 200; SMD = -0.08; 95% C.I., -0.36 to 0.19)  There is evidence suggesting that there is unlikely to be a clinically
1	53	
Charopiani Zonig		
03 Citalonram 60mg vs	c3	·
1	30	
Cimiopium iomg		
		adults with OCD (N = 1; n = 198; SMD = 0.04; 95% C.I., -0.24 to 0.32)
02 Citalopram 60mg vs. Citalopram 20mg  03 Citalopram 60mg vs. Citalopram 40mg	s3 s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 20mg of citalopram on impairment of work as measured by the SDS work subscale in adu with OCD (N = 1; n = 202; SMD = -0.04; 95% C.I., -0.32 to 0.24)  There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 40mg of citalopram on impairment of work as measured by the SDS work subscale in adults with OCD (N = 1; n = 198; SMD = 0.04; 95% C.I., -0.24 to 0.32)

03 SSRIs vs. other SSRIs (acute policy of Leaving study early of Fluoxetine vs. Sertraline of Leaving study early due to a sertraline vs. Sertraline of Fluoxetine vs. Sertraline	s4 dverse effe s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and sertraline on the likelihood of leaving the study early in adults with OCD (N = 1; n = 150; RR = 1.05; 95% C.I., 0.64 to 1.73)  ects  The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and sertraline on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 150; RR = 0.70; 95% C.I., 0.34 to 1.46)
03 Fluoxetine vs. Sertraline 02 Leaving study early due to a	dverse effo	there is a clinically important difference between fluoxetine and sertraline on the likelihood of leaving the study early in adults with OCD (N = 1; n = 150; RR = 1.05; 95% C.I., 0.64 to 1.73) $\stackrel{\text{ects}}{}$ The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and sertraline on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 150; RR = 0.70; 95% C.I., 0.34 to
	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and sertraline on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 150; RR = 0.70; 95% C.I., 0.34 to
03 Fluoxetine vs. Sertraline		there is a clinically important difference between fluoxetine and sertraline on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 150; RR = 0.70; 95% C.I., 0.34 to
	1.	
03 Non-responders	4	
01 Citalopram vs. Paroxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between citalopram and paroxetine on the likelihood of treatment response, defined as a 35% or greater reduction on the Y-BOCS and a score of 3 or less on the CGI global improvement subscale, in adults with OCD (N = 1; n = 20; RR = 1.15; 95% C.I., 0.55 to 2.39)
04 Fluvoxamine vs. Paroxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and paroxetine on the likelihood of treatment response, defined as a 35% or greater reduction on the Y-BOCS and a score of 3 or less on the CGI global improvement subscale, in adults with OCD (N = 1; n = 19; RR = 0.72; 95% C.I., 0.28 to 1.88)
04 Non-remitters	•	•
01 Fluoxetine vs. Sertraline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and sertraline on the likelihood of remission, defined as a score of 11 or less on the Y-BOCS, in adults with OCD ( $N = 1$ ; $n = 150$ ; $RR = 1.2$ ; 95% C.I., 0.98 to 1.47)
05 Y-BOCS	<u> </u>	/
02 Fluoxetine vs. Sertraline	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between fluoxetine and sertraline on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 1$ ; $n = 148$ ; SMD = -0.01; C.I., -0.34 to 0.31)
03 Fluvoxamine vs. Citalopram	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and citalopram on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = 1; n = 21; SMD = $-0.36$ ; 95% C.I., $-1.23$ to $0.50$ )
04 Fluvoxamine vs. Paroxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and paroxetine on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = 1; n = 19; SMD = $-0.62$ ; 95% C.I., $-1.55$ to $0.31$ )
05 Paroxetine vs. Citalopram 06 NIMH-OC	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and citalopram on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = 1; n = 20; SMD = 0.19; 95% C.I., - 0.69 to 1.07)

01 Fluoxetine vs. Sertraline	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between fluoxetine and sertraline on obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = 1; n = 148; SMD = $0.07$ ; 95% C.I., $-0.25$ to $0.40$ )
02 Fluvoxamine vs. Citalopram	s2x	There is limited evidence suggesting a difference favouring fluvoxamine over citalopram on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = 1; n = 21; SMD = $-1.22$ ; 95% C.I., $-2.17$ to $-0.27$ )
03 Fluvoxamine vs. Paroxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine and paroxetine on obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = 1; n = 19; SMD = $-0.77$ ; 95% C.I., $-1.71$ to $0.17$ )
04 Paroxetine vs. Citalopram	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and citalopram on obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD ( $N = 1$ ; $n = 20$ ; SMD = -0.40; 95% C.I., -1.29 to 0.49)
07 Hamilton Rating Scale for De	pression	
01 Fluoxetine vs. Sertraline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and sertraline on depressive symptoms as measured by the Hamilton Rating Scale for Depression in adults with OCD (N = 1; n = 148; SMD = $0.18$ ; 95% C.I., $-0.14$ to $0.50$ )
04 SSRIs vs. Clomipramine (acu	te phase)	
01 Adverse events	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on the likelihood of reporting adverse events in adults with OCD (N = $7$ ; n = $1037$ ; RR = $0.95$ ; $95\%$ C.I., $0.89$ to $1$ )
02 Attempted suicide		,
01 Sertraline vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between sertraline and clomipramine on the likelihood of attempting suicide in adults with OCD ( $N = 1$ ; $n = 168$ ; $RR = 0.32$ ; $95\%$ C.I., $0.03$ to $2.99$ )
03 Leaving study early	s2x	There is limited evidence suggesting a difference favouring SSRIs over clomipramine on the likelihood of leaving the study early in adults with (N = $10$ ; n = $1139$ ; RR = $0.72$ ; $95\%$ C.I., $0.59$ to $0.88$ )
04 Leaving study early due to adverse events	s2x	There is limited evidence suggesting a difference favouring SSRIs over clomipramine on the likelihood of leaving the study early due to adverse events in adults with OCD (N = 8; n = 1095; RR = 0.62; 95% C.I., 0.46 to 0.84)
05 Non-responders (OCD)	s3	There is evidence suggesting there is unlikely to be a clinically important difference between SSRIs and clomipramine on treatment response (OCD) ( $N = 9$ ; $n = 1019$ ; $RR = 1.02$ ; $95\%$ C.I., $0.89$ to $1.17$ )
06 Y-BOCS	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between fluoxetine and clomipramine on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 7$ ; $n = 739$ ; SMD = 0.14; 95% C.I., -0.01 to 0.29)

07 NIMH-OC	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 3$ ; $n = 666$ ; SMD = 0.08; 95% C.I., -0.08 to 0.23)
08 Comprehensive Psychopathol	logical Ratir	
01 Fluoxetine vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and clomipramine on obsessive-compulsive symptoms as measured by the CPRS obsessive-compulsive subscale in adults with OCD (N = 1; n = 54; SMD = $0.37$ ; 95% C.I., $-0.17$ to $0.91$ )
09 Hamilton Rating Scale for Depression	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on depressive symptoms as measured by the Hamilton Rating Scale for Depression in adults with OCD (N = 3; n = 344; SMD = $0.14$ ; 95% C.I., - $0.07$ to $0.35$ )
10 Montgomery-Asberg Depression Rating Scale	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on depressive symptoms as measured by the Montgomery-Asberg Depression Rating Scale in adults with OCD (N = 3; n = 356; SMD = $-0.03$ ; 95% C.I., $-0.25$ to $0.19$ )
11 Depression (HRSD; MADRS)	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on depressive symptoms as measured by the HRSD or MADRS in adults with OCD $(N = 4; n = 592; SMD = 0.01; 95\% C.I., -0.16 to 0.17)$
12 Clinical Anxiety Scale		
01 Fluvoxamine vs. Clomipramine	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between fluvoxamine and clomipramine on anxiety symptoms as measured by the Clinical Anxiety Scale in adults with OCD ( $N = 1$ ; $n = 217$ ; SMD = 0.07; 95% C.I., -0.20 to 0.33)
13 Covi Anxiety Scale	•	
01 Fluoxetine vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and clomipramine on anxiety symptoms as measured by the Covi Anxiety Scale in adults with OCD ( $N = 1$ ; $n = 54$ ; SMD = 0.0; 95% C.I., -0.54 to 0.54)
14 Anxiety (Clinical Anxiety Scale; Covi Anxiety Scale)	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on anxiety symptoms as measured by the Clinical Anxiety Scale or Covi Anxiety Scale in adults with OCD (N = 2; n = 271; SMD = $0.05$ ; 95% C.I., - $0.18$ to $0.29$ )
15 Clinical Global Impresssions: global improvement	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on global improvement of illness as measured by the CGI global improvement subscale in adults with OCD (N = $2$ ; n = $271$ ; SMD = $0.00$ ; C.I., - $0.24$ to $0.24$ )
16 Clinical Global Impression: severity of illness *HETEROGENEITY*	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on the severity of illness as measured by the CGI severity of illness subscale in adults with OCD (N = 3; n = 559; SMD = $0.11$ ; 95% C.I., - $0.06$ to $0.28$ )

17 Symptom Checklist -90			
01 Paroxetine vs. Clomipramine	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between paroxetine and clomipramine on psychological distress as measured by the Symptom Checklist-90 in adults with OCD ( $N = 1$ ; $n = 243$ ; SMD = 0.06; C.I., -0.21 to 0.34)	
Subanalysis: Non-responders (OCD) (CGI or Y-BOCS) in patients with comorbid depression	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SSRIs and clomipramine on treatment response as measured by the CGI or Y-BOCS in adults with OCD with comorbid depression ( $N = 2$ ; $n = 282$ ; $RR = 1.16, 95\%$ C.I., $0.88$ to $1.51$ )	
Subanalysis: Y-BOCS in patients with comorbid depression	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on OCD symptoms (Y-BOCS) in patients with comorbid depression (K = $3$ ; N = $192$ ; SMD = $0.22$ ; $95\%$ CI, $-0.06$ to $0.51$ ). I	
Subanalysis: Hamilton Rating Scale for Depression in patients with comorbid depression	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on depressive symptoms (HDRS) in patients with comorbid depression (K = $2$ ; N = $141$ ; SMD = $0.16$ ; $95\%$ CI, $-0.08$ to $0.4$ ). I	
05 SSRIs/Clomipramine vs. non-	-SRIs (acute	phase)	
01 Adverse effects			
01 Sertraline vs. Desipramine	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and desipramine on the likelihood of reporting adverse effects in adults with OCD (N = 1; n = $166$ ; RR = $1.01$ ; $95\%$ C.I., $0.97$ to $1.05$ ). I	
02 Leaving study early due to adverse events	s2x	There is limited evidence suggesting a difference favouring SSRIs/Clomipramine over non-SRIs on the likelihood of leaving the study early due to adverse events in adults with OCD ( $N = 5$ ; $n = 279$ ; $RR = 0.51$ , $95\%$ C.I., $0.28$ to $0.95$ ). I	
03 Non-responders (OCD)	I.		
• , , ,	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between sertraline and desipramine on the likelihood of treatment response, defined as $40\%$ or greater reduction on the Y-BOCS in adults with OCD (N = 1; n = $166$ ; RR = $1.13$ ; $95\%$ C.I., $0.83$ to $1.54$ ). I	
04 Y-BOCS	s2x	There is limited evidence suggesting a difference favouring SSRIs/Clomipramine over non-SRIs on reducing obsessive-compulsive symptoms, as measured by the Y-BOCS in adults with OCD (N =4; n = 258; SMD = $-0.3$ ; 95% C.I., $-0.54$ to $-0.05$ ). I	
05 NIMH-OC	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SSRIs/Clomipramine and non-SRIs on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = $3$ n = $218$ ; SMD = $-0.22$ ; $95\%$ C.I., $-0.49$ to $-0.05$ ). I	
06 SSRIs vs. placebo (continuation phase)			
01 Leaving study early	s2x	There is limited evidence suggesting a difference favouring the continuation of SSRIs over placebo on the likelihood of leaving the study early in adults with OCD (N = $4$ ; n = $278$ ; RR = $0.61$ ; 95% C.I., $0.46$ to $0.81$ )	

02 Leaving study early due to	s1x	There is evidence suggesting a difference favouring the continuation
adverse events		of SSRIs over placebo on the likelihood of leaving the study early due to adverse events in adults with OCD (N = 3; n = 249; RR = 0.26; 95% C.I., 0.11 to 0.63)
03 Non-responders		
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and placebo on the likelihood of treatment response, defined as a 35% or greater reduction on the Y-BOCS, in adults with OCD ( $N = 1$ ; $n = 29$ ; $RR = 1.22$ ; $95\%$ C.I., $0.51$ to $2.89$ )
04 Y-BOCS		
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and placebo on obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 2$ ; $n = 98$ ; SMD = -0.08; 95% C.I., -0.50 to 0.34)
05 Hamilton Rating Scale for De	epression	
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and placebo on depressive symptoms as measured by the Hamilton Rating Scale for Depression in adults with OCD ( $N = 1$ ; $n = 69$ ; SMD = -0.11; 95% C.I., -0.58 to 0.36)
06 SF-36: social functioning		., , ,
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and placebo on social functioning as measured by the SF-36 social functioning subscale in adults with OCD (N = 1; n = 68; SMD = $0.15$ ; 95% C.I., $-0.32$ to $0.63$ )
07 SSRIs vs. placebo (discontinu	ation phase	
01 Death		
01 Sertraline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation and discontinuation of sertraline on the likelihood of death in adults with OCD ( $N = 1$ ; $n = 223$ ; $RR = 0.35$ ; 95% C.I., 0.01 to 8.46)
02 Leaving study early		
01 Sertraline	s2x	There is limited evidence suggesting a difference favouring the continuation of sertraline over discontinuation on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 223$ ; $RR = 0.58$ ; 95% C.I., 0.42 to 0.82)
03 Leaving study early due to a	dverse effect	s
01 Sertraline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation and discontinuation of sertraline on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 223; RR = $0.44$ ; 95% C.I., $0.16$ to $1.20$ )
04 Relapse	1	
01 Sertraline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation and discontinuation of sertraline on the likelihood of relapse, defined as a Y-BOCS increase by 5 points, Y-BOCS total score>=20 and CGI increase by 1 point, in adults with OCD (N = 1; n = 223; RR = 0.70; 95% C.I., 0.20 to 2.40)

05 Y-BOCS		
01 Sertraline	s2x	There is limited evidence suggesting a difference favouring the continuation of sertraline over discontinuation on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 1$ ; $n = 221$ ; $SMD = -0.37$ ; $95\%$ C.I., $-0.63$ to $-0.10$ )
06 NIMH-OC		
01 Sertraline	s2x	There is limited evidence suggesting a difference favouring the continuation of sertraline over discontinuation on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = 1; n = 221; SMD = $-0.53$ ; 95% C.I., $-0.80$ to $-0.26$ )
07 Qol Enjoyment and Satisfac	tion	
01 Sertraline	s2x	There is limited evidence suggesting a difference favouring the continuation of sertraline over discontinuation on improving quality of life as measured by the Quality of Life Enjoyment and Satisfaction scale in adults with OCD (N = 1; n = 215; SMD = -0.53; 95% C.I., -0.80 to $-0.26$ )
08 SSRIs at different doses (co	ntinuation	phase)
01 Leaving study early		
01 40mg Fluoxetine v 20mg Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of $40 \text{mg}$ of fluoxetine and $20 \text{mg}$ of fluoxetine on the likelihood of leaving the study early in adults with OCD (N = 2; n = 82; RR = 1.47; 95% C.I., 0.68 to 3.19)
02 60mg Fluoxetine v 20mg	s4	The evidence is inconclusive and so it is not possible to determine if
Fluoxetine		there is a clinically important difference between the continuation of 60mg of fluoxetine and 20mg of fluoxetine on the likelihood of leaving the study early in adults with OCD (N = 2; n = 90; RR = $0.82$ ; 95% C.I., $0.34$ to $1.97$ )
03 60mg Fluoxetine v 40mg Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of 60mg of fluoxetine and 40mg of fluoxetine on the likelihood of leaving the study early in adults with OCD (N = 2; n = 90; RR = 0.56; 95% C.I., 0.26 to 1.24)
02 Leaving study early due to	adverse ef	fects
01 40mg Fluoxetine v 20mg Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of 40mg of fluoxetine and 20mg of fluoxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 38; RR = 2.71; 95% C.I., 0.12 to 62.70)
02 60mg Fluoxetine v 20mg Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of 60mg of fluoxetine and 20mg of fluoxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; $n = 41$ ; $RR = 2.38$ ; 95% C.I., 0.10 to 55.06)
03 60mg Fluoxetine v 40mg Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of 60mg of fluoxetine and 40mg of fluoxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 43; RR = 0.87; 95% C.I., 0.06 to 13.02)
03 Non-responders	1	,

01 Fluoxetine 40mg v Fluoxetine 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of $40 \text{mg}$ of fluoxetine and $20 \text{mg}$ of fluoxetine on the likelihood of treatment response, defined as $35\%$ or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = $44$ ; RR = $0.94$ ; $95\%$ C.I., $0.57$ to $1.54$ )
02 Fluoxetine 60mg v Fluoxetine 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of 60mg of fluoxetine and 20mg of fluoxetine on the likelihood of treatment response, defined as 35% or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = 49; RR = 0.7; 95% C.I., 0.4 to 1.21)
03 Fluoxetine 60mg v Fluoxetine 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of 60mg of fluoxetine and 40mg of fluoxetine on the likelihood of treatment response, defined as 35% or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = 47; RR = 0.74; 95% C.I., 0.41 to 1.32)
04 Y-BOCS		
01 Fluoxetine 40mg v Fluoxetine 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of 40mg of fluoxetine and 20mg of fluoxetine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 1$ ; $n = 44$ ; $SMD = -0.06$ ; $95\%$ C.I., $-0.65$ to $0.53$ )
02 Fluoxetine 60mg v Fluoxetine 20mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of 60mg of fluoxetine and 20mg of fluoxetine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 1$ ; $n = 49$ ; $SMD = -0.43$ ; $95\%$ C.I., $-1.00$ to $0.13$ )
03 Fluoxetine 60mg v Fluoxetine 40mg	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of 60mg of fluoxetine and 40mg of fluoxetine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 1$ ; $n = 47$ ; $SMD = -0.42$ ; $95\%$ C.I., $-1.00$ to $0.17$ )
09 SSRIs v Clomipramine (contir	nuation phas	e)
01 Leaving study early		
01 Fluoxetine 60mg vs. Clomipramine 200mg (non- responders)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and clomipramine on the likelihood of leaving the study early in adults with OCD who failed to respond to acute treatment with the same drug ( $N = 1$ ; $n = 22$ ; $RR = 1.14$ ; 95% C.I., 0.39 to 3.36)
02 Fluoxetine 20mg vs. Clomipramine 100mg (responders)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and clomipramine on the likelihood of leaving the study early in adults with OCD who responded to acute treatment with the same drug ( $N = 1$ ; $n = 24$ ; $RR = 0.59$ ; $95\%$ C.I., $0.19$ to $1.83$ )
02 Leaving study early due to ad	verse effects	
01 Fluoxetine 60mg vs. Clomipramine 200mg (non- responders)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and clomipramine on the likelihood of leaving the study early due to adverse effects in adults with OCD who failed to respond to acute treatment with the same drug ( $N = 1$ ; $n = 22$ ; $RR = 3.00$ ; 95% C.I., 0.16 to 55.72)

