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The effectiveness and tolerability of trauma-focused psychotherapies for psychotic symptoms: A systematic review of trauma-focused psychotherapies

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Abstract

Introduction: Psychological trauma is an established risk factor for psychosis. Trauma-focused psychotherapies (TFPT) have been suggested as a potential treatment for reducing psychotic symptoms in those who have experienced trauma. We therefore sought to investigate the effectiveness, tolerability, and acceptability of TFPT for psychotic symptoms.

Methods: We conducted a systematic review of studies of any form of TFPT that measured psychotic symptoms across a broad range of diagnoses.

Results: From 2584 papers initially identified, 17 studies (857 participants) met eligibility criteria. TFPT were found to be well tolerated, with very few adverse events. Acceptability was also high, with a mean dropout rate of 20%.

Conclusions: Whilst the evidence of effectiveness for TFPT in reducing psychotic symptoms is weak, we found tentative evidence in favour of exposure-based interventions. Methodologically rigorous trials investigating the efficacy of TFPT for the treatment of psychotic symptoms are needed to assess this promising intervention.

KEYWORDS

psychosis, psychotherapy, PTSD, schizophrenia, trauma

1 | INTRODUCTION

1.1 | Psychotic symptoms

Psychotic symptoms are characterised by delusions, hallucinations, and paranoia (positive symptoms) as well as difficulties with thinking, blunted emotions, and low motivation (negative symptoms) (Jablensky, 2010). Psychotic symptoms can occur in a range of disorders including schizophrenia spectrum disorders, bipolar disorder, major depressive disorder, dissociative disorders and borderline personality disorder and can therefore be considered a transdiagnostic phenomenon (Buckley et al., 2008). The life expectancy of people with psychotic symptoms is significantly reduced (Saha et al., 2007) due to suicide and greater health problems (Hannerz et al., 2001; Yuen et al., 2014).

1.2 | Psychosis and trauma

Exposure to psychological trauma has consistently been associated with increased risk of psychotic experiences. The evidence fulfils the Bradford Hill criteria (Hill, 1965) supporting the hypothesis of a

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causative relationship between trauma and psychosis, including temporal relationships (Kelleher et al., 2013), dose-response relationships (Croft et al., 2019) and plausible biological mechanisms (Howes & Murray, 2014).

Traumatic experiences can also give rise to post-traumatic stress disorder (PTSD), and there is a growing body of evidence showing a relationship between PTSD and psychotic symptoms in people with psychosis who have experienced trauma (Bloomfield et al., 2021). PTSD is a risk factor for the development of psychotic symptoms (Okkels et al., 2016), and around 39% of people with psychosis experience concurrent PTSD (Mueser et al., 2010). Within trauma survivors, auditory hallucinations have been proposed to be a type of posttraumatic intrusion related to a traumatic memory (Peach et al., 2018; Steel, 2015). This is consistent with studies that have shown that hallucinatory content is often linked to experiences of trauma (Hardy et al., 2005; Onyeama et al., 2011; Peach et al., 2020). Within this framework, an intrusive trauma memory may not be experienced as a memory and is instead misattributed in a psychotic way (e.g. as a voice). Indeed, there are broad similarities between dominant cognitive theories of PTSD (Ehlers & Clark, 2000) and trauma-induced psychotic symptoms (Morrison et al., 2003).

1.3 | Trauma-focused psychotherapies

Given the links between traumatic experiences, psychotic symptoms and PTSD, there is growing interest in trauma-focused psychotherapies (TFPT) for psychotic symptoms. TFPT are a family of therapies developed to treat PTSD that explicitly focus on reprocessing memories of traumatic experiences (Schnurr, 2017). Some TFPT may utilise cognitive techniques only, some may use exposure, and some may use a combination of the two. Broadly, TFPT that utilise exposure are thought to work by promoting emotional habituation and reprocessing of traumatic memories via repeated exposure to the traumatic event and related cues. Cognitive therapies such as trauma-focused CBT (TFCBT) additionally explicitly focus on restructuring peri-traumatic (such as 'I'm going to die') and posttraumatic (such as 'I should have coped better') appraisals, often utilising learnings gained from exposure. In the UK, National Institute for Clinical Excellence guidelines recommend the use of TFCBT and eve movement desensitisation and reprocessing (EMDR) in the treatment of PTSD (National Institute for Health and Clinical Excellence, 2018). Though the literature remains in its infancy, two recent reviews found that TFPT can safely reduce PTSD symptoms in those with psychosis (Sin & Spain, 2016; Swan et al., 2017).

1.4 | The need for improved research and outcomes

There is little research into whether TFPT can reduce psychotic symptoms themselves. The only review to date (Brand et al., 2018) found small effects for TFPT on positive symptoms of psychosis. There remains therefore a pressing need for more research to

improve treatments and outcomes for people with psychotic symptoms and trauma histories. We sought to address this by systematically reviewing the current evidence for the efficacy, tolerability, and acceptability of TFPT on psychotic symptoms. To differentiate this review from previously published work and to draw together disparate research, we have not limited the review to any specific type of TFPT. Additionally, as psychotic symptoms in the context of trauma are likely a transdiagnostic phenomenon (Buckley et al., 2008), we have not limited the review to any specific diagnosis.

2 | MATERIALS & METHODS

2.1 | Search strategy and selection criteria

We pre-registered our review with PROSPERO (CRD42020202135). We followed the preferred reporting items for systematic reviews and meta-analysis guidelines (PRISMA) (Moher et al., 2009). We included any study of the efficacy of a TFPT with a quantitative outcome measure for psychotic symptoms. We defined TFPTs as any psychological intervention from any modality of psychotherapy that had an explicit focus on past traumatic memories and/or was described as 'trauma-focused'. We distinguished between TFCBT that would be in-line with the principles of Ehlers & Clark (2000) by facilitating memory reprocessing through exposure, and trauma-informed CBT (TICBT) that did not include exposure. Interventions could be delivered in any setting in a group or individual format. We defined psychotic symptoms as hallucinations, delusions and/or paranoia.

We included studies on adults over the age of 18. Otherwise, we placed no limits on the population to be included. We included any study design that offered a TFPT, including case reports, case series and randomised controlled trials. We did not limit our criteria to any specific diagnoses. For example, dissociative identity disorder was included due to research highlighting elevated levels of psychotic symptoms (Foote & Park, 2008). We excluded studies that were not reported in English, were not published in peer-reviewed journals, and studies of non-clinical populations.

We used Ovid to systematically search medical and psychological databases (MEDLINE and PsycINFO) and ProQuest to search PTSDPubs from the earliest possible date to the date of the search. We searched a psychotic symptoms term (e.g. hallucinat* OR delus*) AND a trauma-focused term (e.g. trauma* OR neglect*) AND a psychotherapy term (e.g. 'Exposure therapy' OR EMDR). See Supporting Information S1 for the full list of search terms by database and screening methodology.

Reviewer 1 (R1) imported articles generated from the search into a reference management software (EndNote 20) (The EndNote Team, 2013) and checked for duplicates which were removed from the review. R1 and reviewer 2 (R2) then screened for inclusion by title and abstract first, then by reading the full text, with any disagreements resolved by discussion, facilitated through the Rayyan platform (Ouzzani et al., 2016).

