Schema therapy for patients with chronic depression: A single case series study

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ABSTRACT
Background and objectives: This study tested the effectiveness of schema therapy (ST) for patients with chronic depression.
Methods: Twelve patients with a diagnosis of chronic depression participated. The treatment protocol consisted of 60 sessions, with the first 55 sessions offered weekly and the last five sessions on a biweekly basis. A single case series A–B–C design, with 6 months follow-up was used. Baseline (A) was a wait period of 8 weeks. Baseline was followed by introduction to ST and bonding to therapist (phase B) with individually tailored length of 12–16 sessions, after which further ST was provided (phase C) up to 60 sessions (included the sessions given as introduction). Patients were assessed with Hamilton Rating Scale for Depression three times during baseline, at the end of phase B, then every 12 weeks until the end of treatment and at 6 months follow-up. Secondary outcome measures were the Hamilton Rating Scale for Anxiety and the Young Schema Questionnaire.
Results: At the end of treatment 7 patients (approximately 60%) remitted or satisfactorily responded. The mean HRSD dropped from 21.07 during baseline to 9.40 at post-treatment and 10.75 at follow-up. The effects were large and the gains of treatment were maintained at 6-month follow-up. Only one patient dropped out for reasons not related to treatment.
Limitations: The lack of control group, the small sample and the lack of a multiple baseline case series.
Conclusions: This preliminary study supports the use of ST as an effective treatment for chronic depression.

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1. Introduction

Approximately 20% of all depressed individuals develop a chronic course (Arnow & Constantino, 2003; Gilmer et al., 2005). This implies that 2.5–6% of the adult population in the community suffers from chronic depression (Kessler et al., 2005, 1994). Chronic depression is associated with increased functional impairment (Klein, Schwartz, Rose, & Leader, 2000; Klein, Shankman, & Rose, 2006; Wells, Burnam, Rogers, Hays, & Camp, 1992), higher levels of health care utilization, hospitalization and economic costs (Berndt et al., 2000; Gilmer et al., 2005; Howland, 1993; Klein et al., 2000; Smit et al., 2006) compared with non-chronic forms of depression.

Four types of chronic depression are usually distinguished in the literature: 1) dystymic disorder, 2) chronic major depressive disorder (MDD), 3) double depression (MDD superimposed on a dystymic disorder) and 4) recurrent major depressive disorder with incomplete remission between the episodes (Torpey & Klein, 2008). There are consistent findings supporting the idea that the various manifestations of chronic depression do not represent distinct disorders (Cuijpers et al., 2010; Klein, Shankman, Lewinsohn, Rohde, & Seeley, 2004; Klein et al., 2006; McCullough et al., 2003, 2000). The DSM-5 (American Psychiatric Association, 2013) diagnosis of persistent depressive disorder (dysthymia).
includes both the DSM-IV diagnostic categories of chronic major depression and dysthymia.

It is suggested that the determinants of chronic depression do not necessarily differ qualitatively, but only quantitatively from those of acute depression, with involvement of increased levels of these determinants in chronic forms (Riso, Miyatake, & Thase, 2002). Among the several possible determinants of chronic depression that have been investigated so far, the strongest support has been found for the role of developmental antecedents and early adversity (Bifulco, Brown, Lillie, & Jarvis, 1997; Brown, Craig, & Harris, 2008; Brown, Craig, Harris, Handle, & Harvey, 2007; Brown, Harris, Hepworth, & Robinson, 1994; Brown & Moran, 1994; Klein et al., 2009; Lizardi et al., 1995; Riso et al., 2002). Family problems, anxious personality in childhood and low self-esteem and mastery in early adulthood have been associated with chronicity (Angst, Gamma, Rossler, Ajdacic, & Klein, 2011). A recent meta-analysis reported that childhood maltreatment is associated with elevated risk of developing chronic depression and lack of response during treatment (Nanni, Uher, & Danese, 2012).