02 Fluoxetine 20mg vs. Clomipramine 100mg (responders)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and clomipramine on the likelihood of leaving the study early due to adverse effects in adults with OCD who responded to acute treatment with the same drug ( $N = 1$ ; $n = 24$ ; $RR = 1.18$ ; 95% C.I., 0.08 to 16.78)
12 Sertraline standard dose vs. h	· ·	1 /
01 Leaving study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of a standard dose of sertraline and a high dose of sertraline on the likelihood of leaving the study early in adults with OCD who failed to respond to previous treatment with the drug ( $N = 1$ ; $n = 66$ ; $RR = 0.56$ ; $95\%$ C.I., $0.22$ to $1.38$ )
02 Leaving study early due to adverse events	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of a standard dose of sertraline and a high dose of sertraline on the likelihood of leaving the study early due to adverse effects in adults with OCD who failed to respond to previous treatment with the drug $(N = 1; n = 66; RR = 4.19; 95\% C.I., 0.21 to 84.03)$
03 Non-responder	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of a standard dose of sertraline and a high dose of sertraline on the likelihood of treatment response, defined as a 25% or greater reduction in Y-BOCS, in adults with OCD who failed to respond to previous treatment with the drug ( $N = 1$ ; $n = 66$ ; $RR = 1.11$ ; 95% C.I., 0.77 to 1.61)

## Clomipramine (adults)

Description	Statement level	Statement and Statistics
01 Clomipramine vs. placebo: a	cute phase	
01 Adverse events *HETEROGENEITY*	s1y	There is evidence suggesting a difference favouring placebo over clomipramine on the likelihood of reporting adverse events in adults with OCD (N = 3; n = 877; RR = $1.36$ ; 95% CI, $1.27$ to $1.45$ )
02 Leaving study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on the likelihood of leaving the study early in adults with OCD ( $N = 5$ ; $n = 921$ ; $RR = 1.15$ ; $95\%$ CI, $0.9$ to $1.47$ )
03 Leaving study early due to adverse events	s1y	There is evidence suggesting a difference favouring placebo over clomipramine on the likelihood of leaving the study early due to adverse events in adults with OCD (N = 2; n = 357; RR = 2.35; 95% CI, 1.31 to 4.22)
04 Non-responders (Y-BOCS)	s2x	There is limited evidence suggesting a difference favouring clomipramine over placebo on the likelihood of achieving response, defined as a 25% or greater reduction on the Y-BOCS, in adults with OCD (N = 2; n = 357; RR = 0.79; 95% CI, 0.66 to 0.96)

05 Non-responders (CGI)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on the likelihood of achieving response, defined as "much improved" or "very much improved" on the CGI scale, in adults with OCD ( $N=1; n=44; RR=0.9; 95\%$ C.I., 0.61 to 1.33)
06 Non-responders (all)	s2x	There is limited evidence suggesting a difference favouring clomipramine over placebo on the likelihood of achieving response, defined as a 25% or greater reduction on the Y-BOCS or as "much improved" or "very much improved" on the CGI scale, in adults with OCD (N = 3; n = 401; RR = 0.81; 95% CI, 0.68 to 0.96)
07 Non-remitters	s1x	There is evidence suggesting a difference favouring clomipramine over placebo on the likelihood of achieving remission, defined as a score of 1 to 6 on the NIMH-OC, in adults with OCD (N = 1; n = 520; $RR = 0.54$ ; 95% C.I., 0.48 to 0.61)
08 Y-BOCS HETEROGENEITY therefore down-graded to s2*	s2x	There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing OCD symptoms as measured by the Y-BOCS in adults with OCD (N = 4; n = 487; SMD = -0.93; 95% C.I., -1.07 to -0.78)
09 NIMH-OC *HETEROGENEITY therefore down-graded to s2*	s2x	There is evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = $4$ ; n = $847$ ; SMD = $-0.91$ ; $95\%$ CI, $-1.05$ to $-0.77$ )
10 Leyton Obsessional Inventory – symptom	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory – symptom subscale in adults with OCD (N=1, n=16, SMD = -0.46; 95% C.I., -1.46 to 0.54)
11 Leyton Obsessional Inventory – trait	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory – trait subscale in adults with OCD (N=1, n=16, SMD = -0.50, 95% C.I., -1.49 to 0.50)
12 Leyton Obsessional Inventory – resistance	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory – resistance subscale in adults with OCD (N=1, n=16, SMD = -0.43, 95% C.I., -1.43 to 0.56)
13 Leyton Obsessional Inventory – interference	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory – interference subscale in adults with OCD (N=1, n=16, SMD = -0.18, 95% C.I., -1.16 to 0.80)
14 OCD Scale (CPRS)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on reducing obsessive-compulsive symptoms as measured by the Comprehensive Psychopathological Rating obsessive-compulsive subscale in adults with OCD (N = 1; n = 16; SMD = -0.75; 95% C.I., -1.78 to 0.27)

15 6-item OCD Scale: amelioration	s2x	There is limited evidence suggesting a difference favouring clomipramine over placebo on the amelioration of obsessive symptoms as measured by the 6-item OCD scale in adults with OCD (N = 1; n = 14; SMD = $-1.70$ ; 95% C.I., $-2.99$ to $-0.42$ )
16 OCD (OCD-CPRS; 6-item OCD Scale-amelioration)	s2x	There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the OCD-CPRS or 6-item OCD Scale in adults with OCD (N = 2; n = 30; SMD = $-1.12$ ; 95% C.I., $-1.92$ to $-0.32$ )
17 Self-Rating Obsessive- Compulsive Personality Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on reducing obsessive-compulsive symptoms as measured by the Self-Rating Obsessive-Compulsive Inventory in adults with OCD ( $N = 1$ ; $n = 44$ ; $SMD = -0.55$ ; $95\%$ C.I., $-1.16$ to $0.05$ )
18 Montgomery-Asberg Depression Rating Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on reducing depressive symptoms as measured by the Montgomery-Asberg Depression Rating Scale in adults with OCD (N = 1; n = 193; SMD = -0.24; 95% C.I., -0.52 to 0.05)
19 Clinical Global Impressions: severity of illness	s2x	There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing the severity of illness as measured by the CGI severity of illness subscale ( $N = 1$ ; $n = 193$ ; $SMD = -0.32$ ; $95\%$ C.I., $-0.60$ to $-0.03$ )
20 Symptom Checklist-90	s2x	There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing psychological distress as measured by the Symptom Checklist-90 in adults with OCD (N = 1; n = 151; SMD = $-0.32$ ; 95% C.I., $-0.64$ to $0.00$ )
02 Clomipramine vs. SSRIs: acut	e phase	. ,
01 Adverse events	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between clomipramine and SSRIs on the likelihood of reporting adverse events in adults with OCD ( $N = 7$ ; $n = 1046$ ; $RR = 1.06$ ; $95\%$ CI, 1 to 1.12)
02 Attempted suicide		
01 Clomipramine vs. Sertraline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and sertraline on the likelihood of attempting suicide in adults with OCD ( $N = 1$ ; $n = 168$ ; $RR = 3.15$ ; $95\%$ C.I., $0.33$ to $29.64$ )
03 Leaving study early	S2y	There is limited evidence suggesting a difference favouring SSRIs over clomipramine on the likelihood of leaving the study early in adults with OCD ( $N = 10$ ; $n = 1139$ ; $RR = 1.39$ ; $95\%$ CI, $1.14$ to $1.68$ )
04 Leaving study early due to adverse events	s1y	There is evidence suggesting a difference favouring SSRIs over clomipramine on the likelihood of leaving the study early due to adverse events in adults with OCD ( $N = 9$ ; $n = 1095$ ; $RR = 1.61$ ; 95% CI, 1.19 to 2.18)
05 Non-responders (25% Y-BOCS)	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between clomipramine and SSRIs on the likelihood of achieving response, defined as a 25% or greater reduction on the Y-BOCS, in adults with OCD ( $N = 2$ ; $n = 379$ ; $RR = 1.03$ ; 95% CI, 0.83 to 1.29)
06 Non-responders (35% Y-BOC	S)	

01 Clomipramine vs.	s4	The evidence is inconclusive and so it is not possible to determine if
Fluvoxamine	51	there is a clinically important difference between clomipramine and fluvoxamine on the likelihood of achieving response, defined as a 35% or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = 227; RR = $0.98$ ; 95% C.I., $0.71$ to $1.35$ )
07 Non-responders (CGI; 25% Y-	BOCS)	
01 Clomipramine vs. Fluoxetine	s2x	There is limited evidence suggesting a difference favouring clomipramine over fluoxetine on the likelihood of achieving response, defined as an improvement of at least 2 points on the CGI and a 25% or greater reduction on the Y-BOCS, in adults with OCD $(N = 2; n = 219; RR = 0.81; 95\% C.I., 0.63 to 1.03)$
08 Non-responders (CGI; 35% Y-	BOCS)	
01 Clomipramine vs. Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and fluoxetine on the likelihood of achieving response, defined as "much improved" or "very much improved" on the CGI and a 35% or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = $169$ ; RR = $0.99$ ; $95\%$ C.I., $0.72$ to $1.35$ )
02 Clomipramine vs. Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and fluvoxamine on the likelihood of achieving response, defined as "much improved" or "very much improved" on the CGI and a 35% or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = 165; RR = 0.96; 95% C.I., 0.7 to 1.32)
09 Non-responders (CGI-I)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and SSRIs on the likelihood of achieving response, defined as "minimally improved", "much improved" or "very much improved" on the CGI, in adults with OCD ( $N = 2$ ; $n = 194$ ; $RR = 1.29$ ; $95\%$ C.I., $0.83$ to $1.98$ )
10 Non-responders (all)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and fluoxetine on the likelihood of achieving response as defined by the study in adults with OCD (N = $7$ ; n = $1019$ ; RR = $0.98$ ; $95\%$ C.I., $0.85$ to $1.13$ )
11 Y-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and SSRIs on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = $7$ ; n = $739$ ; SMD = $-0.14$ ; 95% C.I., $-0.29$ to $0.01$ )
12 NIMH-OC	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between clomipramine and SSRIs on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = $3$ ; n = $666$ ; SMD = $-0.08$ ; $95\%$ CI, $-0.23$ to $0.08$ )
13 Comprehensive Psychopatho	logical Ratin	g Scale: obsessive-compulsive
01 Clomipramine vs. Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and fluoxetine on reducing obsessive-compulsive symptoms as measured by the CPRS obsessive-compulsive subscale in adults with OCD (N = 1; n = 54; SMD = $-0.37$ ; 95% C.I., $-0.91$ to $0.17$ )
14 Hamilton Rating Scale for Depression	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between clomipramine and SSRIs on reducing depressive symptoms as measured by the HRSD in adults with OCD ( $N = 3$ ; $n = 344$ ; SMD = -0.14; 95% C.I., -0.35 to 0.07)

15 Montgomery-Asberg	s3	There is evidence suggesting that there is unlikely to be a clinically
Depression Rating Scale		important difference between clomipramine and SSRIs on reducing depressive symptoms as measured by the MADRS in adults with
16 Depression (HRSD; MADRS)	s3	OCD (N = 3; n = 356; SMD = $0.03$ ; 95% C.I., $-0.19$ to $0.25$ ) There is evidence suggesting that there is unlikely to be a clinically important difference between clomipramine and SSRIs on reducing depressive symptoms as measured by the HRSD or MADRS in adults with OCD (N = 4; n = $592$ ; SMD = $-0.01$ ; 95% C.I., $-0.17$ to $0.16$ )
17 Clinical Anxiety Scale		
01 Clomipramine vs. Fluvoxamine	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between clomipramine and fluvoxamine on reducing anxiety symptoms as measured by the Clinical Anxiety Scale in adults with OCD (N = 1; n = 217; SMD = -0.07; 95% C.I., -0.33 to 0.20)
18 Covi Anxiety Scale		
01 Clomipramine v Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and fluoxetine on reducing anxiety symptoms as measured by the Covi Anxiety Scale in adults with OCD ( $N = 1$ ; $n = 54$ ; $SMD = 0$ ; 95% C.I., $-0.54$ to $0.54$ )
19 Anxiety (Clinical Anxiety Scale; Covi Anxiety Scale)	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between clomipramine and SSRIs on reducing anxiety as measured by the Clinical Anxiety Scale or Covi Anxiety Scale in adults with OCD (N = 2; n = 271; SMD = $-0.05$ ; 95% C.I., $-0.29$ to $0.18$ )
20 Clinical Global Impressions: global improvement	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between clomipramine and SSRIs on global improvement as measured by the CGI global improvement scale (N = 2; n = 271; SMD = 0; 95% C.I., -0.24 to 0.24)
21 Clinical Global Impressions: severity of illness *HETEROGENEITY*	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between clomipramine and SSRIs on reducing the severity of illness as measured by the CGI-severity of illness subscale in adults with OCD (N = 3; n = 559; SMD = -0.11; 95% C.I., -0.28 to $0.06$ )
22 Symptom Checklist-90	I	
01 Clomipramine vs. Paroxetine	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between clomipramine and SSRIs on reducing psychological distress as measured by the SCL-90 in adults with OCD ( $N = 1$ ; $n = 243$ ; SMD = -0.06; 95% C.I., -0.34 to 0.21)
03 Clomipramine vs. TCAs		
01 Leaving study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and TCAs on the likelihood of leaving the study early in adults with OCD ( $N = 2$ ; $n = 43$ ; $RR = 0.71$ ; 95% C.I., 0.24 to 2.12)
02 Leaving study early due to ad	verse effects	·
01 Clomipramine vs. Imipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and imipramine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 23; RR = 1.09; 95% C.I., $0.18$ to $6.48$ )
03 Leyton Obsessional Inventory	- symptom	

01 Clomipramine vs. Nortriptyline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and nortriptyline on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - total symptom score in adults with OCD (N = 1; n = 16; SMD = -0.19; 95% C.I., -1.17 to 0.80)
04 Leyton Obsessional Inve	ntory - trait	
01 Clomipramine vs. Nortriptyline	s <b>4</b>	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and nortriptyline on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - trait score in adults with OCD (N = $1$ ; n = $16$ ; SMD = $-0.21$ ; 95% C.I., $-1.19$ to $0.77$ )
05 Leyton Obsessional Inve	ntory - resistar	nce
01 Clomipramine vs. Nortriptyline vs.	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and nortriptyline on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - resistance subscale in adults with OCD ( $N = 1$ ; $n = 16$ ; $SMD = -0.45$ ; $95\%$ C.I., $-1.44$ to $0.55$ )
06 Leyton Obsessional Inve	ntory - interfer	ence
01 Clomipramine vs. Nortriptyline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and nortriptyline on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - interference subscale in adults with OCD (N = 1; n = 16; SMD = $0.31$ ; 95% C.I., - $0.68$ to $1.30$ )
07 OCD Scale (CRPS)	L	
01 Clomipramine vs. Nortriptyline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and nortriptyline on reducing obsessive-compulsive symptoms as measured by the Comprehensive Psychopathological Rating obsessive-compulsive subscale in adults with OCD ( $N = 1$ ; $n = 16$ ; SMD = -0.31; 95% C.I., -1.30 to 0.68)
08 Self-Rating Obsessive Co	ompulsive Pers	onality Inventory
01 Clomipramine vs. Imipramine	s2x	There is limited evidence suggesting a difference favouring clomipramine over imipramine on reducing obsessive-compulsive symptoms as measured by the Self-Rating Obsessive-Compulsive Personality Inventory in adults with OCD (N = 1; n = 16; SMD = $-1.44$ ; 95% C.I., $-2.57$ to $-0.30$ )
09 Self-Rating Obsessional	Neurotic Scale	
01 Clomipramine vs. Imipramine	s2x	There is evidence suggesting a difference favouring clomipramine over imipramine on reducing obsessive-compulsive symptoms as measured by the Self-Rating Obsessional Neurotic Scale in adults with OCD (N = 1; n = 16; SMD = -1.17; 95% C.I., -2.26 to -0.09)
10 Hamilton Rating Scale fo	or Depression	
01 Clomipramine vs. Imipramine	s2x	There is limited evidence suggesting a difference favouring clomipramine over imipramine on reducing depression symptoms as measured by the HRSD in adults with OCD (N = 1; n = 16; SMD = $-1.04$ ; 95% C.I., $-2.11$ to 0.02)
11 Global Evaluation of Eff	icacy	

