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# The Efficacy of Short-Term Psychodynamic Psychotherapy for Depressive Disorders with Comorbid Personality Disorder

Allan Abbass, Joel Town, and Ellen Driessen

The presence of comorbid personality disorder (PD) is one of the factors that can make the treatment of depression unsuccessful. Short-term Psychodynamic Psychotherapy (STPP) has been shown efficacious in the treatment of personality and depressive disorders (DD). However, the efficacy of STPP for comorbid DD and PD has not been systematically evaluated. In this study, data from patients meeting criteria for both DD and PD participating from randomized controlled trials of STPP was collected, systematically reviewed, and meta-analyzed where possible. Eight studies were included, 6 with major depression and 2 with minor depressive disorders. Pre- to post- treatment effects sizes were large ($d$ = 1.00–1.27), suggesting symptom improvement during STPP, and these gains were maintained in follow-ups averaging over 1.5 years. For major depression, no differences were found comparing STPP to other psychotherapies, and STPP was found superior to a wait-list condition in one study. STPP may have had an advantage over other therapy controls in treating minor depression as noted in ratings of general psychopathology. Patients with Cluster A/B and C PD were responsive to STPP, with the majority of all studied patients showing clinically significant change on self-report measures. Within the limits of this study, these findings suggest that STPP warrants consideration as a first line treatment for combined personality disorder and depression. Future research directions are proposed.

Major depression is a common, serious condition that usually does not respond to first line medication treatment (Thase, 2003). Among factors undermining depression treatment, the presence of personality disorder (PD) stands out, potentially doubling the rate of poor outcomes (Newton-Howes, Tyer, & Johnson, 2006). In this setting, personality disorder may impede the treatment alliance with healthcare providers and subsequent outcome. The internalization of rage and self-defeating behavioral patterns, typical in PD, are common in depression, rendering conventional first line approaches less effective (Gilbert, & Irons, 2004). Moreover, PD predisposes to chronic depression and dysthymic disorder, conditions that have worse prognoses and lower treatment response (Garyfallos et al., 1999, Thase, 1999). There is thus a paucity of lit-

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erature supporting medical or psychotherapeutic treatment in patients with comorbid PD and major depression (Newton-Howes et al., 2006).

Short-term psychodynamic psychotherapy (STPP) is a category of brief treatment that focuses on unconscious emotional processes that can impact on a person to produce or exacerbate depression, other symptom disorders, and personality disorders. STPP aims to directly address emotional repression, the turning inward of rage and interpersonal avoidance patterns through the resolution of past and current unconscious conflicts. In doing so it targets proposed mechanisms underlying both depression and interpersonal deficits prominent in PD. Three studies have demonstrated direct treatment intervention-outcome relationships between STPP and subsequent improvements in depressive symptoms (Barber et al., 2005; Gaston et al., 1998; Hilsenroth et al., 2007).

Like other psychological treatments, STPP operates in the substrate of the brain. A recent study found STPP significantly enhanced serotonin binding while Fluoxetine did not (Karlsson et al., 2009); this finding may explain the general finding of maintained gains in long-term follow-up after STPP in depression (Driessen et al., 2010) while relapse is very common upon antidepressant withdrawal.

A number of meta-analyses have supported the efficacy of STPP for general psychiatric symptoms, somatic symptoms, depressive disorders, and personality disorders (Abbass et al., 2006; Abbass et al., 2009; Driessen et al., 2010; Town, Abbass, & Hardy, 2011). In each of these reviews, STPP outperformed minimal treatment and wait list controls. Furthermore, Driessen and colleagues (2010), in a meta-analysis of 23 studies, found that STPP resulted in large depression symptom reductions ($d$ = 1.34) which were maintained in one-year follow-up. Individual STPP was found as efficacious as other psychotherapies at post-treatment and in follow-up. These findings add to the evidence base for STPP and, based on this, we proposed STPP be elevated to the level of first line evidence for the treatment of depression (Abbass & Driessen, 2010). Likewise, Town and colleagues (2011) found STPP had robust and persistent effects in patients with personality disorders. In this review, selecting only well-described randomized controlled trials ($n$ = 8), STPP was found to be superior to wait list controls and comparable to other recommended psychotherapies across symptomatic, interpersonal, and functional domains.

