

## Appendix 17: Clinical evidence statements

[Psychology](#), [Pharmacology](#), [Psychological vs. pharmacological interventions](#), [Combination therapy](#); [Other medical interventions](#)

### Psychological interventions evidence statements

[Psychological vs. control \(OCD\)](#); [Psychological vs. Psychological \(OCD\)](#); [Psychological vs. control \(BDD\)](#); [Psychological vs. Psychological \(BDD\)](#)

#### Psychological vs. control (OCD)

Description	Statement level <sup>1</sup>	Statement
<b>01 Behaviour therapy v Control</b>		
<i>01 Leaving the study early</i>		
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
<i>02 Non-responders (CGI)</i>		
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
<i>03 Y-BOCS</i>		
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

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

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04 <i>Padua Inventory</i>	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 <i>Depression: BDI or HAM-D</i>		
01 ERP vs Anxiety management	s4	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	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 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	s3	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 <i>Maudsley Obsessive-Compulsive Inventory</i>		
01 ERP vs Anxiety management	s2x	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 <i>STAI: Trait</i>		
01 ERP vs Anxiety management	s4	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 <i>STAI: State</i>		
01 ERP vs Anxiety management	s4	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 <i>Work and Social Adjustment Scale</i>		
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

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<i>13 Interference</i>		
01 ERP vs Anxiety Management	s1x	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
<b>03 Cognitive-Behavioural Therapy v Control</b>		
<i>01 Y-BOCS</i>		
01 CBT v Wait-list control (patients with obsessions only)	s2x	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
<i>02 Padua Inventory</i>		
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
<i>03 Current Functioning Assessment</i>		
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
<i>04 Beck Anxiety Inventory</i>		
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
<i>05 Beck Depression Inventory</i>		
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
<i>10 Overvalued Ideas Scale</i>		
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
<i>11 WHOQOL-BREF: physical</i>		
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

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<b>12 WHOQOL-BREF: psychological</b>		
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
<b>13 WHOQOL-BREF: social</b>		
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
<b>14 WHOQOL-BREF: environmental</b>		
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
<b>15 Non-responders (35% Y-BOCS)</b>		
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
<b>10 Children: Individual CBFT vs Wait-list control</b>		
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
<b>11 Children: Group CBFT vs Wait-list control</b>		

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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 Inventory	s2x	There is limited evidence suggesting a difference favouring group 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 Device – Mother's rating	s2x	There is limited evidence suggesting a difference favouring group 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% CI, -1.45 to -0.11). 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 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
<b>01 Behaviour therapy v Cognitive therapy</b>		
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%)		

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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 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

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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		
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; 95% CI, -0.35 to 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

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		follow-up as measured by the Beck Anxiety Inventory (N = 2; n = 144; SMD = -0.13; 95% CI, -0.46 to 0.2). I
<i>08 Symptom Checklist-90</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 psychological distress as measured by the Symptom Checklist (N = 1; n = 57; SMD = 0.22; 95% CI, -0.3 to 0.74). I
<i>09 Anxiety Discomfort Scale: mean of patient, therapist and assessor ratings</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
<i>10 Irrational Belief Inventory</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 irrational beliefs as measured by the Irrational Belief Inventory (N = 1; n = 55; SMD = 0.49; 95% CI, -0.05 to 1.03). I
<i>11 Behavioural Avoidance Test: Avoidance</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 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). 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 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). 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 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
<i>12 Behavioural Avoidance Test: Discomfort</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 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



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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
<i>13 Obsessive Thoughts Checklist: total</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 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
<i>14 ITIQ - intrusive thoughts</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 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
<i>15 ITIQ - interpretation/intrusion</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 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). 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 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 = -

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		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 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
<i>16 ITIQ - responsibility</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 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
<i>17 ITIQ - guilt</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 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
<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

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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
<i>19 Salkovskis Responsibility Scale</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 Salkovskis 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 Salkovskis 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 Salkovskis Responsibility scale (N = 1; n = 48; SMD = -0.09; 95% CI, -0.66 to 0.47). I
<i>20 Quality of life</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 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
<b>06 Behaviour therapy v Rational Emotive therapy</b>		
<i>01 Non-responders (Anxiety/Discomfort scale)</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 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

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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
<i>02 Maudsley Obsessive-Compulsive Inventory</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 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 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
<i>03 Anxiety Discomfort Scale</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 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
<i>04 Irrational Belief test</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 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
<i>05 Self-rating Depression scale</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 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
<i>06 Social Anxiety scale</i>		