01 Clomipramine vs.	s2x	There is limited evidence suggesting a difference favouring
Imipramine	527	clomipramine over imipramine on overall therapeutic effect as measured by the Global Evaluation Efficacy Scale in adults with OCD (N = 1; n = 16; SMD = -1.05; 95% C.I., -2.12 to 0.02)
04 Clomipramine vs. other drugs		100/30 // 2.12 to 0.02)
01 Adverse effects		
01 Clomipramine vs. Venlafaxine	s2y	There is limited evidence suggesting a difference favouring venlafaxine over clomipramine on the likelihood of reporting adverse effects in adults with OCD (N = 1; n = 73; RR = 1.49; 95% C.I., $1.08$ to $2.04$ )
02 Leaving study early		
01 Clomipramine vs. Buspirone	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and buspirone on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 20$ ; $RR = 1$ ; 95% C.I., 0.07 to 13.87)
02 Clomipramine vs. Phenelzine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and phenelzine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 30$ ; $RR = 0.88$ ; $95\%$ C.I., $0.14$ to $5.42$ )
03 Clomipramine vs. Venlafaxine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and venlafaxine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 73$ ; $RR = 3.87$ ; $95\%$ C.I., $0.5$ to $29.77$ )
03 Leaving study early due to ad	verse effects	
01 Clomipramine vs. Buspirone	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and buspirone on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 20; RR = 3; 95% C.I., $0.14$ to $65.90$ )
02 Clomipramine vs. Phenelzine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and phenelzine on the likelihood of leaving the study early due to adverse effects in adults with OCD ( $N = 1$ ; $n = 30$ ; $RR = 2.65$ ; 95% C.I., 0.12 to 60.21)
03 Clomipramine vs. Venlafaxine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and venlafaxine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 73; RR = 6.19; 95% C.I., 0.36 to $107.66$ )
04 Non-responders		
Venlafaxine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and venlafaxine on the likelihood of achieving response, defined as a 35% or greater reduction on the Y-BOCS and "much improved" or "very much improved" on the CGI, in adults with OCD (N = 1; n = $73$ ; RR = $0.88$ ; $95\%$ C.I., $0.61$ to $1.28$ )
05 Y-BOCS	1.	
01 Clomipramine vs. Buspirone	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and buspirone on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = 1; n = 18; SMD = $0.15$ ; 95% C.I., $-0.78$ to $1.07$ )

	Ι.	
02 Clomipramine vs. Venlafaxine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and venlafaxine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = 1; n = 73; SMD = $-0.16$ ; 95% C.I., $-0.64$ to $1.01$ )
06 NIMH-OC		
01 Clomipramine vs. Buspirone	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and buspirone on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = 1; n = 18; SMD = $0.08$ ; 95% C.I., $-0.84$ to $1.01$ )
07 Maudsley Obsessive-Compul	sive Inventor	y
01 Clomipramine vs. Phenelzine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and phenelzine on reducing obsessive-compulsive symptoms as measured by the Maudsley Obsessive-Compulsive Inventory in adults with OCD (N = 1; n = $26$ ; SMD = $0.33$ ; $95\%$ C.I., $-0.44$ to $1.11$ )
08 Hamilton Rating Scale for De	pression	
01 Clomipramine vs. Buspirone	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and buspirone on reducing depressive symptoms as measured by the HRSD in adults with OCD (N = 1; n = 18; SMD = $-0.09$ ; 95% C.I., $-1.02$ to $0.83$ )
02 Clomipramine vs. Phenelzine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and phenelzine on reducing depressive symptoms as measured by the HRSD in adults with OCD (N = 1; n = 26; SMD = $0.68$ ; 95% C.I., -0.11 to $1.48$ )
09 Hamilton Rating Scale for An	xiety	
01 Clomipramine vs. Phenelzine		There is limited evidence suggesting a difference favouring phenelzine over clomipramine on reducing anxiety as measured by the Hamilton Rating Scale for Anxiety in adults with OCD (N = 1; n = 26; SMD = 0.88; 95% C.I., 0.07 to 1.69)
05 Intravenous Clomipramine vs	s. IV placebo	,
01 Leaving study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between intravenous clomipramine and intravenous placebo on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 54$ ; $RR = 0.43$ ; 95% C.I., 0.04 to 4.48)
02 Non-responders	s2x	There is limited evidence suggesting a difference favouring intravenous clomipramine over intravenous placebo on the likelihood of achieving response, defined as "much improved" or "very much improved" on the CGI, in adults with OCD (N = 1; n = 54; RR = 0.79; 95% C.I., 0.66 to 0.96)
03 Y-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between intravenous clomipramine and intravenous placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = 1; n = 51; SMD = -0.14; 95% C.I., -0.70 to 0.41)
04 NIMH-OC	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between intravenous clomipramine and intravenous placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD ( $N = 1$ ; $n = 51$ ; SMD = -0.24; 95% C.I., -0.80 to 0.31)

05 Clinical Global Impressions:	s4	The evidence is inconclusive and so it is not possible to determine if
severity		there is a clinically important difference between intravenous clomipramine and intravenous placebo on reducing illness severity as measured by the CGI severity of illness subscale in adults with OCD (N = 1; n = 51; SMD = $-0.11$ ; 95% C.I., $-0.67$ to $0.44$ )
06 Hamilton Rating Scale for Depression	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between intravenous clomipramine and intravenous placebo on reducing depressive symptoms as measured by the HRSD in adults with OCD (N = 1; n = $51$ ; SMD = $-0.11$ ; 95% C.I., $-0.66$ to $0.45$ )
06 Intravenous Clomipramine v	s. oral Clor	mipramine
01 Adverse effects	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between intravenous clomipramine and oral clomipramine on the likelihood of reporting adverse effects in adults with OCD ( $N = 1$ ; $n = 15$ ; $RR = 0.91$ ; 95% C.I., 0.4 to 2.11)
02 Leaving study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between intravenous clomipramine and oral clomipramine on the likelihood of leaving the study early in adults with OCD (N = 1; n = 15; RR = 3.38; 95% C.I., 0.16 to 71.67)
03 Non-responders	s2x	There is limited evidence suggesting a difference favouring intravenous clomipramine over oral clomipramine on the likelihood of response, defined as a 25% or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = 15; RR = $0.16$ ; 95% C.I., $0.03$ to $1.02$ )
04 Y-BOCS	s2x	There is limited evidence suggesting a difference favouring intravenous clomipramine over oral clomipramine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD (N = 1; n = 15; SMD = -1.26; 95% C.I., -2.40 to -0.12)
07 Clomipramine vs. placebo: co	ntinuation	n phase
01 Adverse effects	s2y	There is limited evidence suggesting a difference favouring continuation treatment with placebo over clomipramine on the likelihood of reporting adverse effects in adults with OCD (N = 1; n = 124; RR = 1.53; 95% C.I., 1.03 to 2.26)
02 Leaving study early due to adverse effects	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between continuation treatment with clomipramine and placebo on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; $n = 124$ ; $RR = 4.73$ ; 95% C.I., 0.3 to 74.65)
03 NIMH-OC	s2x	There is limited evidence suggesting a difference favouring continuation treatment with clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD (N = 1; n = 113; SMD = -1.44; 95% C.I., -2.07 to – 0.82)
04 Physician's Global Evaluation	ns2x	There is limited evidence suggesting a difference favouring continuation treatment with clomipramine over placebo on improving the overall therapeutic effect as measured by the Physician's Global Evaluation in adults with OCD (N = 1; n = 113; SMD = -1.53; 95% C.I., -2.16 to -0.90)
08 Clomipramine vs. SSRIs: con 01 Leaving study early	tinuation p	hase

	1	,
01 Clomipramine 200mg vs. Fluoxetine 60mg (non- responders)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 200mg clomipramine and 60mg fluoxetine on the likelihood of leaving the study early in adults with OCD who previously failed to respond to acute treatment with the same drug (N = 1; n = 22; RR = 0.88; 95% C.I., 0.3 to 2.58)
02 Clomipramine 100mg vs. Fluoxetine 20mg (responders)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between $100 \text{mg}$ clomipramine and $20 \text{mg}$ fluoxetine on the likelihood of leaving the study early in adults with OCD who previously responded to acute treatment with the same drug (N = 1; n = 24; RR = 1.69; 95% C.I., 0.55 to 5.24)
02 Leaving study early due to ac	lverse effects	
01 Clomipramine 200mg vs. Fluoxetine 60mg (non- responders)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 200mg clomipramine and 60mg fluoxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD who previously failed to respond to acute treatment with the same drug $(N = 1; n = 22; RR = 0.33; 95\% C.I., 0.02 to 6.19)$
02 Clomipramine 100mg vs. Fluoxetine 20mg (responders)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between $100mg$ clomipramine and $20mg$ fluoxetine on the likelihood of leaving the study early due to adverse effects in adults with OCD who previously responded to acute treatment with the same drug (N = 1; n = 24; RR = $0.85$ ; $95\%$ C.I., $0.06$ to $12.01$ )
09 Clomipramine same dose vs.	half dose vs.	none: continuation/discontinuation
01 Leaving study early		,
01 Same dose (150mg) vs. half dose (75mg)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between continuation with the same dose and half the dose of clomipramine received during acute treatment on the likelihood of leaving the study early in adults with OCD (N = 1; n = 29; RR = $5.33$ ; $95\%$ C.I., $0.28$ to $102.26$ )
02 Same dose (150mg) vs. none	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between continuation with the same dose of clomipramine received during acute treatment and discontinuation of clomipramine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 28$ ; $RR = 2$ ; 95% C.I., 0.2 to 19.62)
03 Half dose (75mg) vs. none	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between continuation with half the dose of clomipramine received during acute treatment and discontinuation of clomipramine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 29$ ; $RR = 0.31$ ; 95% C.I., 0.01 to 7.09)
02 Relapse (CGI; 25% Y-BOCS)		
01 Same dose (150mg) vs. half dose (75mg)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between continuation with the same dose and half the dose of clomipramine received during acute treatment on the likelihood of relapse, defined as a 25% or greater reduction on Y-BOCS and "much worse" or "very much worse" on CGI, in adults with OCD (N = 1; n = 29; RR = 1.34; 95% C.I., 0.45 to 4)

02 Same dose (150mg) vs. none		There is limited evidence suggesting a difference favouring continuation with the same dose of clomipramine received during acute treatment over discontinuation of clomipramine on the likelihood of relapse, defined as a 25% or greater reduction on Y-BOCS and "much worse" or "very much worse" on CGI, in adults with OCD (N = 1; n = 28; RR = 0.45; 95% C.I., 0.21 to 0.97)
03 Half dose (75mg) vs. none		There is limited evidence suggesting a difference favouring continuation with half the dose of clomipramine received during acute treatment over discontinuation of clomipramine on the likelihood of relapse, defined as a 25% or greater reduction on Y-BOCS and "much worse" or "very much worse" on CGI, in adults with OCD (N = 1; n = 29; RR = 0.34; 95% C.I., 0.14 to 0.82)
04 Continuation vs. discontinuation	s2x	There is limited evidence suggesting a difference favouring continuation with clomipramine over discontinuation on the likelihood of relapse, defined as a 25% or greater reduction on Y-BOCS and "much worse" or "very much worse" on CGI, in adults with OCD (N = 1; n = 43; RR = 0.39; 95% C.I., 0.22 to 0.73)

## Tricyclic antidepressants

Description	Statement level	Statement and Statistics
01 TCAs vs. placebo (adults)		
01 Leyton Obsessional Inventor	y - symptom	
01 Nortriptyline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and placebo on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - symptoms subscale in adults with OCD (N = 1; n = 16; SMD = -0.69; 95% C.I., -1.71 to 0.33)
02 Leyton Obsessional Inventor	y - trait	
01 Nortriptyline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and placebo on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - trait subscale in adults with OCD (N = 1; $n = 16$ ; SMD = -0.42; 95% C.I., -1.41 to 0.58)
03 Leyton Obsessional Inventor	y - resistance	
01 Nortriptyline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and placebo on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - resistance subscale in adults with OCD ( $N = 1$ ; $n = 16$ ; $SMD = -0.02$ ; $95\%$ C.I., $-1.00$ to $0.96$ )
04 Leyton Obsessional Inventor	y - interferenc	ce
01 Nortriptyline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and placebo on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - interference subscale in adults with OCD (N = 1; n = 16; SMD = -0.63; 95% C.I., -1.64 to 0.38)
05 OCD Scale (CPRS)		
01 Nortriptyline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and placebo on reducing obsessive-compulsive symptoms as measured by the Comprehensive Psychopathology Rating Scale in adults with OCD ( $N = 1$ ; $n = 16$ ; SMD = -0.45; 95% C.I., -1.44 to 0.55)

02 TCAs vs. other drugs (adult	s)	
01 Adverse effects	,	
01 Desipramine vs. Sertraline	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between desipramine and sertraline on the likelihood of reporting adverse effects in adults with OCD (N = 1; n = 166; RR = 0.99; 95% C.I., 0.97 to 1.01)
02 Leaving study early		
01 Amitriptyline vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and placebo on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 20$ ; $RR = 2$ ; $95\%$ C.I., $0.21$ to $18.69$ )
02 Desipramine vs. Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between desipramine and fluvoxamine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 40$ ; $RR = 3.32$ ; $95\%$ C.I., $0.76$ to $14.49$ )
03 Desipramine vs. Sertraline	s2y	There is limited evidence suggesting a difference favouring sertraline over desipramine on the likelihood of leaving the study early in adults with OCD (N = 1; n = 166; RR = $2.19$ ; 95% C.I., $1.27$ to $3.79$ )
04 Imipramine vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imipramine and clomipramine on the likelihood of leaving the study early in adults with OCD ( $N = 1$ ; $n = 23$ ; $RR = 1.22$ ; $95\%$ C.I., $0.35$ to $4.28$ )
03 Leaving study early due to a	dverse effe	ects
01 Desipramine vs. Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between desipramine and fluvoxamine on the likelihood of leaving the study early due to adverse effects in adults with OCD ( $N = 1$ ; $n = 40$ ; $RR = 5.5$ ; 95% C.I., 0.28 to 107.78)
02 Desipramine vs. Sertraline	s2y	There is limited evidence suggesting a difference favouring sertraline over desipramine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 166; RR = 2.38; 95% C.I., 1.17 to 4.83)
03 Imipramine vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imipramine and clomipramine on the likelihood of leaving the study early due to adverse effects in adults with OCD (N = 1; n = 23; RR = 0.92; 95% C.I., 0.15 to 5.44)
04 Non-responders (OCD)		
01 Desipramine vs. Sertraline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between desipramine and sertraline on the likelihood of achieving response, defined as a $40\%$ or greater reduction on the Y-BOCS, in adults with OCD (N = 1; n = $166$ ; RR = $0.89$ ; $95\%$ C.I., $0.65$ to $1.21$ )
05 Non-responders (MDD)		
01 Desipramine vs. Sertraline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between desipramine and sertraline on the likelihood of achieving response, defined as a 50% or greater reduction on the HRSD, in adults with OCD (N = 1; n = 166; RR = 1.38; 95% C.I., 0.96 to 1.98)
06 Non-remitters (HRSD>17)		

01 Desipramine vs. Sertraline	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between desipramine and sertraline on the likelihood of remission, defined as a score of 17 or less on the HRSD, in adults with OCD (N = 1; n = 166; RR = 1.27; 95% C.I., 0.98 to 1.65)
07 Y-BOCS	s2y	There is limited evidence suggesting a difference favouring SSRIs over desipramine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 2$ ; $n = 204$ ; SMD = 0.39; 95% C.I., 0.11 to 0.66)
08 NIMH-OC	II.	· ·
01 Desipramine vs. Sertraline	s2y	There is limited evidence suggesting a difference favouring sertraline over desipramine on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in adults with OCD ( $N = 1$ ; $n = 164$ ; SMD = 0.37; 95% C.I., 0.06 to 0.67)
09 Leyton Obsessional Inventor	ry - symptoi	,
01 Nortriptyline vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and clomipramine on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - total symptom score in adults with OCD ( $N = 1$ ; $n = 16$ ; SMD = 0.19; 95% C.I., -0.80 to 1.17)
10 Leyton Obsessional Inventor	ry - trait	
01 Nortriptyline vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and clomipramine on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - trait score in adults with OCD ( $N = 1$ ; $n = 16$ ; SMD = 0.21; 95% C.I., -0.77 to 1.19)
11 Leyton Obsessional Inventor	ry - resistan	се
01 Nortriptyline vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and clomipramine on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - resistance subscale in adults with OCD (N = 1; n = 16; SMD = $0.45$ ; 95% C.I., - $0.55$ to $1.44$ )
12 Leyton Obsessional Inventor	ry - interfere	
01 Nortriptyline vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and clomipramine on reducing obsessive symptoms as measured by the Leyton Obsessional Inventory - interference subscale in adults with OCD ( $N = 1$ ; $n = 16$ ; $SMD = -0.31$ ; $95\%$ C.I., $-1.30$ to $0.68$ )
13 OCD Scale (CPRS)		
01 Nortriptyline vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and clomipramine on reducing obsessive-compulsive symptoms as measured by the Comprehensive Psychopathological Rating obsessive-compulsive subscale in adults with OCD (N = 1; n = 16; SMD = $0.31$ ; 95% C.I., $-0.68$ to $1.30$ )
14 Self-Rating Obsessive Comp	ulsive Perso	onality Inventory
01 Imipramine vs. Clomipramine	s2y	There is limited evidence suggesting a difference favouring clomipramine over imipramine on reducing obsessive-compulsive symptoms as measured by the Self-Rating Obsessive Compulsive Personality Inventory in adults with OCD (N = 1; n = 16; SMD = 1.44; 95% C.I., 0.30 to 2.57)
15 Self-Rating Obsessional Neu	rotic Scale	

01 Imipramine vs.	s2y	There is limited evidence suggesting a difference favouring
Clomipramine		clomipramine over imipramine on reducing obsessive-compulsive symptoms as measured by the Self-Rating Obsessional Neurotic Scale Inventory in adults with OCD ( $N = 1$ ; $n = 16$ ; SMD = 1.17; 95% C.I., 0.09 to 2.26)
16 Hamilton Rating Scale for De	pression	
01 Desipramine vs. Fluvoxamine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between desipramine and fluvoxamine on reducing depressive symptoms as measured by the HRSD in adults with OCD (N = 1; n = $40$ ; SMD = $0.32$ ; 95% C.I., - $0.30$ to $0.94$ )
02 Desipramine vs. Sertraline	s2y	There is limited evidence suggesting a difference favouring sertraline over desipramine on reducing depressive symptoms as measured by the HRSD in adults with OCD (N = 1; n = 164; SMD = $0.33$ ; 95% C.I., $0.02$ to $0.64$ )
03 Imipramine vs. Clomipramine	s2y	There is limited evidence suggesting a difference favouring clomipramine over imipramine on reducing depressive symptoms as measured by the HRSD in adults with OCD (N = 1; $n = 16$ ; SMD = $1.04$ ; 95% C.I., $-0.02$ to $2.11$ )
17 Global Evaluation of Efficacy		
01 Imipramine vs. Clomipramine	s2y	There is limited evidence suggesting a difference favouring clomipramine over imipramine on improving therapeutic effect as measured by the Global Evaluation of Efficacy in adults with OCD $(N = 1; n = 16; SMD = 1.05; 95\% C.I., -0.02 to 2.12)$
03 Desipramine substitution vs.	Clomipram	nine continuation (child/adolescent)
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between substitution of previous clomipramine treatment with desipramine and continuation of previous clomipramine treatment on the likelihood of leaving the study early in children with OCD ( $N = 1$ ; $n = 21$ ; $RR = 3.27$ ; 95% C.I., 0.15 to 72.23)
02 Relapse (Physician's Relapse Scale)	s2y	There is limited evidence suggesting a difference favouring continuation of previous clomipramine treatment over substitution of previous clomipramine treatment with desipramine on the likelihood of relapse as defined by the Physician's Relapse Scale in children with OCD ( $N = 1$ ; $n = 20$ ; $RR = 4.89$ ; 95% C.I., 1.37 to 17.49)
03 Comprehensive Psychopathological Rating Scale: obsessive-compulsive	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between substitution of previous clomipramine treatment with desipramine and continuation of previous clomipramine treatment on reducing obsessive-compulsive symptoms as measured by the Comprehensive Psychopathological Rating Scale - obsessive-compulsive subscale in children with OCD ( $N = 1$ ; $n = 20$ ; SMD = 0.28; 95% C.I., -0.60 to 1.17)
04 NIMH-OC	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between substitution of previous clomipramine treatment with desipramine and continuation of previous clomipramine treatment on improving global functioning as measured by the NIMH-OC in children with OCD (N = 1; n = 20; SMD = 0.44; 95% C.I., -0.45 to 1.34)