2.2 | Data analysis

We defined the primary outcome a priori as quantitatively measured psychotic symptoms, including global measures of psychotic symptoms and measures of hallucinations, delusions, or paranoia. Our secondary outcomes were measures of other domains of psychopathology (depression, anxiety and PTSD) and social functioning. We assigned a level of evidence (OCEBM Levels of Evidence Working Group, 2011) with case reports assigned a level of evidence of 5. For randomised controlled trials (RCT), we assessed risk of bias using the Cochrane Risk of Bias 2 tool (Sterne et al., 2019). For case series, we used the Quality Appraisal Tool for Case Series Studies (Institute of Health Economics, 2014). For case reports, we used the Joanna Briggs Institute Checklist for Case Reports (Moola et al., 2017). R1 and reviewer 3 (R3) undertook a risk of bias and quality assessment. Any discrepancies between reviewers were resolved through discussion and by consensus. If agreement could not be reached, reviewer 5 (R5) was consulted to resolve this.

We extracted data manually from each study paper. R1 extracted the following data for each study: study design, *n*, participant characteristics and clinical presentation, location and setting of the treatment, the type of trauma participants had experienced, intervention type and dose, medications prescribed to participants, control or comparison, primary outcomes, secondary outcomes, adverse events (identified as adverse events reported by the authors and/or symptom exacerbation) and dropout rates. We grouped studies by therapeutic modality.

We considered findings statistically significant when the *p*-value was below 0.05. Where possible, we calculated an effect size (Hedges' *g*; see Supporting Information S1 for formulae) for each study at each time point for each outcome of interest. We chose Hedges' *g* as it has superior properties to Cohen's *d* with small sample sizes (Cumming, 2012).

We undertook a narrative synthesis of quantitative outcomes following SWiM (synthesis without meta-analysis) guidance (Campbell et al., 2020), grouping by modality of psychotherapy, including any relevant information about tolerability or acceptability of these interventions. We followed the GRADE guidance for clinical recommendations (Guyatt et al., 2008).

3 | RESULTS

We screened 2597 studies, identifying 17 studies (n = 857) that quantitatively measured psychotic symptoms in adults undergoing TFPT. Details of the selection process and exclusions at each stage are presented in the PRISMA flowchart (Figure 1; Moher et al., 2009). Table 1 provides a summary of the characteristics, primary outcomes, secondary outcomes, and adverse events of each study.

Risk of bias is summarised in Tables S1–S3. Each RCT held 'some concerns' regarding risk of bias. The case series were mostly of acceptable risk of bias, though two were assessed to be of a higher risk of bias (Brand & Loewenstein, 2013; Trappler & Newville, 2007). The case reports were all assessed to meet an appropriate quality level for inclusion

3.1 | Trauma-informed CBT

Two of three TICBT studies (Mueser et al., 2015; Steel et al., 2016) were RCTs following the same protocol teaching cognitive restructuring to challenge trauma-related thoughts and beliefs, meaning no exposure techniques were used (Ehlers & Clark, 2008). TICBT was not superior to the respective control groups in reducing positive psychotic symptoms. The third study (Trappler & Newville, 2007) was a case series using Cloitre's Skill Training in Affect Regulation preparatory work (Cloitre et al., 2002). It found significant decreases in measures of overall psychotic symptoms, delusions, and paranoia. Only paranoia also significantly decreased in a matched group undergoing supportive psychotherapy.

Regarding secondary outcomes, one of the two controlled studies (Mueser et al., 2015) found a small but statistically significant decrease in PTSD symptoms as compared to control. The noncontrolled study (Trappler & Newville, 2007) found a significant effect for its treatment programme on PTSD symptoms, with no effect found in its matched supportive psychotherapy group. There were no significant effects in these studies on measures of depression, anxiety nor quality of life.

3.2 | Trauma-focused CBT

The TFCBT studies utilised a variety of exposure techniques, such as imagery rescripting and reliving. To qualify as TFCBT, it is necessary that therapeutic techniques intended to process and modify unhelpful peri- or post-traumatic thoughts and feelings, such as cognitive distortions, guilt, and shame, are included. Each included TFCBT study was non-controlled. Due to lack of comprehensive raw data and statistical analysis conducted by the authors, effect sizes were not able to be calculated for these studies. One (Keen et al., 2017) found decreases in auditory hallucinations and delusions scores, 29% and 43% respectively of which remained clinically significant at 6-month follow-up. Of the case reports, two (Callcott et al., 2004; Ward-Brown et al., 2018) found decreases in their measures of psychotic symptoms. The final included case report (McCartney et al., 2019) found a reliable but not clinically significant decrease in auditory hallucinations.

Regarding secondary outcomes, PTSD symptoms decreased in all TFCBT studies. Consistent decreases in depression and anxiety measures were also found. Only one study (Ward-Brown et al., 2018) included a quality-of-life scale, reporting a substantial improvement.

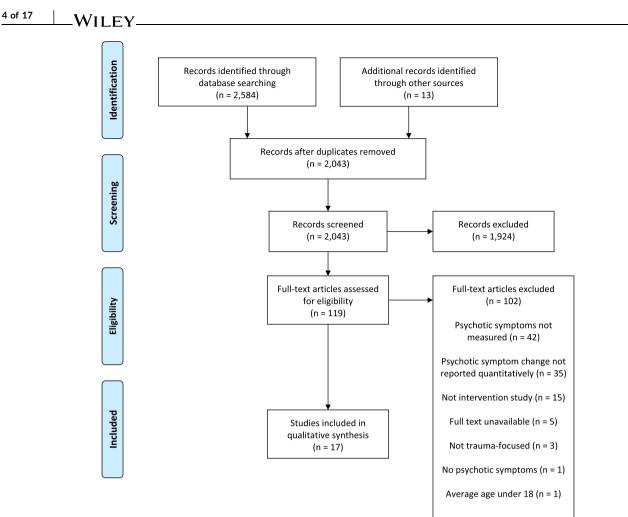


FIGURE 1 Preferred reporting items for systematic reviews and meta-analyses flowchart.

3.3 | Eye movement desensitisation and reprocessing

All the EMDR studies included in this review used the standard eightphase protocol (Shapiro, 2001). This protocol focuses mostly on exposure, although one phase focuses on 'installing' more helpful thoughts and cognitions. Whilst EMDR had the greatest number of included studies, the evidence of effectiveness was mixed. One RCT (de Bont et al., 2016) found EMDR was superior to the control in reducing paranoia following treatment as well as across all time points. EMDR was also associated with greater likelihood of remission from a psychotic disorder following treatment, but this was no longer statistically significant at 6-month follow-up. This contrasts with the other included RCT (Kim et al., 2010), which found no statistically significant differences on psychotic symptoms between EMDR and either of its two control conditions. The case series and case reports provide mixed evidence of small effects on specific symptom domains, particularly hallucinations (Slotema et al., 2019; van den Berg & van der Gaag, 2012) and overall symptoms (van den

Berg & van der Gaag, 2012; Yasar et al., 2017). No study found a specific effect on paranoia.

Regarding secondary outcomes, no controlled study reported on PTSD symptoms. Four studies found large decreases on PTSD symptom measures (de Bont et al., 2013; Slotema et al., 2019; van den Berg & van der Gaag, 2012; Yasar et al., 2017). Regarding depression, the two controlled studies found no superior effect for EMDR over the control following treatment (de Bont et al., 2016; Kim et al., 2010). Uncontrolled studies found decreases in depression measures (van den Berg & van der Gaag, 2012; Yasar et al., 2017). One controlled study measured anxiety finding no effect for EMDR over the controls (Kim et al., 2010). Two uncontrolled studies reported decreases in anxiety measures, reaching significance when significance testing was possible (van den Berg & van der Gaag, 2012). For quality-of-life measures, one controlled study found no superior effect for EMDR over control following intervention or 6month follow-up (de Bont et al., 2016), whilst one uncontrolled study found a significant improvement following treatment and 3-month follow-up (de Bont et al., 2013).