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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 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
<i>07 Hostility and Direction of Hostility Questionnaire: Intrapunitivity</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 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
<i>08 Hostility and Direction of Hostility Questionnaire: Extrapunitivity</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 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
<i>09 Dutch Obsessive-Compulsive Questionnaire</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 rational emotive therapy on reducing obsessive-compulsive symptoms as measured by the Dutch Obsessive-Compulsive Questionnaire] (N = 1; n = 21; SMD = 0.17; 95% CI, -0.69 to 1.03). I
<i>10 Anxiety Disability scale: main OC symptoms (Assessor)</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 rational emotive therapy on reducing anxiety and discomfort as measured by the Anxiety Discomfort scale for main obsessive-compulsive symptoms (N = 1; n = 21; SMD = 0.08; 95% CI, -0.78 to 0.93). I
<b>07 BT v RET + exposure in vivo (post RET alone)</b>		
<i>01 Maudsley Obsessive-Compulsive Inventory</i>		

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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 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
<i>02 Dutch Obsessive-Compulsive Questionnaire</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
<i>03 Anxiety Discomfort scale: main OC symptoms (assessor)</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 anxiety and discomfort as measured by the Anxiety Discomfort scale for main obsessive-compulsive symptoms (N = 1; n = 21; SMD = -0.48; 95% CI, -1.35 to 0.39). I
<i>04 Irrational Belief test</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 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
<i>05 Self-rating Depression scale</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 depression at the end of treatment as measured by the Self-rating Depression scale (N = 1; n = 21; SMD = 0.52; 95% CI, -0.35 to 1.4). I

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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
<b>08 BT v CBT</b>		
01 <i>Leaving the study early</i>	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 <i>Non-remission (Y-BOCS)</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 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
<b>03 Y-BOCS</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 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
<b>08 BDI</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

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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
<b>09 Yogic meditation v Relaxation response + mindfulness meditation</b>		
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
<b>09 Self-exposure v Partner-assisted exposure</b>		
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



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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
<b>10 Imaginal + live ERP v Live ERP</b>		
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

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		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 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
<i>04 Y-BOCS rituals</i>		
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; SMD = -0.18; 95% CI, -0.76 to 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
<i>05 Compulsions checklist</i>		
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

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<i>06 Beck Depression Inventory</i>		
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
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
<b>11 ERP + relapse prevention v ERP + associative therapy</b>		
<i>01 Non-responders (Y-BOCS 50%)</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 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). I
<i>03 Y-BOCS</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 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
<i>04 Obsessive-compulsive symptoms (assessor-rated)</i>		

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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 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
<b>05 Hamilton Depression Scale</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 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
<b>12 Anxiogenic exposure v Neutral thoughts</b>		
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

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06 <i>Main target</i>	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 <i>Beck Depression Inventory</i>	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 <i>Work adjustment</i>	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 <i>Home adjustment</i>	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 <i>Social adjustment</i>	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 <i>Private adjustment</i>	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
<b>13 BTSTEPS + scheduled support v BTSTEPS + requested support</b>		
01 <i>Y-BOCS</i>	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 <i>Target triggers discomfort</i>	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 <i>Work and Social Adjustment Scale</i>	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 <i>Leaving the study early</i>	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
<b>14 Family-based BT v Patient-based BT</b>		
01 <i>MOCI</i>		

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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
<i>02 Zung Self-rating Depression Scale</i>		
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
<i>03 Social adjustment: Occupation</i>		
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
<i>04 Social adjustment: family</i>		
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
<i>05 Social adjustment: household responsibilities</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 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	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

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		= -0.51; 95% CI, -1.24 to 0.22). I
<i>06 Social adjustment: leisure-time activities</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
<b>01 CBT v No treatment/wait-list control</b>		
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
<b>01 BT v CT (mid-treatment)</b>		
<i>01 Non-responders (Y-BOCS non-reliable change)</i>	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
<i>02 Y-BOCS (BDD)</i>	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
<i>03 BDD Examination</i>	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
<i>04 Defects Related Beliefs Test</i>	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
<i>05 Beck Anxiety Inventory</i>	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
<i>06 Beck Depression Inventory</i>	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
<i>07 Body Satisfaction Scale</i>	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
<i>08 Social Avoidance and Distress Scale</i>	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
<i>09 Quality of Life Inventory</i>	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
<b>02 BT v CBT (post-treatment)</b>		
<i>01 Non-responders (Y-BOCS non-reliable change)</i>	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,



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		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
<b>01 SSRIs vs placebo (acute phase)</b>		
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
<b>05 Non-responders (MDD)</b>		
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
<b>06 Non-remitters</b>		

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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 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
<i>09 General Rating Scale - compulsions</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
<i>10 General Rating Scale - obsessions</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 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
<i>11 Maudsley Obsessive Compulsive Inventory</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 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
<i>12 Obsessive-Compulsive Checklist</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 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
<i>13 OCD Scale (CPRS)</i>		
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
<i>14 Beck Depression Inventory</i>		

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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
<i>15 Hamilton Rating Scale for Depression</i>		
	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
<i>16 Montgomery-Asberg Depression Rating Scale</i>		
	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
<i>17 Hamilton Anxiety Scale</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 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
<i>18 Clinical Global Impressions</i>		
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
<i>19 Clinical Global Impressions: severity</i>		
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
<i>20 Sheehan Disability Scale - family</i>		
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
<i>21 Sheehan Disability Scale - social</i>		
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
<i>22 Sheehan Disability Scale - work</i>		