### **SNRIs**

Description	Statement level	Statement and Statistics
01 SNRIs vs. other drugs	L	
01 Adverse effects		
01 Venlafaxine vs. Clomipramine	s2x	There is limited evidence suggesting a difference favouring venlafaxine over clomipramine on the likelihood of reporting adverse effects ( $N = 1$ ; $n = 73$ ; $RR = 0.67$ ; 95% C.I., 0.49 to 0.92)
02 Leaving study early		
01 Venlafaxine vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between venlafaxine and clomipramine on the likelihood of leaving the study early ( $N = 1$ ; $n = 73$ ; $RR = 0.26$ ; 95% C.I., 0.03 to 1.99)
03 Leaving study early due to a	adverse effec	ts
01 Venlafaxine vs. Paroxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between venlafaxine and paroxetine on the likelihood of leaving the study early due to adverse effects ( $N = 1$ ; $n = 150$ ; $RR = 0.33$ ; 95% C.I., 0.07 to 1.6)
02 Venlafaxine vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between venlafaxine and clomipramine on the likelihood of leaving the study early due to adverse effects ( $N = 1$ ; $n = 73$ ; $RR = 0.16$ ; 95% C.I., 0.01 to 2.81)
04 Non-responders	•	
01 Venlafaxine vs. Paroxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between venlafaxine and paroxetine on the likelihood of achieving response, defined as a 35% or more reduction on the Y-BOCS (N = 1; n = 150; RR = 1.12; 95% C.I., $0.86$ to $1.46$ )
02 Venlafaxine vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between venlafaxine and clomipramine on the likelihood of achieving response, defined as a 35% or greater reduction on the Y-BOCS and a CGI score less than or equal to 2 ( $N = 1$ ; $n = 73$ ; $RR = 1.14$ ; 95% C.I., 0.78 to 1.65)
05 Y-BOCS		
01 Venlafaxine vs. Paroxetine	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between venlafaxine and paroxetine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 145; SMD = $0.09$ ; 95% C.I., $-0.23$ to $0.42$ )
02 Venlafaxine vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between venlafaxine and clomipramine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 73; SMD = 0.16; 95% C.I., -0.32 to 0.64)
06 Hamilton Rating Scale for D	epression	

01 Venlafaxine vs. Paroxetine	There is evidence suggesting that there is unlikely to be a clinically important difference between venlafaxine and paroxetine on reducing depressive symptoms as measured by the HRSD (N = 1; n = 145; SMD = $0.14$ ; 95% C.I., $-0.19$ to $0.46$ )
07 Hamilton Anxiety Scale	
01 Venlafaxine vs. Paroxetine	There is evidence suggesting that there is unlikely to be a clinically important difference between venlafaxine and paroxetine on reducing anxiety as measured by the Hamilton Anxiety Scale (N = 1; n = 145; SMD = $-0.03$ ; 95% C.I., $-0.36$ to $0.29$ )

### Monoamino-oxidase inhibitors

Description	Statement level	Statements and Statistics
01 MAOIs vs. placebo		
01 Leaving study early		
01 Phenelzine vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and placebo on the likelihood of leaving the study early in patients with OCD ( $N = 1$ ; $n = 41$ ; $RR = 1.05$ ; 95% C.I., 0.24 to 4.61)
02 Y-BOCS		
01 Phenelzine vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD ( $N = 1$ ; $n = 35$ ; $SMD = -0.36$ ; $95\%$ C.I., $-1.03$ to $0.31$ )
03 NIMH-OC		
01 Phenelzine vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in patients with OCD ( $N = 1$ ; $n = 35$ ; SMD = -0.29; 95% C.I., -0.96 to 0.38)
04 OCD Scale (CPRS)	<b>'</b>	,
01 Phenelzine vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and placebo on reducing obsessive-compulsive symptoms as measured by the Comprehensive Psychopathological Rating Scale - OCD subscale in patients with OCD (N = 1; n = 35; SMD = 0.19; 95% C.I., -0.47 to 0.86)
05 Clinical Global Impression	า	
01 Phenelzine vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and placebo on reducing the severity of illness as measured by the Clinical Global Impressions scale in patients with OCD ( $N = 1$ ; $n = 35$ ; SMD = 0; 95% C.I., -0.66 to 0.66)
02 MAOIs vs. other drugs		
01 Leaving study early		

01 Phenelzine vs.	s4	The evidence is inconclusive and so it is not possible to determine if
Clomipramine		there is a clinically important difference between phenelzine and
		clomipramine on the likelihood of leaving the study early in patients with OCD ( $N = 1$ ; $n = 30$ ; $RR = 1.14$ ; 95% C.I., 0.18 to 7.08)
02 Phenelzine vs. Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and fluoxetine on the likelihood of leaving the study early in patients with OCD ( $N = 1$ ; $n = 43$ ; $RR = 0.86$ ; $95\%$ C.I., $0.22$ to $3.4$ )
02 Leaving study early due to	adverse effe	ets
01 Phenelzine vs.	s4	The evidence is inconclusive and so it is not possible to determine if
Clomipramine		there is a clinically important difference between phenelzine and clomipramine on the likelihood of leaving the study early due to adverse effects in patients with OCD ( $N = 1$ ; $n = 30$ ; $RR = 0.38$ ; 95% C.I., 0.02 to 8.59)
03 Y-BOCS		
01 Phenelzine vs. Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and fluoxetine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD ( $N = 1$ ; $n = 36$ ; SMD = 0.01; 95%
04 NIMH-OC		C.I., -0.64 to 0.67)
	1	
01 Phenelzine vs. Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and fluoxetine on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in patients with OCD ( $N = 1$ ; $n = 36$ ; $SMD = -0.16$ ; $95\%$ C.I., $-0.81$ to $0.50$ )
05 Maudsley Obsessive-Comp	oulsive Inver	itory
01 Phenelzine vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and clomipramine on reducing obsessive-compulsive symptoms as measured by the Maudsley Obsessive-Compulsive Inventory in patients with OCD (N = 1; n = 26; SMD = $-0.33$ ; 95% C.I., $-1.11$ to $0.44$ )
06 OCD Scale (CPRS)		
01 Phenelzine vs. Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and fluoxetine on reducing obsessive-compulsive symptoms as measured by the Comprehensive Psychopathological Rating Scale - OCD subscale in patients with OCD (N = 1; n = 36; SMD = -0.06; 95% C.I., -0.72 to 0.59)
07 Hamilton Rating Scale for I	Depression	
01 Phenelzine vs.	s4	The evidence is inconclusive and so it is not possible to determine if
Clomipramine		there is a clinically important difference between phenelzine and clomipramine on reducing depressive symptoms as measured by the Hamilton Rating Scale for Depression in patients with OCD ( $N = 1$ ; $n = 26$ ; SMD = -0.68; 95% C.I., -1.48 to 0.11)

08 Hamilton Rating Scale for A	nxiety	
01 Phenelzine vs. Clomipramine	s2x	There is limited evidence suggesting a difference favouring phenelzine over clomipramine on reducing anxiety as measured by the Hamilton Anxiety Scale in patients with OCD (N = 1; n = 26; SMD = -0.88; 95% C.I., -1.69 to -0.07)
09 Clinical Global Impression		
01 Phenelzine vs. Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and fluoxetine on reducing the severity of illness as measured by the Clinical Global Impressions scale in patients with OCD (N = 1; n = 36; SMD = $0.10$ ; 95% C.I., $-0.56$ to $0.75$ )

# Anxiolytics

Description	Statement level	Statement and Statistics
01 Anxiolytics vs. placebo	•	
01 Leaving study early		
01 Clonazepam	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on the likelihood of leaving the study early ( $N = 1$ ; $n = 27$ ; $RR = 0.74$ ; 95% C.I., 0.26 to 2.12)
02 Leaving study early due to	adverse effect	ts
01 Clonazepam	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on the likelihood of leaving the study early due to adverse effects ( $N = 1$ ; $n = 27$ ; $RR = 0.88$ ; 95% C.I., 0.18 to 4.41)
03 Non-responders		
01 Clonazepam	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on the likelihood of achieving response, defined as "much improved" or "very much improved" on the CGI - Improvement subscale ( $N = 1$ ; $n = 27$ ; $RR = 1.18$ ; $95\%$ C.I., $0.84$ to $1.64$ )
04 Y-BOCS	- 1	
01 Clonazepam	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 25; SMD = -0.22; 95% C.I., -1.04 to 0.60)
05 NIMH-OC		
01 Clonazepam	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC ( $N = 1$ ; $n = 25$ ; $SMD = 0.05$ ; $95\%$ C.I., $-0.77$ to $0.87$ )
06 Hamilton Rating Scale for 1	Depression	

01 Clonazepam	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on reducing depressive symptoms as measured by the HRSD $(N = 1; n = 25; SMD = -0.10; 95\% C.I., -0.92 to 0.72)$
07 Hamilton Anxiety Scale		
01 Clonazepam	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on reducing anxiety as measured by the Hamilton Anxiety Scale ( $N = 1$ ; $n = 25$ ; SMD = -0.16; 95% C.I., -0.98 to 0.66)
02 Anxiolytics vs. other drugs		
01 Leaving study early		
01 Buspirone vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between buspirone and clomipramine on the likelihood of leaving the study early ( $N = 1$ ; $n = 20$ ; $RR = 1$ ; 95% C.I., 0.07 to 13.87)
02 Leaving study early due to a	dverse effe	cts
01 Buspirone vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between buspirone and clomipramine on the likelihood of leaving the study early due to adverse effects (N = 1; n = 20; RR = 0.33; 95% C.I., 0.02 to 7.32)
03 Y-BOCS	1	
01 Buspirone vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between buspirone and clomipramine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 18; SMD = -0.15; 95% C.I., -1.07 to 0.78)
04 NIMH-OC		
01 Buspirone vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between buspirone and clomipramine on reducing obsessive-compulsive symptoms as measured by the NIMH-OC ( $N = 1$ ; $n = 18$ ; $SMD = -0.08$ ; $95\%$ C.I., $-1.01$ to $0.84$ )
05 Hamilton Rating Scale for De	epression	
01 Buspirone vs. Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between buspirone and clomipramine on reducing depressive symptoms as measured by the HRSD ( $N = 1$ ; $n = 18$ ; SMD = 0.09; 95% C.I., -0.83 to 1.02)

## Other Pharmacological interventions

Description	Statement level	Statement and Statistics
01 Inositol vs. placebo		
01 Y-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between inositol and placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in adults with OCD ( $N = 1$ ; $n = 13$ ; $SMD = -0.29$ ; $95\%$ C.I., $-1.38$ to $0.81$ )
02 Oxytocin vs. placebo		

01 Adverse effects	s2y	There is limited evidence suggesting a difference favouring placebo over oxytocin on the likelihood of reporting adverse effects in adults with OCD ( $N = 1$ ; $n = 12$ ; $RR = 6.00$ ; 95% C.I., 1.00 to 35.91)
03 Hamilton Depression Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between oxytocin and placebo on reducing depressive symptoms as measured by the Hamilton Depression Scale in adults with OCD (N = 1; n = 12; SMD = $0.38$ ; 95% C.I., -0.77 to 1.52)
04 Self Rating Depression Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between oxytocin and placebo on reducing depressive symptoms as measured by the Self Rating Depression Scale in adults with OCD (N = 1; n = 12; SMD = 0; 95% C.I., -1.13 to 1.13)
05 Hamilton Anxiety Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between oxytocin and placebo on reducing anxiety as measured by the Hamilton Anxiety Scale in adults with OCD (N = 1; n = 12; SMD = $0.22$ ; 95% C.I., - $0.92$ to $1.36$ )
06 State Anxiety Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between oxytocin and placebo on reducing anxiety as measured by the State Anxiety Inventory in adults with OCD (N = 1; n = 12; SMD = $-1.05$ ; 95% C.I., $-2.29$ to $0.19$ )
07 General Symptom Index	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between oxytocin and placebo on reducing general symptoms as measured by the General Symptom Index in adults with OCD (N = 1; n = 12; SMD = 0; 95% C.I., -1.13 to 1.13)

Augmentation strategies

Description	Statement	Statement and Statistics
	level	
01 Buspirone vs. placebo		
01 Non-responders		
01 Fluvoxamine+Buspirone v Fluvoxamine+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine + buspirone and fluvoxamine + placebo on the likelihood of response in patients with OCD (N = 1; n = 33; RR = 1.04; 95% C.I., 0.80 to 1.36)
02 Y-BOCS	1	
01 Fluvoxamine+Buspirone v Fluvoxamine+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine + buspirone and fluvoxamine + placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD (N = 1; n = 33; SMD = $0.49$ ; 95% C.I., $-0.21$ to $1.19$ )
03 Hamilton Depression Rating	g Scale	

01 Fluvoxamine+Buspirone v Fluvoxamine+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine + buspirone and fluvoxamine + placebo on reducing depressive symptoms as measured by the Hamilton Depression Rating Scale in patients with OCD (N = 1; n = 33; SMD = $0.64$ ; 95% C.I., -0.07 to 1.35)
04 Hamilton Anxiety Scale		
01 Fluvoxamine+Buspirone v Fluvoxamine+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine + buspirone and fluvoxamine + placebo on reducing anxiety symptoms as measured by the Hamilton Anxiety Scale in patients with OCD (N = 1; n = 33; SMD = 0.48; 95% C.I., -0.22 to 1.18)
02 Citalopram+Clomipramine	vs. Citalopra	am
01 Non-responders (Y-BOCS 35%)	s2x	There is limited evidence suggesting a difference favouring citalopram + clomipramine over citalopram on the likelihood of response, defined as a reduction of 35% or greater on the Y-BOCS, in patients with OCD (N = 1; n = 16; RR = $0.06$ ; 95% C.I., $0.00$ to $0.94$ )
02 Y-BOCS	s2x	There is limited evidence suggesting a difference favouring citalopram + clomipramine over citalopram on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD (N = 1; n = 16; SMD = -2.15; 95% C.I., -3.46 to -0.84)
03 Hamilton Depression Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between citalopram + clomipramine and citalopram on reducing depressive symptoms as measured by the Hamilton Depression Scale in patients with OCD (N = 1; $n = 16$ ; SMD = -0.33; 95% C.I., -1.33 to 0.67)
03 Desipramine vs. placebo		· ·
01 Y-BOCS at 6 weeks		
01 SSRI+Desipramine v SSRI+Placebo	s2x	There is limited evidence suggesting a difference favouring SSRIs + desipramine over SSRIs + placebo on reducing obsessive-compulsive symptoms at 6 weeks as measured by the Y-BOCS in patients with OCD ( $N = 1$ ; $n = 23$ ; SMD = -1.97; 95% C.I., -3.00 to -0.93)
02 Y-BOCS at 10 weeks	<u></u>	
01 SSRI+Desipramine v SSRI+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SSRIs + desipramine and SSRIs + placebo on reducing obsessive-compulsive symptoms at $10$ weeks as measured by the Y-BOCS in patients with OCD (N = 1; n = 23; SMD = -0.80; 95% C.I., -1.66 to 0.06)
04 Haloperidol vs. placebo		
01 Non-responders	<del></del>	
01 Fluvoxamine+Haloperidol v Fluvoxamine+Placebo	rs2x	There is limited evidence suggesting a difference favouring fluvoxamine+haloperidol over fluvoxamine+placebo on the likelihood of response in patients with OCD (N = 1; n = 34; RR = 0.59; 95% C.I., 0.40 to 0.88)
02 Y-BOCS		
01 Fluvoxamine+Haloperidol v Fluvoxamine+Placebo	,s2x	There is limited evidence suggesting a difference favouring fluvoxamine + haloperidol over fluvoxamine + placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD (N = 1; n = 34; SMD = -2.20; 95% C.I., -3.07 to – 1.32)
05 Inositol vs. placebo		
01 Y-BOCS		