D et al.	47%	Wil	EY ^{5 of}
Dropouts	TICBT: 22/92 = 24% Brief treatment: 4/ 88 = 5%	TICBT: 4/27 = 15%	ž
Adverse effects	ž	Ϋ́	ž
Secondary outcomes	Linear regression (across post-treatment, 6 and 12 months): CAPS: Significant decrease in TICBT group as compared to brief treatment across post-treatment, 6 and 12-month; F = 6.51, (p = 0.01), Hedges' g = -0.29 Hedges' g of CBT versus brief treatment following treatment: -0.26 Hedges' g of CBT versus brief treatment at 12-month: -0.25 Hedges' g of CBT versus brief treatment at 12-month: -0.25 Hedges' g of CBT versus brief treatment at 12-month: -0.26 Hedges' g of CBT versus brief treatment at 12-month: -0.26 Hedges' g of CBT versus brief treatment, 6 and 12-month to brief treatment across post-treatment, 6 and 12-month brief treatment across post-treatment, 6 and 12-month	LMM analysis (pre to 6 and 12 months): CAPS-S: Non statistically significant increase in TICBT group as compared to TAU following treatment; and 6 compared to TAU following treatment; non statistically significant increase in TICBT group as compared to TAU following treatment and 6-month follow-up BAI: Non statistically significant decrease in TICBT group as compared to TAU following treatment and 6-month follow-up as compared to TAU following treatment and 6-month follow-up to TAU following treatment and 6-month follow-up as compared to TAU following treatment and 6-month following treatment and 6-month follow-up as compared to TAU following treatment and 6-month follow-up as compared to TAU following treatment and 6-month following treatment and 6-month following treatment and 6-month following to TAU following to TAU following treatment and 6-month following to TAU follow	Witcoxon signed rank test (pre and post): IES: Significant decrease in TICBT group following treatment: z = -3.47 ($p = 0.003$), $r = -0.74$. Non statistically significant decrease in the supportive psychotherapy group
	Linear regression (across post-treatment, 6 and 12 months): PANSS: Baseline mean Post-treatment mean TICBT 65.75 62.25 Brief 7 67.18 6.1.33 Non statistically significant decrease in TICBT group as compared to brief treatment, 6 and 12-month	LMM analysis (baseline to post-treatment and 6 months): PANSS positive: Baseline mean Post-treatment mean T/CIEF 19:1 17.8 TAU 19:3 19.8 Non statistically significant decrease in TICBT group as compared to TAU following treatment and 6-month follow-up Post-treatment mean PANSS negative: Baseline mean Post-treatment mean TAU 16.3 16.4 PANSS negative: 16.3 16.4 TAU 15.3 16.4 PANS negative: 16.3 16.4 TAU 15.3 16.4 TAU 15.3 16.4 Significant decrease in TICBT group as compared to TAU stolowing treatment tean for the tack of tack): Post-treatment mean NR NR
v	Linear regression (across post-treatment, 6 and 12 months): PANSS: Baseline mean Post-treatment TICBT 65.75 62.25 Brief T 67.18 61.33 Non statistically significant decrease in TICBT group as comp brief treatment, across post-treatment, 6 and 12-month	analysis (baseline to post-treatment and 6 months): SS positive: Baseline mean Post-treatment BA 19.1 17.8 U 19.1 17.8 U 18.3 19.8 U 18.3 19.8 U 18.3 19.8 U 18.3 19.8 TAU following treatment and 6-month follow-up as comparated to the mean Post-treatment SS negative: Baseline mean Post-treatment EF 16.3 15 U 15.3 16.4 Mand decrease in TICBT group as compared to TAU follow-up freatment: t = 2.31 (p = 0.03), Hedges' g = 0.45. Non state significant decrease in TICBT group as compared to TAU follow-up ATS-AHRS: Baseline mean Post-treatment March decrease in TICBT group as compared to TAU follow-up 16.8 16.8 March decrease in TICBT group as compared to TAU follow-up 16.8 16.8 March decrease in TICBT group as compared to TAU follow-up 16.8 16.8 March decrease in TICBT group as compared to TAU follow-up 16.8 16.8 March decrease in TICBT group as compared to TAU follow-up 16.8	Wilcoxon signed rank test (pre and post): BPRS total: Baseline mean TICBT NR Sup. Psy. NR
Primary outcomes	Linear regression PANSS: TICBT Brief T Non statistically s brief treatm	LUMM analysis (baselin PANSS positive: Ba TAU 18 Non statistically signifi TAU 1601owing tre PANSS negative: TAU 16 TAU 15 Significant decrease in treatment: t = 2.0 significant decrease in treatment: t = 2.1 Significant decrease in treatme	Wilcoxon signed BPRS total: TICBT Sup. Psy.
Treatment and dose	TICBT (breathing retraining, psychoeducation and cognitive restructuring) Minimum: 6 Mean. NR Mean. NR Mean. NR Length of time: NR	TCBT (psychoeducation, cognitive restructuring) Maani 12.3 Maani 14 Length of time: NR Length of time: NR	Group TICBT (emotion regulation and behavior/ coping strategies to trauma triggers) Minimum: NR Average: NR Maximum: 12 Length of time: 12 weeks
Population, including diagnosis, location and setting	Diagnoses of schizophrenia, schizoaffective disorder, major dession or bipolar disorder (DSN-Hv criteria), plus diagnosis of severe PTSD (pased on CAPS). USA; 3 inpatient services and 2 outpatient services.	Schizophrenia, schizo- affective disorder or schizophray criteria); plus (DSM-IV criteria); UK: 2 outpatient services	Diagnosis of schizophrenia or schizoaffective disorder IDSM-14 virteraia, juis PTSD (IOSN-1V virteria), USA; 3 inpatient services
Design (level of evidence), <i>n</i>	Multicentre RCT (1b) <i>n</i> = 201	Multicentre RCT (1b) $n = 61$	Multicentre case series (4) n = 24
Author	TICBT Mueser et al. (2015)	Steel et al. (2017)	Trappler and Newville (2007)

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	Adverse effects Dropouts																	None: No adverse events or 0/9 = 0% symptom exacerbation An increase in voice frequency N/A occurred at mid-treatment, but this was not appraised negatively by the	participant, with low distress. This resolved later in treatment
	Secondary outcomes Ar																	Mean charge: No PDS: 80% showed decrease following treatment, 25% showed reliable charge (RC) at 6-month follow-up Mean at baseline; 23.38 Mean af ollowing treatment, 23.38 Mean af ormonth follow-up; 23.45 BD-II mean following treatment, 40% showed reliable charge (RC) at 6-month follow-up) BD-II mean tabseline; 24.5 BD-II mean tabseline; 24.5 BD-II mean tabseline; 24.5 BD-II mean tabseline; 24.5 BD-II mean tabseline; 24.6 Mean at 6-month follow-up Mean at 6-month follow-up. Mean at 6-month follow-up. 20.33 Mean 46-month follow-up. 20.33 M	Below level of probable PTSD following treatment DASS: Baseline: 40 Post-intervention: 24 6-month follow-up: 31 No reliable or clinically significant change
	Primary outcomes 5	Significant decrease in TICBT group following treatment: $z = -4.20$ ($p < 0.001$), $r = -0.9$. Non statistically significant decrease in the supportive psychotherapy group	BPRS subscale—hallucinatory behaviour:	Baseline mean Post-treatment mean	TICBT NR NR	Sup. Psy. NR NR	Non statistically significant decrease in TCBT group following treatment. Non statistically significant decrease in the supportive psychotherapy group	BPRS subscale-unusual thought content:	Baseline mean Post-treatment mean	TICBT NR NR	Sup. Psy. NR NR	Significant decrease in TICBT group following treatment: $z=-2$ ($\rho=0.046,r=-0.43$. Non statistically significant decrease in the supportive psychotherapy group	BPRS subscale—suspiciousness:	TICBT NR NR	Sup. Psy. NR NR	Significant decrease in TICBT group following treatment: $z = -4.24$ ($p < 0.001$), $r = -0.9$. Significant decrease in the supportive psychotherapy group following treatment: $z = -2.07$ ($p = 0.039$), r = -0.44		Mean change: PSYRATS-AHRS: Baseline mean post-treatment mean TFCBT 2956 20.5 63% showed decrease following treatment. 29% showed reliable change (RCI) at 6-month follow-up) PSYRATS-DRS: Baseline mean post-treatment mean TFCBT 13.57 8.33 67% showed decrease following treatment. 4.3% showed reliable change (RCI) at 6-month follow-up) PSYRATS-AHRS: Baseline score post-treatment score	TFCBT 36 23 E Reliable but not clinically significant change following treatment E P P
led)	(evel ence). Population, including diagnosis, location and setting Treatment and dose																	 Schlzophrenia spectrum P. Garder or PTSD or cognitive restructuring, psychotic depresion (ICD- psychotic symptoms Psychotic symptoms 	
TABLE 1 (Continued)	Design (level of evidence), Author n																TFCBT	Keen et al. (2017) Case series (4) $n = 9$ (4) $n = 9$ (4) $n = 9$ (4) $n = 9$ (4) $n = 9$	

