Journal of Psychosomatic Research xxx (xxxx) xxx

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

Short-term psychodynamic psychotherapy for functional somatic disorders: A systematic review and meta-analysis of within-treatment effects

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ARTICLE INFO

Keywords:
Short-term psychodynamic psychotherapy
Somatoform disorders
Somatization
Somatic symptom disorders
Medically unexplained symptoms
Functional somatic disorders
Within-treatment effects
Systematic review
Meta-analysis

ABSTRACT

Objective: A recent meta-analysis of 17 randomized, controlled trials (RCTs) showed that Short-term Psychodynamic Psychotherapy (STPP) for functional somatic disorders (FSD) reduced somatic symptoms compared to wait list, minimal treatment, and treatment-as-usual controls. A clinically important yet unanswered question is how much improvement patients experience within STPP treatment.

Methods: Following a systematic search, we identified STPP trials presenting data at baseline and post-treatment/follow-up. Meta-analyses determined the magnitude of changes in somatic symptoms and other outcomes from before to after STPP, and analyses examined effect sizes as a function of study, therapy, and patient variables.

Results: We identified 37 trials (22 pre-post studies and 15 RCTs) totaling 2094 patients treated an average of 13.34 sessions for a range of FSD. Across all studies, somatic symptoms improved significantly from pre-treatment to short-term follow-up with a large effect size (SMD = −1.07), which was maintained at long-term follow-up (SMD = −0.90). After excluding two outlier studies, effects at short- and medium-term follow-up remained significant but were somewhat reduced in magnitude (e.g., short-term SMD = −0.73). Secondary outcomes including anxiety, depression, disability, and interpersonal problems had medium to large effects. Effects were larger for studies of STPP that were longer than 12 sessions or used an emotion-focused type of STPP, and for chronic pain or gastrointestinal conditions than for functional neurological disorders.

Conclusions: STPP results in moderate to large improvements in multiple outcome domains that are sustained in long-term follow-up. STPP is an effective treatment option for FSD and should be included in treatment guidelines.

1. Introduction

Functional somatic disorders (FSD) have been variously labeled over the years and have included diagnoses such as most somatoform, psychophysiological, psychosomatic, and somatic symptom disorders, as well as "medically unexplained" symptoms. A recent consensus definition views functional somatic disorders as an umbrella term that includes conditions characterised by persistent and troublesome physical symptoms that are accompanied by impairment or disability and that reflect the complex interaction of biological and psychosocial factors and the integration of bodily and brain functions and dysfunctions [15]. Although FSD are neither purely somatic nor purely mental, psychosocial trauma, intrapsychic conflicts, and disturbed emotion regulation are elevated in these disorders and believed to contribute substantially to them [9,29,35]. These conditions are very common in health care settings, resulting in substantial burdens to patients and health care systems [20,41,58]. Outside of treatments like medications, rehabilitation, behavioral interventions and physical therapies, psychotherapies play a

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https://doi.org/10.1016/j.jpsychores.2021.110473
Received 15 December 2020; Received in revised form 23 March 2021; Accepted 24 March 2021
Available online 26 March 2021
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prominent role in the treatment of FSD. The most commonly studied psychological approaches for FSD are cognitive-behavioral and related interventions, and meta-analytic reviews of these interventions reveal varying but often small effects, both for specific syndromes like chronic pain [78] and for mixed FSD populations [56,74]. Therefore, expansion of treatment options for this population is needed.

Short-term psychodynamic psychotherapy (STPP) describes a class of related therapies that typically last 40 or fewer sessions and share a focus on emotional and relational processes linked to development, unresolved conflicts, and past adverse experiences. Most STPP approaches are guided by conceptual frameworks such as the 2-triangles model [54,55] and emphasize unconscious processes (thoughts, fantasies, and feelings) tied to adverse life events. The range of techniques used in STPP include support, interpretation, clarification of intrapsychic patterns, challenges to defenses, and eliciting the experience and expression of feelings related to conflicted relationships in the past and present. Different types of STPP vary in their focus; for example, some therapies (e.g., Psychodynamic-Interpersonal Therapy; PIT), target primarily patient insight or understanding of intrapsychic and interpersonal conflicts, whereas other therapies (e.g., Intensive Short-term Psychodynamic Therapy; ISTDP; Emotional Awareness and Expression Therapy; EAET) are emotion-focused, emphasizing in-session emotional activation, experiencing, and expression.

The efficacy of STPP has been studied in over 250 randomized controlled trials (RCTs) [47], and reviews and meta-analyses of these trials conclude that STPP improves numerous conditions, including depression [24], anxiety [38], personality disorders [72], and common mental disorders [7]. We recently examined the efficacy of STPP for FSD, conducting a meta-analysis of 17 RCTs [6]. Compared to minimal or no treatment (i.e., waitlist, treatment-as-usual, or minimal contact), STPP resulted in lower somatic symptoms, with large effect sizes (standardized mean difference, SMD) at post-treatment (SMD = −0.84) and long-term follow-up (6 or more months, SMD = −1.00), and generally large effects on various secondary outcomes.