01 SRI+Inositol v SRI+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SRIs + Inositol and SRIs + placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD (N = 1; n = 10; SMD = $-0.24$ ; 95% C.I., $-1.51$ to $1.04$ )
06 Lithium vs. placebo		
01 Non-responders	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine + lithium and fluvoxamine + placebo on the likelihood of response, defined as a reduction of 35% or greater on the Y-BOCS, in patients with OCD (N = 2; n = 30; RR = 0.82; 95% C.I., 0.62 to 1.08)
02 Y-BOCS change score *HETEROGENEITY	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine + lithium and fluvoxamine + placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD ( $N = 2$ ; $n = 30$ ; $SMD = -0.29$ ; $95\%$ C.I., $-1.07$ to $0.50$ )
03 Hamilton Depression Scale change score *HETEROGENEITY	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine + lithium and fluvoxamine + placebo on reducing depressive symptoms as measured by the Hamilton Depression Scale in patients with OCD (N = 2; n = 30; SMD = $-0.15$ ; 95% C.I., $-0.93$ to $0.63$ )
04 Hamilton Anxiety Scale change score	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluvoxamine + lithium and fluvoxamine + placebo on reducing anxiety symptoms as measured by the Hamilton Anxiety Scale in patients with OCD (N = $2$ ; n = $30$ ; SMD = $0.41$ ; $95\%$ C.I., $-0.34$ to $1.15$ )
07 Nortriptyline vs. placebo		,
01 Y-BOCS		
01 Clomipramine+Nortriptyline v Clomipramine+Placebo	s2x	There is limited evidence suggesting a difference favouring clomipramine + nortriptyline over clomipramine + placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD (N = 1; n = 30; SMD = -1.90; 95% C.I., -2.78 to -1.02)
08 Olanzapine vs. placebo	1	,
01 Leaving the study early		
01 Fluoxetine+Olanzapine v Fluoxetine+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine + olanzapine and fluoxetine + placebo on the likelihood of leaving the study early in patients with OCD (N = 1; n = 44; RR = 2.50; 95% C.I., 0.54 to 11.54)
02 Leaving the study early due	to adverse ef	fects
	s4	The evidence is inconclusive and so it is not possible to determine if
Fluoxetine+Placebo		there is a clinically important difference between fluoxetine + olanzapine and fluoxetine + placebo on the likelihood of leaving the study early due to adverse effects in patients with OCD (N = 1; n = 44; RR = $1.00$ ; 95% C.I., $0.15$ to $6.48$ )
03 Non-responders (Y-BOCS 25	5%)	
01 Fluoxetine+Olanzapine v Fluoxetine+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine + olanzapine and fluoxetine + placebo on the likelihood of response, defined as a reduction of 25% or greater on the Y-BOCS, in patients with OCD ( $N = 1$ ; $n = 44$ ; $RR = 1.00$ ; 95% C.I., 0.61 to 1.64)

04 Y-BOCS change score		
01 Fluoxetine+Olanzapine v Fluoxetine+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine + olanzapine and fluoxetine + placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD (N = 1; n = $44$ ; SMD = $-0.29$ ; 95% C.I., $-0.89$ to $0.30$ )
09 Pindolol vs. placebo		
01 Leaving the study early		
01 Paroxetine + Pindolol v Paroxetine + Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine + pindolol and paroxetine + placebo on the likelihood of leaving the study early in patients with OCD (N = 1; n = 16; RR = 0.20; 95% C.I., 0.01 to 3.61)
02 Leaving the study early du	e to adver	se effects
01 Paroxetine + Pindolol v Paroxetine + Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine + pindolol and paroxetine + placebo on the likelihood of leaving the study early due to adverse effects in patients with OCD (N = 1; n = 16; RR = 0.20; 95% C.I., 0.01 to 3.61)
03 Y-BOCS		
01 Paroxetine+Pindolol v Paroxetine+Placebo	s2x	There is limited evidence suggesting a difference favouring paroxetine + pindolol over paroxetine + placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD (N = 1; n = 14; SMD = -2.15; 95% C.I., -3.56 to $-0.73$ )
04 MADRS		
01 Paroxetine+Pindolol v Paroxetine+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine + pindolol and paroxetine + placebo on reducing depressive symptoms as measured by the MADRS in patients with OCD (N = 1; n = 14; SMD = $0.04$ ; 95% C.I., $-1.02$ to $1.10$ )
05 Hamilton Anxiety Scale		01125 0101, 90% 011, 110 <b>2</b> to 1110)
01 Paroxetine+Pindolol v Paroxetine+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine + pindolol and paroxetine + placebo on reducing anxiety symptoms as measured by the Hamilton Anxiety Scale in patients with OCD (N = $1$ ; n = $14$ ; SMD = $-0.31$ ; 95% C.I., $-1.37$ to $0.76$ )
10 Quetiapine vs. placebo		
01 Adverse effects		
01 SRI + Quetiapine v SRI + Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SRIs + quetiapine and SRIs + placebo on the likelihood of reporting adverse effects in patients with OCD (N = 1; n = 27; RR = 2.09; 95% C.I., 0.85 to 5.16)
03 Non-responders (Y-BOCS 3	30%)	<b>'</b>
01 SRI + Quetiapine v SRI +	s2x	There is limited evidence suggesting a difference favouring SRIs +
Placebo		quetiapine over SRIs + placebo on the likelihood of response, defined as a reduction of 30% or greater on the Y-BOCS, in patients with OCD $(N = 1; n = 27; RR = 0.29; 95\% C.I., 0.12 to 0.65)$
04 Y-BOCS	·	·

01 SRI+Quetiapine v SRI+Placebo	s2x	There is limited evidence suggesting a difference favouring SRIs + quetiapine over SRIs + placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD (N = 1;
		n = 27; SMD = -2.06; 95% C.I., -3.02 to -1.10)
05 CGI-severity of illness		,
01 SRI+Quetiapine v SRI+Placebo	s2x	There is limited evidence suggesting a difference favouring SRIs + quetiapine over SRIs + placebo on improving obsessive-compulsive symptoms as measured by the CGI severity of illness subscale in patients with OCD ( $N = 1$ ; $n = 27$ ; SMD = -1.15; 95% C.I., -1.98 to -0.33)
11 Risperidone vs. placebo		
01 Adverse effects	•	
01 SRI+Risperidone v SRI+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SRIs + risperidone and SRIs + placebo on the likelihood of reporting adverse effects in patients with OCD ( $N = 2$ ; $n = 52$ ; $RR = 0.99$ ; 95% C.I., 0.77 to 1.28)
02 Leaving the study early	<b>-</b>	
01 SRI+Risperidone v SRI+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SRIs + risperidone and SRIs + placebo on the likelihood of leaving the study early in patients with OCD (N = 2; n = 52; RR = 0.70; 95% C.I., 0.16 to 3.01)
03 Leaving the study early due	e to advers	e effects
01 SRI+Risperidone v SRI+Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SRIs + risperidone and SRIs + placebo on the likelihood of leaving the study early due to adverse effects in patients with OCD ( $N = 1$ ; $n = 36$ ; $RR = 2.43$ ; 95% C.I., 0.11 to 55.89)
04 Non-responders	s2x	There is limited evidence suggesting a difference favouring SRIs + risperidone over SRIs + placebo on the likelihood of response in patients with OCD (N = 2; n = 52; RR = $0.74$ ; 95% C.I., $0.60$ to $0.91$ )
05 Y-BOCS change score		
01 SRI + Risperidone v SRI + Placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SRIs + risperidone and SRIs + placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD (N = 2; n = 52; SMD = -0.37; 95% C.I., -0.92 to 0.19)
06 Hamilton Depression Scale		
01 SRI + Risperidone v SRI + Placebo	s2x	There is limited evidence suggesting a difference favouring SRIs + risperidone over SRIs + placebo on reducing depressive symptoms as measured by the Hamilton Depression Scale in patients with OCD (N = 1; n = 33; SMD = $-1.08$ ; 95% C.I., $-1.82$ to $-0.34$ )
07 Hamilton Anxiety Scale	<u> </u>	
01 SRI + Risperidone v SRI + Placebo	s2x	There is limited evidence suggesting a difference favouring SRIs + risperidone over SRIs + placebo on reducing anxiety symptoms as measured by the Hamilton Anxiety Scale in patients with OCD (N = $1$ ; n = $33$ ; SMD = $-1.09$ ; $95\%$ C.I., $-1.83$ to $-0.35$ )
12 All antipsychotics v placebo	)	

01 Adverse effects *HETEROGENEITY	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between antipsychotic augmentation and placebo on the likelihood of reporting adverse effects in patients with OCD (N = 3; n = 79; RR = 1.19; 95% C.I., 0.89 to 1.58)
02 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between antipsychotic augmentation and placebo on the likelihood of leaving the study early in patients with OCD ( $N = 4$ ; $n = 123$ ; $RR = 1.34$ ; 95% C.I., 0.49 to 3.67)
03 Leaving the study early due to adverse effects	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between antipsychotic augmentation and placebo on the likelihood of leaving the study early due to adverse effects in patients with OCD ( $N = 2$ ; $n = 80$ ; $RR = 1.31$ ; 95% C.I., 0.27 to 6.35)
04 Non-responders *HETEROGENEITY	s2x	There is limited evidence suggesting a difference favouring antipsychotic augmentation over placebo on the likelihood of response in patients with OCD (N = $5$ ; n = $157$ ; RR = $0.66$ ; $95\%$ C.I., $0.54$ to $0.81$ )
05 Y-BOCS *HETEROGENEITY	s1x	There is evidence suggesting a difference favouring antipsychotic augmentation over placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in patients with OCD (N = 5; $n = 157$ ; SMD = -0.84; 95% C.I., -1.19 to -0.50)
06 Hamilton Depression Scale		, ,
01 SRI + Risperidone v SRI + Placebo	s2x	There is limited evidence suggesting a difference favouring SRIs + risperidone over SRIs + placebo on reducing depressive symptoms as measured by the Hamilton Depression Scale in patients with OCD (N = 1; $n = 33$ ; SMD = -1.08; 95% C.I., -1.82 to -0.34)
07 Hamilton Anxiety Scale		
01 SRI + Risperidone v SRI + Placebo	s2x	There is limited evidence suggesting a difference favouring SRIs + risperidone over SRIs + placebo on reducing anxiety symptoms as measured by the Hamilton Anxiety Scale in patients with OCD ( $N = 1$ ; $n = 33$ ; SMD = -1.09; 95% C.I., -1.83 to -0.35)
08 CGI: severity of illness		· · · · · · · · · · · · · · · · · · ·
01 SRI+Quetiapine v SRI+Placebo	s2x	There is limited evidence suggesting a difference favouring SRIs + quetiapine over SRIs + placebo on improving obsessive-compulsive symptoms as measured by the CGI severity of illness subscale in patients with OCD (N = 1; n = $27$ ; SMD = $-1.15$ ; 95% C.I., $-1.98$ to $-0.33$ )

## SSRIs (children and adolescents)

1	Statement level	Statistics
01 SSRI vs. placebo: acute phase	9	
01 Adverse events		There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and placebo on the likelihood of reporting adverse effects in children or adolescents with OCD (N = 4; n = $473$ ; RR = $1.09$ ; $95\%$ C.I., $0.99$ to $1.19$ )

02 Serious adverse events	s4	The evidence is inconclusive and so it is not possible to determine if
		there is a clinically important difference between SSRIs and placebo on the likelihood of serious adverse events in children or adolescents with OCD (N = 1; n = 207; RR = 3.21; 95% C.I., 0.34 to 30.39)
03 Leaving study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SSRIs and placebo on the likelihood of leaving the study early in children or adolescents with OCD (N = 7; n = 732; RR = 1.02; 95% C.I., 0.8 to 1.3)
04 Leaving study early due to adverse effects	s1y	There is evidence suggesting a difference favouring placebo over SSRIs on the likelihood of leaving the study early due to adverse effects (N = 6; n = 732; RR = 3.05; 95% C.I., 1.55 to 6.00)
05 Non-responders (25% CY-B	OCS)	
01 Fluvoxamine	s1x	There is evidence suggesting a difference favouring placebo over SSRIs on the likelihood of achieving response, defined as a 25% or greater reduction on the CY-BOCS in children or adolescents with OCD ( $N = 2$ ; $n = 327$ ; $RR = 0.7$ ; $95\%$ C.I., $0.58$ to $0.86$ )
06 Non-responders (40% Y-BO	CS)	
01 Fluoxetine	s2x	There is limited evidence suggesting a difference favouring fluoxetine over placebo on the likelihood of achieving response, defined as a $40\%$ or greater reduction on the CY-BOCS, in children or adolescents with OCD (N = 1; n = $103$ ; RR = $0.68$ ; $95\%$ C.I., $0.50$ to $0.92$ )
07 Non-responders (all)	s1x	There is evidence suggesting a difference favouring SSRIs over placebo on the likelihood of achieving response, defined as a 25% $/40\%$ or greater reduction on the CY-BOCS, in children or adolescents with OCD (N = 3; n = 430; RR = 0.7; 95% C.I., 0.59 to 0.83)
08 Non-responders (CGI Global Improvement)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on the likelihood of response, defined as "much improved" or "very much improved" on CGI Global Improvement (N = 1; n = 207; RR = 0.82; 95% C.I., 0.66 to 1.02)
09 Remission (CY-BOCS<11)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between sertraline and placebo on the likelihood of remission, defined as a score less than 11 on the CY-BOCS ( $N = 1$ ; $n = 56$ ; $RR = 0.81$ ; 95% C.I., 0.66 to 1)
10 CY-BOCS	s2x	There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured by the CY-BOCS in children or adolescents with OCD (N = $7$ ; n = $718$ ; SMD = $-0.43$ ; 95% C.I., $-0.58$ to $-0.28$ )
11 NIMH-OC	s2x	There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in children or adolescents with OCD (N = 4; n = 453; SMD = -0.38; 95% C.I., -0.57 to -0.19)
12 Child Obsessive Compulsiv	e Impact Scal	e (Parent Version)
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on reducing the social impact of OCD as measured by the parent-rated Child Obsessive Compulsive Impact Scale in children or adolescents with OCD (N = 1; n = 43; SMD = -0.41; 95% C.I., -1.02 to 0.19)

13 Leyton Obsessional Inventor	v (Child Vers	sion): interference
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on reducing OCD-related interference as measured by the child-rated Leyton Obsessional Inventory - interference subscale in children or adolescents with OCD (N = 1; n = 13; SMD = -0.02; 95% C.I., -1.11 to 1.07)
14 Leyton Obsessional Inventor	y (Child Vers	ion): resistance
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on reducing OCD-related resistance as measured by the child-rated Leyton Obsessional Inventory - resistance subscale in children or adolescents with OCD (N = 1; n = 13; SMD = -0.12; 95% C.I., -1.21 to 0.97)
15 Leyton Obsessional Inventor	y (Child Vers	sion): symptoms
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on reducing obsessive-compulsive symptoms as measured by the child-rated Leyton Obsessional Inventory - symptom subscale in children or adolescents with OCD (N = 1; n = 13; SMD = 0.23; 95% C.I., -0.87 to 1.32)
16 Children or adolescents 'Dep	pression Ratir	g Scale (Revised Version)
01 Fluvoxamine	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between fluvoxamine and placebo on reducing depressive symptoms as measured by the Children or adolescents' Depression Rating Scale in children or adolescents with OCD (N = 1; n = 120; SMD = $0.04$ ; 95% C.I., $-0.32$ to $0.40$ )
17 Hamilton Rating Scale for D	epression	
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on reducing depressive symptoms as measured by the Hamilton Depression Rating Scale in children or adolescents with OCD (N = 1; n = 43; SMD = 0.29; 95% C.I., -0.31 to 0.89)
18 Depression (CDRS; HRSD)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SSRIs and placebo on reducing depressive symptoms as measured by the Children or adolescents' Depression Rating Scale or Hamilton Depression Rating Scale in children or adolescents with OCD (N = 2; n = 163; SMD = 0.10; 95% C.I., -0.20 to 0.41)
19 Multidimensional Anxiety S	cale for Child	ren or adolescents
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on reducing anxiety as measured by the Multidimensional Anxiety Scale for Children or adolescents in children or adolescents with OCD (N = 1; n = $103$ ; SMD = $-0.25$ ; 95% C.I., $-0.67$ to $0.17$ )
20 Revised Children or adolesc	ents' Manifest	Anxiety Scale

01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if
of Huovenic	OT.	there is a clinically important difference between fluoxetine and placebo on reducing anxiety as measured by the Revised Children or adolescents' Manifest Anxiety Scale in children or adolescents with OCD ( $N = 1$ ; $n = 11$ ; SMD = 0.34; 95% C.I., -0.86 to 1.53)
21 Anxiety (MASC; RCMAS)		
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on reducing anxiety as measured by the Multidimensional Anxiety Scale for Children or adolescents or Revised Children or adolescents' Manifest Anxiety Scale in children or adolescents with OCD (N = 2; n = 114; SMD = -0.19; 95% C.I., -0.58 to 0.21)
22 Children or adolescents' Glo	bal Assessme	ent Scale
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on improving global functioning as measured by the Children or adolescents' Global Assessment Scale in children or adolescents with OCD (N = 1; n = 13; SMD = 0.69; 95% C.I., -0.44 to 1.83)
23 Systolic blood pressure - su	pine	
01 Sertraline vs. Placebo at 1 week	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on systolic blood pressure (supine) in children or adolescents with OCD ( $N = 1$ ; $n = 187$ ; SMD = 0.08; 95% C.I., -0.21 to 0.36)
02 Sertraline vs. Placebo at 4 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on systolic blood pressure (supine) in children or adolescents with OCD ( $N = 1$ ; $n = 187$ ; SMD = 0.09; C.I., -0.19 to 0.38)
03 Sertraline vs. Placebo at 12 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on systolic blood pressure (supine) in children or adolescents with OCD ( $N = 1$ ; $n = 187$ ; SMD = -0.04; 95% C.I., -0.32 to 0.25)
24 Systolic blood pressure - sta	nding	
01 Sertraline vs. Placebo at 1 week	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on systolic blood pressure (standing) in children or adolescents with OCD (N = 1; n = $187$ ; SMD = $0.09$ ; 95% C.I., -0.20 to $0.38$ )
02 Sertraline vs. Placebo at 4 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on systolic blood pressure (standing) in children or adolescents with OCD (N = 1; n = $187$ ; SMD = $-0.09$ ; 95% C.I., $-0.37$ to $0.20$ )
03 Sertraline vs. Placebo at 12 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on systolic blood pressure (standing) in children or adolescents with OCD (N = 1; n = $187$ ; SMD = $-0.12$ ; 95% C.I., $-0.41$ to 0.17)
25 Diastolic blood pressure - su	Î	
01 Sertraline vs. Placebo at 1 week	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on diastolic blood pressure (supine) in children or adolescents with OCD (N = 1; n = 187; SMD = $0.04$ ; 95% C.I., $-0.24$ to $0.33$ )