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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
<i>23 Symptom Checklist-90</i>		
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
<i>Subanalysis: Y-BOCS in patients with comorbid depression</i>	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
<i>Subanalysis: Non-responders (OCD) (CGI or Y-BOCS) in patients with comorbid depression</i>	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
<b>02 SSRIs at different doses (acute phase)</b>		
<i>01 Adverse effects</i>		
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
<i>02 Leaving study early</i>		
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)

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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)

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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)
<i>03 Leaving study early due to adverse events</i>		
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% C.I., 0.61 to 3.11)

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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 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)
<i>04 Non-responders</i>		
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)



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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)
<i>05 Y-BOCS</i>		
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)

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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)
<i>06 NIMH-OC</i>		
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)
<i>07 Hamilton Rating Scale for Depression</i>		
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)
<i>08 Montgomery-Asberg Depression Rating Scale</i>		
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)

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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 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 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 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 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 Fluoxetine 40mg	s3	There is evidence suggesting that there is unlikely to be a clinically 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 = 0.00; 95% C.I., -0.38 to 0.38)
<i>09 Sheehan Disability Score - family</i>		
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 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 Citalopram 20mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 20mg of citalopram on 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 Citalopram 40mg	s3	There is evidence suggesting that there is unlikely to be a clinically 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)
<i>10 Sheehan Disability Score - social</i>		
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 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)

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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 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 Citalopram 40mg	s3	There is evidence suggesting that there is unlikely to be a clinically important difference between 60mg and 40mg of citalopram on impairment of social life as measured by the SDS social subscale in adults with OCD (N = 1; n = 198; SMD = -0.21; 95% C.I., -0.49 to 0.07)
<i>11 Sheehan Disability Score - work</i>		
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 impairment of work as measured by the SDS work subscale in adults with OCD (N = 1; n = 200; SMD = -0.08; 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 impairment of work as measured by the SDS work subscale in adults with OCD (N = 1; n = 202; SMD = -0.04; 95% C.I., -0.32 to 0.24)
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 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)
<b>03 SSRIs vs other SSRIs (acute phase)</b>		
<i>01 Leaving study early</i>		
03 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 leaving the study early in adults with OCD (N = 1; n = 150; RR = 1.05; 95% C.I., 0.64 to 1.73)
<i>02 Leaving study early due to adverse effects</i>		
03 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 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)
<i>03 Non-responders</i>		
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)

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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)
<i>04 Non-remitters</i>		
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)
<i>05 Y-BOCS</i>		
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	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)
<i>06 NIMH-OC</i>		
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)

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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)
<i>07 Hamilton Rating Scale for Depression</i>		
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)
<b>04 SSRIs vs Clomipramine (acute phase)</b>		
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)
<i>02 Attempted suicide</i>		
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)
<i>08 Comprehensive Psychopathological Rating Scale: obsessive compulsive</i>		

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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 <i>Hamilton Rating Scale for Depression</i>	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 <i>Montgomery-Asberg Depression Rating Scale</i>	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 <i>Depression (HRSD; MADRS)</i>	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 <i>Clinical Anxiety Scale</i>		
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 <i>Covi Anxiety Scale</i>		
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 <i>Anxiety (Clinical Anxiety Scale; Covi Anxiety Scale)</i>	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 <i>Clinical Global Impressions: global improvement</i>	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)

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16 <i>Clinical Global Impression: severity of illness</i> <b>*HETEROGENEITY*</b>	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 <i>Symptom Checklist -90</i>		
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)
<i>Subanalysis: Non-responders (OCD) (CGI or Y-BOCS) in patients with comorbid depression</i>	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)
<i>Subanalysis: Y-BOCS in patients with comorbid depression</i>	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
<i>Subanalysis: Hamilton Rating Scale for Depression in patients with comorbid depression</i>	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
<b>05 SSRIs/Clomipramine vs non-SRIs (acute phase)</b>		
01 <i>Adverse effects</i>		
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 <i>Leaving study early due to adverse events</i>	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 <i>Non-responders (OCD)</i>		
01 Sertraline vs Desipramine	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



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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
<b>06 SSRIs vs placebo (continuation phase)</b>		
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 adverse events	s1x	There is evidence suggesting a difference favouring the continuation 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)
<b>03 Non-responders</b>		
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)
<b>04 Y-BOCS</b>		
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)
<b>05 Hamilton Rating Scale for Depression</b>		
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)
<b>06 SF-36: social functioning</b>		
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)
<b>07 SSRIs vs placebo (discontinuation phase)</b>		
<b>01 Death</b>		
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)
<b>02 Leaving study early</b>		