EID ET AL.			21% in NDR 2015)	_WILEY
Dropouts	NA	۲ ۲	EMDR: 11/53 = 21% PE: 13/53 = 25% No statistically significant difference in dropout between EMDR and PE (van den Berg et al., 2015)	
Adverse effects	۳	Ϋ́Ζ	None: Fewer participants in the trauma-focused conditions experienced symptom exacerbition or adverse events as compared to the TAU condition	
Secondary outcomes	IES: Baseline: 41 Post-Intervention: 10	IES-R: Baseline: 71 Post-intervention: 25 -month follow-up: 8 CAPS: CAPS: CAPS: Baseline: 87 bost-intervention: 44 6-month follow-up: 4 Baseline: 13 Post-intervention: 13 Post-intervention: 13 Baseline: 25 For the follow-up: 1 Baseline: 25 Post-intervention: 13 Post-intervention: 13 Post-intervention: 13 Post-intervention: 13 Post-intervention: 2 For the follow-up: 0 Post-intervention: 7 C-month follow-up: 0 Post-intervention: 7 C-month follow-up: 0 Post-intervention: 7 C-month follow-up: 0	PE versus TAU LMM analysis LMM analysis BDI-III: Significant decrease in PE group as compared to TAU following treatment: 1175 = -3.64 ($\beta < 0.001$); Hedges' $g = 0.77$ Significant decrease in PE group as compared to TAU at 6-month rollow-up; 1175 = -2.92 ($\beta = 0.0001$); Hedges' $g = 0.64$ Fe superior to TAU over time: = -3.52 ($\beta = 0.0001$) No significant changes on BDI-II between 6 and 12-month follow-up (van den Beng et al. 2018) Non statistically Significant threases in PE group as compared to TAU following treatment, 6-month follow-up, and across all time points PSF: Significant decrease between 6- and 12-month follow-up in PE group: t = 4.31, ($\rho < 0.001$)	
	Post-treatment score 22 Post-treatment	Post-treatment score	Et versus TAU LMM analysis PSYRATS-AHRS: PSYRATS-AHRS: Baseline mean PE 21.7 18.8 TAU 21.7 18.8 TAU 23.3 24.2 Non statistically significant decrease in PE group as compared to TAU at 6-month following treatment and across all time points: non statistically significant increase in PE group as compared to TAU at 6-month follow-up GPTs: Baseline mean Post-treatment mean	PE 888 67.3 TAU 838 67.3 FaU 838 82.7 Significant decrease in PE group as compared to TAU following treatment: $t = -2.86$ ($p = 0.005$), Hedges $g = 0.62$ Significant decrease in PE group as compared to TAU at 6-month follow-up: $t = -2.46$, ($p = 0.015$), Hedges $g = 0.54$ Fe superior to TAU acos all time points: $t = -3.03$ ($p = 0.003$) No significant changes on GPTS between 6- and 12-month follow-up (van den Berg et al., 2018)
	Baseline score 60 Baseline 8	Baseline score	Baseline mean 21.7 23.3 1.7 23.3 2.3 2.3 2.3 2.3 2.3 2.3 2.3 1.5 2.5 2.5 2.5 1.5 2.5 2.5 2.5 2.5 2.5 2.5 2.5 2.5 2.5 2	888 67.3 U 83.8 67.3 freamt decrease in PE group as compared to TAU fol freamt decrease in PE group as compared to TAU at freamt decrease in PE group as compared to TAU at follow the decrease in PE group as compared to TAU at follow to the 2.28.6 ($p = 0.005$), Hedges' $g = 0.54$ for follow the acrease all time points: $t = -3.03$ ($p = 3.015$) ($p = 3.015$ ($p = 3.015$) ($p = 3.015$) ($p = 3.015$) gridficant changes on GPTS between $6 - and 12$ -mor (van den Berg et al., 2018)
Primary outcomes	CPRS: TFCBT SANS: TFCBT	PSYRATS-AHRS: TFCBT	PE versus TAU LMM analysis PSYRATS-AHRS: PE TAU Non statistically sig follow-up follow-up follow-up follow-up	PE 888 TAU 838 Significant decrease in PE group as co treatment: t = -2.86 (p = 0.005 Significant decrease in PE group as co follow-up: t = -2.46. (p = 0.015 Follow-up: t = -2.46. (p = 0.015 PE superior to TAU across all time pr PE superior to TAU across all time pr No significant changes on GPTs betw (van den Berg et al., 2018)
Treatment and dose	TFCBT (exposure, imagery rescripting cognitive restructuring) 17 sessions Length of time: NR	TFCBT and EMDR (coping strategies, imagery rescripting, prolonged in- vivo exposure via EMDR, reliving) 33 sessions 33 sessions Length of time: 1 year Length of time: 1 year	EMDR or PE Minimum: 8 Mean (E): 7.1 Mean (EMDR): 7.8 Maximum: 8 weeks Length of time: 8 weeks	
Population, including diagnosis, location and setting Treatment and dose	Schizophrenia (ICD-10 criteria): plus PTSD (ICD-10 criteria): UK; outpatient service	First-episode psychosis (meeting criteria for EIP service): UK; outpatient service	Lifetime diagnosis of a psychotic disorder (MINI psychotic disorder (MINI pus criteria) plus criteria PTSD (DSN-IV-TX fracteria on the CAPS). Netherlands, 13 outpatient services	
Design (level of evidence), I n	Case report (5) n = 1	Case report (5) $n = 1$	Multicentre I RCT (1b) n = 155	
Author	Calcott et al. (2004)	Ward-Brown et al. (2018)	EMDR de Bont et al. (2016)	