02 Sertraline vs. Placebo at 4 weeks	s3	(There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on diastolic blood pressure (supine) in children or adolescents with OCD N = 1; n = $187$ ; SMD = $-0.09$ ; $95\%$ C.I., $-0.37$ to $0.20$ )
03 Sertraline vs. Placebo at 12 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on diastolic blood pressure (supine) in children or adolescents with OCD (N = 1; n = $187$ ; SMD = $-0.08$ ; $95\%$ C.I., $-0.37$ to $0.21$ )
26 Diastolic blood pressure - st	anding	
01 Sertraline vs. Placebo at 1 week	s3	(There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on diastolic blood pressure (standing) in children or adolescents with OCD N = 1; $n = 187$ ; SMD = -0.01; 95% C.I., -0.30 to 0.28)
02 Sertraline vs. Placebo at 4 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on diastolic blood pressure (standing) in children or adolescents with OCD (N = $1$ ; n = $187$ ; SMD = $-0.02$ ; $95\%$ C.I., $-0.31$ to $0.27$ )
03 Sertraline vs. Placebo at 12 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on diastolic blood pressure (standing) in children or adolescents with OCD (N = $1$ ; n = $187$ ; SMD = $-0.08$ ; 95% C.I., $-0.36$ to $0.21$ )
27 Heart rate – supine		
01 Sertraline vs. Placebo at 1 week	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on heart rate (supine) in children or adolescents with OCD (N = 1; n = 187; SMD = $-0.19$ ; 95% C.I., $-0.47$ to $0.10$ )
02 Sertraline vs. Placebo at 4 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on heart rate (supine) in children or adolescents with OCD (N = 1; n = 187; SMD = $-0.10$ ; 95% C.I., $-0.39$ to $0.18$ )
03 Sertraline vs. Placebo at 12 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on heart rate (supine) in children or adolescents with OCD (N = 1; n = 187; SMD = $-0.09$ ; 95% C.I., $-0.38$ to $0.19$ )
28 Heart rate - standing	<u>-</u>	,
01 Sertraline vs. Placebo at 1 week	s2x	There is limited evidence suggesting a difference favouring sertraline over placebo on heart rate (standing) in children or adolescents with OCD (N = 1; n = 187; SMD = $-0.34$ ; 95% C.I., $-0.63$ to $-0.06$ )
02 Sertraline vs. Placebo at 4 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on heart rate (standing) in children or adolescents with OCD ( $N = 1$ ; $n = 187$ ; SMD = -0.05; 95% C.I., -0.33 to 0.24)
03 Sertraline vs. Placebo at 12 weeks	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between sertraline and placebo on heart rate (standing) in children or adolescents with OCD ( $N = 1$ ; $n = 187$ ; SMD = -0.08; 95% C.I., -0.37 to 0.21)
29 Suicidality (Columbia reclassification - "Outcome 3: definitive suicidal behaviour/ideation")	s2	It is possible that SSRIs when compared to placebo increase the risk of suicidal behaviour/ ideation (K = 4; N = 616; RR = 1.81; 95% C.I., 0.46 to 7.13). I
02 SSRI vs. placebo: continuation	on phase	
01 Adverse effects		

01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and placebo on the likelihood of reporting adverse effects in children or adolescents with OCD ( $N = 1$ ; $n = 18$ ; $RR = 4.67$ ; 95%
		C.I., 0.28 to 78.68)
02 Leaving study early	1	
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and placebo on the likelihood of leaving the study early in children or adolescents with OCD ( $N = 1$ ; $n = 19$ ; $RR = 1.85$ ; 95% C.I., 0.09 to 40.05)
03 CY-BOCS		
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and placebo on reducing obsessive-compulsive symptoms as measured by the CY-BOCS in children or adolescents with OCD (N = 1; n = 18; SMD = $-0.69$ ; 95% C.I., $-1.67$ to $0.30$ )
04 NIMH-OC		
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in children or adolescents with OCD $(N = 1; n = 18; SMD = -0.69; 95\% C.I., -1.67 to 0.29)$
05 Child Obsessive Compu	lsive Impact S	Scale (Parent version)
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and placebo on reducing the social impact of OCD as measured by the parent-rated Child Obsessive Compulsive Impact Scale in children or adolescents with OCD (N = 1; n = 18; SMD = -0.97; 95% C.I., -1.98 to 0.04)
06 Hamilton Rating Scale fo	or Depression	1
01 Fluoxetine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between the continuation of fluoxetine and placebo on reducing depressive symptoms as measured by the Hamilton Depression Rating Scale in children or adolescents with OCD (N = 1; n = 18; SMD = -0.41; 95% C.I., -1.37 to 0.55)
03 SSRIs vs. placebo (discor	ntinuation)	
01 Adverse events	s3	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on the likelihood of reporting adverse events in children or adolescents with OCD who were previously responsive to paroxetine $N = 1$ ; $n = 193$ ; $RR = 0.94$ ; $95\%$ C.I., $0.81$ to $1.09$ )
02 Serious adverse events	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on the likelihood of reporting serious adverse events in children or adolescents with OCD who were previously responsive to paroxetine ( $N = 1$ ; $n = 193$ ; $RR = 1.03$ ; $95\%$ C.I., $0.15$ to $7.18$ )

03 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on the likelihood of leaving the study early in children or adolescents with OCD who were previously responsive to paroxetine $(N = 1; n = 193; RR = 0.84; 95\% C.I., 0.68 to 1.06)$
04 Leaving the study early due to adverse events	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on the likelihood of leaving the study early due to adverse events in children or adolescents with OCD who were previously responsive to paroxetine ( $N = 1$ ; $n = 193$ ; $RR = 0.75$ ; 95% C.I., 0.32 to 1.78)
04 Non-responders (25% CY-BOCS)	s2x	There is limited evidence suggesting a difference favouring paroxetine over placebo on the likelihood of response, defined as a 25% or greater reduction on the CY-BOCS events in children or adolescents with OCD who were previously responsive to paroxetine $(N = 1; n = 193; RR = 0.86; 95\% C.I., 0.75 to 0.99)$
05 Non-responders (CGI < "much improved")	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on the likelihood of response defined as "much improved" or "very much improved" on the Clinical Global Impression scale in children or adolescents with OCD who were previously responsive to paroxetine ( $N = 1$ ; $n = 193$ ; $RR = 0.78$ ; $95\%$ C.I., $0.59$ to $1.04$ )
06 Relapse (CGI Global Improvement score increase)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on the likelihood of relapse, defined as (a) an increase in CGI Global Improvement score by 1 point for 2 consecutive visits, (b) an increase in CGI Global Improvement score by >=2 points at any single visit, or (c) a CGI Global Improvement Score >=5 at any time in children or adolescents with OCD who were previously responsive to paroxetine (N = 1; n = 193; RR = 0.79; 95% C.I., 0.56 to 1.13)
07 CY-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on reducing obsessive-compulsive symptoms as measured by the CY-BOCS in children or adolescents with OCD who were previously responsive to paroxetine (N = 1; n = 190; WMD = 3.3; 95% C.I., 0.83 to 5.77)
08 Hamilton Rating Scale for Depression	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on reducing depression as measured by the Hamilton Rating Scale for Depression in children or adolescents with OCD who were previously responsive to paroxetine (N = 1; n = 190; WMD = 1.2; 95% C.I., -0.29 to 2.69)
09 Hamilton Rating Scale for Anxiety	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on reducing anxiety as measured by the Hamilton Rating scale for Anxiety in children or adolescents with OCD who were previously responsive to paroxetine (N = 1; n = 190; WMD = 1.3; 95% C.I., -0.24 to 2.84)
10 Global Assessment of Functioning	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between paroxetine and placebo on the likelihood of improving global functioning as measured by the Global Assessment of Functioning scale in children or adolescents with OCD who were previously responsive to paroxetine (N = 1; n = 191; WMD = -2.3; 95% C.I., -5.74 to 1.14)

#### Clomipramine (children and adolescents)

Description	Statement level	Statement and Statistics
01 Clomipramine vs. placebo	·	
01 Leaving study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on the likelihood of leaving the study early in children with OCD (N = 2; n = 76; RR = $2.48$ ; 95% C.I., $0.62$ to $9.93$ )
02 Leaving study early due to adverse effects	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on the likelihood of leaving the study early due to adverse effects in children with OCD ( $N = 2$ ; $n = 76$ ; $RR = 3.86$ ; 95% C.I., 0.45 to 32.8)
03 Y-BOCS	s2x	There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS in children with OCD (N = 1; n = 16; SMD = -0.94; 95% C.I., -1.99 to 0.11)
04 NIMH-OC	s2x	There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in children with OCD (N = $1$ ; n = $16$ ; SMD = $-0.93$ ; $95\%$ C.I., $-1.98$ to $-0.12$ )
02 Clomipramine continuation	vs. Desipran	nine substitution
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between continuation of previous clomipramine treatment and substitution of previous clomipramine treatment with desipramine on the likelihood of leaving the study early in children with OCD (N = 1, n = 21, RR = 0.31, 95% C.I., 0.01 to 6.74)
02 Relapse (Physician's Relapse Scale)	es2x	There is limited evidence suggesting a difference favouring continuation of previous clomipramine treatment over substitution of previous clomipramine treatment with desipramine on the likelihood of relapse, as defined by the Physician's Relapse Scale, in children with OCD (N = 1; n = 20; RR = 0.2; 95% C.I., 0.06 to 0.73)
03 Comprehensive Psychopathological Rating Scale: obsessive-compulsive	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between continuation of previous clomipramine treatment and substitution of previous clomipramine treatment with desipramine on reducing obsessive-compulsive symptoms as measured by the Comprehensive Psychopathological Rating Scale - obsessive-compulsive subscale in children with OCD ( $N = 1$ ; $n = 20$ ; $SMD = -0.28$ ; $95\%$ C.I., $-1.17$ to $0.60$ )
04 NIMH-OC	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between continuation of previous clomipramine treatment and substitution of previous clomipramine treatment with desipramine on reducing obsessive-compulsive symptoms as measured by the NIMH-OC in children with OCD ( $N = 1$ ; $n = 20$ ; $SMD = -0.44$ ; $95\%$ C.I., $-1.34$ to $0.45$ )

## Pharmacological interventions (BDD)

Description	Statement level	Statement and Statistics
01 Fluoxetine vs. placebo		
01 Adverse effects	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on the likelihood of reporting adverse effects in adults with BDD ( $N = 1$ ; $n = 67$ ; $RR = 1.29$ ; 95% C.I., 0.96 to 1.75)
02 Leaving study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between fluoxetine and placebo on the likelihood of leaving the study early in adults with BDD ( $N = 1$ ; $n = 67$ ; $RR = 0.58$ ; 95% C.I., 0.15 to 2.24)
04 Non-responders	s2x	There is limited evidence suggesting a difference favouring fluoxetine over placebo on the likelihood of response, defined as a 30% or greater reduction in BDD-YBOCS scores in adults with BDD (N = 1; n = 67; RR = $0.58$ ; 95% C.I., $0.39$ to $0.85$ )
05 BDD-YBOCS	s2x	There is limited evidence suggesting a difference favouring fluoxetine over placebo on reducing BDD symptoms as measured by the BDD-YBOCS in adults with BDD (N = 1; n = $67$ ; SMD = $-0.60$ ; 95% C.I., $-1.09$ to $-0.11$ )
06 BDD-NIMH	s2x	There is limited evidence suggesting a difference favouring fluoxetine over placebo on reducing BDD symptoms as measured by the BDD-NIMH in adults with BDD (N = 1; n = 67; SMD = -0.49; 95% C.I., -0.97 to 0.00)
07 Hamilton Rating Scale for Depression	s2x	There is limited evidence suggesting a difference favouring fluoxetine over placebo on reducing depressive symptoms as measured by the HRSD in adults with BDD (N = 1; n = 67; SMD = $-0.67$ ; 95% C.I., $-1.16$ to $-0.18$ )
08 Global Assessment of Functioning Scale	s2x	There is limited evidence suggesting a difference favouring fluoxetine over placebo on improvement in global functioning as measured by the Global Assessment Functioning Scale in adults with BDD ( $N = 1$ ; $n = 67$ ; SMD = -0.83; 95% C.I., -1.33 to -0.33)
09 Social & Occupational Functioning Scale	s2x	There is limited evidence suggesting a difference favouring fluoxetine over placebo on improvement in social and occupational functioning as measured by the Social and Occupational Functioning Scale in adults with BDD ( $N = 1$ ; $n = 67$ ; $SMD = -0.68$ ; $95\%$ C.I., $-1.17$ to $-0.19$ )
02 Clomipramine vs. Desiprar	nine	
01 BDD-YBOCS at 8 weeks	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and desipramine on reducing BDD symptoms as measured by the BDD-YBOCS in adults with BDD ( $N = 1$ ; $n = 23$ ; $SMD = -0.75$ ; $95\%$ C.I., $-1.60$ to $0.10$ )
02 NIMH-BDD at 8 weeks	s2x	There is limited evidence suggesting a difference favouring clomipramine over desipramine on reducing BDD symptoms as measured by the NIMH-BDD scale in adults with BDD ( $N = 1$ ; $n = 23$ ; SMD = -1.46; 95% C.I., -2.40 to -0.52)
03 HAM-D at 8 weeks	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and desipramine on reducing depressive symptoms as measured by the Hamilton Rating Scale for Depression in adults with BDD ( $N = 1$ ; $n = 23$ ; SMD = -0.73; 95% C.I., -1.58 to 0.12)

## Psychological vs. pharmacological interventions evidence statements

Description	Statement level	Statement and Statistics
01 ERP vs. Clomipramine	1	
01 Y-BOCS or Compulsive Checklist	s2	There is limited evidence suggesting a difference favouring exposure and response prevention over clomipramine on reducing obsessive-compulsive symptoms as measured on the Y-BOCS or the Compulsive Checklist (N = 2; n = 68; SMD = -0.67; 95% CI, -1.16 to -0.17). I
02 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure and response prevention and clomipramine on the likelihood of leaving the study early ( $N = 1$ ; $n = 84$ ; $RR = 1.02$ ; 95% CI, 0.62 to 1.67). I
03 Non-responders (CGI)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure and response prevention and clomipramine on the likelihood of response, defined as 'much improved' or 'very much improved' on the CGI ( $N = 1$ ; $n = 84$ ; $RR = 1.33$ ; 95% CI, 0.92 to 1.92). I
02 Children: CBT vs. Clomipra	mine	
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and Clomipramine on the likelihood of leaving the study early $(N = 1; n = 23; RR = 2.36; 95\% CI, 0.11 to 52.41)$ . I
02 Non-responders (CY-BOCS 30%)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and Clomipramine on the likelihood of response, defined as a 30% or more reduction on the CY-BOCS ( $N = 1$ ; $n = 23$ ; $RR = 0.77$ ; 95% CI, 0.3 to 1.94). I
03 CY-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and Clomipramine on reducing obsessive-compulsive symptoms as measured on the Y-BOCS (N = 1; n = 22; SMD = $-0.79$ ; 95% CI, $-1.66$ to 0.09). I
04 Leyton Inventory- Child Version	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and Clomipramine on reducing obsessive-compulsive symptoms as measured by the Leyton Inventory (CV) (N = 1; n = 22; SMD = $-0.47$ ; 95% CI, $-1.32$ to $0.38$ ). I
05 Child Behaviour Checklist	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and Clomipramine on reducing behavioural and emotional problems as measured by the CBC (N = 1; n = 19; SMD = $-0.24$ ; 95% CI, $-1.16$ to 0.67). I
06 Children's Depression Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and Clomipramine on reducing depressive symptoms as measured by the CDI ( $N = 1$ ; $n = 20$ ; SMD = -0.29; 95% CI, -1.18 to 0.59). I
02 Children: CBT v Sertraline		•

01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and Sertraline on the likelihood of leaving the study early ( $N = 1$ ; $n = 56$ ; $RR = 1.5$ ; 95% CI, 0.27 to 8.3). I
02 Remission (CY-BOCS < 11)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and Sertraline on the likelihood of remission, defined as a score of less than or equal to 10 on the CY-BOCS ( $N = 1$ ; $n = 56$ ; $RR = 0.77$ ; 95% CI, 0.54 to 1.1). I
03 CY-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and Sertraline on reducing obsessive-compulsive symptoms as measured on the CYBOCS ( $N = 1$ ; $n = 56$ ; $SMD = -0.27$ ; $95\%$ CI, $-0.79$ to $0.26$ ). I

# Combination therapy evidence statements

Description	Statement level	Statement and Statistics
01 ERP + SRI v ERP	•	
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy + SRI and behaviour therapy on the likelihood of leaving the study early ( $N = 3$ ; $n = 160$ ; $RR = 1.18$ ; 95% CI, 0.75 to 1.85). I
02 Leaving the study early due to adverse effects	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention and placebo on the likelihood of leaving the study early due to adverse effects ( $N = 1$ ; $n = 40$ ; $RR = 2$ ; 95% CI, 0.2 to 20.33). I
03 Non-responders (Y-BOCS 35%)	s2x	There is limited evidence suggesting a difference favouring multimodal behaviour therapy + fluvoxamine over multimodal behaviour therapy + placebo on the likelihood of treatment response, defined as a 35% or greater reduction on the Y-BOCS (N = 1; n = 49; RR = 0.31; 95% CI, 0.10 to 1.00). I
04 Non-responders (CGI)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy + clomipramine and behaviour therapy on the likelihood of response, defined as "much improved" or "very much improved" on the Clinical Global Improvement scale (N = 1; n = 70; RR = 0.71; 95% CI, 0.41 to 1.23). I
06 Global criterion of non-impr	ovement (du	ration of rituals)
01 ERP+Fluvoxamine v ERP+placebo: post-treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention and placebo on the likelihood of improvement, defined as a reduction in duration of rituals per day greater than 30% at the end of treatment ( $N = 1$ ; $n = 40$ ; $RR = 0.64$ ; $95\%$ CI, $0.37$ to $1.13$ ). I
02 ERP+Fluvoxamine v ERP+placebo: 6 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention and placebo on the likelihood of improvement, defined as a reduction in duration of rituals per day greater than 30% at 6 months follow-up ( $N = 1$ ; $n = 40$ ; $RR = 0.79$ ; 95% CI, 0.48 to 1.28). I