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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)
<i>03 Leaving study early due to adverse effects</i>		
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)
<i>04 Relapse</i>		
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 $\geq$ 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)
<i>05 Y-BOCS</i>		
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)
<i>06 NIMH-OC</i>		
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)
<i>07 Qol Enjoyment and Satisfaction</i>		
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)
<b>08 SSRIs at different doses (continuation phase)</b>		
<i>01 Leaving study early</i>		
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 in adults with OCD (N = 2; n = 82; RR = 1.47; 95% C.I., 0.68 to 3.19)
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 in adults with OCD (N = 2; n = 90; RR = 0.82; 95% C.I., 0.34 to 1.97)

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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)
<i>02 Leaving study early due to adverse effects</i>		
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)
<i>03 Non-responders</i>		
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 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)
<i>04 Y-BOCS</i>		
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)

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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)
<b>09 SSRIs v Clomipramine (continuation phase)</b>		
<i>01 Leaving study early</i>		
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)
<i>02 Leaving study early due to adverse effects</i>		
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)
<b>12 Sertraline standard dose vs high dose (non-responders)</b>		

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01 <i>Leaving study early</i>	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 <i>Leaving study early due to adverse events</i>	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 <i>Non-responder</i>	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
<b>01 Clomipramine vs placebo: acute phase</b>		
01 <i>Adverse events</i> <b>*HETEROGENEITY*</b>	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 <i>Leaving study early</i>	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 <i>Leaving study early due to adverse events</i>	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 <i>Non-responders (Y-BOCS)</i>	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)

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05 <i>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 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 <i>Non-responders (all)</i>	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 <i>Non-remitters</i>	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 <b>HETEROGENEITY therefore down-graded to s2*</b>	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 <b>*HETEROGENEITY therefore down-graded to s2*</b>	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 <i>Leyton Obsessional Inventory – symptom</i>	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 <i>Leyton Obsessional Inventory – trait</i>	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 <i>Leyton Obsessional Inventory – resistance</i>	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 <i>Leyton Obsessional Inventory – interference</i>	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)

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14 <i>OCD Scale (CPRS)</i>	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 <i>6-item OCD Scale: amelioration</i>	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 <i>OCD (OCD-CPRS; 6-item OCD Scale-amelioration)</i>	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 <i>Self-Rating Obsessive-Compulsive Personality Inventory</i>	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 <i>Montgomery-Asberg Depression Rating Scale</i>	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 <i>Clinical Global Impressions: severity of illness</i>	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 <i>Symptom Checklist-90</i>	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)
<b>02 Clomipramine vs SSRIs: acute phase</b>		
01 <i>Adverse events</i>	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 <i>Attempted suicide</i>		
01 <i>Clomipramine vs Sertraline</i>	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)

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03 <i>Leaving study early</i>	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 <i>Leaving study early due to adverse events</i>	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 <i>Non-responders (25% Y-BOCS)</i>	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 <i>Non-responders (35% Y-BOCS)</i>		
01 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 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 <i>Non-responders (CGI; 25% Y-BOCS)</i>		
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 <i>Non-responders (CGI; 35% Y-BOCS)</i>		
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 <i>Non-responders (CGI-I)</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)



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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 <i>Comprehensive Psychopathological Rating Scale: obsessive-compulsive</i>		
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 <i>Hamilton Rating Scale for Depression</i>	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 <i>Montgomery-Asberg Depression Rating Scale</i>	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 MADRS in adults with OCD (N = 3; n = 356; SMD = 0.03; 95% C.I., -0.19 to 0.25)
16 <i>Depression (HRSD; MADRS)</i>	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 or MADRS in adults with OCD (N = 4; n = 592; SMD = -0.01; 95% C.I., -0.17 to 0.16)
17 <i>Clinical Anxiety Scale</i>		
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 <i>Covi Anxiety Scale</i>		
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)

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19 <i>Anxiety (Clinical Anxiety Scale; Covi Anxiety Scale)</i>	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 <i>Clinical Global Impressions: global improvement</i>	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 <i>Clinical Global Impressions: severity of illness</i> <b>*HETEROGENEITY*</b>	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 <i>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)
<b>03 Clomipramine vs TCAs</b>		
01 <i>Leaving study early</i>	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 <i>Leaving study early due to adverse effects</i>		
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 <i>Leyton Obsessional Inventory - symptom</i>		
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 <i>Leyton Obsessional Inventory - trait</i>		
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 - trait score in adults with OCD (N = 1; n = 16; SMD = -0.21; 95% C.I., -1.19 to 0.77)
05 <i>Leyton Obsessional Inventory - resistance</i>		

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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)
<i>06 Leyton Obsessional Inventory - interference</i>		
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)
<i>07 OCD Scale (CRPS)</i>		
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)
<i>08 Self-Rating Obsessive Compulsive Personality Inventory</i>		
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)
<i>09 Self-Rating Obsessional Neurotic Scale</i>		
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)
<i>10 Hamilton Rating Scale for Depression</i>		
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)
<i>11 Global Evaluation of Efficacy</i>		
01 Clomipramine vs Imipramine	s2x	There is limited evidence suggesting a difference favouring 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)
<b>04 Clomipramine vs other drugs</b>		
<i>01 Adverse effects</i>		
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)