There is individual study evidence to suggest that the presence of comorbid depression and personality disorder may render STPP a valid treatment option. However, the efficacy of STPP for comorbid PD and depressive disorders (DD) has not been systematically examined. Given the potential that STPP can be of value in this challenging group of patients, we herein report the methods and results of such a review.

## METHODS

### Selection of Studies

With the recent elaborate literature searches for the meta-analyses of STPP for depression and personality disorders just completed (Driessen et al., 2010) and Town, Abbass, and Hardy (2011), we decided to use all studies included in these two meta-analyses as our main body of literature. As different inclusion criteria were used in these two meta-analyses, we applied a new set of inclusion criteria to this collection of studies in order to ensure consistency between the literature being reviewed. We included studies if they met the following criteria: a) STPP was delivered in an individual format; b) studies utilized a randomized controlled design, that is, those that incorporate a comparison or control group for evaluating the effects and random assignment to treatment

group; c) participants met specified criteria for either major depression or another DSM depressive disorder and personality disorder; d) depression was measured using standardized measures and raw data was available; and e) STPP was provided without pharmacotherapy. These studies were then reviewed in detail for outcome data on patients meeting the criteria for both PD and DD. When this data was not reported, study authors were contacted to send us separated data of patients with both conditions and further categorized by PD cluster. Outcome measures of interest were depression measures, general symptom measures, and measures of interpersonal dysfunction. Authors were then asked to extract raw patient data specifically for the purposes of clinical change calculations on depression measures only.

**Assessing Clinical Change**

The assessment of clinical change on depression measures was calculated based on the recommendations of Jacobsen and Truax (1991). Clinical significance (CS) was established with reference to normative data reported in the respective manual for the measure in question. In each case, patients' longest follow-up measurement was examined and those below the non-clinical cut-off threshold were deemed clinical significant. Next, the reliable change index (RCI)[1] was calculated to ensure that the magnitude of change was reliable. Based on Jacobsen, Follette, and Revenstorf's (1984) criteria, individual patient's response to treatment within each study was categorized either as *Recovered* (passed CS normative and RCI criteria), *Improved* (passed CS criteria alone), *Unchanged* (failed to pass CS criteria), or *Deteriorated* (passed RCI criteria in the negative direction).

**Meta-Analysis**

We conducted different meta-analyses, assessing the pre- to post-treatment change and the post-treatment to follow-up change in the STPP conditions and assessing the comparison of STPP with alternative treatments at post-treatment and follow-up. Therefore, different effect sizes (*d)* were computed for each of the primary studies. The pre- to post-treatment STPP effect size was calculated by subtracting the average post-treatment score from the average pre-treatment score and dividing the result by the pooled standard deviations of both groups. The effect size of STPP at follow-up was calculated by subtracting the average follow-up score from the average post-treatment score and dividing the result by the pooled standard deviations of both groups. The comparative effect sizes of STPP with other treatments at post-treatment and follow-up were calculated by subtracting the average score of the alternative condition from the average score of the STPP condition and dividing the result by the pooled standard deviations of both conditions. Effect sizes of 0–0.32 are assumed to be small; effect sizes of 0.33–0.55 are considered moderate; and effect sizes of 0.56–1.2 are considered large (Lipsey & Wilson, 1993). We used depression, general psychopathology, and interpersonal functioning as outcome measures. Only instruments explicitly measuring these constructs were used in the calculation of effect sizes. If more than one instrument was used to assess one outcome measure, the mean effect size from the different measures was computed for the study.