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	Adverse effects Dropouts												None: No participant showed Emdr : 2/15 = 13% any exacerbation of PMR : 1/15 = 7%	ithdraw	due to a worsening or their significant condition difference in rate								
	Secondary outcomes		EMDR versus TAU LMM analysis (ITT)	BDI-II: Non statistically significant decrease in EMDR group as compared to TAU following treatment, 6-month follow-up and across all time points	No significant changes on BDI-II between 6 and 12-month follow-up (van den Berg et al., 2018)	PSP: Non statistically significant increase in EMDR group as compared to TAU following treatment, 6-month follow-up and across all	time points Paired sample t-test: PSP: Significant decrease in EMDR group as compared to TAU between 6 and 12-month follow-up in EMDR: t = 2.08 (p = 0.044)						Repeated measures ANOVA: HAM-D: Non statistically significant decrease in EMDR group as	compared to PMR and TAU HAM-A: Non statistially significant decrease in EMDR group as									
		EMDR superior to TAU following treatment: OR = 3.17 (p = 0.013) PE participants non statistically significantly more likely than TAU participants to be in remission at 6-month follow-up FE superior to TAU across all time points: OR = 2.325 (p = 0.020) No significant changes in the number of participants in remission at 12-month follow-up (van den Berg et al., 2018)			Post-treatment mean 16.8	24.2	Non statistically significant decrease in EMDR group as compared to TAU following treatment and 6-month follow-up. Non statistically significant increase in EMDR group as compared to TAU across all time points		Post-treatment mean	68	82.7	Significant decrease in EMDR group as compared to TAU following treatment: $t = -2.68$ ($\mu = 0.008$), Hedges' $g = 0.57$ Non stratistically significant decrease in EMDR group as compared to TAU at 6-month follow-up. Non stratistically significant decrease in EMDR group as compared to Significant changes on any measure between 6 and 12-month follow-up (van den Berg et al., 2018) GEI sensitivity analysis (odds ratio): Remission from psychotic disorders (SCI-SR-PANS): Remission from psychotic disorders (SCI-SR-PANS): Remission from psychotic disorders (SCI-SR-PANS): EMDR participants significantly more likely than TAU participants to be in remission at 6-month follow-up and across all time points: No significant changes in the number of participants to remission at 12-month follow-up (van den Berg et al., 2018)		Post-treatment mean	62.7	61.7	67.2	Non statistically significant decrease in EMDR group as compared to PMR and TAU		Post-treatment mean	12.2	12.9	15.4
	comes	rior to TAU following trea ints non statistically signif pants to be in remission a to TAU across all time po nt changes in the numbe nth follow-up (van den B	sis (ITT)		Baseline mean 24.5	23	statistically significant decrease in EMDR group as con TAU following treatment and 6-month follow-up. Non statistically significant increase in EMDR group as con TAU across all time points		Baseline mean	82.7	83.8	Significant decrease in EMDR group as compared to TAI treatment: t = -2.68 (p = 0.008), Hedges' g = 0.57 Non statistically significant decrease in EMDR group as. TAU at 6 -month follow-up EMDR superior to TAU across all time points: t = -2.38 No significant changes on any measure between 6 and follow-up (van den Berg et al., 2018) GEE sensitivity analysis (odds ratio): EMDR aparticipants significantly more likely than TAU pa participants to be in remission at 6-month follow-u all time points No significant changes in the number of participants in 12-month follow-up (van den Berg et al., 2018) 12-month follow-up (van den Berg et al., 2018)	Repeated measures ANOVA:	l: Baseline mean	73.1	69.8	76.8	statistically significant decrease PMR and TAU	tive:	Baseline mean	16.9	15.9	18.8
	Primary outcomes	EMDR super PE participa partici PE superior No significa 12-mo	EMDR versus TAU LMM analysis (ITT)	PSYRATS-AHRS:	EMDR	TAU	Non statisti TAU fc statisti TAU a	GPTS:		EMDR	TAU	Significant d reatm Non statistic TAU an EMDR super Significa Be liot Pe in r EMDR partic partici all tim No significa 12-mo	Repeated m	PANSS total:	EMDR	PMR	TAU	Non statistio PMR a	PANSS positive:		EMDR	PMR	TAU
	el 9. Population, including diagnosis, location and setting Treatment and dose												Diagnosis of schizophrenia EMDR (DSM-1V criteria), inpatient Minimum: 3	stay of over 1 week; South Average: NR Korea; 1 inpatient service Maximum: 3	LENGTH OF TIME: 5 WEEKS								
(Continued)	Design (level of evidence), n												RCT (2b) n = 45										
TABLE 1 (0	Author												Kim et al. (2010)										

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																												(Continues)	
	Dropouts								15/47 = 32%			5/27 = 19%																(Con	
	Adverse effects								EMDR treatment was completed by 68% of the sample EMDR treatment was	experienced as stressful with an increase of instability in 4	participants, leading to dropout	3 participants reported increased stress or PTSD	symptoms. In these cases,	one session was dedicated to coning skills which was	enough to help them regain	control and motivation for treatment	2 participants contacted their case manager to discuss	increased arousal, which later resolved	1 participant had a single relance into drug use	after leaving the home	years No other advorce avents were	reported							
	Secondary outcomes								t-test for dependent samples (pre to post): PDS: significant decrease following treatment: $t = 7.94$, $(p < 0.001)$, Hedges' $g = 2.26$			Paired sample t-test (pre to post): CAPs: Significant decrease following treatment: $t = 7.26$ ($n = 0.000$).	Hedges' g = 1.49	PSS-SR : Significant decrease following treatment: $t = 6.23$ ($p = 0.000$), Hedges' $a = 1.28$	BDI-II : Significant decrease following treatment: $t = 4.81$ ($p = 0.000$),	Hedges' $g = 0.99$ BAI: Significant decrease following treatment: $t = 4.4$ ($p = 0.000$),	Hedges $g = 0.91$												
	Primary outcomes	Non statistically significant increase in EMDR group as compared to PMR: non statistically significant decrease in EMDR group as compared to TAU	PANSS negative:	Baseline mean Post-treatment mean	EMDR 18.7 16.2	18.5		Non statistically significant decrease in EMDR group as compared to PMR and TAU	Wilcoxon signed rank test last observation carried forward (pre to post): PSYRATS-AHRS (median):	Baseline mean Post-treatment mean EMDR 0 0	Significant decrease following treatment: $Z = -2.12$ ($p = 0.034$), Hedges' $g = 0.2$ Without observation carried forward: Non statistically significant decrease following treatment	Wilcoxon signed rank tests (pre to post):	PSYRATS-AHRS:	Baseline mean Post-treatment mean	EMDR NR NR	Significant decrease following treatment: $z = -2.17$ ($p < 0.030$), r = 0.33	PSYRATS-DRS:	Baseline mean Post-treatment mean		Significant decrease following treatment: $z = -2.02$ ($p < 0.043$).		Reseline mean Doct-treatment mean		Significant decrease following treatment: $z = -2.67$ ($p < 0.008$), $r = 0.40$	Paired sample t-test:	Baseline mean Post-treatment mean	EMDR 72.1 65.6	GPTS. Non statistically significant decrease following treatment	
	Population, including diagnosis, location and setting. Treatment and dose Pr	N	PA			L	L	Z	y disorder (DSM- EMDR. Participants were riterial; Juus PTSD undergoing other therapies V-TV criteria); simultaneously in TAU lands; 1 outpatient (psychodynamic		supporting essions (39%), family therapy (7%) or Sig other therapy (5%) Minimum: 2 Median: 4 Maximum: 15	PTSD (criteria not reported); EMDR, focused on trauma W blus a lifetime that caused current PTSD	ipectrum Minimum: NR	4	Length of time: 6 weeks	Sig	Sd		Ξ	Sie	ž	2	Ξ	Sie	Pa		Ε	5	
(Continued)	Design (level of evidence), Pop n dia								Case series Per $(4) n = 47$ (1)			Multicentre PTS case n	(4)																
TABLE 1 (Co	Author								Slotema et al. (2019)			van den Berg and van der	Gaag (2012)																