Our meta-analysis of STPP for FSD, however, examined only RCTs and used only post-intervention data, comparing STPP to controls. Such between-condition effects from RCTs ("controlled effects") are viewed as the gold standard for intervention meta-analyses. It is important, however, to consider what such meta-analyses do—and do not—accomplish. Between-condition effect sizes from RCTs provide the most theoretically and scientifically valuable information about the unique or specific effects of a treatment. They accomplish this by "subtracting" the effects of the control condition and its many non-specific factors, such as repeated assessment, trial participation, and especially naturalistic change over time.

There are, however, limitations to RCTs and the meta-analyses that summarize them. Generalizability to the larger population of treatment-seeking patients and front-line practitioners is limited due to selection bias into RCTs (e.g., willingness to be randomized) as well as the unique characteristics and context of those providing therapy. Data from RCTs may also be biased by negative reactions to being assigned to the control condition as well as variations in what is offered to controls. For example, "waitlist controls" may artificially enhance treatment effects [60], and "minimal contact" or "treatment-as-usual" controls vary widely among trials and among patients within trials. Yet, most meta-analyses of RCTs collapse these control conditions, and meta-analyses may also include various "active controls" into their overall effect size estimates. Such variation in the control / comparison conditions complicates interpretation of the effect sizes obtained from between-condition meta-analyses of RCTs.

In addition, there is a question of great clinical importance that remains unanswered by between-condition RCT meta-analyses. Patients and clinicians typically wish to know how much improvement occurs when receiving a given treatment, not how much improvement relative to some control condition. This clinical question is best answered not by randomization and comparison controls, but rather by examining change in patients from before to after treatment (i.e., pre-post or within-treatment effect sizes). Most between-condition meta-analyses of RCTs use only the post-treatment or follow-up data rather than change from baseline, due in part because of the unknown test-retest reliability of the outcome measures [22]. Data on change over time within treatment can be extracted from the treatment arms of RCTs, and importantly, the literature usually has numerous "pre-post," "clinical cohort," or "uncontrolled naturalistic" studies for a given treatment. Such studies likely reflect actual clinical practice better than RCTs, but they usually are excluded from meta-analyses.

Studies rarely report how pre-post or within-treatment effect sizes compare to between-condition RCT effect sizes. There are three possibilities. First, it is certainly possible that a treatment's pre-post effect is larger than its controlled effect because the latter removes change that occurs due to non-treatment factors. Second, the pre-post effect may be similar in magnitude to the controlled effect when no change occurs in the control condition, as might happen with highly stable disorders. Finally, the pre-post effect may be smaller than the controlled effect if, for example, higher quality therapy is provided in RCTs than naturalistic studies due to better therapist training, treatment adherence, supervision, or even more homogeneous patient selection. Therefore, it is important to evaluate pre-post effect sizes and compare them with controlled, between-condition effect sizes from RCTs.

In summary, meta-analyses of pre-post comparisons offer a complementary perspective to meta-analyses of RCTs of a treatment's effects. In this paper, we complement our review of controlled effects of STPP for FSD [6] by examining the uncontrolled effects of STPP for FSD over time—from pre-treatment to follow-up. We obtained data not only from the treatment arms of RCTs analyzed in Abbass et al. [6] but also from a larger number of pre-post, naturalistic, and non-randomized trials of STPP for FSD. In addition to determining the effect size of STPP on somatic symptoms (primary outcome) and many secondary outcomes, analyses also tested predictors of treatment effect sizes on somatic symptoms at short-term follow-up (where we have the greatest number of studies), including whether or not the data came from an RCT, various methodological features of the studies, the length of therapy, the specific type of STPP, whether or not therapy was emotion-focused, and the type of FSD (e.g., chronic pain, gastrointestinal, neurological). We also conducted sensitivity analyses to address concerns about unknown pre-post reliability of measures as well as other potential confounds.

2. Methods

2.1. Study registration

Our research plan was published on the PROSPERO website (PROSPERO 2017 CRD42017083235) prior to commencing this study. We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) recommendations for the background, search strategy, methods, results, discussion, and conclusions [32].

2.2. Selection criteria

We searched for all studies using STPP to treat adult patients with FSD. STPP was defined as a treatment that: 1) is verbal, in-person, provided in either individual or group formats, and in any setting; 2) targets psychodynamic processes and is informed by major developers of STPP (e.g., David Malan, Habib Davanloo, James Mann, Peter Sifneos, among others); and 3) is 40 or fewer standard-length sessions, but more than a single session. With respect to FSD, we included studies of DSM-IV somatoform disorders, pain disorders, and other conditions that would likely meet DSM-5 criteria for a somatic symptom and related disorder, such as irritable bowel syndrome. Importantly, we excluded studies of conditions with known structural pathological or disease processes such as cancer, cardiovascular disease, or autoimmune disease. In addition, included studies had to provide usable data from both baseline and post-

treatment/follow-up and include at least one of our study outcome variables.

