ORIGINAL ARTICLE

Effectiveness of a cognitive-behavioral group intervention for comorbid anxiety and mood disorders in partially responder outpatients

Roberto Truzoli, Cecilia Rovetta, Marta Romanò, Caterina Viganò, Gabriella Ba

Department of Biomedical and Clinical Science, University of Milan, Milan, Italy.

Correspondence to: Prof. Truzoli Roberto, A.O. Luigi Sacco-Polo Universitario, Psichiatria II, via G.B. Grassi, 74 20157 Milano, Italia; e-mail: roberto.truzoli@unimi.it

Key words:

cognitive-behavioral group intervention; reliable change index; unwanted effects; anxiety and mood disorders comorbidity; effectiveness

Act Nerv Super Rediviva 2015; 57(4): 89-97

ANSR570415A01

© 2015 Act Nerv Super Rediviva

Abstract

OBJECTIVE: The purpose of this study was to evaluate the effectiveness of a short-term Cognitive-Behavioral Group Intervention for comorbid Anxiety e Mood Disorders in partially pharmacotherapy responder outpatients and to test the feasibility of this intervention as complementary treatment in psychiatric settings.

DESIGN: Thirty-three participants have been assessed for the overall psychopathological symptoms, depression and anxiety before and after an 8 weeks group intervention, while following their pharmacological treatment as usual. The contents of the cognitive-behavioral group intervention have been psychoeducation, relaxation techniques, life-style management, emotional regulation techniques, meditative techniques, cognitive restructuring, addressed to support the patients in reducing their symptoms and learning self-regulatory and self-management skills.

Statistically and clinically significant change analyses have been conducted, included the evaluation of the unwanted effects.

RESULTS: The outcomes have shown a significant reduction in the overall symptoms and the patients reported significant decrease of both Depression and Anxiety. Clinical significance outcomes are similar or higher than those available in scientific literature. The analysis showed that the unwanted effects had been amenable to extra-therapy factors.

CONCLUSION: These outcomes support the effectiveness of this multi-component group intervention. Symptoms of Anxiety and Depression can be modulated and reduced by learning self-management and self-regulation skills. This short-term training offers a cost-effective tool in treating the most common and co-occurrent psychiatric disorders claimed in public health settings.

Introduction

Anxiety and Mood Disorders are among the prevalent psychiatric conditions (Mathers & Loncar 2006) and the pharmacological therapy is highly common in the treatment of these disorders (Hollingworth *et al* 2010). However, the psychopharmacological approach

has been noted to have a variable set of outcomes and particularly: the depressive symptoms show a partial reduction high around 50% (Pigott *et al* 2010) and the anxiety symptoms frequently show only a partial response either (Dell'Osso *et al* 2010). Given the prevalence of Anxiety and Depression and the noticed significant degree of partial or no response, the psy-

chological interventions are often integrated to pharmacological therapy in the treatment of these conditions. Many psychological intervention for Depression or Anxiety, such as CBT are well tested and evidence-based (Cuijpers *et al* 2013; Deacon & Abramowitz 2004).

In addition, the co-occurrence of Depression and Anxiety in clinical settings is the rule rather than the exception (Kessler *et al* 2005), following the existing high rates of comorbidity (Kaufman & Charney 2000; Kessler *et al* 1994; Lecrubier 1998a, 1998b): there are evidences that commonly patients with a main diagnosis of Anxiety Disorder also meet criteria for at least one additional Anxiety or Mood Diagnosis (DiNardo & Barlow 1990; Kessler *et al* 2010); the most recurrent comorbidity is with a Major Depressive Disorder (MDD), and 50 to 60% of the patients with a MDD diagnosis report a lifetime history of Anxiety Disorder (Kaufman & Charney 2000).