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<i>02 Leaving study early</i>		
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)
<i>03 Leaving study early due to adverse effects</i>		
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)
<i>04 Non-responders</i>		
01 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 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)
<i>05 Y-BOCS</i>		
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)

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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)
<i>06 NIMH-OC</i>		
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)
<i>07 Maudsley Obsessive-Compulsive Inventory</i>		
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)
<i>08 Hamilton Rating Scale for Depression</i>		
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)
<i>09 Hamilton Rating Scale for Anxiety</i>		
01 Clomipramine vs Phenelzine	s2y	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)
<b>05 Intravenous Clomipramine vs IV placebo</b>		
01 <i>Leaving study early</i>	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 <i>Non-responders</i>	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)

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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 <i>Clinical Global Impressions: severity</i>	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 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 <i>Hamilton Rating Scale for Depression</i>	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)
<b>06 Intravenous Clomipramine vs oral Clomipramine</b>		
01 <i>Adverse effects</i>	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 <i>Leaving study early</i>	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 <i>Non-responders</i>	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)
<b>07 Clomipramine vs placebo: continuation phase</b>		
01 <i>Adverse effects</i>	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)

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02 <i>Leaving study early due to adverse effects</i>	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 <i>Physician's Global Evaluation</i>	s2x	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)
<b>08 Clomipramine vs SSRIs: continuation phase</b>		
<i>01 Leaving study early</i>		
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 100mg clomipramine and 20mg 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)
<i>02 Leaving study early due to adverse effects</i>		
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)
<b>09 Clomipramine same dose vs half dose vs none: continuation/discontinuation</b>		
<i>01 Leaving study early</i>		

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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)
<i>02 Relapse (CGI; 25% Y-BOCS)</i>		
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	s2x	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	s2x	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
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<b>01 TCAs vs placebo (adults)</b>		
<i>01 Leyton Obsessional Inventory - symptom</i>		
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)
<i>02 Leyton Obsessional Inventory - trait</i>		
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)
<i>03 Leyton Obsessional Inventory - resistance</i>		
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)
<i>04 Leyton Obsessional Inventory - interference</i>		
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)
<i>05 OCD Scale (CPRS)</i>		
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)
<b>02 TCAs vs other drugs (adults)</b>		
<i>01 Adverse effects</i>		
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)
<i>02 Leaving study early</i>		

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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)
<i>03 Leaving study early due to adverse effects</i>		
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)
<i>04 Non-responders (OCD)</i>		
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)
<i>05 Non-responders (MDD)</i>		
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)

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06 <i>Non-remitters (HRSD&gt;17)</i>		
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 <i>NIMH-OC</i>		
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 <i>Leyton Obsessional Inventory - symptom</i>		
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 <i>Leyton Obsessional Inventory - trait</i>		
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 <i>Leyton Obsessional Inventory - resistance</i>		
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 <i>Leyton Obsessional Inventory - interference</i>		
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 <i>OCD Scale (CPRS)</i>		

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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)
<i>14 Self-Rating Obsessive Compulsive Personality Inventory</i>		
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)
<i>15 Self-Rating Obsessional Neurotic Scale</i>		
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 Obsessional Neurotic Scale Inventory in adults with OCD (N = 1; n = 16; SMD = 1.17; 95% C.I., 0.09 to 2.26)
<i>16 Hamilton Rating Scale for Depression</i>		
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)
<i>17 Global Evaluation of Efficacy</i>		
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)
<b>03 Desipramine substitution vs Clomipramine continuation (child/adolescent)</b>		

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01 <i>Leaving the study early</i>	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 <i>Relapse (Physician's Relapse Scale)</i>	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 <i>Comprehensive Psychopathological Rating Scale: obsessive-compulsive</i>	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 <i>NIMH-OC</i>	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
<b>01 SNRIs vs other drugs</b>		
<i>01 Adverse effects</i>		
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)
<i>02 Leaving study early</i>		
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)
<i>03 Leaving study early due to adverse effects</i>		

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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)
<i>04 Non-responders</i>		
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)
<i>05 Y-BOCS</i>		
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)
<i>06 Hamilton Rating Scale for Depression</i>		
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 depressive symptoms as measured by the HRSD (N = 1; n = 145; SMD = 0.14; 95% C.I., -0.19 to 0.46)
<i>07 Hamilton Anxiety Scale</i>		
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 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**

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Description	Statement level	Statements and Statistics
<b>01 MAOIs vs placebo</b>		
<i>01 Leaving study early</i>		
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)
<i>02 Y-BOCS</i>		
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)
<i>03 NIMH-OC</i>		
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)
<i>04 OCD Scale (CPRS)</i>		
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)
<i>05 Clinical Global Impression</i>		
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)
<b>02 MAOIs vs other drugs</b>		
<i>01 Leaving study early</i>		
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 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)