2.3. Search strategy

We updated an earlier search [3,4] that covered the published literature to 2008; for the current review, we searched for all studies (no language restriction) published from January 2006 through May 2020. All studies from the recent review [6] were evaluated for inclusion in this review. We searched the following data bases: PubMed, Web of Science, EMBASE, the Cochrane Central Register of Controlled Trials (CENTRAL) and PsycINFO. Combinations of the following terms were used: 1) psychotherapy, psychoanalytic, psychodynamic, dynamic, or short-term therapy; AND 2) clinical trial, randomized controlled trial, or naturalistic study; AND 3) a long list of various FSD symptoms and conditions (chest pain, pain, somatoform disorder, medically unexplained symptoms, psychogenic pain, conversion disorder, somatosensory disorder, urethral syndrome, fibromyalgia, functional neurological disorder, functional movement disorder, psychogenic non-epileptic seizures, non-epileptic attack disorder, headache, migraine, irritable bowel, dyspepsia, dermatitis, inflammatory dermatosis, laryngospasm, pharyngospasm, hysteria, hypochondriasis, tics, Tourette's, tinnitus, temporomandibular syndrome, bruxism, abdominal pain, leg pain, foot pain, back pain, muscle tension, muscular disorder, muscle strain, arm pain, hand pain, chronic fatigue syndrome, fatigue, alexithymia, somatic symptom disorder, somatization disorder, functional somatic symptom, functional somatic syndrome, functional somatic disorder). In addition, we searched prospective trial registries for unpublished ongoing research (e.g., http://www.controlled-trials.com, https://clinicaltrials.gov/) and an internet database of controlled and comparative outcome studies on psychological treatments of somatic symptom disorders (http://www.psychotherapyrcts.org).

2.4. Selection and data extraction

Two reviewers (PL, CD) screened titles and abstracts to confirm eligibility. Full-text versions of studies were then examined for inclusion/exclusion by pairs of reviewers (PL/AA and JT/LR). Disagreement between authors was discussed toward reaching consensus; when consensus could not be reached, a third author (SK) was consulted.

Descriptive data from selected studies were extracted and tabulated by pairs of reviewers. These data included type of FSD (pain, gastrointestinal, neurological, or mixed somatic symptoms), the study design (RCT or pre-post/uncontrolled), number and gender of patients receiving STPP, type of STPP, treatment duration, and follow-up time-points. Reviewers also recorded whether or not the therapy was video/audio recorded, manualized, evaluated for adherence, and focused on emotion (vs. insight).

Raw data for effect sizes for each outcome measure were extracted separately by a reviewer (HH) who has no affiliation with STPP. Data entry was spot checked by two others (AA, SK). For the current analyses, pre-treatment and available post-treatment / follow-up data were extracted for the STPP condition only. The outcome categories were as follows: somatic symptoms (primary outcome), anxiety, depression, general symptoms, interpersonal problems, physical function, disability, quality of life, health care use, and health care cost. Post-intervention outcomes were categorized into three time-points: short-term (< 3 months), medium-term (3 to 6 months), and long-term (> 6 months).

2.5. Quality ratings

Given that two different study designs were included in this meta-analysis, two quality assessment approaches were used. For RCTs, we used the Cochrane Risk of Bias tool to rate the methodological characteristics of studies. For the pre-post/case series studies, we used a modified version of the Newcastle Ottawa Rating Scale [76] (See Online Supplement Fig. 1), which notes the representativeness of the sample, ascertainment of diagnosis, comparability to any controls and measurement of outcomes. Ratings were done independently by a pair of reviewers, and differences in ratings were discussed to reach consensus.

2.6. Data analyses

Separate meta-analyses were conducted for each outcome measure and at each of the three follow-up time points, when data were available from at least two studies for the outcome and time point. Effect sizes using SMD were calculated using RevMan, WinPepi and Comprehensive Meta-analysis. We defined effect sizes as small (SMD of 0.20–0.49), medium (SMD of 0.5–0.79) and large (SMD of $\geq$0.8) [37]. Significance was assessed using 95% confidence intervals, and heterogeneity by using I^2 statistic. A value of greater than 50% for the I^2 statistic indicates heterogeneity. The random-effects model was used for all the analyses because we could not definitively exclude between-study variation even in the absence of statistical heterogeneity.

We adjusted for the fact that pre-test and post-test scores are not independent from each other by using the correlation between the two when original studies reported such values. Such correlations were rarely available, however, so we used a default correlation of $r = 0.59$, which is the median within-group correlation reported from a meta-analysis of 811 correlations stemming from 123 intervention trials [12].

Note that two of the 37 studies in this review—both conducted by one team [16,17]. Both of these studies reported very high STPP-related reductions in somatic symptoms (over 5 SDs) as well as outlying values for reductions in depression, anxiety, and general psychiatric symptoms at the two time points they were assessed (short-and medium-term); no other studies even approached such extreme values. Therefore, we present results for both the full sample of studies and also after excluding these two outlier studies from short- and medium-term follow-up. Note that long-term follow-up effects did not change because these two studies did not have long-term follow-up data.

We undertook subgroup analyses of studies to determine correlates of effect sizes. In particular, we examined effect sizes based on several features: whether or not the study was an RCT, had adherence ratings, used video or audio review, used a manual for therapy; whether or not therapy was more than 12 sessions and was emotion-focused; the specific type of STPP; and the type of FSD treated. To have a large enough sample size for reliable inferences, these subgroup analyses were conducted on only the 24 studies that assessed the primary outcome of somatic symptoms and only at the short-term follow-up.