Several studies have emphasized the close relationship between chronic depression and Axis II personality disorders (Garfallo et al., 1999; Klein et al., 1995; Maddux et al., 2009; Pepper et al., 1995; Riso et al., 1996, 2002). Among dysthymic patients the rates of personality disorders tend to be high, up to 65% (Klein et al., 1995; Riso et al., 2002). Cluster C personality traits in patients with chronic depression predict poor outcome in a naturalistic study at 5 (Hayden & Klein, 2001) and 10-year follow-up (Klein, Shankman, & Rose, 2008).

According to the cognitive theory of depression negative core beliefs or cognitive schemas represent key vulnerability factors to depression (Beck, 1976; Beck, Rush, Shaw, & Emery, 1979). Young, influenced by cognitive and attachment theory, elaborated the schema concept (Young, 1994; Young, Klosko, & Weishaar, 2003) and proposed that Early Maladaptive Schemas (EMS) are broad, pervasive, trait-like, cognitive and emotional self-defeating patterns, regarding oneself and one’s personal relationships (Young et al., 2003). EMS are hypothesized to develop as a result of toxic childhood experiences and unmet core emotional needs, and to underlie the development of psychopathology and chronic psychological disorders (Young et al., 2003). To date 18 EMS have been identified and grouped in five domains: disconnection and rejection; impaired autonomy and performance; other directedness; over-vigilance and inhibition; and impaired limits (Young et al., 2003). EMS remain stable over time (Renner et al., 2013; Wang, Halvorsen, Eisemann, & Waterloo, 2010) and relate to depressive symptoms in depressed patients (Halvorsen et al., 2009; Hawke, Provencher, & Arntz, 2011; Petrocelli, Glaser, Calhoun, & Campbell, 2001). EMS of the domains impaired autonomy & performance and disconnection & rejection relate to depressive symptoms severity (Renner, Lobbestael, Peeters, Arntz, & Huibers, 2012), and EMS of the domains impaired autonomy & performance and over-vigilance & inhibition distinguish patients with chronic depression from patients with non-chronic major depressive disorder (Riso et al., 2003). The emotional deprivation schema mediates the relation between physical abuse and anhedonic depressive symptoms whereas social isolation and self-sacrifice schemas mediate the relation between emotional maltreatment and anhedonic depressive symptoms (Lumley & Harkness, 2008).

In conclusion, the evidence so far suggests that EMSs play a role in chronic depression.

A schema model for chronic depression has been described proposing the interplay between distal factors (early adversity, personality pathology), which are mediated by proximal factors (EMS) triggered by life events (loss, failure) and maintained by avoidant coping strategies (Renner, Arntz, Leeuw, & Huibers, 2013).

Pharmacological (Kocsis, 2003; Kocsis et al., 2009) and psychotherapeutic (Keller et al., 2000; Markowitz, 1994) interventions have been developed for the treatment of chronic depression. Cognitive Behavior Analysis System of Psychotherapy (CBASP) (McCullough, 2000) is a model incorporating cognitive behavioral and interpersonal techniques, which was developed for the treatment of chronically depressed patients. Studies have supported its effectiveness (Keller et al., 2000; Schatzberg et al., 2005) suggesting equivalence to pharmacotherapy (Keller et al., 2000; Kocsis, 2009) and superiority to pharmacotherapy for chronically depressed patients with a history of childhood trauma (Klein et al., 2009; Nemeroff et al., 2003). Although the initial effects of the implementation of CBASP were good, when it comes to long-term effects, CBASP does not seem to do better than continued antidepressant medication (Gelenberg et al., 2003; Kocsis et al., 2009; Renner, Arntz et al., 2013).

A meta-analysis examining the effects of psychotherapy on chronic depression reported that the length of the studied psychotherapies may not be sufficient to treat dysthymia (Imel, Malterer, McKay, & Wampold, 2008). Indeed, the efficacy of psychotherapeutic interventions increases with the number of sessions (Cuijpers et al., 2010). Klein et al. (2008) suggests that chronically depressed patients with comorbid personality disorders may require a modified and more intensive course of treatment.