Ormel *et al* (1994) had investigated the form and the frequency of psychiatric disorders in primary care patients spread around 14 different countries, highlighting a rate of about 45% of patients with a current Anxiety disorder and a comorbid Depressive disorder; whereas the rate of patients with a Depressive disorder's diagnosis and a comorbid current Anxiety disorder was about 40%. Furthermore, the co-occurrence of Depression and Anxiety is higher than among the Anxiety Disorders different diagnosis themselves.

The clinical effects of such a high comorbidity are of an extensive nature: beside the evidence that patients with comorbid psychiatric disorders show a greater symptom severity and are expected of a chronic course of illness (Weisberg *et al* 2012), Kaufman & Charney (2000) and Kamaradova *et al* (2014) highlighted that comorbid Anxiety-Depression patients are less likely to respond to the pharmacological therapy; and Andrews *et al* (2002) show that the same comorbidity is associated with increased distress, disability and mental health service utilization.

Given all those reasons (rates of comorbidity; partial or no-response; complex clinical concerns), there is a body of evidence in favor of the efficacy and effectiveness of targeting the comorbidity in psychological intervention instead of the main diagnosis first.

Although the efficacy of diagnosis-specific anxiety treatments is very well established Norton & Price 2007), Tyrer et al (1988) showed that identical pharmacological and Cognitive Behavioral Therapies did not differentially impact patients with varied Anxiety Disorder diagnosis. Subsequently, several researchers (Moses & Barlow 2006; Norton 2006; 2008) suggest that anxiety disorders either share a common underlying factor or are superficially different manifestations of the same pathology. Out of this conceptualization a trans-diagnostic, broad-spectrum intervention has been designed to tailor treatment to the alleged core pathology underlying Anxiety disorder (Barlow et al 2004; Erickson et al 2007; Norton & Philipp 2008) that

has shown to be effective even for comorbid Anxiety-Depression disorders. In order to compare the efficacy of a trans-diagnostic CBT intervention for anxiety and depression to similar published efficacy trials, McEvoy & Nathan (2007) defined a benchmark: in a sample of 143, with 30 Anxiety disorders, 38 Depressive disorders and 75 comorbid Anxiety-Depression disorders, the clinically significant change indices were highly comparable to those obtained in methodologically similar diagnosis-specific treatment studies.

Regardless of trans-diagnostic intervention, several studies have indicated that even a specific-diagnosis psychological intervention (e.g. Generalized Anxiety Disorder – GAD) has its outcome in a significant reduction in comorbid conditions (i.e., Borkovec *et al* 1995). Overall, outcomes from studies of mixed-diagnosis groups have been encouraging (Erickson 2003; Norton & Hope 2005).

Finally it should be emphasized that treat broad diagnostic areas within which are placed different diagnoses can be notable because there is not always diagnostic agreement at a given time and the diagnosis may evolve over time.

At least two explanations are possible for why treatment of a main disorder might lead to the elimination of comorbid conditions: a) treatment generalization may occur because of client application of coping skills learned in therapy to other problems not specifically targeted by the interventions; and b) anxiety and mood disorders share certain overlapping features (restlessness, irritability, asthenia, attention deficit, sleep disorders, muscle tension, pain) in diagnostic criteria; thus, improvement on those features in the principal disorder would necessarily have an impact on determinations of the presence or severity of criterial symptoms for comorbid conditions (Brown & Barlow 1992).

A third explanation implicit in the above rationales may underlay both. Different psychological processes may be basic to many or most of the emotional disorders. If this is the case, then most of the anxiety and mood's complains share at a fundamental level the same underlying psychological mechanism (or mechanisms). Treatments that affect basic mechanism, would impact on any disorder sharing that mechanism (Borkovec 1994; Sanderson & Barlow 1990).

It should also highlighted that several potential benefits account for requiring on one program only compared to numerous disorder-specific programs: the patients with different disorders may be treated with the same program thus reducing waiting list, and the comorbid patients may concurrently learn to manage multiple disorders.

