TECHNICAL COMMENTARY

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Pathways to care

Introduction

The help-seeking efforts made by an individual and their families when symptoms of PTSD are apparent, and the clinical services made available as a result of these efforts, are collectively known as 'pathways to care'. Pathways to care can also encompass service structures that have not been actively sought by the individual.

Method

We have included only systematic reviews (systematic literature search, detailed methodology with inclusion/exclusion criteria) published in full text, in English, from the year 2010 that report results separately for people with PTSD. Reviews were identified by searching the databases MEDLINE, EMBASE, and PsycINFO. When multiple copies of reviews were found, only the most recent version was included. We prioritised reviews with pooled data for inclusion.

Review reporting assessment was guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist that describes a preferred way to present a meta-analysis[1]. Reviews with less than 50% of items checked have been excluded from the library. Note that early reviews may have been guided by less stringent reporting checklists than the PRISMA, and that some reviews may have been limited by journal guidelines.

Evidence was graded using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) Working Group approach where high quality evidence such as that gained from randomised controlled trials (RCTs) may be downgraded to moderate or low if review and study quality is limited, if there is inconsistency in results, indirect comparisons, imprecise or sparse data and high probability of reporting bias. It may also be downgraded if risks associated with the intervention or other matter under review are high. Conversely, low quality evidence such as that gained from observational studies may be upgraded if effect sizes are large or if there is a dose dependent response. We have also taken into account sample size and whether results are consistent, precise and direct with low associated risks (see end of table for an explanation of these terms)[2]. The resulting table represents an objective summary of the available evidence, although the conclusions are solely the opinion of staff of NeuRA (Neuroscience Research Australia).

Results

We found one systematic review that met our inclusion criteria[3].

- Moderate to low quality evidence found the most commonly reported barriers to initiation of evidence-based interventions for PTSD were inflexibility of manualised approaches, fear of increasing client distress, problems working with comorbidities, and a lack of training and support for clinicians.

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Finch J, Ford C, Grainger L, Meiser-Stedman R

A systematic review of the clinician related barriers and facilitators to the use of evidence-informed interventions for post-traumatic stress

Journal of Affective Disorders 2020; 263: 175-86

View review abstract online

Comparison	**Barriers and facilitators to evidence-based interventions.**
Summary of evidence	**Moderate to low quality evidence (unclear sample size, unable to assess consistency or precision, direct) finds the most commonly cited barriers identified include inflexibility of manualised approaches, fear of increasing client distress, working with comorbidities and a lack of training and support.**

Intervention-level barriers and facilitators

Barriers

5 studies found that intervention manual components were too rigid, and that an individualised approach was preferable.

3 studies found that there was difficulty adapting the treatment intervention for a group-based approach.

2 studies found that the evidence-informed intervention was not generalisable to all populations and that it disregards individual/social/cultural needs.

1 study found that the treatment length was too inflexible.

Facilitators

5 studies found the guideline flexibility within approach and use of a variety of modules.

3 studies found a robust research base and theoretical depth 2 studies found an ability to adapt approach to meet client's individual needs.

Client-level barriers and facilitators

Barriers

9 studies found that client comorbidities such as substance use and suicidality were barriers to care.

7 studies found that concerns about re-traumatising clients or client decompensating due to the intervention were barriers to care.

6 studies found that low treatment adherence and other treatment preferences were barriers to care.

5 studies found that prioritising client needs, if other needs or crises are present, were barriers to care.

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2 studies found that client cognitive impairments were barriers to care.

2 studies found that not engaging family and caregivers in the intervention could be barriers to care.

Facilitators

1 study found that good quality of the therapeutic relationship was a facilitator to care.

1 study found that patient preference for the treatment approach and motivation to engage were facilitators to care.

1 study found that clients' access to a support network was a facilitator to care.

Clinical-level barriers and facilitators

Barriers

11 studies found that a lack of training in the treatment approach or evidence-informed interventions for trauma were barriers to care.

7 studies found that emotional burden of trauma work or clinician burnout were barriers to care.

4 studies found that an uncertainty of how to acknowledge trauma or when to use exposure appropriately were barriers to care.

4 studies found that competing responsibilities were barriers to care.

3 studies found that a lack of knowledge about evidence-informed interventions was a barrier to care.

3 studies found that clinicians' lack of confidence was a barrier to care.

2 studies found that fewer years of experience was a barrier to care.

2 studies found that psychodynamic/humanistic orientation was a barrier to care.

Facilitators

10 studies found that increased clinical experience was a facilitator to care.

9 studies found that endorsement of treatment manuals and belief in treatment credibility were facilitators to care.

8 studies found that having received additional training or expressed interest in additional training were facilitators to care.

6 studies found that clinician confidence was a facilitator to care.

5 studies found that awareness of evidence-informed interventions and increased engagement in continued professional development were facilitators to care.

5 studies found that clinician CBT orientation was a facilitator to care.

5 studies found that personal experience of treatment effectiveness was a facilitator to care.

4 studies found that receiving additional support and supervision was a facilitator to care.

4 studies found that an approach that is consistent with familiar clinical style was a facilitator to care.

4 studies found that being a younger therapist or having fewer years' experience were facilitators to care.