The above-mentioned literature suggests the crucial causal role of early adversity, EMS and comorbid personality disorders in the development of chronic depression. Moreover the effectiveness of the existed treatment interventions remains limited. A qualitatively different psychotherapeutic intervention lengthier and more intensive, which focuses on underlying psychological factors like childhood adversity and schemas might lead to improvement of treatment of chronic depression.

Schema therapy (ST) has been developed as the clinical implication of Young (1994) schema theory. It is an integrative therapy, which combines elements of cognitive behavior therapy, attachment theory, object relations theory and emotional-focused models and was developed for the treatment of patients with chronic emotional difficulties (Young et al., 2003). ST is an effective treatment for patients with borderline personality disorder (BPD) (Farrell, Shaw, & Webber, 2009; Giesen-Bloo et al., 2006; Nardon et al., 2009; Nordahl & Nyaester, 2005) and for patients with cluster C personality disorders, including comorbid depression (Bamelis, Evers, Spinholven, & Arntz, 2013). Recently a randomized clinical trial compared ST and CBT for patients with a current major depressive episode, in a protocol of weekly sessions for six months and monthly sessions for another six months (Carter et al., 2013). No difference was found between the two therapies. Brewin et al. (2009) tested the use of imagery rescripting (a core technique of ST) as a stand-alone treatment for chronically depressed patients with intrusive memories and found large treatment effects, maintained at one-year follow-up. Renner, Arntz et al. (2013) currently conduct a single case series study of ST for chronic depression testing the model described above.

To the best of the authors’ knowledge so far no study has been published on the application of schema therapy in chronic depression. The aim of this study is to examine the effectiveness of schema therapy in a sample of chronically depressed patients.

2. Methods

2.1. Participants

Inclusion criteria were a primary diagnosis of DSM-IV chronic depression, age 18–65 years and a score of 15 or higher on the 24-item Hamilton Rating Scale for Depression (HRSD24)
Exclusion criteria were a secondary diagnosis of bipolar or cyclothymic disorder, schizophrenia or other psychotic disorder, borderline or antisocial PD, eating disorder, obsessive compulsive disorder, psychiatric disorders secondary to medical conditions, mental retardation, severe addiction needed detoxification and presence of current suicidal ideation.

Patients could be on antidepressant medication, with the treatment remaining stable, concerning dosage and medication, for at least 1 month before the first baseline assessment.

In case of severe suicidal ideation or medical conditions needing hospitalization, the participant would be excluded from the trial and appropriate treatment would be provided. This did not happen.

Patient flow is presented in Fig 1. Twelve patients participated, all women, age 26–56 years old.

### 2.2. Design

A single case series A-B-C design, with 6 months follow-up was used. Baseline (A) was a wait period of 8 weeks during which the primary outcome (HRSD) was taken three times every 4 weeks. After baseline, an introduction to ST and bonding to therapist was provided (phase B), with individually tailored length of 12 to maximally 16 sessions, after which assessment was repeated and ST was provided (phase C) up to 60 sessions (included the sessions given as introduction). We assessed effects of phase B separately like was done in Weertman and Arntz (2007) and Arntz, Sofi, and van Breukelen (2013).

All the participants were outpatients of the Women’s Mental Health Clinic of the 1st Department of Psychiatry, Eginition Hospital, Athens Medical School. Therapists of the unit referred patients to the study, based on a clinical diagnosis of chronic depression.

Patients were assessed using the Structured Clinical Interview for DSM-IV Axis I (SCID-I, First, Spitzer, Gibbon, & Williams, 2002) and Axis II (SCID-II, First, Gibbon, Spitzer, Williams, & Benjamin, 1997) Disorders. They were further screened using the HRSD; patients had to have a score of 15 or higher to be eligible for the study. All the screening interviews were conducted by one experienced psychiatrist.