To calculate the pooled mean effect sizes, we used the computer program Comprehensive Meta-analysis (version 2.2.021; Biostat, Englewood, NJ). As considerable heterogeneity of the included studies was expected, we computed the pooled mean effect

1. Formula used to calculate reliable change index was: RCI = pre-treatment depression score minus longest follow-up depression score / standard error of difference between the two scores. The criterion level for reliable change was set at 1.96 times standard error of change.

sizes using the random effects model. In the random effects model the included studies are seen as a sample drawn from a population of studies, rather than replications of each other, so that not only the random error within the studies, but also the true variations of effect sizes from one study to the next are taken into account. Consequently, the random effects model results in broader 95%-confidence intervals (95% CI) and more conservative results.

As an indicator of homogeneity, we calculated the *Q*-statistic. A significant *Q*-value rejects the null hypothesis of homogeneity. We also calculated the $I^2$-statistic, which is an indicator of heterogeneity in percentages. A value of 0% indicates no observed heterogeneity, and larger values show increasing heterogeneity, with 25% indicating low, 50% indicating moderate, and 75% indicating high heterogeneity (Higgins, Thompson, Deeks, & Altman, 2003).

## RESULTS

### Inclusion of Studies

A total of 21 RCTs were identified from the reviews by Driessen and colleagues (2010) and Town, Abbass, and Hardy (2011) (13 and 8 studies, respectively), although in one case the data reported came from the same sample (Hardy et al., 1995; Shapiro et al., 1994). We contacted all authors of these 21 studies. The requisite raw data for effect size calculation was either no longer available or not accessible in 5 studies (Carrington, 1979; Hellerstein et al., 1998; Munroe-Blum & Marziali, 1995; Thompson, 1987; Winston et al., 1994). Four studies were excluded due to the lack of a formalized measure of personality disorder in the depressed study sample (Barkham et al., 1999; Gallagher & Thompson, 1982; Liberman & Eckman, 1981) or the absence of a depression measure in the personality disorder sample (Emmelkamp et al., 2006). Salimen and colleagues (2008) excluded patients with a personality disorder, and Morris (1975) reported an STPP group treatment, therefore both were excluded from the meta-analysis. In total, 8 studies were included in the meta-analysis.

### Study Characteristics

Based on the 8 studies, data from 166 participants who received an STPP was included in the meta-analysis (Table 3). The mean treatment length across studies was typically <40 sessions (range 8-80); however, the therapy format in one study (Lehto et al., 2007) involved twice weekly sessions, so the average number of sessions was 80. The quality of studies can be considered as moderate: studies utilized randomized comparative treatment designs; all STPP treatments were manualized; all but one study had adherence checks; in most cases diagnoses were made using versions of the Diagnostic Statistical Manual; and diagnoses were confirmed using a standardized interview method in all but two studies (De Jonghe et al., 2004; Lehto et al., 2008). Severity of depression was most commonly measured using the Hamilton Rating Scale for Depression (HRSD), the Beck Depression Inventory (BDI), and the Symptom Checklist Depression scale (SCL-90-D). STPP treatments can be further subcategorized according to primary therapist techniques existing on an expressive-supportive continuum (Luborsky, 1984). Studies included in the meta-analysis describe STPPs as reflecting a cross-section of these methods.

To our knowledge, data on treatment response rate for the subsample of patients with DD and PD have not been reported in the eight RCTs identified. Based on examination of the mean depression ratings at longest follow-up, on average ratings reached the normal range in most studies (Abbass et al., 2008; Maina, Forner, & Bogetto, 2005; Thyme et al., 2007; Svartberg, Stiles, &