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Service-level barriers and facilitators	
Barriers 14 studies found that a lack of time for, or access to, training were barriers to care. 10 studies found that a lack of resources within an organisation was a barrier to care. 9 studies found that a lack of time to provide treatment or high caseloads were barriers to care. 8 studies found that a lack of support or flexibility within an organisation were barriers to care. 2 studies found that a lack of supervision was a barrier to care. Facilitators 5 studies found that good access to high quality training was a facilitator to care. 5 studies found that access to resources including administration was a facilitator to care. 3 studies found that support to include the approach in one's schedule was a facilitator to care. 2 studies found that strong leadership and management support were facilitators to care. 2 studies found that access to support and supervision were facilitators to care.	
Consistency in results‡	Unable to assess; no measure of consistency is reported.
Precision in results§	Unable to assess; no CIs are reported.
Directness of results‖	Direct

Explanation of acronyms

CBT = cognitive behavioural therapy, CI = confidence interval

Explanation of technical terms

* Bias has the potential to affect reviews of both RCT and observational studies. Forms of bias include; reporting bias – selective reporting of results; publication bias - trials that are not formally published tend to show less effect than published trials, further if there are statistically significant differences between groups in a trial, these trial results tend to get published before those of trials without significant differences; language bias – only including English language reports; funding bias - source of funding for the primary research with selective reporting of results within primary studies; outcome variable selection bias; database bias - including reports from some databases and not others; citation bias - preferential citation of authors. Trials can also be subject to bias when evaluators are not blind to treatment condition and selection bias of participants if trial samples are small[4].

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† Different effect measures are reported by different reviews.

Prevalence refers to how many existing cases there are at a particular point in time. Incidence refers to how many new cases there are per population in a specified time period. Incidence is usually reported as the number of new cases per 100,000 people per year. Alternatively some studies present the number of new cases that have accumulated over several years against a person-years denominator. This denominator is the sum of individual units of time that the persons in the population are at risk of becoming a case. It takes into account the size of the underlying population sample and its age structure over the duration of observation.

Reliability and validity refers to how accurate the instrument is. Sensitivity is the proportion of actual positives that are correctly identified (100% sensitivity = correct identification of all actual positives) and specificity is the proportion of negatives that are correctly identified (100% specificity = not identifying anyone as positive if they are truly not).

Weighted mean difference scores refer to mean differences between treatment and comparison groups after treatment (or occasionally pre to post treatment) and in a randomised trial there is an assumption that both groups are comparable on this measure prior to treatment. Standardised mean differences are divided by the pooled standard deviation (or the standard deviation of one group when groups are homogenous) that allows results from different scales to be combined and compared. Each study's mean difference is then given a weighting depending on the size of the sample and the variability in the data. Less than 0.4 represents a small effect, around 0.5 a medium effect, and over 0.8 represents a large effect[4].

Odds ratio (OR) or relative risk (RR) refers to the probability of a reduction (< 1) or an increase (> 1) in a particular outcome in a treatment group, or a group exposed to a risk factor, relative to the comparison group. For example, a RR of 0.75 translates to a reduction in risk of an outcome of 25% relative to those not receiving the treatment or not exposed to the risk factor. Conversely, a RR of 1.25 translates to an increased risk of 25% relative to those not receiving treatment or not having been exposed to a risk factor. A RR or OR of 1.00 means there is no difference between groups. A medium effect is considered if RR > 2 or < 0.5 and a large effect if RR > 5 or < 0.2[5]. lnOR stands for logarithmic OR where a lnOR of 0 shows no difference between groups. Hazard ratios measure the effect of an explanatory variable on the hazard or risk of an event.

Correlation coefficients (eg, r) indicate the strength of association or relationship

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between variables. They can provide an indirect indication of prediction, but do not confirm causality due to possible and often unforseen confounding variables. An r of 0.10 represents a weak association, 0.25 a medium association and 0.40 and over represents a strong association. Unstandardised (*b*) regression coefficients indicate the average change in the dependent variable associated with a 1 unit change in the independent variable, statistically controlling for the other independent variables. Standardised regression coefficients represent the change being in units of standard deviations to allow comparison across different scales.

‡ Inconsistency refers to differing estimates of effect across studies (i.e. heterogeneity or variability in results) that is not explained by subgroup analyses and therefore reduces confidence in the effect estimate. I^2 is the percentage of the variability in effect estimates that is due to heterogeneity rather than sampling error (chance) - 0% to 40%: heterogeneity might not be important, 30% to 60%: may represent moderate heterogeneity, 50% to 90%: may represent considerable heterogeneity and over this is considerable heterogeneity. I^2 can be calculated from Q (chi-square) for the test of heterogeneity with the following formula[4];

$$I^2 = \left(\frac{Q - df}{Q}\right) \times 100\%$$

§ Imprecision refers to wide confidence intervals indicating a lack of confidence in the effect estimate. Based on GRADE recommendations, a result for continuous data (standardised mean differences, not weighted mean differences) is considered imprecise if the upper or lower confidence limit crosses an effect size of 0.5 in either direction, and for binary and correlation data, an effect size of 0.25. GRADE also recommends downgrading the evidence when sample size is smaller than 300 (for binary data) and 400 (for continuous data), although for some topics, these criteria should be relaxed[6].

‖ Indirectness of comparison occurs when a comparison of intervention A versus B is not available but A was compared with C and B was compared with C that allows indirect comparisons of the magnitude of effect of A versus B. Indirectness of population, comparator and/or outcome can also occur when the available evidence regarding a particular population, intervention, comparator, or outcome is not available and is therefore inferred from available evidence. These inferred treatment effect sizes are of lower quality than those gained from head-to-head comparisons of A and B.

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