After the screening the patients that were eligible for the study entered phase A. An HRSD score of 15 or higher in the assessments of phase A was mandatory for them to continue in phase B with the initiation of the treatment (no participant was excluded for this reason). Additionally the patients who received therapy were assessed pre-treatment on the battery of the instruments of the study. Assessment during treatment was conducted by the administration of the HRSD and the other instruments used in the study at 12th–16th sessions, 24th session, 36 session, 48 session, post-treatment (60th session) and follow-up (6 months after the termination). The first assessment during treatment was conducted on session 12th–16th to give therapists the flexibility to complete the first stage of the treatment including assessment, education and bonding with the patient.

The baseline, pre-, during, post-treatment and follow-up assessments were conducted by an experienced psychiatrist (other from the one conducted the screening interviews).

The study was approved by the Medical Ethics Committee. All patients signed informed consent.

### 2.3. Outcome measures

The main outcome measure was the 24-item HRSD. Following previous studies on chronic depression (Keller et al., 2000; Kocsis et al., 2009) we defined remission as an HRSD score of no more than 8 at post-treatment and follow-up assessments and satisfactory response (but not remission) as a reduction of 50% in the HRSD and a score of 15 or less, but of more than 8 at post-treatment and follow-up assessments.

A second outcome measure was the Hamilton Rating Scale for Anxiety. A score of 14 has been suggested as the threshold for clinically significant anxiety.

A third, treatment-specific measure, was the Young Schema Questionnaire YSQ, Long form, 3rd version (YSQ-L3). The YSQ-L3 is a 232-item self-report inventory that assesses the 18th schemas proposed by Young (2003). We used the sum of the scores of the schemas of each of the five domains, defined as YSQ1–5 respectively.

### 2.4. Treatment protocol

The schema therapy treatment protocol consisted of 60 sessions of 50 min duration each. The first 55 sessions were offered weekly and the last five sessions on a biweekly basis. The whole treatment was offered over a 20 months period.
Taken into account recent developments in ST for PD that focus more on schema modes than schemas (Arntz, 2012) and the close relation between chronic depression and PDs we followed a mode approach of ST similar to that described by Arntz (2012) for application of ST in patients with cluster C PDs. Whereas schemas are trait-like constructs, schema modes are state-like, and denote the current affective-cognitive-behavioral state of the person. Schema modes can be understood as the combination of an activated schema and specific coping (Lobbestael, Arntz, & Arntz, 2013). First the main effect of time was found to be optimal and was used in these analyses. The analytic structure for the repeated part was determined comparing ARMA11, AR1, and AR1 heterogeneous structures. The last one was used. The optimal covariance structure for the repeated part, and time effect was non-significant, it was deleted as predictor. Additionally, adherence to ST for methods and techniques used was excellent (mean = 0.87; SD = 0.15) and no non-ST techniques were observed.

### 2.6. Statistical analysis

SPSS version 21 mixed regression was used to analyze the HRSD results, using all available data. For the separate analysis of the three HRSD baseline assessments an unstructured covariance structure for the repeated part was used. For the other analyses, involving more repeated assessments, the optimal covariance structure for the repeated part was determined comparing ARMA11, AR1, and AR1 heterogeneous structures. The last one was found to be optimal and was used in these analyses. The analytic strategy for analyzing treatment effects was similar to those described in Arntz et al. (2013). First the main effect of time was tested (i.e., the linear time effect), by entering assessments (starting with zero) as covariate. In the second step, condition and time-within-treatment (centered) were added. Condition had three levels: baseline (reference), introduction (the assessment immediately after the introduction phase), and treatment (the four assessments after 24, 36, 48 and 60 sessions). If the general linear time effect was non-significant, it was deleted as predictor. Addition of random intercepts and slopes was tried but estimations failed to converge. The 6 months follow-up was separately tested against baseline, also by means of mixed regression, with an unstructured covariance structure for the repeated part, and time within baseline (centered) as covariate.

For the HRSA, only one baseline assessment was available, just before introduction to treatment. Therefore no change during baseline could be assessed, but the other analyses were similar as with the HRSD.