TABLE 1 (C	(Continued)								
Author	Design (level of evidence), n	Population, including diagnosis, location and setting Treatment and dose	Treatment and dose	Primary outcomes	ues		Secondary outcomes	Adverse effects	Dropouts
de Bont et al. (2013)	Case series (4) <i>n</i> = 10	Under treatment for current psychotic symptoms: plus PTSD (DSM-IV criteria): Netherlands, 1 outpatient service	PE or EMDR Minimum: NR Mean (PE): 9 Mean (EMDR): 11.5 Mean (EMDR): 11.5 Length of time: NR Length of time: NR	Wilcoxon pairwise test: PSYRATS-AHRS (estima Base Base EMDR 14.5 Non statistically significa follow-up follow-up PSYRATS-DRS (estimate Base EMDR 5.68	oxon pairwise test: RATS-AHIRS (estimated marginal means): Baseline mean ADR 14.54 14.54 14.54 14.54 14.54 statistically significant decrease following follow-up follow-up Baseline mean Baseline mean	Wiccoxon pairwise test: PSYRATS-AHRS (estimated marginal means): Baseline mean Post-treatment mean EMDR 14.54 10.67 Non statistically significant decrease following treatment and 3-month follow-up PSYRATS-DRS (estimated marginal means): Baseline mean Post-treatment mean Baseline mean Post-treatment mean EMDR 5.68 1.49	Wilcoxon pairwise test: PSS-SR: Significant decrease following treatment: $r = 0.73$ ($p < 0.001$) and 3-month follow- $p (p < 0.001$) and 3-month follow-up ($p < -0.02$) r = 0.49 and 3-month follow-up: $Z = -2.52$ ($p = 0.012$), $r = 0.63Q = 0.28$), $r = 0.69$ and 3-month follow-up: $Z = -2.19(p = 0.028), r = 0.69 and 3-month follow-up: Z = -2.37(p = 0.028), r = 0.63 and 3-month follow-up: Z = -2.37(p = 0.018), r = 0.75SF: Non statistically significant increase following treatment and 3-month follow-up$	None: No participants showed an increase in symptoms or deterioration in social functioning or clinically adverse events	Emdr: 1/5 = 20% P.E. 1/5 = 20%
Yaşar et al. (2017)	Case report (5) n = 1	Schizophrenia (criteria not reported): Turkey; inpatient service	EMDR 2 sessions Length of time: 2 weeks	Non statistically follow-up PANSS: EMDR BPRS: EMDR	/ significant decrease follo, Baseline score 78 Baseline score 37	Non statistically significant decrease following treatment and 3-month follow-up PANSs: Baseline score 5-month follow-up score EMDR 78 34 BPRS: Baseline score 5-month follow-up score EMDR 37 3	CAPS: Baseline: 96 5-month follow-up: 12 IES-R: Baseline: 25 5-month follow-up: 15 BOI: Baseline: 30 Post-intervention: 16	Ϋ́	e viz
Other Brand and Loeverstein (2013)	Case series (4) n = 237	DID (criteria not reported); 19 Phasic trauma treatment for countries: many outpatient DID services Average: NR Average: NR Maximum: NR Length of time: NR	Phasic trauma treatment for DID Minimur: NR Averge: NR Maximur: NR Length of time: NR	ANOVA (across SCL-90-R-heart Baseline mean 1.89	ANOVA (across 6-, 18- and 30-month follow-up): SCL-90-R-hearing voices item: Baseline mean 189	ilow-up): 6-month mean 1.63	CDS: CDS: Baseline: 16 5-month follow-up: 6 Baseline: 37 Post-intervention: 24 5-month follow-up: 4 N/A	¥	斑
Paulik et al. (2019) Case series (4) <i>n</i> = 11	(d) n = 12 (d) n = 12	Currently hearing voices; plus experiencing PTSD symptoms that appear directly to indirectly linked to the voices (no symptom threshold). Australia: 1 outpatient service	Significant decrease across all time points: F = 340 (p = 002) Hedges's at 6-month of treatment: 0.16 Hedges's at 16-month of treatment: 0.25 Hedges's at 18-month of treatment: 0.25 Hedges's at 18-month of treatment: 0.25 Minimum: NR Minimum: NR Minimum: NR LMM analysis over time (baseline, mid-intervention, post-treatment): Average: NR Average: NR FSYRATS-AHRS distress: Maximum: 10 Average: NR Baseline mean Average: NR Baseline mean Analysis over time (taseline, mid-intervention, post-treatment): Average: NR Analysis over time (taseline, mid-intervention, post-treatment): Average: NR Analysis over time (taseline, mid-intervention, post-treatment): Average: NR Analysis over time (taseline, mid-intervention, post-treatment): 16 Analysis over time (tase across all time points: t = -333 (p = 001) Hedges's following treatment: 0.69 No significant increase from post-treatment to 3-month follow-up	Significant decrease acros Hedges' g at 6-month of Hedges' g at 30-month of Hedges' g at 30-month of Hedges' g at 30-month of Hedges' g at 30-month of LMM analysis over time PSYRATS-AHRS distress Baseline mean 16 16 Significant decrease acros Hedges' g following treat No significant increase fro	Significant decrease across all time points: $F = 3.40$ ($p = 0.02$) Hedges' g at 6-month of treatment: 0.16 Hedges' g at 30-month of treatment: 0.25 Hedges' g at 30-month of treatment: 0.22 LMM analysis over time (baseline, mid-intervention, post-treatme PSYRATS-AHIS distress: Baseline mean Post-treatment me 16 12 Significant decrease across all time points: $t = -3.33$ ($p = 0.01$) Hedges' g following treatment: 0.69 No significant increase from post-treatment to 3-month follow-up	<pre>% F = 3.40 (p = 0.02) % % % % % % % % % % % % % % % % % % %</pre>	LMM analysis over time (baseline, mid-intervention, post-treatment): PSS: significant decrease over time: $t = -3.62$ ($p = 0.005$), Hedges' g = 0.74 decrease over time: $t = -3.62$ ($p = 0.005$), Hedges' DASS depression: Non statistically significant decrease across all time points DASS anxiety. Non statistically significant decrease across all time points	Two patients reported an initial 1/12 = 8% increase in intrusions which lasted 1 week. No other adverse events occurred	1/12 = 8%

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	Dropouts													n or long- 0/24 = 0%	vents were																(Continues)			
	Adverse effects														<i>p</i> < U.UU.1 iasting adverse events were reported																			
	Secondary outcomes													Paired t-test (baseline and post):	CAPS: significant decrease following treatment: $t = 4.2$, $(p < 0.001)$ Hedges' $g = 0.65$																			
			Post-treatment mean	6	Significant decrease across all time points: $t = -7.47$ ($p < 0.001$). Hedges $g = 0.74$	No signincant increase from post-treatment to 3-month follow-up BAVQ (malevolence):	Post-treatment mean	80	Significant decrease across all time points: t = 2.22 (p = 0.033), Hedges' g = 0.34		Post-treatment mean	10	Non statistically significant decrease across all time points	post):		Post-treatment mean	69.8	Non statistically significant increase following treatment		Post-treatment mean	14.3	Non statistically significant decrease following treatment		Post-treatment mean	23.9	Non statistically significant increase following treatment		Post-treatment mean	31.6	Non statistically significant increase following treatment				
	Primary outcomes	PSYRATS-AHRS frequency:	Baseline mean	6	Significant decrease across Hedges' g = 0.74	No significant increase froi BAVQ (malevolence):	Baseline mean	6	Significant decrease across $g = 0.34$	BAVQ (omnipotence):	Baseline mean	11	Non statistically significant	Paired t-test (baseline and post):	PANNS total:	Baseline mean	68.6	Non statistically significant	PANNS positive:	Baseline mean	14.4	Non statistically significant	PANNS negative:	Baseline mean	22.9	Non statistically significant	PANNS global:	Baseline mean	31.4	Non statistically significant				
	Treatment and dose													3-h video testimony	session over 1-2 sessions																			
	Population, including diagnosis, location and setting Treatment and dose													Schizophrenia diagnosis,	noiocaust survivors; Israel; 2 inpatient	services																		
.E 1 (Continued)	Design (level of evidence), n														case series $(4) n = 24$																			
TABLE	Author													Strou																				