We undertook several sensitivity analyses. Given that meta-analyses of within-condition data can be biased by the fact that the correlations between pre and post measures of the outcome variable are usually unknown but are often lower than $r = 0.59$ (our default value), we also tested a low ($r = 0.2$) test-retest value for the 24 studies that assessed somatic symptoms at short-term follow-up. We also explored any heterogeneity further through sensitivity analyses of the effect of omitting each study in turn. When multiple measures were used for the same outcome, we examined the effect of substituting one for the other. A few of the studies have small overlap of patients for certain outcome measures [2,26,46,65], so we assessed the effect of omitting each study in turn. Finally, we tested for publication bias for our primary outcome (somatic symptoms) using funnel plot asymmetry (where low p values suggest publication bias).

3. Results

3.1. Description of included studies

Our search identified 546 titles through bibliographic databases and 267 through other sources such as the ISRCTN trial registry (Online Supplementary Fig. 2). After screening and full-text review, we retained a total of 37 studies including 2094 patients receiving STPP (Table 1).

Table 1
Description of studies.

1st Author, year [reference]	Patient Group	n	STPP Model	Sessions	Longest follow-up (Months)	RCT	Adherence rated	Video/ audio review	Manual or guide	Emotional experience	< 12 sessions
Abbass 2008 [2]	Headache	29	ISTDP	19.7	36	No	No	Yes	Yes	Yes	No
Abbass 2009 [34]	MUS in emergency	50	ISTDP	3.8	12	No	Yes	Yes	Yes	Yes	Yes
Alessiani 2020 [10]	Chronic migraine	96	Unclear	12	12	No	No	No	No	No	Yes
Alteri 2009 [11]	Headache	13	Unclear	8	12	No	No	No	No	No	–
Bassett 1985 [13]	Chronic pain	14	Unclear	12	12	Yes	No	Yes	No	No	No
Burger 2016 [14]	Chronic pain	72	EAET	5	6	No	No	No	Yes	Yes	Yes
Chavooshi 2016 [16]	Medically unexplained pain	23	ISTDP	20	3	Yes	Yes	Yes	Yes	Yes	No
Chavooshi 2017a [17]	Medically unexplained pain	177	ISTDP	16	3	Yes	Yes	Yes	Yes	Yes	No
Chavooshi 2017b [18]	Medically unexplained pain	42	ISTDP	13	12	Yes	Yes	Yes	Yes	Yes	No
Chirco 2015 [19]	Bruxism	5	ISTDP	20	12	Yes	No	Yes	Yes	Yes	No
Creed 2003 [21]	Severe IBS	85	PIT	8	12	Yes	Yes	No	Yes	No	Yes
Faramarzi 2015 [25]	Functional dyspepsia	24	SEP	16	12	Yes	No	No	Yes	No	No
Flibotte 2012 [26]	Fibromyalgia	67	ISTDP	7.2	Post	No	Yes	Yes	Yes	Yes	Yes
Hamilton 2000 [28]	Chronic dyspepsia	31	PIT	8	12	Yes	Yes	Yes	Yes	No	Yes
Hawkins 2004 [30]	Chronic back pain	47	ISTDP	8	12	No	No	No	Yes	Yes	Yes
Hecke 2008 [31]	Psychosomatic	34	SASB	25	12	No	No	No	Yes	No	No
Hinson 2006 [33]	Functional movement disorders	10	ISTDP	12	Post	No	No	No	Yes	Yes	No
Junkert-Tress 2001 [36]	Somatoform disorders	24	TLDP	25	60	No	No	Yes	Yes	No	No
Lilliengren 2020 [46]	Chronic Pain	228	ISTDP	6.1	36	No	Yes	Yes	Yes	Yes	Yes
Limburg 2019 [49]	Functional vertigo and dizziness	98	Mix	~24	6	No	No	No	No	No	No
Lumley 2008 [51]	Fibromyalgia	10	Mix	10	3	No	No	Yes	Yes	Yes	Yes
Lumley 2017 [53]	Fibromyalgia	79	EAET	8	6	Yes	Yes	Yes	Yes	Yes	Yes
Monsen 2000 [57]	Chronic pain	20	ACTM	33	12	Yes	No	No	Yes	Yes	No
Petolicco 2017 [62]	Chronic migraine	117	Unclear	8	6	No	No	No	No	No	Yes
Reuber 2007 [63]	Functional neurological disorders	91	PIT	6	6	No	No	No	Yes	No	Yes
Russell 2016 [64]	Pseudoseizures	28	ISTDP	3.6	36	No	Yes	Yes	Yes	Yes	Yes
Russell 2017 [65]	Functional Neurological	11	ISTDP	11.7	Post	No	No	No	Yes	Yes	Yes
Sattel 2012 [66]	Multisomatoform disorder	107	PIT	12	9	Yes	Yes	Yes	Yes	No	No
Schaerfert 2013 [67]	MUS	170	PIT	12	9	Yes	No	No	Yes	No	No
Scheidt 2013 [68]	Fibromyalgia with depression	24	Unclear	25	12	Yes	Yes	No	Yes	No	No
Selders 2015 [69]	MUS	57	DIT	20	Post	No	No	No	No	No	No
Thakur 2017 [71]	IBS	36	EAET	3	2.5	Yes	Yes	Yes	Yes	Yes	Yes
Tschuschke 2007 [73]	Somatoform disorders	50	Unclear	20	12	No	No	No	No	No	No
Ventegodt 2008 [75]	Somatoform disorders	31	Mix	20	Post	No	No	No	No	Yes	No
Williams 2018 [77]	Functional neurological disorders	44	BAPIT	11.9	Post	No	No	No	Yes	Yes	Yes
Yarns 2020 [79]	Chronic pain	28	EAET/ ISTDP	8	3	Yes	Yes	Yes	Yes	Yes	Yes
Yasky 2016 [80]	Psychosomatic	22	SEP	15	Post	No	No	No	No	–	No