## Table 1: Sociodemographic and clinical characteristics of the participants.

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age</th>
<th>Marital status</th>
<th>Education (years)</th>
<th>Chronic depression diagnosis (SCID-I)</th>
<th>Years of onset of depression</th>
<th>Medication</th>
<th>Personality disorder (PD) diagnosis (SCID-II)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>47</td>
<td>Married</td>
<td>12</td>
<td>Chronic MDD</td>
<td>2</td>
<td>SSRI</td>
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<td>2</td>
<td>26</td>
<td>Single</td>
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<td>6</td>
<td>54</td>
<td>Married 2 children</td>
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Four therapists participated in the trial. All were psychologists with MSc degrees, one had also a PhD degree. All therapists were trained in the training program of the Greek Society of Schema Therapy and were certified on advanced level by the International Society of Schema Therapy (2013) (www.isst-online.com).

### 2.5. Therapists and treatment integrity

Treatment integrity was monitored by means of supervision. Therapists were provided with 3 h group supervision weekly during the first 12 months of the treatment and bi-weekly during the last eight months of the treatment. The supervisor was an experienced psychiatrist and schema therapist, accredited by the ISST.

All sessions were audiotaped. One audiotape between sessions 16 and 50, from each of the 12 patients, was randomly selected and rated by the supervisor, using the Schema Therapist Rating Scale (Young, 2005). An adequate level of competency was defined as a mean score of 4 or 4.5 for standard or advanced level certified therapists respectively (Nadort, Genderen, & Behary, 2012). A cut off score of 4.5 was chosen, as therapists were all certified in advanced level. Adherence to ST for methods and techniques used was excellent (mean = 5.12; SD = 0.15) and no non-ST techniques were observed.

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For the YSQ, only assessment just before introduction to treatment, after 24 sessions, and post-treatment were available. Because of the skewed distributions, changes were tested with mixed gamma regression with a log-link, using an unstructured repeated part. Assessment was entered as factor, with the baseline-24 sessions and the baseline-post-treatment assessment contrasts.

Three versions of Cohen’s $d$ as effect size for the final change as result of treatment were calculated based on the mean change in estimated means from the mixed regression analysis from the (average) baseline assessment(s) to the 60 months assessment, respectively the half year follow-up, and three SD estimates: (1) the baseline SD; (2) the pooled standard deviation of the assessments involved in the pertinent change; the SD of the change score. All necessary statistics were derived from the mixed regression results.

3. Results

Of 16 patients referred for the study, 4 (25%) were not eligible for participation: 1 did not meet inclusion criteria (had no chronic depression), 3 met exclusion criteria (1 bipolar disorder, 1 psychotic disorder, 1 BPD). 12 patients (75%) were included in the study. Of the 12 patients who began treatment one participant (8.33%) dropped out at session 50 due to business reasons (had to move to another city). No participant was excluded during treatment due to need of hospitalization or suicidal ideation.

At post-treatment assessment five out of the twelve patients (41.6%) fully remitted (participants 2, 4, 5, 6, 8) and another two patients (16.6%) responded satisfactorily (participants 9, 11). Of the fully remitted patients, at the follow-up assessment one relapsed (participant 6) and the others maintained the result while in the same period of the 6-month follow-up, one patient (7) who had not responded by the end of the treatment, exceeded satisfactory response and also one patient (9) who had responded satisfactorily by the end of the treatment, exceeded remission, leading to five remitters and an additional three responders at follow-up.

Table 2 presents the estimated means from mixed regression analysis for HRSD and HRSA.

3.1. Depressive symptoms: HRSD

3.1.1. Change in depression during baseline

Using an unstructured covariance structure for analyzing the three baseline assessments, it was found that there was a significant linear increase in HRSD scores during baseline, $B = 0.52$, $se = 0.186$, $t (11) = 2.81$, $P = 0.017$. Thus, during wait, depression symptoms increased.