TABLE 1. Depression Outcome Data for Studies Examining STPP for Co-Morbid Depressive Disorder and Personality Disorder

| | | | STPP scores | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Pre | | | Post | | | FU | | | | |
| First Author (year) | Depression Scale | STPP Model | Mean | SD | N | Mean | SD | N | Mean | SD | N | Pre-post ES | Post follow-up ES |
| Abbass (2008) | BSI-D | ISTDP (Davanloo, 2000) | 25.1 | 9.3 | 10 | 4 | 3.9 | 10 | 0.5 | 0.5 | 10 | 2.42** | -0.45 |
| De Jonghe (2004)/ Dekker (2010) | HAM-17 | SPSP (De Jonghe, 1994) | 17.8 | 3.6 | 60 | 11.1 | 7.2 | 60 | 9.6 | 6.9 | 60 | 1.11** | 0.22 |
| | SCL-90-D | | 4.6 | 1.1 | 59 | 3.1 | 1.7 | 60 | 2.7 | 1.6 | 60 | | |
| Hardy (1995) | BDI | PI (Hobson, 1985) | 25.1 | 9.3 | 13 | 15.1 | 9.8 | 13 | 12.8 | 11 | 13 | 1.05* | 0.22 |
| Lehto (2007) | HAM-17 | Unclear | 17.2 | 6.9 | 10 | 9.8 | 5.6 | 10 | | | | 1.43** | |
| | HAM-29 | | 27.6 | 8.2 | 10 | 14.3 | t7.7 | 10 | | | | | |
| Maina (2005) | HAM-17 | STDP (Malan, 1979) | 10.5 | 2.7 | 4 | 6 | 2.7 | 4 | 4.5 | 2.5 | 4 | 1.68* | 0.57 |
| Svartberg (2004) | BDI | AR-STDP (McCullough-Vaillant, 1997) | 23.9 | 5.8 | 7 | 11.6 | 6.9 | 7 | 9.6 | 10.2 | 7 | 1.94** | 0.26 |
| Thyme (2007) | BDI | TLP (Mann, 1973) | 22 | 7.6 | 21 | 13.4 | 11 | 21 | 10.7 | 7.2 | 21 | 1.15** | 0.2 |
| | SCL-90-D | | 2.3 | 0.6 | 21 | 1.2 | 0.9 | 21 | 1.1 | 0.8 | 21 | | |
| Vinnars (2005) | SCL-90-D | SE (Luborsky, 1984) | 29.5 | 11 | 36 | 17.9 | 12 | 27 | | | | 1.02** | 0.01 |

*$p < .05$; **$p < .01$.

Seltzer, 2004; Vinnars et al., 2005) and approached the cut-off threshold in the remaining two (Hardy et al., 1995; Lehto et al., 2007). Table 2 reports reliably and clinically significant change on depression measures for the five RCTs which selected only patients with comorbid major depression and PD. Forty-four percent (57/131) of patient outcomes examined met recommended criteria for "recovery" (Jacobsen et al., 1984) and significantly more showed clinically significant change. Chi-squared ($x^2$)analysis, not including the "deteriorated" response category due to low observed values, revealed no association between treatment response and PD cluster ($x^2$ (2) = 5.40; $p > .05$).

### Meta-Analyses

Table 1 summarizes data from depression measures extracted for those patients treated in each study with comorbid diagnoses of DD and PD. Data from 141 patients receiving STPP and 64 receiving a treatment comparison revealed large treatment effects at long-term follow-up in both conditions. Data from two studies treating PD and minor depressive disorders demonstrated large effect sizes following STPP, in both cases greater than that seen in the treatment comparison, with a statistically significant difference between Brief Dynamic Therapy and a non-STPP ($t = 3.11$; df = 7; $p = 0.017$; Maina, personal correspondence, 2010).