RCT: Randomized Controlled Trial, MUS: Medically Unexplained Symptoms, ISTDP: Intensive Short-term Dynamic Psychotherapy, PIT: Psychodynamic Interpersonal Therapy, TLDP: Time Limited Dynamic Psychotherapy, SEP: Supportive Expressive Therapy, SASB: Structural Analysis of Social Behavior, DIT: Dynamic Interpersonal

Therapy, EAET: Emotional Awareness and Expression Therapy. ACTM: Affect Consciousness Treatment Model, BAPIT: Brief Augmented Psychodynamic Interpersonal Therapy.

Fifteen (40.5%) of the 37 studies were RCTs of STPP, whereas the other 22 (59.5%) were pre-post or naturalistic cohort studies. Several of these 22 studies also presented data from a separate, non-randomized comparison group, but the STPP treatments were essentially a cohort study and are treated as such in these analyses.

As shown in Tables 1, 18 studies (48.6%) were of pain-related conditions (chronic pain, fibromyalgia, head pain), 9 studies (24.3%) were of mixed somatic symptom conditions, 6 studies (16.2%) were of functional neurological disorders, and 4 studies (10.8%) were of functional gastrointestinal disorders. Most of the studies ($k = 28$, 75.7%) followed a specific STPP model: 12 studies (32.4%) tested Intensive Short-term Dynamic Psychotherapy (ISTDP) [8,23]; 5 studies (13.5%) used Psychodynamic-Interpersonal Therapy (PIT) [34], 4 studies (10.8%) tested Emotional Awareness and Expression Therapy (EAET) [52], 2 studies (5.4%) implemented Supportive Expressive Therapy [50], and 5 studies (13.5%) used other STPP approaches. Nine studies (24.3%) were of short-term psychodynamic models that were either linked to multiple STPP theorists or not linked to a specific STPP theorist but otherwise met criteria as STPP.

Treatments averaged 13.3 sessions (SD = 7.2, range: 3–33), and 22 of the studies (59.5%) had 12 or fewer therapy sessions, whereas 15 studies (40.5%) had therapy longer than 12 sessions. Most studies (k = 30; 80.1%) had follow-up evaluations beyond post treatment, and among these studies, the longest follow-up assessment averaged 13.3 months (SD 12.6, range 2.5–60). Most studies (k = 34, 91.9%) took place in outpatient settings, but 3 studies (8.1%) were on inpatient units. Thirty-one studies (83.8%) provided individual therapy whereas the other 6 (16.2%) provided group or combination interventions; 20 studies (54.0%) used an emotion-focused form of STPP.

3.2. Study quality

The overall quality of the RCT studies was moderate as determined by pairwise, independent Cochrane Risk of Bias ratings [20]. Eight of the 15 RCT studies (53.3%) had blinded measurement of some outcomes (6 did not, 1 unclear), 10 (66.7%) had adequate allocation concealment (4 unclear, 1 did not), 11 (73.3%) had random sequence generation (2 did not, 2 were unclear), and 12 (80.0%) had complete outcome data or adjustments to correct for missing data such as intention to treat methods (1 did not, 1 unclear). It was not possible to determine if outcome reporting was complete due to lack of published protocols except for 3 studies that did appear complete. Blinding of either therapists or patients is not possible in psychotherapy research so this was rated as absent in each case (Online Supplement Table 1). For the 22 pre-post/cohort studies, based on a modified Newcastle Ottawa Rating System, 16 studies (72.7%) were rated low quality, 4 were rated good, and 2 were rated fair (Online Supplement Table 2). Other measures revealed variability of study rigour. Most of the 37 studies (k = 28; 75.6%) used a manual to guide therapy, 17 studies (45.9%) had audio/video review, and 14 studies (37.8%) were rated for adherence.

3.3. Effects of STPP on somatic symptoms

As shown in Table 2 (and Supplementary Fig. 3), reductions in somatic symptoms from before to after STPP were large in magnitude at all three post-treatment time points: short-term (i.e., less than 3 months, $k = 24$; SMD = −1.07), medium-term (3 to 6 months, $k = 13$; SMD = −0.92), and long-term (over 6 months: $k = 10$; SMD = −0.90). Excluding the two outlier studies reduced the effect sizes at short-term (SMD = −0.73, 95% CI = −0.90, −0.56, $p < .0001$) and at medium-term (SMD = −0.61, 95% CI = −0.76, −0.46, $p < .0001$). The large long-term effect remained unchanged at SMD = −0.90 because the two outlier studies did not contribute data at long-term.

Table 2
Meta-analyses of studies examining the effects of STPP for functional somatic disorders.