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Author	Design (level of evidence), <i>n</i>	Design (level of evidence), Population, including <i>n</i> diagnosis, location and setting Treatment and dose		Primary outcomes		Secondary outcomes	Adverse effects	Dropouts
Arens (2014)	Case report (5) $n = 1$	Case report Combat PTSD and (5) <i>n</i> = 1 hallucinations: USA: 1 outpatient service	Trauma management therapy (15 VR assisted imaginal and in-vivo exposure with 14 group social and	No. auditory hallucinations per week: Baseline	Post-treatment	CAPS Baseline: 91 Baseline: 21 3-month follow-up: 33	A slight increase in hallucinations in the first week of treatment, which resolved in the following	N/A
			emotional skill sessions) Length of time: 3 weeks	/ No. visual hallucinations per week:	H	PCL-M: Baseline: 64 Post-intervention: 28	weeks	
				Baseline	Post-treatment			
				2	7	Baseline: 6.4 Post-intervention: 1.3		
						3-month follow-up: 1.1 No. social activities per week:		
						Baseline: 0 Post-intervention: 5		
						3-month follow-up: 0		

Scale: PE, prolonged exposure: PSP, , randomised controlled trial; SANS, Scale; HAM-D, Hamilton Rating RCT. Diagnostic Anxiety Posttraumatic Diagno , Quality of Life Scale; HAM-A, Hamilton Scale Social Adjustment r version; PDS, F ng Scale; QLS, C Scale; **Thoughts** Rating Checklist-Military and bioi Work Par Del NSAS PSYRATS-DRS Scale: PTSD PCL-M, GPTS, Scale; I disorder; : Syndrome Scale nations Rating S Social F dentity Hallucinations SFS. Checklist-90-Revised: and Negative dissoc nptom Rating Scale—Auditory DID, Positive Symptom Questionnaire-45.2; PANSS, SCL-90-R. PANSS ę Outcome -Report; PSYRATS-AHRS, for symptoms of Remission not reported; OQ-45.2 CPRS, enia; Scale-Self Schizoph for Revised; NR, PTSD Symptor Scale for Clinical Scaleession t of Events S Depre Calgary Impact Scale; SCI-SR-PANSS. CDSS, IES-R, Perceived Events Scale; | Schizoph PSS, Scale for ession Rating Scale; IES, Impact of scale; PTSD f, red Per Social -Admini and nal for Depre Clinic Scale

3.4 | Prolonged Exposure

Two studies used PE (Foa et al., 2007), a protocol which focuses solely on exposure without a cognitive component. One RCT (de Bont et al., 2016) found a superior effect for PE over control in reducing paranoia following treatment, which sustained at 6 and 12-month follow-up (van den Berg et al., 2018). In addition, PE participants were significantly more likely to be in remission from a psychotic disorder at follow-up. No significant impact for PE over the control for auditory hallucinations was found. A study that combined participants that had undergone PE or EMDR as one treatment group has been reported above (de Bont et al., 2013).

Regarding secondary outcomes, PE resulted in statistically significant decreases on depression scores following treatment and 6month follow-up compared to the control, but not on social functioning scores (de Bont et al., 2016).

3.5 | Other interventions

One case series investigated the effects of 'phasic trauma treatment' for Dissociative Identity Disorder (Brand & Loewenstein, 2013) (incorporating an exposure component), described in the Guidelines for Treating Dissociative Identity Disorder in Adults (International Society for the Study of Trauma and Dissociation, 2011). They found this treatment had a significant impact on a hearing voices item, with effect sizes growing larger the longer the person was in treatment.

There were no secondary outcomes reported relevant to this review within this study.

One case series investigated the effects of imagery rescripting (Paulik et al., 2019). This intervention had a beneficial impact on voice hearing, with auditory hallucination distress and frequency, and belief in voice malevolence seeing significant decreases following treatment, sustained at 3-month follow-up. However, belief in voice omnipotence did not significantly decrease following treatment.

This study reported on PTSD symptomology, finding a significant decrease following treatment. No significant effect was found on depression or anxiety measures (Paulik et al., 2019).

One case series investigated the effects of a one-off video interview regarding a personal experience of the Holocaust, with a focus on details about losses and grief experienced (Strous et al., 2005). There was no effect on psychotic nor PTSD symptoms.

One case report (Arens, 2014) looked at the effectiveness of trauma management therapy (Turner et al., 2005) (TMT) in a combat veteran. TMT incorporates in vivo and virtual reality assisted imaginal exposure sessions together with group social and emotional coping skills. The number of self-reported auditory and visual hallucinations declined from 7 to 1, and 2 to 0 respectively by 3-month follow-up. Two measures of PTSD symptomology showed a decrease at 3-month follow-up.

3.6 | Tolerability and acceptability

Regarding tolerability, 41% (7/17) of all studies and 100% (3/3) of the non-exposure based protocols did not report on adverse events or harm. Of the exposure-based studies, 29% (4/14) did not report on harm, 36% (5/14) reported that there were no instances of adverse events or harm, with one (de Bont et al., 2016) reporting that fewer participants in the active condition (EMDR) experienced symptom exacerbation or adverse events than in the control condition. 29% (4/14) reported a brief symptom exacerbation early in treatment that resolved later, sometimes with brief (i.e. one session or one conversation) additional support provided (e.g. psychoeducation about increased arousal when starting a new intervention). Only one reported exacerbation that may not have resolved; in this case stress and an increase in instability leading to dropouts in 4/47 (9%) of their participants, with only 32/47 (68%) completing EMDR treatment (Slotema et al., 2019).

Regarding acceptability, 17% (2/12) of studies did not report on dropouts (five studies have not been included in this calculation due to being case reports). We amalgamated dropout rates within treatment modality (excluding again case reports) and found a dropout rate of 22% (26/119 participants) within TICBT studies, 0% (0/9) within TFCBT studies, 23% (34/147) within EMDR studies, 24% (14/58) within PE studies and 3% for other interventions (1/36 participants). In total, this results in a dropout rate of 22% (26/119 participants) for non exposure-based protocols and 20% (49/250 participants) for exposure-based protocols.

4 | DISCUSSION

In our systematic review with broad inclusion criteria, we found only 17 studies of TFPT in psychosis, with the literature largely comprised of case series. Nevertheless, several well-controlled studies indicate that TICBT, which does not utilise exposure, is not effective at reducing psychotic symptoms. Regarding those approaches that do use exposure to facilitate trauma memory reprocessing, the majority of the high-quality evidence results from the large multicentre RCT of de Bont et al. (2016). Though this study provided evidence in favour of PE and EMDR, the study protocol included psychotic symptoms as a secondary outcome. Whilst included case studies and case reports further support the idea that exposure-based interventions may be effective, the low robustness of the evidence base means that the effectiveness of TFPT for psychotic symptoms is equivocal.

Our finding of greater evidence for the use of exposure as compared to non exposure-based interventions in reducing psychotic symptoms is in keeping with a previous review (Brand et al., 2018). In our review, 11 of the 14 studies utilising exposure found a positive impact for TFPT on at least one psychotic symptom, whilst one of three of the studies not utilising exposure did. This provides support for the view that the inclusion of trauma memory reprocessing is necessary to address psychotic symptoms in the context of trauma. This is also in-keeping with a process analysis of intervention sessions which concluded that exposure is required to treat trauma symptoms in patients with psychosis (Steel et al., 2016).