### STPP for Comorbid PD and Major Depression

We could compare the STPP pre- to post-treatment depression change in 6 studies, totaling 141 subjects (Table 3). The mean pooled effect size was 1.13 (95% CI:

TABLE 1. (continued)

| | Comparison group scores | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Pre | | | Post | | | FU | | | Between group ES | |
| Format | Mean | SD | N | Mean | SD | N | Mean | SD | N | STPP vs comp post-treatment | STPP vs comp Follow-up |
| W/L control | 16.4 | 4.3 | 5 | 13.2 | 7.3 | 5 | | | | 1.76** | |
| n/a | | | | | | | | | | | |
| CBT | 25 | 5.4 | 14 | 12.1 | 7.2 | 14 | 13 | 7.3 | 14 | -0.35 | 0.03 |
| n/a | | | | | | | | | | | |
| Supportive Therapy | 12 | 2.8 | 5 | 14.5 | 12.7 | 5 | 8.8 | 1.6 | 5 | 0.58 | 2.08* |
| CBT | 21.8 | 12.6 | 12 | 14.5 | 12.7 | 12 | 8.8 | 8.6 | 12 | 0.27 | -0.08 |
| Art Psychotherapy | 22 | 7.5 | 18 | 14.4 | 7.4 | 18 | 12.9 | 9.4 | 18 | 0.26 | 0.39 |
| | 2.2 | 0.7 | 18 | 1.5 | 0.9 | 18 | 1.5 | 0.8 | 18 | | |
| Psychodynamic TAU | 32.3 | 9.8 | 38 | 16.9 | 11.4 | 26 | | | | -0.08 | |

0.87–1.39). The effect size was 1.34 for all measures of depression (95% CI: 0.95–1.73). Mean effect sizes for general psychopathology and measures of interpersonal functioning were 1.00 (95% CI: 0.67-1.33) and 1.27 (95% CI: 0.76-1.79), respectively. All these pooled mean effect sizes were significant and indicate large pre- to post-treatment improvement in the STPP conditions.

We compared the post-treatment STPP depression scores with the scores at follow-up (Table 3). We calculated the change between post-treatment and longest follow-up which averaged 19.4 months. The effect sizes were all small and non-significant, suggesting no notable improvement or deterioration in long-term follow-up.

STPP could be compared to a wait list control group in only 1 of these studies. In Abbass and colleagues (2008), the effect sizes of BSI-D changes were 2.37 and 0.54 for the STPP and control groups, respectively.

STPP was compared with other psychotherapies in 3 studies (Table 3). The other psychotherapies consisted of cognitive behavioral therapy ($n$ = 2) and another variety of psychodynamic therapy ($n$ = 1). The pooled mean effect size for the between therapy difference at post-treatment was –0.04 (95% CI:–0.44 to–0.36), indicating essentially no difference between STPP and the other therapies. Similar results were found in measures of depression, general psychopathology, and interpersonal functioning. Interpersonal functioning did show a non-significant improvement with an ES of 0.24.

Two studies compared STPP with other psychotherapies in follow-ups averaging 18 months (Table 3). The mixed effect size was –0.15 (95% CI: –0.29–0.19), a non-significant difference. Similar results

TABLE 2: Response Rates Between Personality Clusters For Stpp For Co-Morbid Major Depressive Disorder And Personality Disorder

| First Author (Year) | N | Depression Scale | PD Clusters A & B | | | | PD Clusters C & NOS | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Recovered | Improved | Deteriorated | Unchanged | Recovered | Improved | Deteriorated | Unchanged |
| Abbass (2008) | 15 | BSI-D | 6(86%) | 0(0%) | 1(14%) | 0(0%) | 8(100%) | 0(0%) | 0(0%) | 0(0%) |
| De Jonghe (2004)/Dekker (2010) | 60 | HAM-17 | 14(33%) | 1(2%) | 2(5%) | 25(60%) | 8(44%) | 0(0%) | 2(12%) | 8(44%) |
| Hardy(1995) | 13 | BDI | – | – | – | – | 2(15%) | 4(31%) | 0(0%) | 7(54%) |
| Lehto (2007) | 10 | HAM–17 | 0 (0%) | 1 (33%) | 0(0%) | 2(67%) | 0(0%) | 3(43%) | 0(0%) | 4(57%) |
| Svartberg (2004) | 7 | BDI | – | – | – | – | 2(29%) | 4(57%) | 0(0%) | 1(14%) |
| Vinnars (2005) | 26 | SCL-90-D | 2(50%) | 1(25%) | 0(0%) | 1(25%) | 15(68%) | 1(5%) | 0(0%) | 6(27%) |
| Total | 131 | – | 22(40%) | 3(5%) | 3(5%) | 28(50%) | 35(47%) | 12(16%) | 2(2%) | 26(35%) |