Comparison	# Studies	*n*	SMD [95% CI]	Significance
Pre to < 3 months Post-tx				
Somatic symptoms	24	1059	−1.07 [−1.40, −0.74]	<0.0001
Depression	16	766	−1.25 [−1.72, −0.78]	<0.0001
Anxiety	15	560	−0.64 [−0.93, −0.35]	0.0001
General symptoms	19	866	−0.85 [−1.20, −0.50]	<0.0001
Physical dysfunction	6	235	−0.98 [−1.51, −0.45]	<0.0001
Interpersonal problems	6	172	−0.66 [−0.88, −0.44]	<0.0001
Disability	6	176	−1.07 [−1.50, −0.64]	<0.0001
Global dysfunction	3	90	−1.32 [−1.81, −0.83]	<0.0001
Pre to 3–6 months Post-tx				
Somatic symptoms	13	809	−0.92 [−1.27, −0.57]	<0.0001
Depression	9	725	−1.66 [−2.35, −0.97]	<0.0001
Anxiety	7	455	−0.64 [−0.93, −0.35]	<0.0001
General symptoms	9	559	−1.05 [−1.76, −0.34]	0.003
Physical dysfunction	4	263	−0.30 [−0.44, −0.16] (a)	<0.0001
Disability	6	176	−0.96 [−1.31, −0.61]	<0.0001
Global dysfunction	3	90	−0.81 [−1.16, −0.46] (a)	<0.0001
Pre to > 6 months Post-tx				
Somatic symptoms	10	534	−0.90 [−1.23, −0.57]	<0.0001
Depression	5	341	−0.66 [−0.91, −0.41]	<0.0001
Anxiety	5	341	−0.88 [−1.23, −0.53]	0.0001
General symptoms	8	431	−0.59 [−0.81, −0.37]	<0.0001
Physical dysfunction	4	195	−0.91 [−1.24, −0.58]	0.0003
Disability	3	62	−1.71 [−2.53, −0.89]	<0.0001
Health care contacts	4	241	−0.39 [−0.62, −0.17]	0.0007
Health care costs	3	78	−0.32 [−0.44, −0.20] (a)	<0.0001

Note: (a) $I^2 \leq 50\%$; Negative values of effect estimates favor STPP.

3.4. Effects of STPP on secondary outcomes

As shown in Table 2 (and Supplementary Fig. 3), STPP led to significant reductions in depression, anxiety, and general psychiatric symptoms at all three follow-up time points. For the full set of studies, these effects were typically large in magnitude at each time. Removing the two outlier studies reduced the short-term effects to medium in magnitude on depression (SMD = −0.74, 95% CI: −0.96, −0.53; p < .0001), anxiety (SMD = −0.50, 95% CI: −0.75, −0.26; p < .0001), and general symptoms (SMD = −0.59, 95% CI: −0.70, −0.48; p < .0001). Similarly, the effects on these outcomes at medium-term follow-up were

somewhat reduced after exclusion of the two outliers: depression (SMD = −0.51, 95% CI: −0.71, −0.31; p < .0001), anxiety (SMD = −0.41, 95% CI: −0.55, −0.28; p < .0001), and general symptoms (SMD = −0.38, 95% CI: −0.48, −0.28; p < .0001).

Also as shown in Table 2, the effects of STPP on reducing physical dysfunction were significant and large at short-term and long-term follow-up, although small at medium-term. Effects on disability were large at all three time points, and effects on global dysfunction were large at short- and medium-term follow-ups. Interpersonal problems were assessed in 6 studies at short-term follow-up only, and the reduction was medium in magnitude. Finally, a handful of studies assessed STPP effects on health care contacts and costs at long-term follow-up only; there were significant, small magnitude effects on these outcomes.

3.5. Subgroup analyses

Meta-analyses of predictors of effect size—analyses of subgroups of studies—were conducted only on the primary outcome of somatic symptoms and only at the short-term follow-up, where the largest number of studies were found ($k = 24$). The results of these subgroup meta-analyses are shown in Table 3, which presents results both including and excluding the two outlier studies. Of note, studies with and studies without all of the subgroup features had effect sizes that were significant and typically at least medium in magnitude. Prior to exclusion of the two outliers, studies that were adherence-rated or over 12 sessions in duration had significantly larger effects than studies without these features, studies of chronic pain had larger effects than those of neurological conditions, and studies of ISTDP had larger effects than those of PIT. Studies that were RCTs, conducted audio/video review, used a therapy manual, or were emotion-focused, had numerically larger effects than studies without these features, but not significantly so, due primarily to the substantial heterogeneity. When the subgroup analyses were repeated excluding the two outliers, there were no significant differences as a function of subgroup. However, when based on a clinically meaningful effect that is at least small (> 0.20 SD), therapy longer than 12 sessions was clinically more effective than shorter therapies, studies of chronic pain or gastrointestinal disorders were clinically larger than those of functional neurological disorders, and both ISTDP and EAET yielded clinically larger effects than PIT.

3.6. Sensitivity analyses, heterogeneity, and publication bias

Sensitivity analyses using a correlation of 0.2 between pre and post measures of somatic symptoms for the 24 studies at short-term follow-up indicated that the effect size changed only slightly, from −1.07 to −1.05 (95% CI: −1.36, −071; $p < .0001$), suggesting the obtained effect sizes are minimally biased by assuming a correlation of $r = 0.59$. Sensitivity analyses also examined the effect of substituting one measure for another when multiple instruments were used for the same outcome. The results show that this made little difference to the findings. Omitting any study with patient overlap with another study also made little difference to the findings.