3.1.2. Treatment effects on depression

The model with time as single predictor revealed a significant effect of time, $B = -1.50$, $se = 0.271$, $t(27.40) = -5.53$, $p < 0.001$ (based on first order autoregressive covariance structure with heterogeneous variances). However, after adding condition and time-within-treatment as predictors, overall time became non-significant, $B = 0.50$, $se = 0.63$; $t (23.64) = 0.81$, $p = 0.43$ (based on first order autoregressive covariance structure with heterogeneous variances). After deleting the overall time effect, the condition effect was significant, $F (2, 21.14) = 22.29$, $p < 0.001$. The assessment after the introduction phase showed significantly lower HRSD scores than during baseline ($m = 20.87$ vs. $m = 10.87$, $t (8.92) = 4.78$, $p < 0.001$). The mean HRSD score during treatment ($m = 12.29$) also differed significantly from baseline, $t (24.32) = 5.83$, $p < 0.001$. During treatment there was a significant linear decrease in HRSD scores, $B = -1.94$, $se = 0.854$, $t (23.25) = -2.28$, $P = 0.032$. At post-test the estimated HRSD mean was 9.40 (SD 7.17), significantly lower than during baseline ($m = 20.87$, $SD = 3.61$). Effect size estimates (Cohen’s $d$) were 2.01 (pooled SD); 2.84 (baseline SD); and 2.02 (SD of change score).

3.1.3. Six months follow-up vs. baseline

The six months follow-up (estimated mean = 10.75) differed significantly from baseline, $t (10.62) = 4.57$, $p = 0.001$. Effect size estimates (Cohen’s $d$) were 2.24 (pooled SD); 2.60 (baseline SD); and 1.40 (SD of change score).

3.2. Anxiety symptoms: HRSA

3.2.1. Change in anxiety during treatment

The linear time effect was significant, $B = -1.49$, $se = 0.478$, $t (27.90) = -3.12$, $p = 0.004$. However, after entering condition, the time effect became non-significant, $B = -1.00$, $se = 0.83$, $t (23.83) = -1.20$, $p = 0.24$. After deleting the general time effect, condition was significant, $F (2, 19.90) = 5.67$, $p = 0.011$. Introduction differed significantly from baseline, $t (18.14) = -2.37$, $p = 0.029$, as was the average during treatment, $t (20.53) = -3.26$, $p = 0.004$. The change during treatment failed to reach significance, $p = 0.24$. The estimated post-treatment mean was 9.56, compared with the baseline mean 17.29. Effect size estimates (Cohen’s $d$) were 1.22 (pooled SD); 1.35 (baseline SD); and 1.04 (SD of change score).

3.2.2. Six months follow-up vs. baseline

The six months follow-up (estimated mean = 9.63) differed significantly from baseline, $t (11.05) = 2.84$, $p = 0.016$. Effect size estimates (Cohen’s $d$) were 1.33 (pooled SD); 1.36 (baseline SD); and 0.83 (SD of change score).

Table 3 presents the estimated means from mixed regression analysis for HRSD and HRSA.

3.3. Maladaptive schema’s: YSQ domain scores

3.3.1. Changes in domain scores during treatment

Table 3 gives an overview of the results of the mixed gamma regression for the five YSQ domain scores and the total YSQ sum. As can be seen, the change from baseline to 24 sessions was modest and NS in the majority of the contrasts, whereas the change from baseline to post-treatment was large and highly significant.

The scores on the standardized measures at baseline, Pretreatment, 12th–16th session, 24th session, 36th session, 46th session, Post-treatment and 6-month follow-up are presented in Fig. 2 (YSQ scales were divided by 10 for better presentation).

4. Discussion

This study found that sixty sessions of ST produced statistically and clinically significant improvements in the symptomatology of chronically depressed female outpatients. This improvement concerns depression and anxiety as measured by...
examining the emotional process in the experiential therapy of depression have found that high levels of emotional arousal in mid-therapy are predictive of reduction in depressive symptomatology and are related to positive outcome (Missirlian, Toukmanian, Warwar, & Greenberg, 2005; Pos, Greenberg, & Warwar, 2009). This emotional processing phase could explain the finding of our study that all patients who exhibit remission or satisfactory response showed this pattern of seeming deterioration at this phase of treatment.