TABLE. 3 Meta-Analyses of Studies Examining the Effects of STPP for Comorbid Major Depression and Personality Disorder

| Comparison | *N* | *d* | 95% CI | Z | Q | $I^2$ |
|---|---|---|---|---|---|---|
| STPP pre- to post-treatment change | | | | | | |
| All outcome measures (mixed) | 6 | 1.13 | 0.87 ~ 1.39 | 8.53** | 4.89 | 0 |
| Depression | 6 | 1.34 | 0.95 ~ 1.73 | 6.75** | 8.58 | 41.72 |
| General psychopathology | 5 | 1 | 0.67 ~ 1.33 | 5.91** | 5.31 | 24.63 |
| Interpersonal functioning | 3 | 1.27 | 0.76 ~ 1.79 | 4.85** | 0.52 | 0 |
| STPP post-treatment to follow-up change[a] | | | | | | |
| All outcome measures (mixed) | 5 | 0.12 | -0.13 ~ 0.37 | 0.93 | 1.12 | 0 |
| Depression | 5 | 0.1 | -0.15 ~ 0.35 | 0.78 | 3.01 | 0 |
| General psychopathology | 4 | 0.08 | -0.17 ~ 0.34 | 0.64 | 1.88 | 0 |
| Interpersonal functioning | 3 | 0.24 | -0.23 ~ 0.72 | 1 | 0.21 | 0 |
| STPP vs. other psychotherapy at post-treatment | | | | | | |
| All outcome measures (mixed) | 3 | -0.04 | -0.44 ~ 0.36 | -0.19 | 1.37 | 0 |
| Depression | 3 | -0.09 | -0.49 ~ 0.31 | -0.45 | 1.02 | 0 |
| General psychopathology | 3 | -0.06 | -0.47 ~ 0.34 | -0.31 | 1.15 | 0 |
| Interpersonal functioning | 2 | 0.16 | -0.79 ~ 1.11 | 0.33 | 2.45 | 59.22 |
| STPP vs. other psychotherapies at follow-up[b] | | | | | | |
| All outcome measures (mixed) | 2 | -0.15 | -0.78 ~ 0.47 | -0.48 | 0.26 | 0 |
| Depression | 2 | -0.02 | -0.63 ~ 0.60 | -0.06 | 0.03 | 0 |
| General psychopathology | 2 | -0.39 | -1.14 ~ 0.36 | -1.02 | 1.37 | 27.25 |
| Interpersonal functioning | 2 | -0.02 | -0.64 ~ 0.60 | -0.07 | 0.26 | 0 |

*Note*: STPP = short-term psychodynamic psychotherapy * $p < .05$; ** $p < .01$; Italic numbers indicate a non-significant trend ($p < .10$) [a] Post-treatment to longest follow-up (mean follow-up period 21.3 months) [b] Post-treatment to longest follow-up (mean follow-up period 18.0 months)

were found in measures of depression, general psychopathology, and interpersonal functioning. Overall, in these 2 studies there was no evidence of superiority of STPP or the other psychotherapy controls.

### STPP for Comorbid PD and Minor Depression

Two studies involved STPP to treat depressive disorders other than major depression. Pre- to post-treatment effects were large, ranging from 1.23 for depression to 2.79 for general psychopathology. Post-treatment to follow-up averaging 4.5 months (Table 4) showed small non-significant trends toward improvement (ES ranging from 0.24 to 0.28).