There was evidence of heterogeneity ($I^2 > 50\%$) in 20 of the 23 analyses presented in Table 2. When we explored this further through sensitivity analyses of excluding the two outlier studies [16,17], heterogeneity was no longer significant for medium-term anxiety ($I^2 = 46\%$). Similarly, the results for long-term depression were no longer heterogeneous ($I^2 = 0\%$) on excluding a third study [18]. Removal of other single studies did not affect heterogeneity.

Finally, we used a funnel plot to assess possible effects of publication bias on our primary outcome. Egger's regression asymmetry test was positive (intercept −4.55, 90% C.I., −6.69 to −2.41, $p = .01$), indicating possible publication bias. We did not use trim and fill given this method performs poorly in the setting of heterogeneity [32]. We found similar results for Egger's regression asymmetry test in the case of depression (−7.11, 90% C.I., −10.41 to −4.11, $p = .006$). However, the test was non-significant in the case of anxiety (−2.50, 90% C.I., −4.23 to −0.76, $p = .076$) and general psychiatric symptoms (−4.08, 90% C.I., −6.68 to −1.47, $p = .051$). When excluding the two outlier studies [16,17], the test for publication bias was no longer significant for somatic symptoms (intercept −2.00, 90% C.I., −3.35 to −0.65, $p = .062$) and continued to

Table 3
Subgroup analyses of STPP effects on short-term somatic symptoms (including and excluding two outlier studies).

Variable	# Studies	Effect Estimate	Significance
Data from RCT?			
Yes	13	−1.52 [−2.15,	<0.0001
Yes (no outliers)	11	−0.89]	<0.0001
No	11	−0.80 [−1.07, −0.52]	<0.0001
		−0.67 [−0.90, −0.43]	
Adherence rated?			
Yes	11	−1.59 [−2.23,	<0.0001
Yes (no outliers)	9	−0.94]	<0.0001
		−0.77 [−1.04, −0.51]	
No	13	−0.69 [−0.94, −0.45]	<0.0001
Audio/video used?			
Yes	12	−1.42 [−2.04,	<0.0001
Yes (no outliers)	10	−0.80]	<0.0001
		−0.68 [−0.93, −0.43]	
No	12	−0.78 [−1.02, −0.53]	<0.0001
Therapy manual used?			
Yes	20	−1.19 [−1.57,	<0.0001
Yes (no outliers)	18	−0.81]	<0.0001
No	4	−0.75 [−0.94, −0.57]	0.0004
		−0.57 [−1.10, −0.04]	
Emotion-focused STPP?			
Yes	15	−1.40 [−1.92,	<0.0001
Yes (no outliers)	13	−0.87]	<0.0001
No	8	−0.79 [−1.02, −0.56]	<0.0001
		−0.65 [−0.95, −0.35]	
Therapy >12 sessions?			
Yes	9	−1.88 [−2.85,	<0.0001
Yes (no outliers)	7	−0.90]	<0.0001
No	15	−0.98 [−1.47, −0.48]	<0.0001
		−0.70 [−0.89, −0.50]	
Type of FSD			
Chronic pain	13	−1.51 [−2.09,	<0.0001
Chronic pain (no outliers)	11	−0.93]	<0.0001
Gastrointestinal	3	−0.71 [−0.87, −0.55]	<0.0001
Neurological	4	−0.88 [−1.10, −0.65]	0.01
Mixed	4	−0.48 [−0.86, −0.10]	
		−0.35 [−0.49, −0.21]	
Type of therapy			
ISTDP	9	−1.98 [−3.03,	<0.0001
ISTDP (no outliers)	7	−0.92]	<0.0001
EAET	4	−0.81 [−1.07, −0.56]	<0.0001
PIT	3	−0.80 [−1.13, −0.46]	0.0006
		−0.41 [−0.64, −0.18]	

ISTDP = Intensive Short-term Dynamic Psychotherapy; EAET = Emotional awareness and Expression Therapy; PIT = Psychodynamic-Interpersonal Therapy.

not be significant for anxiety and general symptoms: it was only significant for depression. (See Online Supplement Figs. 4–7). Thus the bulk of all markers of possible publication bias disappeared with removal of 2 outliers.

4. Discussion

This meta-analysis of 37 trials of STPP for FSD indicates that STPP leads to large reductions in somatic symptoms following treatment as well as medium or large improvements in most other secondary outcomes, including depression, anxiety, general psychiatric symptoms, disability, and physical function. Notably, these effects are durable, lasting beyond 6 months with no signs of decrement or reversal. FSDs commonly result in chronic functional impairment and long-term excess costs to patients and health and insurance providers. For this reason, the findings of significant, sustained reductions in health care cost, disability and physical dysfunction are also important. Further, such measures go beyond subjective patient symptom reports, strengthening the evidence in support of STPP. The findings of this meta-analysis provide valuable information regarding the clinically relevant question of expected effects from engaging in STPP: patients and providers can predict substantial and lasting improvements in somatic symptoms and other outcomes.