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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)
<i>02 Leaving study early due to adverse effects</i>		
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 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)
<i>03 Y-BOCS</i>		
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% C.I., -0.64 to 0.67)
<i>04 NIMH-OC</i>		
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)
<i>05 Maudsley Obsessive-Compulsive Inventory</i>		
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)
<i>06 OCD Scale (CPRS)</i>		
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)
<i>07 Hamilton Rating Scale for Depression</i>		
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 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)



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<i>08 Hamilton Rating Scale for Anxiety</i>		
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)
<i>09 Clinical Global Impression</i>		
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
<b>01 Anxiolytics vs placebo</b>		
<i>01 Leaving study early</i>		
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)
<i>02 Leaving study early due to adverse effects</i>		
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)
<i>03 Non-responders</i>		
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)
<i>04 Y-BOCS</i>		
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)
<i>05 NIMH-OC</i>		

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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)
<i>06 Hamilton Rating Scale for Depression</i>		
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)
<i>07 Hamilton Anxiety Scale</i>		
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)
<b>02 Anxiolytics vs other drugs</b>		
<i>01 Leaving study early</i>		
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)
<i>02 Leaving study early due to adverse effects</i>		
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)
<i>03 Y-BOCS</i>		
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)
<i>04 NIMH-OC</i>		
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)
<i>05 Hamilton Rating Scale for Depression</i>		

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01 Bupirone vs Clomipramine	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between bupirone 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)
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**Other Pharmacological interventions**

Description	Statement level	Statement and Statistics
<b>01 Inositol vs placebo</b>		
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)
<b>02 Oxytocin vs placebo</b>		
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)

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07 <i>General Symptom Index</i>	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)
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**Augmentation strategies**

Description	Statement level	Statement and Statistics
<b>01 Buspirone vs placebo</b>		
<i>01 Non-responders</i>		
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)
<i>02 Y-BOCS</i>		
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)
<i>03 Hamilton Depression Rating Scale</i>		
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)
<i>04 Hamilton Anxiety Scale</i>		
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)
<b>02 Citalopram+Clomipramine vs Citalopram</b>		
<i>01 Non-responders (Y-BOCS 35%)</i>	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)
<i>02 Y-BOCS</i>	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)

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<i>03 Hamilton Depression Scale</i>		
	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)
<b>03 Desipramine vs placebo</b>		
<i>01 Y-BOCS at 6 weeks</i>		
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)
<i>02 Y-BOCS at 10 weeks</i>		
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)
<b>04 Haloperidol vs placebo</b>		
<i>01 Non-responders</i>		
01 Fluvoxamine+Haloperidol v Fluvoxamine+Placebo	s2x	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)
<i>02 Y-BOCS</i>		
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)
<b>05 Inositol vs placebo</b>		
<i>01 Y-BOCS</i>		
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)
<b>06 Lithium vs placebo</b>		

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01 <i>Non-responders</i>	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 <i>Y-BOCS change score</i> <b>*HETEROGENEITY</b>	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 <i>Hamilton Depression Scale change score</i> <b>*HETEROGENEITY</b>	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 <i>Hamilton Anxiety Scale change score</i>	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)
<b>07 Nortriptyline vs placebo</b>		
01 <i>Y-BOCS</i>		
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)
<b>08 Olanzapine vs placebo</b>		
01 <i>Leaving the study early</i>		
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 <i>Leaving the study early due to adverse effects</i>		
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 due to adverse effects in patients with OCD (N = 1; n = 44; RR = 1.00; 95% C.I., 0.15 to 6.48)
03 <i>Non-responders (Y-BOCS 25%)</i>		

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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)
<i>04 Y-BOCS change score</i>		
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)
<b>09 Pindolol vs placebo</b>		
<i>01 Leaving the study early</i>		
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)
<i>02 Leaving the study early due to adverse effects</i>		
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)
<i>03 Y-BOCS</i>		
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)
<i>04 MADRS</i>		
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)
<i>05 Hamilton Anxiety Scale</i>		
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)
<b>10 Quetiapine vs placebo</b>		

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<i>01 Adverse effects</i>		
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)
<i>03 Non-responders (Y-BOCS 30%)</i>		
01 SRI + Quetiapine v SRI + Placebo	s2x	There is limited evidence suggesting a difference favouring SRIs + 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)
<i>04 Y-BOCS</i>		
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)
<i>05 CGI-severity of illness</i>		
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)
<b>11 Risperidone vs placebo</b>		
<i>01 Adverse effects</i>		
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)
<i>02 Leaving the study early</i>		
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)
<i>03 Leaving the study early due to adverse effects</i>		
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)
<i>04 Non-responders</i>	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)



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<i>05 Y-BOCS change score</i>		
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)
<i>06 Hamilton Depression Scale</i>		
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)
<i>07 Hamilton Anxiety Scale</i>		
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)
<b>12 All antipsychotics v placebo</b>		
01 Adverse effects <b>*HETEROGENEITY</b>	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 <b>*HETEROGENEITY</b>	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 <b>*HETEROGENEITY</b>	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)
<i>06 Hamilton Depression Scale</i>		