Both these studies also compared STPP to other psychotherapies; however, neither were manualized. Post-treatment differences were non-significant, but there was a trend toward significant benefits of STPP over other therapies in general psychopathology (ES 0.54, 95% CI -0.04-1.12). In follow-up, large but statistically non-significantly superior effects were seen in each measure (ES 1.06-1.32).

## DISCUSSION

This review supports STPP as a reasonable treatment option for depression in the setting of personality disorder. Pre-treatment to post-treatment effect sizes were large across multiple measures and sustained in long-term follow-up. Remission was achieved and sustained in 44% of patients, a result exceeding that of conventional antidepressant therapies in non-PD depressed populations (Thase, 2003). Unfavorable outcomes for depressed patients with the more difficult to treat personality clusters have been reported (Sato et al., 1994); however, benefits to patients with clusters B and A were observed, comparable to those seen in clusters C and NOS.

However, the limitations of this study are substantial and suggest the findings need be interpreted with caution. First, a small number of studies, with relatively small samples, were able to be included. Second and related, data for PD and depression patients was difficult to obtain from some of the studies due to the age of the data or other problems in access. This introduces a bias toward more recent active researchers' publications. Third, intention to treat analyses were not performed in each case, so that final values may favor the treatment. Fourth, we did not perform tests for publication bias due to the small number of studies. Fifth, the measures, samples, and methodologies were not consistent, limiting the interpretation of grouped data. Finally, although we included RCTs only, patients were generally randomized based on either the presence of DD or the presence of PD only. Therefore, subsamples of randomized studies were used to calculated post-treatment effect sizes comparing STPP with other conditions, and we cannot be sure that baseline differences between the participants in the different conditions did not influence outcome data.

Sustained gains over time and trends toward improved gains may well be due to sleeper effects. In this case, interpersonal gains would theoretically take time to modify social structures and secondary psychological health. As seems to be typical, the few studies comparing STPP with other formal treatments showed no significant differences, except in one measure in those with minor depression.

This review did not examine the literature on STPP provided concurrently with antidepressants: however, there is data, based on several studies, to support STPP in combination for patients with depression and PD. In an RCT, Burnand and colleagues (Burnand et al., 2002) studied STPP versus clomipramine alone in a sample of patients with major depression, 46% of whom had concurrent

TABLE 4. Meta-Analyses Of Studies Examining the Effects of STPP for Comorbid Minor Depression and Personality Disorder

| Comparison | *N* | *d* | 95% CI | *Z* | *Q* | $I^2$ |
|---|---|---|---|---|---|---|
| STPP pre- to post-treatment change | | | | | | |
| All outcome measures (mixed) | 2 | 1.95 | -0.14 ~ 4.04 | *1.82* | *3.30* | 69.73 |
| Depression | 2 | 1.23 | 0.62 ~ 1.84 | 3.97** | 0.35 | 0 |
| General psychopathology | 2 | 2.79 | -1.02 ~ 6.60 | 1.43 | 7.09* | 85.89 |
| STPP post-treatment to follow-up change[a] | | | | | | |
| All outcome measures (mixed) | 2 | 0.24 | -0.32 ~ 0.80 | 0.84 | 0.65 | 0 |
| Depression | 2 | 0.26 | -0.30- ~ 0.81 | 0.9 | 0.23 | 0 |
| General psychopathology | 2 | 0.28 | -0.54 ~ 1.10 | 0.66 | 1.42 | 29.57 |
| STPP vs. other psychotherapy at post-treatment | | | | | | |
| All outcome measures (mixed) | 2 | 0.38 | -0.19 ~ 0.96 | 1.31 | 0.03 | 0 |
| Depression | 2 | 0.31 | -0.26 ~ 0.89 | 1.08 | 0.19 | 0 |
| General psychopathology | 2 | 0.54 | -0.04 ~ 1.12 | *1.83* | 0.05 | 0 |
| STPP vs. other psychotherapies at follow-up[a] | | | | | | |
| All outcome measures (mixed) | 2 | 1.19 | -0.61 ~ 2.99 | 1.29 | 4.11* | 75.66 |
| Depression | 2 | 1.06 | -0.56 ~ 2.69 | 1.28 | *3.61* | 72.3 |
| General psychopathology | 2 | 1.32 | -0.62 ~ 3.26 | 1.34 | 4.46* | 77.58 |