03 ERP+Fluvoxamine v	s4	The evidence is inconclusive and so it is not possible to determine if
ERP+placebo: 1 year follow-up		there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention and placebo on the likelihood of improvement, defined as a reduction in duration of rituals per day greater than 30% at 1 year follow-up ( $N = 1$ ; $n = 40$ ; $RR = 0.92$ ; 95% CI, 0.54 to 1.56). I
07 Y-BOCS	s2x	There is limited evidence suggesting a difference favouring behaviour therapy + SRI over behaviour therapy on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = $3$ ; n = $126$ ; SMD = $-0.37$ ; 95% CI, $-0.72$ to $-0.01$ ). I
08 Padua Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention on reducing obsessive-compulsive symptoms as measured by the Padua Inventory ( $N = 1$ ; $n = 37$ ; SMD = -0.62; 95% CI, -1.28 to 0.04). I
09 Depression scales (post treatment)	s2x	There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing depressive symptoms at the end of treatment (N = $4$ ; n = $137$ ; SMD = $-0.73$ ; 95% CI, $-1.08$ to $-0.38$ ). I
10 Hamilton Depression Rating	scale (follow	-up)
01 ERP + Fluvoxamine v ERP + placebo (6 months follow-up)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention and placebo on reducing depressive symptoms at 6 months follow-up as measured by the Hamilton Depression scale (N = 1; n = 26; SMD = -0.17; 95% CI, -0.94 to 0.6). I
02 ERP + Fluvoxamine v ERP + placebo (1 year follow-up)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention and placebo on reducing depressive symptoms at 1 year follow-up as measured by the Hamilton Depression scale ( $N = 1$ ; $n = 23$ ; SMD = 0.28; 95% CI, -0.54 to 1.1). I
11 Symptom Checklist-90	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention on reducing psychological distress as measured by the Symptom Checklist-90 (N = 1; n = 37; SMD = -0.54; 95% CI, -1.20 to 0.11). I
12 Anxiety Discomfort Scale: patient's ratings	s2x	There is limited evidence suggesting a difference favouring exposure with response-prevention + fluvoxamine over exposure with response-prevention on reducing anxiety and discomfort as measured by the patient-rated Anxiety Discomfort Scale (N = 1; n = 37; SMD = $-0.73$ ; 95% CI, $-1.4$ to $-0.06$ ). I
13 Anxiety Discomfort Scale: therapist's ratings	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention on reducing anxiety and discomfort as measured by the therapist-rated Anxiety Discomfort Scale (N = 1; n = 37; SMD = -0.6; 95% CI, -1.26 to 0.06). I
14 Anxiety Discomfort Scale assessor's ratings	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention on reducing anxiety and discomfort as measured by the assessor-rated Anxiety Discomfort Scale ( $N = 1$ ; $n = 37$ ; SMD = -0.58;

		95% CI, -1.24 to 0.08). I
15 Clinical Anxiety Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between multimodal behaviour therapy + fluvoxamine and multimodal behaviour therapy + placebo on reducing anxiety symptoms as measured by the Clinical Anxiety Scale (N = 1; n = 49; SMD = -0.21; 95% CI, -0.78 to 0.35). I
16 Global Assessment Scale	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between multimodal behaviour therapy + fluvoxamine and multimodal behaviour therapy + placebo on improving functioning as measured by the Global Assessment Scale ( $N = 1$ ; $n = 49$ ; SMD = -0.21; 95% CI, -0.77 to 0.35). I
17 CGI - therapist rating	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between multimodal behaviour therapy + fluvoxamine and multimodal behaviour therapy + placebo on improving functioning as measured by the therapist-rated Clinical Global Improvement scale (N = 1; n = 49; SMD = -0.27; 95% CI, -0.84 to 0.29). I
18 CGI - patient rating	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between multimodal behaviour therapy + fluvoxamine and multimodal behaviour therapy + placebo on improving functioning as measured by the patient-rated Clinical Global Improvement scale ( $N = 1$ ; $n = 49$ ; $SMD = -0.48$ ; $95\%$ CI, $-1.04$ to $0.09$ ). I
19 Compulsive activity checklist (post treatment)	s2x	There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing compulsive symptoms as measured by the Compulsive Activity Checklist at the end of treatment (N = $2$ ; n = $51$ ; SMD = $-0.55$ ; $95\%$ CI, $-1.12$ to $0.01$ ). I
20 Compulsive activity checkli	st (follow-up)	
01 ERP + Fluvoxamine v ERP + placebo (1 year follow-up)	Fs4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy + SRIs and behaviour therapy on reducing compulsive symptoms as measured by the Compulsive Activity Checklist at 1 year follow-up ( $N = 1$ ; $n = 23$ ; SMD = -0.47; 95% CI, -1.3 to 0.36). I
21 Target rituals (assessor): time (post-treatment)	s2x	There is limited evidence suggesting a difference favouring BT +SRIs over BT on reducing time spent in rituals at the end of treatment ( $N = 2$ ; $n = 51$ ; SMD = -0.81; 95% CI, -1.38 to -0.23). I
22 Target rituals (assessor): time (follow-up)	s2x	There is limited evidence suggesting a difference favouring behaviour therapy + SRIs over behaviour therapy on reducing time spent in rituals at the end of treatment (N = $2$ ; n = $51$ ; SMD = $-0.72$ ; $95\%$ CI, $-1.29$ to $-0.15$ ). I
01 ERP + Fluvoxamine v ERP + placebo (6 months follow-up)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention + placebo on reducing time spent in rituals at 6 months follow-up ( $N = 1$ ; $n = 26$ ; $SMD = -0.17$ ; $95\%$ CI, $-0.94$ to $0.60$ ). I
02 ERP + Fluvoxamine v ERP + placebo (1 year follow-up)	+s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention + placebo on reducing time spent in rituals at 1 year follow-up ( $N = 1$ ; $n = 23$ ; $SMD = 0.03$ ; $95\%$ CI, $-0.78$ to $0.85$ ). I

23 Target rituals (assessor):	s2x	There is limited evidence suggesting a difference favouring BT + SRIs
discomfort (post-treatment)		over BT on reducing discomfort due to rituals at end of treatment (N = 2; n = 51; SMD = -0.88; 95% CI, -1.46 to -0.30). I
24 Target rituals (assessor): disc	comfort (follo	w-up)
01 ERP + Fluvoxamine v ERP +	s4	The evidence is inconclusive and so it is not possible to determine if
placebo (6 months follow-up)		there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention + placebo on reducing discomfort due to rituals at 6 months follow-up ( $N = 1$ ; $n = 26$ ; $SMD = -0.11$ ; $95\%$ CI, $-0.88$ to $0.66$ ). I
02 ERP + Fluvoxamine v ERP + placebo (1 year follow-up)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with
		response-prevention + fluvoxamine and exposure with response-prevention + placebo on reducing discomfort due to rituals at 1 year follow-up ( $N = 1$ ; $n = 23$ ; SMD = 0.05; 95% CI, -0.77 to 0.87). I
25 Target rituals (assessor): dur	ation per day	
01 ERP + Fluvoxamine v ERP + placebo (post treatment)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention + placebo on reducing duration of rituals per day at end of treatment ( $N = 1$ ; $n = 31$ ; $SMD = -0.64$ ; $95\%$ CI, $-1.37$ to $0.08$ ). I
02 ERP + Fluvoxamine v ERP +	·s4	The evidence is inconclusive and so it is not possible to determine if
placebo (6 months follow-up)		there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention + placebo on reducing duration of rituals per day at 6 months follow-up ( $N = 1$ ; $n = 26$ ; SMD = -0.25; 95% CI, -1.03 to 0.52). I
03 ERP + Fluvoxamine v ERP + placebo (1 year follow-up)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response-prevention + fluvoxamine and exposure with response-prevention + placebo on reducing duration of rituals per day at 1 year follow-up ( $N = 1$ ; $n = 23$ ; $SMD = -0.21$ ; $95\%$ CI, $-1.04$ to $0.61$ ). I
26 Behavioural avoidance test: discomfort (post treatment)	s2x	There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing discomfort as measured by the Behavioural Avoidance Test discomfort subscale at end of treatment (N = 2; n = $51$ ; SMD = $-0.57$ ; 95% CI, $-1.14$ to $-0.01$ ). I
27 Behavioural avoidance test:	discomfort (fo	ollow-up)
01 ERP + Fluvoxamine v ERP + placebo (1 year follow-up)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response prevention + fluvoxamine and exposure with response-prevention + placebo on reducing discomfort as measured by the Behavioural Avoidance test discomfort subscale at 1 year follow-up $(N = 1; n = 23; SMD = -0.14; 95\% CI, -0.96 to 0.68)$ . I
03 ERP + Clomipramine v Clon	nipramine	
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy plus clomipramine and clomipramine on the likelihood of leaving the study early ( $N = 1$ ; $n = 80$ ; $RR = 1$ ; 95% CI, 0.59 to 1.67). I
03 Non-responders (CGI)	s2x	There is limited evidence suggesting a difference favouring behaviour therapy plus clomipramine over clomipramine on the likelihood of response, defined as "much improved" or "very much improved" on the Clinical Global Improvement scale ( $N = 1$ ; $n = 80$ ; $RR = 0.53$ ; 95% CI, 0.33 to 0.87). I

05 Y-BOCS	s2x	There is limited evidence suggesting a difference favouring behaviour
		therapy plus clomipramine over clomipramine on reducing obsessive compulsive symptoms as measured by the Y-BOCS (N = 1; n = 46; SMD = -0.95; 95% CI, -1.57 to -0.33). I
04 ERP + Fluvoxamine v Anti-I	ERP + Fluvox	amine
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on the likelihood of leaving the study early ( $N = 1$ ; $n = 40$ ; $RR = 0.57$ ; $95\%$ CI, $0.20$ to $1.65$ ). I
02 Leaving the study early due to adverse effects	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on the likelihood of leaving the study early due to adverse effects (N = 1; n = 40; RR = 1.00; 95% CI, 0.16 to 6.42). I
03 Global criterion of non-impr	ovement (du	ration of rituals)
01 Post-treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on the likelihood of improvement, defined as a reduction in duration of rituals per day greater than $30\%$ at end of treatment (N = 1; n = $40$ ; RR = $0.69$ ; $95\%$ CI, $0.39$ to $1.24$ ). I
02 6 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on the likelihood of improvement, defined as a reduction in duration of rituals per day greater than 30% at 6 months follow-up (N = 1; n = 40; RR = 0.73; 95% CI, 0.46 to 1.17). I
03 1 year follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on the likelihood of improvement, defined as a reduction in duration of rituals per day greater than 30% at 1 year follow-up ( $N = 1$ ; $n = 40$ ; $RR = 0.79$ ; 95% CI, 0.48 to 1.28). I
04 Hamilton Depression Scale	•	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing depressive symptoms as measured by the Hamilton Rating Scale for Depression at the end of treatment ( $N = 1$ ; $n = 29$ ; $SMD = -0.20$ ; $95\%$ CI, $-0.94$ to $0.53$ ). I
02 6 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing depressive symptoms as measured by the Hamilton Rating Scale for Depression at 6 months follow-up ( $N = 1$ ; $n = 25$ ; SMD = -0.09; 95% CI, -0.88 to 0.70). I
03 1 year follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing depressive symptoms as measured by the Hamilton Rating Scale for Depression at 1 year follow-up ( $N = 1$ ; $n = 22$ ; SMD = 0.50; 95% CI, -0.36 to 1.35). I
05 Target rituals (assessor): tim	e	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing time spent in rituals at the end of treatment ( $N=1$ ; $n=29$ ; $SMD=-0.56$ ; $95\%$ CI, -1.31 to 0.19). I

	1 .	
02 6 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing time spent in rituals at 6 months follow-up (N= 1; n = 25; SMD = -0.45; 95% CI, -1.25 to 0.35). I
03 1 year follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing time spent in rituals at 1 year follow-up ( $N=1$ ; $n=22$ ; $SMD=-0.17$ ; $95\%$ CI, $-1.01$ to $0.67$ ). I
06 Target rituals (assessor):	discomfort	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing discomfort due to rituals at the end of treatment (N= 1; n = 29; SMD = -0.11; 95% CI, -0.84 to 0.62). I
02 6 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing discomfort due to rituals at 6 months follow-up (N= 1; n = 25; SMD = -0.11; 95% CI, -0.90 to 0.69). I
03 1 year follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing discomfort due to rituals at 1 year follow-up ( $N=1$ ; $n=22$ ; $SMD=0.18$ ; $95\%$ CI, -0.66 to 1.03). I
07 Target rituals (assessor):	duration per	day
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing duration of rituals per day at the end of treatment ( $N=1$ ; $n=29$ ; $SMD=-0.06$ ; $95\%$ CI, $-0.79$ to $0.68$ ). I
02 6 months follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing duration of rituals per day at 6 months follow-up (N= 1; n = 25; SMD = -0.03; 95% CI, -0.82 to 0.76). I
03 1 year follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing duration of rituals per day at 1 year follow-up (N= 1; n = 22; SMD = -0.17; 95% CI, -1.01 to 0.67). I
08 Behavioural avoidance t	est: discomfo	rt
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing discomfort as measured by the Behavioural Avoidance test discomfort scale at the end of treatment ( $N = 1$ ; $n = 29$ ; SMD = -0.34; 95% CI, -1.08 to 0.40). I
02 1 year follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing discomfort as measured by the Behavioural Avoidance test discomfort scale at 1 year follow-up ( $N = 1$ ; $n = 22$ ; SMD = -0.01; 95% CI, -0.85 to 0.83). I
09 Compulsive activity che	cklist	
01 Post treatment	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing compulsive symptoms as measured by the Compulsive Activity Checklist at the end of

		treatment (N = 1; n = 29; SMD = -0.44; 95% CI, -1.18 to 0.30). I
02 1 year follow-up	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and Anti-ERP + fluvoxamine on reducing compulsive symptoms as measured by the Compulsive Activity Checklist at 1 year follow-up ( $N = 1$ ; $n = 22$ ; SMD = -0.31; 95% CI, -1.15 to 0.54). I
05 CT + Fluvoxamine v CT		
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between cognitive therapy + fluvoxamine and cognitive therapy on the likelihood of leaving the study early ( $N = 1$ ; $n = 49$ ; $RR = 1.74$ ; 95% CI, 0.75 to 4.03). I
02 Y-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between cognitive therapy + fluvoxamine and cognitive therapy on reducing obsessive-compulsive symptoms as measured by the Y-BOCS ( $N = 1$ ; $n = 33$ ; SMD = 0.25; 95% CI, -0.44 to 0.94). I
03 Padua Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between cognitive therapy + fluvoxamine and cognitive therapy on reducing obsessive-compulsive symptoms as measured by the Padua Inventory ( $N = 1$ ; $n = 33$ ; SMD = -0.25; 95% CI, -0.95 to 0.44). I
04 Beck Depression Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between cognitive therapy + fluvoxamine and cognitive therapy on reducing depressive symptoms as measured by the Beck Depression Inventory (N = 1; n = 33; SMD = 0.13; 95% CI, -0.56 to 0.82). I
05 Symptom Checklist-90	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CT + fluvoxamine and CT on reducing psychological distress as measured by the Symptom Checklist-90 (N = 1; n = 33; SMD = -0.03; 95% CI, -0.72 to 0.66). I
06 Anxiety Discomfort Scale: patient's ratings	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between cognitive therapy + fluvoxamine and cognitive therapy on reducing anxiety and discomfort as measured by the patient-rated Anxiety Discomfort Scale ( $N = 1$ ; $n = 33$ ; SMD = 0.06; 95% CI, -0.63 to 0.75). I
07 Anxiety Discomfort Scale: therapist's ratings	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between cognitive therapy + fluvoxamine and cognitive therapy on reducing anxiety and discomfort as measured by the therapist-rated Anxiety Discomfort Scale ( $N = 1$ ; $n = 33$ ; SMD = 0.05; 95% CI, -0.64 to 0.74). I
08 Anxiety Discomfort Scale: assessor's ratings	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between cognitive therapy + fluvoxamine and cognitive therapy on reducing anxiety and discomfort as measured by the assessor-rated Anxiety Discomfort Scale ( $N = 1$ ; $n = 33$ ; SMD = 0.16; 95% CI, -0.53 to 0.85). I
08 ERP + Fluvoxamine v CT + I	Fluvoxamine	
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response prevention + fluvoxamine and cognitive therapy + fluvoxamine on the likelihood of leaving the study early $(N = 1; n = 1)$

		52; RR = 0.86; 95% CI, 0.43 to 1.7). I
02 Y-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response prevention + fluvoxamine and cognitive therapy + fluvoxamine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 32; SMD = -0.48; 95% CI, -1.19 to 0.23). I
03 Padua Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response prevention + fluvoxamine and cognitive therapy + fluvoxamine on reducing obsessive-compulsive symptoms as measured by the Padua Inventory ( $N = 1$ ; $n = 32$ ; $SMD = -0.01$ ; $95\%$ CI, $-0.71$ to $0.68$ ). I
04 Beck Depression Inventory	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response prevention + fluvoxamine and cognitive therapy + fluvoxamine on reducing depressive symptoms as measured by the Beck Depression Inventory ( $N = 1$ ; $n = 32$ ; $SMD = -0.1$ ; $95\%$ CI, $-0.8$ to $0.6$ ). I
05 Symptom Checklist-90	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and CT + fluvoxamine on reducing psychological distress as measured by the Symptom Checklist-90 (N = 1; n = 32; SMD = -0.15; 95% CI, -0.85 to 0.55). I
06 Anxiety Discomfort Scale: patient's ratings	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response prevention + fluvoxamine and cognitive therapy + fluvoxamine on reducing anxiety and discomfort as measured by the patient-rated Anxiety Discomfort Scale (N = 1; n = 32; SMD = 0; 95% CI, -0.7 to 0.7). I
07 Anxiety Discomfort Scale: therapist's ratings	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response prevention + fluvoxamine and cognitive therapy + fluvoxamine on reducing anxiety and discomfort as measured by the therapist-rated Anxiety Discomfort Scale (N = 1; n = 32; SMD = 0.06; 95% CI, -0.64 to 0.76). I
08 Anxiety Discomfort Scale: assessor's ratings	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure with response prevention + fluvoxamine and cognitive therapy + fluvoxamine on reducing anxiety and discomfort as measured by the assessor-rated Anxiety Discomfort Scale (N = 1; n = 32; SMD = 0.05; 95% CI, -0.64 to 0.75). I
09 Children: ERP + Fluvoxamiı		
01 Non-responder (Y-BOCS rel		
01 At 43 weeks	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and fluvoxamine alone on the likelihood of response 43 weeks after the beginning of treatment ( $N = 1$ ; $n = 10$ ; $RR = 0.14$ ; 95% CI, 0.01 to 2.21). I