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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)
<i>07 Hamilton Anxiety Scale</i>		
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)
<i>08 CGI: severity of illness</i>		
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)**

Description	Statement level	Statistics
<b>01 SSRI vs placebo: acute phase</b>		
<i>01 Adverse events</i>	s3	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)
<i>02 Serious adverse events</i>	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)
<i>03 Leaving study early</i>	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)
<i>04 Leaving study early due to adverse effects</i>	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)
<i>05 Non-responders (25% CY-BOCS)</i>		
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)
<i>06 Non-responders (40% Y-BOCS)</i>		

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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 Compulsive Impact 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 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 Inventory (Child Version): 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 Inventory (Child Version): 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)

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<i>15 Leyton Obsessional Inventory (Child Version): symptoms</i>		
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)
<i>16 Children or adolescents' Depression Rating Scale (Revised Version)</i>		
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)
<i>17 Hamilton Rating Scale for Depression</i>		
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)
<i>19 Multidimensional Anxiety Scale for Children or adolescents</i>		
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)
<i>20 Revised Children or adolescents' Manifest Anxiety Scale</i>		
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 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)
<i>21 Anxiety (MASC; RCMAS)</i>		
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)
<i>22 Children or adolescents' Global Assessment Scale</i>		

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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)
<i>23 Systolic blood pressure - supine</i>		
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)
<i>24 Systolic blood pressure - standing</i>		
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)
<i>25 Diastolic blood pressure - supine</i>		
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)

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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)
<i>26 Diastolic blood pressure - standing</i>		
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)
<i>27 Heart rate - supine</i>		
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)
<i>28 Heart rate - standing</i>		
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)

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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
<b>02 SSRI vs placebo: continuation phase</b>		
<i>01 Adverse effects</i>		
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)
<i>02 Leaving study early</i>		
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)
<i>03 CY-BOCS</i>		
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)
<i>04 NIMH-OC</i>		
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)
<i>05 Child Obsessive Compulsive Impact Scale (Parent version)</i>		
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)
<i>06 Hamilton Rating Scale for Depression</i>		
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)
<b>03 SSRIs vs placebo (discontinuation)</b>		

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01 <i>Adverse events</i>	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 <i>Serious adverse events</i>	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 <i>Leaving the study early</i>	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 <i>Leaving the study early due to adverse events</i>	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 <i>Non-responders (25% CY-BOCS)</i>	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 <i>Non-responders (CGI &lt; "much improved")</i>	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 <i>Relapse (CGI Global Improvement score increase)</i>	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 $\geq 2$ points at any single visit, or (c) a CGI Global Improvement Score $\geq 5$ 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 <i>CY-BOCS</i>	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)



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08 <i>Hamilton Rating Scale for Depression</i>	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 <i>Hamilton Rating Scale for Anxiety</i>	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 <i>Global Assessment of Functioning</i>	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
<b>01 Clomipramine vs placebo</b>		
01 <i>Leaving study early</i>	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 <i>Leaving study early due to adverse effects</i>	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 <i>Y-BOCS</i>	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 <i>NIMH-OC</i>	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)
<b>02 Clomipramine continuation vs Desipramine substitution</b>		

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01 <i>Leaving the study early</i>	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 <i>Relapse (Physician's Relapse Scale)</i>	s2x	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 <i>Comprehensive Psychopathological Rating Scale: obsessive-compulsive</i>	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 <i>NIMH-OC</i>	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
<b>01 Fluoxetine vs placebo</b>		
01 <i>Adverse effects</i>	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 <i>Leaving study early</i>	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 <i>Non-responders</i>	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)

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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)
<b>02 Clomipramine vs Desipramine</b>		
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
<b>01 ERP vs Clomipramine</b>		
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
<b>02 Children: CBT v Clomipramine</b>		
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

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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
<b>02 Children: CBT v Sertraline</b>		
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 CY-BOCS (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
<b>01 ERP + SRI v ERP</b>		
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-improvement (duration 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

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		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 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 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

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13 <i>Anxiety Discomfort Scale: therapist's ratings</i>	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 <i>Anxiety Discomfort Scale assessor's ratings</i>	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 <i>Clinical Anxiety Scale</i>	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 <i>Global Assessment Scale</i>	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 <i>CGI - therapist rating</i>	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 <i>CGI - patient rating</i>	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 <i>Compulsive activity checklist (post treatment)</i>	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 <i>Compulsive activity checklist (follow-up)</i>		
01 <i>ERP + Fluvoxamine v ERP + placebo (1 year follow-up)</i>	s4	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 <i>Target rituals (assessor): time (post-treatment)</i>	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 <i>Target rituals (assessor): time (follow-up)</i>	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

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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 <i>Target rituals (assessor): discomfort (post-treatment)</i>	s2x	There is limited evidence suggesting a difference favouring BT + SRIs 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 <i>Target rituals (assessor): discomfort (follow-up)</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 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 <i>Target rituals (assessor): duration per day</i>		
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 + 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 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 <i>Behavioural avoidance test: discomfort (post treatment)</i>	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 <i>Behavioural avoidance test: discomfort (follow-up)</i>		
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