*Note*: STPP=short-term psychodynamic psychotherapy * $p < .05$; ** $p < .01$; Italic numbers indicate a non-significant trend ($p < .10$) [a] Post-treatment to longest follow-up (mean follow-up period 4.5 months)

personality disorders. They found greater remission rates and noted more money was saved through reduced hospital use and disability payments than the STPP treatment actually cost. In an RCT, Maina and colleagues (2007) found superior long-term HAM-D remission and response rates (87.5% and 75%) in patients provided with STPP plus medication versus supportive therapy plus medications (25% and 12.5%): the supportive therapy-medication combination showed deterioration over the course of this study while the STPP group showed further gains (rate of PD was not provided in this study, but PD was not an exclusion criteria). In an RCT, Kool and colleagues (2003) found that a brief supportive format of STPP in combination with antidepressants was superior to medication alone in a sample with major depression and PD. In a case series, Abbass (2006) found 8 of 10 patients with PD and treatment-resistant depression remitted and had sustained gains using an emotion-focused variety of STPP, Davanloo's Intensive Short-term Dynamic Psychotherapy (2000). This treatment was 13.6 sessions, and costs were offset through hospital and medication use reduction. Burnand and colleagues (2002) and Abbass (2006) provide further evidence to support the cost-effectiveness of STPP in depression and other common mental disorders (Abbass, 2003).

STPP showed significant treatment effects in a complex population, including the more severe PD diagnoses. This is of note given that research has suggested that PD clusters A and B have a negative effect on major depression (Corruble, Ginestet, & Guelfi, 1996) and these patients report poorer quality of life and a greater number of suicide attempts (Breiger et al., 2002).

Indeed, there is a lack of literature supporting treatment in patients with comorbid PD and depression. Following the unexpected finding that personality disorders did adversely affect treatment response in some patients receiving Interpersonal Psychotherapy (IPT), Joyce and colleagues (2007) questioned the selection of other dynamic therapies for this population. In contrast, the positive findings in this set of studies appear to distinguish STPP from IPT and suggest that STPP warrants consideration as a first line treatment for this complex population, as was alluded to in a recent set of depression treatment guidelines (Parikh et al., 2009). Indications of between-treatment differences in response in the presence of comorbid personality disorder (Joyce et al., 2007) supports the assumption that therapies work by different underlying mechanisms, thus emphasizing the need for further research around treatment-specific change mechanisms.

To consolidate this concept, further study is however warranted. Formal study with dually diagnosed populations, measurement of relapse, and remission and objective ratings by blinded reviewers should be employed in such studies. Research into which elements in the process appear beneficial should be elucidated with prospective studies using dismantling or such other methods as detailed case series designs.

## CONCLUSION

STPP is a brief psychotherapeutic intervention with a modest evidence base to support its consideration in major depression with PD. It lacks significant adverse effects, side effects, and toxicities, as well as the adverse effects of somatic treatments. Thus, it should ethically be considered first, prior to more invasive treatments (Malhi et al., 2009). Moreover, evidence to support STPP's cost effectiveness in this very costly condition should not be ignored. Further research is warranted into the specific mechanisms of action, magnitude of effects and limitations of utility of this method. However, within the limits of this study, our findings suggest that STPP warrants consideration, based on recent depression guidelines criteria (Malhi et al., 2009; Parikh et al., 2009), as a first line treatment option for comorbid PD and major depression.

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