A crucial point to consider regarding the studies in this review is that they were primarily oriented towards treating PTSD. This means the interventions were often not targeted to traumatic memories that may be directly or indirectly related to psychotic symptoms. This is important considering research which has shown relationships between the content of psychotic symptoms in trauma survivors and experiences of trauma (Hardy et al., 2005; McCarthy-Jones et al., 2012; Onyeama et al., 2011; Peach et al., 2020; Vila-Badia et al., 2021).

Several included studies which did show a positive effect of TFPT on psychotic symptoms described clear links between trauma and psychotic symptoms in their participants (Arens, 2014; Yasar et al., 2017). Paulik et al. (2019) also chose to focus their trauma intervention on traumas that appeared related to psychotic phenomena, and reported the largest effect sizes in the review (g = 0.69for distress and g = 0.74 for frequency of voices following treatment), maintained at 3 months. Across all participants, trauma memory intrusions and voices reduced concurrently, suggesting the two symptom domains may be related by common underlying processes. This accords with theories suggesting that similar mechanisms are involved in PTSD and trauma-induced psychotic symptoms (Bloomfield et al., 2021; Morrison et al., 2004) and that, in the context of trauma, auditory hallucinations may be a type of post-traumatic intrusion related to a memory (Steel, 2015). Therefore, when a patient is able to construct a complete memory of the traumatic event through exposure during TFPT, the memory may stop being retrieved involuntarily through intrusions such as voices.

It may therefore be important psychotherapeutically to differentiate between focussing on a trauma memory that pre-dates psychosis (in which case the trauma may be relevant to the aetiology of the psychosis) and a trauma memory that took place after the onset of psychosis. In the present review, many of the included studies included substantial proportions of post-psychosis onset trauma (e.g. 18% (Steel et al., 2016), 18% (de Bont et al., 2016), 30% (van den Berg & van der Gaag, 2012)) which may have contributed to some of the null results. Future research will benefit from directly addressing this question.

The use of exposure for people with psychotic symptoms has been a concern for some clinicians, who believe that exposure may cause harm by exacerbating psychotic symptoms (Cragin et al., 2017). In this review, though some studies did report a temporary increase in distress and/or symptoms, this is a typical response to exposure in trauma treatment as it is designed to elicit and facilitate the therapeutic processing of distress (Foa et al., 2002). Overall, the majority of those that reported on harm reported no harm, with one study reporting fewer adverse events in the TFPT group than the control (de Bont et al., 2016). Furthermore, there was a similar dropout rate between non exposurebased (22%; 26/119 participants) and exposure-based (20%; 49/ 250 participants) protocols. These dropout rates are comparable to those of CBTp (Lincoln et al., 2008) (16%). This indicates that TFPTs, and exposure specifically, have acceptable levels of tolerability and acceptability. This finding accords with van den Berg et al. (2015) who reviewed adverse events during PE or EMDR with people with psychosis, finding that these treatments were associated with significantly less symptom exacerbation and adverse events than waitlist conditions.

It is important to consider the limitations of the existent literature. There were only four controlled studies included in this review, only two of which used an intervention meeting the criteria of TFPT (Ehlers & Clark, 2000). The methodological quality of studies was impacted by low sample sizes, lack of blinding and other methodological issues. As many studies did not specifically target psychotic symptoms, the inclusion criteria of studies did not always necessitate the high levels of psychotic symptoms in all participants, or for psychotic symptoms to be present at all (e.g. de Bont et al., 2013; Slotema et al., 2019). This means the studies may have had low power to detect and may fall victim to floor effects.

Our review has a number of strengths. We pre-registered our review and adhered to the robust a priori criteria. We took a broad view of psychotic symptoms and did not limit inclusion criteria to any diagnosis, syndrome, or trauma type. We calculated Hedges' *g* where possible (a more reliable measure of effect size for small sample sizes) (Cumming, 2012). The broad range of studies included in the review and the use of risk of bias measures appropriate to each also represents a strength.

A limitation of our review is that it has not been possible to meta-analyse the available data as we did not meet our a priori criteria due to the lack of controlled studies and variation in psychotherapeutic intervention. This has also rendered it not possible to differentiate further between types of TFPT that use exposure, for example, those that include a significant cognitive component compared to those that do not. There remains a risk of selection bias in this review as some forms of therapy would likely involve reappraisal of a trauma memory (e.g. learning that a traumatic experience was not their fault) and so could be described as trauma-focused according to our broad criteria, but would not necessarily have been picked up our search.

Several clinical recommendations arise from this review. Given the evidence of a relationship between trauma and psychosis, there is a clear need for clinicians to consider and assess for histories of trauma and PTSD in people with psychotic symptoms (strong recommendation).

TFPT with exposure should not be withheld by default from all patients for fears of safety or causing harm. Consistent with previous reviews (Sin & Spain, 2016; Swan et al., 2017), we found that TFPT is effective in reducing PTSD symptoms in people with psychosis, and we also found that TFPT may reduce psychotic symptoms. Given that we further found TFPT to be well tolerated and acceptable, there appears to be little justification for withholding TFPT from people with psychosis (conditional recommendation).

Several research recommendations arise from this review. Most pressingly, more high-quality research is needed, specifically with psychotic symptoms as a primary outcome and on patients with trauma histories. We look forward to the findings of controlled research which is underway (Peters, 2020; Valiente-Gómez et al., 2020). Future research will benefit from a standardisation of the measure used for psychotic symptoms to enable direct comparison between studies: there have been recent (albeit controversial) movements to do so for non-psychotic measures (The Lancet Psychiatry, 2020). The diversity of measures currently used in the literature serves as a barrier to understanding, obfuscating potentially promising findings. Furthermore, future research can compare outcomes for different types of TFPT that do utilise exposure. For example, TFCBT, which follows exposure with cognitive techniques to restructure and reappraise peri- and post-traumatic cognitions, may differ from PE, which does not. Finally, future research can consider the question of whether targeting the memory reprocessing intervention to traumas that appear related to the psychotic symptoms is most effective. Such a study could also investigate if there is a difference in efficacy between people that identify a direct (e.g. hallucination as a trauma memory), indirect (content is thematically linked) or no association between the trauma and the psychotic symptoms. Researchers such as Peach et al. (2020) and Hardy et al. (2005) have suggested that direct associations indicate a key role of posttraumatic intrusions, whilst indirect associations suggest a larger role of beliefs and emotions. This therefore indicates different treatment protocols may be more effective depending on the type of links that can be made between trauma and psychotic symptoms.

5 | CONCLUSION

The evidence base of effectiveness for TFPT in traumatised people for reducing psychotic symptoms is currently weak. There is tentative evidence in favour of exposure-based interventions which provides support for the view that the inclusion of trauma memory reprocessing may be necessary to treat psychotic symptoms in trauma survivors. Further well-controlled studies of TFPT for psychotic symptoms are needed. Our analysis indicated that TFPT was well tolerated, and acceptability levels were comparable to other psychological interventions for psychotic symptoms. Therefore, there is little to suggest that TFPT for PTSD should be withheld from people with psychotic symptoms.

AUTHOR CONTRIBUTIONS

Jordan Reid: Conceptualization; data curation; formal analysis; investigation; methodology; project administration; supervision; writing – original draft; writing – review & editing. Charles Cole: Data curation; formal analysis; investigation; validation. Nabeela Malik: Data curation; formal analysis; investigation; validation. Vaughan Bell: Conceptualization; methodology. Michael Bloomfield: Conceptualization; funding acquisition; methodology; supervision.

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CONFLICT OF INTEREST STATEMENT

The authors have declared that there are no conflicts of interest in relation to the subject of this study.

DATA AVAILABILITY STATEMENT

The authors confirm that the data supporting the findings of this study are available within the article and its supplementary materials.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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