02 At 52 weeks	s4	The evidence is inconclusive and so it is not possible to determine if
02 11 32 WCCR3	51	there is a clinically important difference between ERP + fluvoxamine
		and fluvoxamine alone on the likelihood of response 52 weeks after
		the beginning of treatment (N = 1; n = 10; RR = 0.20; 95% CI, 0.01 to 3.35). I
03 At 2 years	s4	The evidence is inconclusive and so it is not possible to determine if
		there is a clinically important difference between ERP + fluvoxamine
		and fluvoxamine alone on the likelihood of response 2 years after the
		beginning of treatment (N = 1; n = 10; RR = 0.20; 95% CI, 0.01 to 3.35).
02 CY-BOCS		<u> </u>
01 At 43 weeks	s4	The evidence is inconclusive and so it is not possible to determine if
		there is a clinically important difference between ERP + fluvoxamine
		and fluvoxamine alone on reducing obsessive-compulsive symptoms
		as measured by the CY-BOCS 43 weeks after the beginning of
		treatment (N = 1; n = 10; SMD = -1.44; 95% CI, -2.93 to 0.04). I
02 At 52 weeks	s2x	There is limited evidence suggesting a difference favouring ERP +
		fluvoxamine over fluvoxamine alone on reducing obsessive-
		compulsive symptoms as measured by the CY-BOCS 52 weeks after
		the beginning of treatment (N = 1; n = 10; SMD = -1.50; 95% CI, -3.00 to 0.00). I
03 At 2 years	s4	The evidence is inconclusive and so it is not possible to determine if
		there is a clinically important difference between ERP + fluvoxamine
		and fluvoxamine alone on reducing obsessive-compulsive symptoms
		as measured by the CY-BOCS 2 years after the beginning of treatment
		(N = 1; n = 10; SMD = -1.23; 95% CI, -2.65 to 0.19). I
03 NIMH-GOCS	1	
01 At 43 weeks	s4	The evidence is inconclusive and so it is not possible to determine if
		there is a clinically important difference between ERP + fluvoxamine
		and fluvoxamine alone on reducing obsessive-compulsive symptoms
		as measured by the NIMH-GOCS 43 weeks after the beginning of treatment ( $N = 1$ ; $n = 10$ ; $SMD = -0.54$ ; $95\%$ CI, $-1.81$ to $0.74$ ). I
02 At 52 weeks	s4	The evidence is inconclusive and so it is not possible to determine if
02 III 32 WCCR3	34	there is a clinically important difference between ERP + fluvoxamine
		and fluvoxamine alone on reducing obsessive-compulsive symptoms
		as measured by the NIMH-GOCS 52 weeks after the beginning of
		treatment (N = 1; n = 10; SMD = -0.63; 95% CI, -1.92 to 0.66). I
03 At 2 years	s4	The evidence is inconclusive and so it is not possible to determine if
		there is a clinically important difference between ERP + fluvoxamine
		and fluvoxamine alone on reducing obsessive-compulsive symptoms
		as measured by the NIMH-GOCS 2 years after the beginning of
		treatment (N = 1; n = 10; SMD = $-0.81$ ; 95% CI, $-2.13$ to 0.51). I
09 Children: CBT + Sertraline v	1	
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if
		there is a clinically important difference between CBT + sertraline and
		Sertraline on the likelihood of leaving the study early (N = 1; n = 56; RR = 1; 95% CI, 0.22 to 4.54). I
02 Leaving the study early due	s4	The evidence is inconclusive and so it is not possible to determine if
to adverse events		there is a clinically important difference between CBT + sertraline and
		Sertraline on the likelihood of leaving the study early due to adverse
		effects (N = 1; n = 56; RR = 1; 95% CI, 0.07 to 15.21). I

02 Remission (CY-BOCS < 11)	s2	There is limited evidence suggesting a difference favouring CBT + sertraline over Sertraline on the likelihood of relapse, defined as a score of less than or equal to 10 on the CY-BOCS (N = 1; n = 56; RR = 0.59; 95% CI, 0.38 to 0.92). I
03 CY-BOCS	s2	There is limited evidence suggesting a difference favouring CBT + sertraline over Sertraline on reducing the severity of obsessive-compulsive symptoms as measured by the CY-BOCS (N = 1; n = 56; SMD = $-0.59$ ; 95% CI, $-1.13$ to $-0.05$ ). I
09 Children: CBT + Sertraline v	CBT	
01 Leaving the study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT + sertraline and CBT on the likelihood of leaving the study early (N = 1; n = 56; RR = $1$ ; 95% CI, 0.22 to 4.54). I
02 Remission (CY-BOCS < 11)	s2	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT + sertraline and CBT on the likelihood of relapse, defined as a score of less than or equal to 10 on the CY-BOCS (N = 1; n = $56$ ; RR = $0.76$ ; $95\%$ CI, $0.47$ to $1.26$ ). I
03 CY-BOCS	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT + sertraline and CBT on reducing the severity of obsessive-compulsive symptoms as measured by the CY-BOCS ( $N = 1$ ; $n = 56$ ; $RR = -0.3$ ; 95% CI, -0.83 to 0.22). I

#### Other medical interventions evidence statements

Description	Statement level	Statement and Statistics
01 Stereotactic anterior capsulo	tomy vs. cing	ulotomy
01 CPRS @ 3 months: change	s4	
score		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between capsulotomy and cingulotomy on reducing psychopathological symptoms as measured by the CPRS ( $N = 1$ ; $n = 4$ ; $SMD = -5.79$ ; $95\%$ CI, $-38.62$ to $27.03$ ). I
02 CPRS @ 6 months: change	s4	
score		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between capsulotomy and cingulotomy on reducing psychopathological symptoms as measured by the CPRS ( $N = 1$ ; $n = 4$ ; $SMD = -0.3$ ; $95\%$ CI, $-2.89$ to $2.29$ ). I
03 CPRS @ 12 months: change	s4	
score		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between capsulotomy and cingulotomy on reducing psychopathological symptoms as measured by the CPRS ( $N = 1$ ; $n = 4$ ; $SMD = 0.46$ ; $95\%$ CI, $-2.78$ to $3.7$ ). I
04 HAM-D @ 3 months:	s4	
change score		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between capsulotomy and cingulotomy on reducing depressive symptoms as measured by the HAM-D ( $N = 1$ ; $n = 4$ ; $SMD = 0.19$ ; 95% CI, -2.05 to 2.43). I

05 HAM-D @ 6 months:	s4	
change score	31	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between capsulotomy and cingulotomy on reducing depressive symptoms as measured by the HAM-D ( $N = 1$ ; $n = 4$ ; $SMD = 0$ ; $95\%$ CI, $-1.96$ to $1.96$ ). I
06 HAM-D @ 12 months:	s4	
change score		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between capsulotomy and cingulotomy on reducing depressive symptoms as measured by the HAM-D (N = 1; n = 4; SMD = $0.48$ ; 95% CI, $-2.89$ to $3.86$ ). I
07 HAM-A @ 3 months: change score	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between capsulotomy and cingulotomy on reducing anxiety symptoms as measured by the HAM-A ( $N = 1$ ; $n = 4$ ; SMD = 0.5; 95% CI, -2.94 to 3.95). I
08 HAM-A @ 6 months:	s4	
change score		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between capsulotomy and cingulotomy on reducing anxiety symptoms as measured by the HAM-A ( $N = 1$ ; $n = 4$ ; SMD = 0.38; 95% CI, -2.54 to 3.31). I
09 HAM-A @ 12 months:	s4	
change score		The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between capsulotomy and cingulotomy on reducing anxiety symptoms as measured by the HAM-A ( $N = 1$ ; $n = 4$ ; SMD = 0.47; 95% CI, -2.84 to 3.79). I
02 Repetitive transcranial mag	netic stim	ulation: active vs. placebo
01 Adverse effects	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between active RTMS and placebo RTMS on the likelihood of reporting adverse effects (N = 1; n = 18; RR = 2.45; 95% CI, 0.11 to 53.25). I
02 Non-responders (Y-BOCS 40%)	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between active RTMS and placebo RTMS on the likelihood of response, defined as a reduction of $40\%$ or greater on the Y-BOCS, (N = 1; n = 18; RR = 0.91; 95% CI, 0.61 to 1.37). I
03 Y-BOCS: change score	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between active RTMS and placebo RTMS on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 18; SMD = -0.45; 95% CI, -1.39 to 0.5). I
04 HAM-D: change score	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between active RTMS and placebo RTMS on reducing depressive symptoms as measured by the HAM-D ( $N = 1$ ; $n = 18$ ; $SMD = -0.17$ ; $95\%$ CI, $-1.1$ to $0.76$ ). I
03 Repetitive transcranial mag	netic stim	ulation: right vs. left
01 Y-BOCS @ post-treatment	1.	
01 Right prefrontal vs. left prefrontal	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between right prefrontal RTMS and left prefrontal RTMS on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 12; SMD = 0.03; 95% CI, -1.1 to 1.16). I
02 BDI @ post-treatment		

01 Right prefrontal vs. left prefrontal	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between right prefrontal RTMS and left prefrontal RTMS on reducing depressive symptoms as measured by the BDI ( $N = 1$ ; $n = 12$ ; $SMD = 0.48$ ; $95\%$ CI, $-0.67$ to $1.64$ ). I
03 MADRS @ post-treatment		
01 Right prefrontal vs. left prefrontal	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between right prefrontal RTMS and left prefrontal RTMS on reducing depressive symptoms as measured by the MADRS (N = 1; n = 12; SMD = $0.95$ ; 95% CI, - $0.27$ to $2.18$ ). I
04 STAI-S @ post-treatment		
01 Right prefrontal vs. left prefrontal	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between right prefrontal RTMS and left prefrontal RTMS on reducing anxiety symptoms as measured by the STAI-S (N = 1; n = 12; SMD = $0.04$ ; 95% CI, -1.1 to 1.17). I
05 Y-BOCS @ 1 month follow	-up	
01 Right prefrontal vs. left prefrontal	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between right prefrontal RTMS and left prefrontal RTMS on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 12; SMD = -0.64; 95% CI, -1.81 to 0.54). I
06 BDI @ 1 month follow-up		
01 Right prefrontal vs. left prefrontal	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between right prefrontal RTMS and left prefrontal RTMS on reducing depressive symptoms as measured by the BDI ( $N = 1$ ; $n = 12$ ; $SMD = 0.06$ ; $95\%$ CI, $-1.07$ to $1.19$ ). I
07 MADRS @ 1 month follow	√-up	
01 Right prefrontal vs. left prefrontal	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between right prefrontal RTMS and left prefrontal RTMS on reducing depressive symptoms as measured by the MADRS (N = 1; n = 12; SMD = 0.67; 95% CI, -0.51 to 1.85). I
08 STAI-S @ 1 month follow-	up	
01 Right prefrontal vs. left prefrontal	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between right prefrontal RTMS and left prefrontal RTMS on reducing anxiety symptoms as measured by the STAI-S ( $N = 1$ ; $n = 12$ ; $SMD = -0.05$ ; $95\%$ CI, $-1.18$ to $1.08$ ). I
09 Non-responders (Y-BOCS	40%) @ 1 r	nonth follow-up
01 Right prefrontal vs. left prefrontal	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between right prefrontal RTMS and left prefrontal RTMS on the likelihood of response, defined as a reduction of $40\%$ or greater on the Y-BOCS, (N = 1; n = 12; RR = 1; 95% CI, 0.2 to 4.95). I
04 Plasma exchange vs. IV in	nmunoglob	oulin vs. placebo (child/adolescent: at 1 month after start of treatment)
01 Adverse effects		

01 Plasma exchange vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and placebo on the likelihood of reporting adverse effects ( $N = 1$ ; $n = 20$ ; $RR = 3.5$ ; 95% CI, 0.95 to 12.9). I
02 IV immunoglobulin vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between IV immunoglobulin and placebo on the likelihood of reporting adverse effects (N = 1; n = 20; RR = 3; 95% CI, 0.79 to 11.44). I
03 Plasma exchange vs. IV immunoglobulin	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on the likelihood of reporting adverse effects $(N = 1; n = 20; RR = 1.17; 95\% CI, 0.61 to 2.23)$ . I
02 Leaving study early	<b>'</b>	
02 IV immunoglobulin vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between IV immunoglobulin and placebo on the likelihood of leaving the study early ( $N = 1$ ; $n = 20$ ; $RR = 3$ ; 95% CI, 0.14 to 65.9). I
03 Plasma exchange vs. IV immunoglobulin	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on the likelihood of leaving the study early $(N = 1; n = 20; RR = 0.33; 95\% CI, 0.02 to 7.32)$ . I
03 Y-BOCS*		
01 Plasma exchange vs. placebo	s2x	There is limited evidence suggesting a difference favouring plasma exchange over placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 12; SMD = -2.24; 95% CI, -3.83 to -0.66). I
02 IV immunoglobulin vs. placebo	s2x	There is limited evidence suggesting a difference favouring IV immunoglobulin over placebo on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 13; SMD = -1.57; 95% CI, -2.88 to -0.26). I
03 Plasma exchange vs. IV immunoglobulin	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 11; SMD = 0.04; 95% CI, -1.15 to 1.23). I
04 NIMH-Global Severity*		
01 Plasma exchange vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and placebo on reducing global severity of symptoms as measured by the NIMH-Global Severity scale (N = 1; n = 12; SMD = -1.15; 95% CI, -2.43 to 0.13). I
02 IV immunoglobulin vs. placebo	s2x	There is limited evidence suggesting a difference favouring IV immunoglobulin over placebo on reducing global severity of symptoms as measured by the NIMH-Global Severity scale (N = 1; n = 13; SMD = -1.26; 95% CI, -2.5 to -0.03). I

03 Plasma exchange vs. IV immunoglobulin	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on reducing global severity of symptoms as measured by the NIMH-Global Severity scale (N = 1; n = 11; SMD = 0; 95% CI, -1.19 to 1.19). I
05 CGI-Symptom Severity*		
01 Plasma exchange vs. placebo	s2x	There is limited evidence suggesting a difference favouring plasma exchange over placebo on reducing symptom severity as measured by the CGI-Symptom Severity scale (N = 1; n = 12; SMD = $-1.43$ ; 95% CI, $-2.78$ to $-0.09$ ). I
02 IV immunoglobulin vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between IV immunoglobulin and placebo on reducing symptom severity as measured by the CGI-Symptom Severity scale (N = 1; n = 13; SMD = $-1.06$ ; 95% CI, $-2.25$ to 0.14). I
03 Plasma exchange vs. IV immunoglobulin	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on reducing symptom severity as measured by the CGI-Symptom Severity scale (N = 1; n = 11; SMD = -0.26; 95% CI, -1.46 to 0.93). I
06 Global Assessment Scale*		
01 Plasma exchange vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and placebo on improving global functioning as measured by the Global Assessment Scale (N = 1; n = 12; SMD = $-0.93$ ; 95% CI, $-2.16$ to 0.31). I
02 IV immunoglobulin vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between IV immunoglobulin and placebo on improving global functioning as measured by the Global Assessment Scale ( $N = 1$ ; $n = 13$ ; $SMD = -0.94$ ; $95\%$ CI, $-2.11$ to $0.23$ ). I
03 Plasma exchange vs. IV immunoglobulin	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on improving global functioning as measured by the Global Assessment Scale (N = 1; n = 11; SMD = -0.05; 95% CI, -1.23 to 1.14). I
07 NIMH-Anxiety*	T	
01 Plasma exchange vs. placebo	s2x	There is limited evidence suggesting a difference favouring plasma exchange over placebo on reducing anxiety symptoms as measured by the NIMH-Anxiety scale (N = 1; n = 12; SMD = $-1.43$ ; 95% CI, $-2.77$ to $-0.09$ ). I
02 IV immunoglobulin vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between IV immunoglobulin and placebo on reducing anxiety symptoms as measured by the NIMH-Anxiety scale ( $N = 1$ ; $n = 13$ ; $SMD = -1.12$ ; $95\%$ CI, $-2.32$ to $0.09$ ). I

03 Plasma exchange vs. IV immunoglobulin	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on reducing anxiety symptoms as measured by the NIMH-Anxiety scale ( $N = 1$ ; $n = 11$ ; SMD = -0.56; 95% CI, -1.79 to 0.66). I
08 NIMH-Depression*		μο 0.00). 1
01 Plasma exchange vs. placebo	s2x	There is limited evidence suggesting a difference favouring plasma exchange over placebo on reducing depressive symptoms as measured by the NIMH-Depression scale (N = 1; n = 12; SMD = -1.62; 95% CI, -3.02 to -0.23). I
02 IV immunoglobulin vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between IV immunoglobulin and placebo on reducing depressive symptoms as measured by the NIMH-Depression scale (N = 1; n = 13; SMD = -1.14; 95% CI, -2.35 to 0.07). I
03 Plasma exchange vs. IV immunoglobulin	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on reducing depressive symptoms as measured by the NIMH-Depression scale (N = 1; n = 11; SMD = -0.69; 95% CI, -1.93 to 0.55). I
09 NIMH-OC*		
01 Plasma exchange vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC scale ( $N = 1$ ; $n = 12$ ; SMD = -1.04; 95% CI, -2.29 to 0.22). I
02 IV immunoglobulin vs. placebo	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between IV immunoglobulin and placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC scale (N = 1; n = 13; SMD = -1.08; 95% CI, -2.28 to 0.12). I
03 Plasma exchange vs. IV immunoglobulin	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on reducing obsessive-compulsive symptoms as measured by the NIMH-OC scale (N = 1; n = 11; SMD = -0.05; 95% CI, -1.23 to 1.14). I
05 Plasma exchange vs. IV in	 nmunoglob	ulin (child/adolescent: at 1 year after start of treatment)
01 Leaving study early	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on the likelihood of leaving the study early (N = 1; n = 19; RR = 4.55; 95% CI, 0.25 to 83.7). I
02 Y-BOCS*	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (N = 1; n = 11; SMD = -0.24; 95% CI, -1.43 to 0.96). I

03 Global Assessment Scale*	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between plasma exchange and IV immunoglobulin on improving global functioning as measured by the Global Assessment Scale ( $N = 1$ ; $n = 11$ ; SMD = -0.95; 95% CI, -2.23 to 0.34). I
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