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<b>03 ERP + Clomipramine v Clomipramine</b>		
01 <i>Leaving the study early</i>	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 <i>Non-responders (CGI)</i>	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 <i>Y-BOCS</i>	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
<b>04 ERP + Fluvoxamine v Anti-ERP + Fluvoxamine</b>		
01 <i>Leaving the study early</i>	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 <i>Leaving the study early due to adverse effects</i>	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
<b>03 Global criterion of non-improvement (duration of rituals)</b>		
01 <i>Post-treatment</i>	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 <i>6 months follow-up</i>	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 <i>1 year follow-up</i>	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
<b>04 Hamilton Depression Scale</b>		
01 <i>Post treatment</i>	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 <i>6 months follow-up</i>	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 <i>1 year follow-up</i>	s4	The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine

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		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
<i>05 Target rituals (assessor): time</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 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
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
<i>06 Target rituals (assessor): discomfort</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 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
<i>07 Target rituals (assessor): duration per day</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 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
<i>08 Behavioural avoidance test: discomfort</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 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

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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
<i>09 Compulsive activity checklist</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 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
<b>05 CT + Fluvoxamine v CT</b>		
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

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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
<b>08 ERP + Fluvoxamine v CT + Fluvoxamine</b>		
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 = 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
<b>09 Children: ERP + Fluvoxamine v Fluvoxamine alone</b>		
01 Non-responder (Y-BOCS reliable change)		

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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 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). I
<b>02 CY-BOCS</b>		
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
<b>03 NIMH-GOCS</b>		
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 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
<b>09 Children: CBT + Sertraline v Sertraline</b>		
01 <i>Leaving the study early</i>	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

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02 <i>Leaving the study early due to adverse events</i>	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 due to adverse effects (N = 1; n = 56; RR = 1; 95% CI, 0.07 to 15.21). I
02 <i>Remission (CY-BOCS &lt; 11)</i>	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 <i>CY-BOCS</i>	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
<b>09 Children: CBT + Sertraline v CBT</b>		
01 <i>Leaving the study early</i>	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 <i>Remission (CY-BOCS &lt; 11)</i>	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 <i>CY-BOCS</i>	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
<b>01 Stereotactic anterior capsulotomy vs cingulotomy</b>		
01 <i>CPRS @ 3 months: change score</i>	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 psychopathological symptoms as measured by the CPRS (N = 1; n = 4; SMD = -5.79; 95% CI, -38.62 to 27.03). I
02 <i>CPRS @ 6 months: change score</i>	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 psychopathological symptoms as measured by the CPRS (N = 1; n = 4; SMD = -0.3; 95% CI, -2.89 to 2.29). I
03 <i>CPRS @ 12 months: change score</i>	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 psychopathological symptoms as measured by the CPRS (N = 1; n = 4; SMD = 0.46; 95% CI, -2.78 to 3.7). I

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04 HAM-D @ 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 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: 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 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: 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 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: 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 = 0.38; 95% CI, -2.54 to 3.31). I
09 HAM-A @ 12 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 = 0.47; 95% CI, -2.84 to 3.79). I
<b>02 Repetitive transcranial magnetic stimulation: active vs placebo</b>		
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
<b>03 Repetitive transcranial magnetic stimulation: right vs left</b>		

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<i>01 Y-BOCS @ post-treatment</i>		
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
<i>02 BDI @ post-treatment</i>		
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
<i>03 MADRS @ post-treatment</i>		
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
<i>04 STAI-S @ post-treatment</i>		
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
<i>05 Y-BOCS @ 1 month follow-up</i>		
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
<i>06 BDI @ 1 month follow-up</i>		
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



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<i>07 MADRS @ 1 month follow-up</i>		
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
<i>08 STAI-S @ 1 month follow-up</i>		
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
<i>09 Non-responders (Y-BOCS 40%) @ 1 month follow-up</i>		
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
<b>04 Plasma exchange vs IV immunoglobulin vs placebo (child/adolescent: at 1 month after start of treatment)</b>		
<i>01 Adverse effects</i>		
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
<i>02 Leaving study early</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 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
<i>03 Y-BOCS*</i>		
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

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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
<i>04 NIMH-Global Severity*</i>		
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
<i>05 CGI-Symptom Severity*</i>		
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
<i>06 Global Assessment Scale*</i>		
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

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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
<i>07 NIMH-Anxiety*</i>		
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
<i>08 NIMH-Depression*</i>		
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
<i>09 NIMH-OC*</i>		
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

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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
<b>05 Plasma exchange vs IV immunoglobulin (child/adolescent: at 1 year after start of treatment)</b>		
01 <i>Leaving study early</i>	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 <i>Y-BOCS*</i>	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 <i>Global Assessment Scale*</i>	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