Relatively little is known about how effect sizes from before to after a treatment compare to effect sizes obtained from comparisons of treatment to randomized control conditions, which is the gold standard meta-analytic approach. The current analyses found that the large reduction in somatic symptoms within-STPP is comparable to that obtained when STPP is compared to no-treatment conditions (waiting list, minimal contact, or treatment as usual) in a recent meta-analysis [6]. Similarly, the current subgroup meta-analysis found that (after removing two outliers) the pre-post effects of STPP when conducted in an RCT were similar to the STPP effects from pre-post/naturalistic cohort studies. One might have expected that the within-treatment effects from uncontrolled studies would be larger than the between-condition effects from controlled studies, given that the latter remove improvements due to the passage of time and other nonspecific factors. Such differences between controlled and uncontrolled effect sizes are expected for many disorders that show improvement without treatment. This lack of differences in between-condition and within-STPP effects may be due to FSD being relatively stable over several months without treatment, meaning that patients in no-treatment control conditions would improve little or not at all.

FSDs are sometimes subclassified according to the primary organ system or somatic symptom, and our review found that over half of the FSD populations were of chronic pain, with smaller numbers of gastrointestinal, neurological, and mixed presentations. Analyses of these FSD subtypes suggested that STPP for chronic pain or gastrointestinal disorders had large effect sizes, whereas STPP had only medium effects for neurological disorders. Although the small number of studies of gastrointestinal and neurological disorders suggests caution in interpretation, this finding is consistent with clinical observations that functional neurological disorders are particularly challenging to treat in brief therapies, as such patients have significant difficulties regulating emotions, experiencing cognitive-perceptual disruption with emotional activation [64,65].

STPP varies in several ways, including duration of treatment and the focus of therapy. Consistent with other reviews [43], the current analyses suggest that longer therapy—operationalized here as over 12 sessions—yields somewhat larger effects than shorter therapies, although even shorter treatments had significant, medium/large magnitude benefits. Regarding the focus of therapy, an earlier meta-analysis of STPP for FSD [3,4] suggested that STPPs that focus on emotional activation and expression yielded larger benefits than STPPs that target primarily insight. The current, much larger meta-analysis finds some support for this proposal. Therapies rated as relatively emotion-focused yielded substantially larger effects on somatic symptoms than non-emotion-focused therapies, although this difference was due primarily to two outlier studies of a highly emotion-focused therapy, ISTDP. When specific types of STPP were compared, however, and after excluding outliers, the emotion-focused therapies of ISTDP and EAET yielded clinically larger effects (by 0.40 SD) than did the mostly insight-focused PIT. The differential effect of these treatment models is consistent with findings of a Cochrane review of STPP for common mental disorders [7]. Such findings are consistent with a growing literature attesting to the value of emotional processing [44,45,59] and meta-analyses showing that patients' emotional expression is a strong predictor of positive therapy outcomes [61].

The improved symptoms and functioning following STPP were maintained through follow-up beyond 6 months. Meta-analyses of STPP for other disorders have also found sustained or increased benefits in follow-up [5,7,24,48]. STPP may yield important relational and personality changes that prevent relapse after treatment [70]. There is some evidence sustained benefits may be seen in other treatments, including CBT [27,40,74].

There are limitations of the literature and our review of it. First, the quality of studies was often subpar, a wide range of outcome measures was used, most analyses had heterogeneity, and the findings may have been influenced by publication bias. Although we tried to minimize some of these limitations via sensitivity analyses, results should be interpreted with caution. Second, we found evidence of possible publication bias. However, it is important to note that the test for publication bias was no longer significant on somatic measures after removing the two outlier studies. This is because such outliers inflate the amount of residual heterogeneity in the meta-analytic distribution. The resulting increased heterogeneity can be mistakenly attributed to publication bias [39]. Third, the current analyses were pre-post and not compared to control conditions, which limits conclusions about the specific benefits of STPP. However, our internal analyses comparing data from naturalistic studies versus RCTs, as well as the separate between-condition meta-analysis of STPP [6], strengthens the conclusion that STPP has large benefits beyond several non-specific factors. Comparisons with active controls—not just with no or minimal treatment—are needed to further test the specificity of STPP. Fourth, STPP has several variants, the boundaries distinguishing STPP from other therapies are not definitive, and it is not optimal to classify treatments based on theorists or the brief descriptions of the therapies that are provided in articles. Finally, FSD is a heterogeneous category, which limits conclusions for any specific syndrome or disorder. It should be noted, however, that co-morbidity, chronic overlapping conditions, and multiple somatic symptoms are extraordinarily common [1,42,81], suggesting that an umbrella category such as FSD has validity and utility.

5. Conclusion

This systematic review and meta-analysis offer further evidence that STPP is both an effective and efficacious treatment for diverse functional and somatic symptom disorders, yielding large magnitude, durable effects from before treatment to follow-up beyond 6 months. The effects of STPP for FSD compare quite favorably to effects of cognitive-behavioral and related interventions for FSD [56,74,78], suggesting that STPP should be included in treatment guidelines for these common clinical presentations, and maybe the preferred treatment approach for some patients, especially those with chronic pain or functional gastrointestinal disorders. Future research should directly compare STPP to other evidence-based approaches for FSD, test individual differences as predictors or moderators of treatment outcomes, and examine therapeutic mechanisms of various treatments for FSD.

Conflicts of interest

None

Acknowledgements

This research was supported by the Dalhousie University Department of Psychiatry and the Nova Scotia Department of Health and Wellness.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychores.2021.110473.

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