Comparing the results of our study to those of CBASP studies. Schema Therapy seems to be a promising new treatment. In the study by Keller et al. (2000) the overall rates of response for the intention-to-treat sample was 48% for CBASP group and 73% for the combined-treatment group (nefazodone plus CBASP). Mean HRSD scores dropped from 26.4 to 15.1 in the CBASP group and from 27.4 to 9.7 in the combined treatment group (21.07 – 9.4 in our study). A more recent study from the same group (Kocsis et al., 2009) found that the addition of CBASP or Brief Supportive Psychotherapy (BSP) in chronically depressed patients already treated with an algorithm of pharmacotherapy produced an overall rate of response of 37.5% with the other 62.5% remained depressed. Mean HRSD dropped from 19.5 to 11.2 in the group received CBASP and from 19.4 to 12.8 in the group received BSP. In difference with the CBASP studies, we provided a much lengthier protocol of 60 sessions of psychotherapy. Moreover over the control of the antidepressant medication was beyond the scope of our study, but all of the participants were already on antidepressant medication, which had to be stable at least three months before the start of treatment and had not responded to it. This concept is closer to the arms of the CBASP studies where psychotherapy has been added (Kocsis et al., 2009) or provided in combination with medication (Keller et al., 2000). Direct comparisons are needed to assess in what aspects the two approaches might yield different outcomes.

Our study along with the study by Carter et al. (2013) supports the suggestion of using ST for depressed patients. In the study of Carter, the focus of interest was a current major depressive episode, but 68% of the participants had also a diagnosis of chronic depression. The presence of chronic depression did not impact the outcome of the study. These results could be interpreted as an indication for the use of CBT for chronic depression. On the other hand the study of Carter et al. lack of a follow-up assessment and as mentioned in the introduction the existing psychotherapeutic interventions suffer from low rates of maintenance of their effects.

Regarding the EMS as measured with the YSQ inventory, in our study, a statistically significant reduction was observed in the scores of the five domains of the YSQ. Reduction of depression and anxiety seems to precede reduction in the YSQ. This is similar with the finding by Renner et al. (2013). An explanation for this could be
Fig. 2. Scores on standardized measures for HRSD, HRSA, YSQ1–5, for each participant (participants 1–12).
Participant 6

Participant 7

Participant 8

Participant 9

Participant 10

Fig. 2. (continued).
that the YSQ, as a trait-instrument, might be not very suitable to assess early changes in schemas. Alternatively, therapeutic changes take first place at emotional/symptom level, before they are shown at schema level.

The reduction observed at the post-treatment assessment in YSQ was especially manifest in the domains Impaired Autonomy & Performance and Over-vigilance & Inhibition that characterize chronically depressed patients (Riso et al. 2003) and the domain of Disconnection & Rejection that is closely related to depressive symptomatology (Renner et al. 2012). These changes at schema level could explain the maintenance of the results in the 6-month follow-up, but larger studies are needed to empirically test this.

A number of factors limit the generalization of the results of this study. One main limitation concerns the lack of control group. A second one concerns the small number of the sample. A third limitation is a weakness of the design, which does not include a baseline phase of variable length, randomized over participants (“multiple baseline case series”). With such a design, especially when it is concurrent (participants start at the same moment), better experimental control is achieved. On the other hand the use of four therapists is strength of the study supporting the feasibility of the treatment by different therapists. The third limitation is a weakness of the design, which does not include a baseline phase of variable length, randomized over participants (“multiple baseline case series”). With such a design, especially when it is concurrent (participants start at the same moment), better experimental control is achieved. On the other hand the use of four therapists is strength of the study supporting the feasibility of the treatment by different therapists.

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duction of the assessments by an independent rater, the supervision of therapists and the control for therapy adherence are other strengths of this trial. The present study should be considered as an indication and a preliminary test of the effectiveness of ST for chronic depression alone or in combination with pharmacotherapy.


declaration of interest

All the authors explicitly state that there is no interest to be declared.


definitions

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