Furthermore, the implementation of psychological interventions in publically funded healthcare systems has usually very specific economic ties. As possible drawback, the prevalence of one to-one intervention rather than group intervention in public service and the related economic impact of such an intensive treat-

ments (Gunter & Whittal 2010) may prove restrictive in terms of offering any adjunct psychological intervention for the partial responder patients. Although the evidence in support of CBT as an empirically based therapy is strong, trained therapists are in short supply and the organization of treatments in many Psychiatric Units remains at best *ad hoc* (Richards & Broglin 2011).

Otherwise, there is a range of treatments overcoming some of the drawback of above and these treatments can stand as possible complementary interventions in mental health settings. For example, the use of Relaxation-based approaches has grown, as these approaches can easily be applied in group settings which may prove to be cost-effective (Borkovec & Costello 1993; Jorm et al 2008); in many case, the focus is based on teaching a specific relaxation technique (e.g., Progressive Relaxation – PR). A meta-analysis (Jorm et al 2008) revealed that in the treatment of depressive patients the most common relaxation techniques (such as PR, imagery, Autogenic Training, etc.) are more effective than none or minimal treatment; however, they were not as effective as psychological treatment, and data on clinicianrated depressive symptoms were less conclusive.

As highlighted by Jacobson & Truax (1991) in order to evaluate the effectiveness of clinical researches in naturalistic settings it is important to take into account the clinical significance of the outcomes. Clinical significance analysis gives the researchers accurate information about the patients' response to the treatment: which and how many subjects improve, recover, don't change or even worsen. Deteriorated meaning a patient's scale score had reliably moved in a negative direction during the course of therapy as judged by the Reliable Change Index (RCI) value; No change meaning a patient's scale score had not changed reliably in any direction over the course of therapy; Improved meaning a patient's scale score had reliably changed in a positive direction over the course of therapy as judged by the RCI value; and Recovered meaning a patient's scale score had improved reliably as judged by the RCI, as well as having moved from within the range of the dysfunctional distribution to within the range of the functional distribution.

In this point of view, in a study on the effectiveness of a CBT group intervention in a population of 143 heterogeneous groups McEvoy & Nathan (2007) have found 5% deteriored, 9% unchanged, 59% improved, 27% recovered on the BDI. Öst & Breitholtz (2000) in a study comparing CBT (N=18) and Applied Relaxation (N=15) as group treatments for GAD have found respectively: 56% vs 53% Improved on the HAM-A, and 67% vs 53% Improved on the Severity Rating scale. Bright & Baker (1999) have compared CBT (N=26) and Mutual Support Intervention (N=28) as group treatments for a population of depressed patients and the outcome on the BDI was respectively 3.8% Deteriored patients , 26.9% Unchanged, 69.2% Improved vs 3.6% Deteriored, 39.3% Unchanged, 57.1% Improved.

In the field of one-to-one psychotherapy (Hansen *et al* 2002) the gathered outcomes of several Clinics on the Outcome Questionnaire (OQ-45) have shown: 8.2% Deteriored (496 patients), 56.8% Unchanged (3.448 patients), 20.9% Improved (1.272 patients), 14.1% Recovered (865 patients).

Psychotherapy is not free of side effects or unwanted effects (Linden & Schermuly-Haupt 2014) but only few study have been focused on this feature of the clinical practice. Lieberman et al (1973) and Lunnen & Ogles (1998) reported a deterioration rate of 10% among clients. In their re-analysis of the National Institute of Mental Health Collaborative Depression Study, Ogles et al (1995) found that from 2% to 13% (M = 6%) of participants worsened. In a population of outpatients, Strupp et al (1969) found that Deteriorated patients were not more significantly disturbed at the beginning of the therapy than patients who subsequently had more positive outcomes. The same was true in the study of Lunnen & Ogles (1998). Of course, the finding that some patient report an increase in symptoms while in treatment it is not a sufficient indicator that the deterioration was cause by the treatment; however how Lambert & Bergin (1994) write: "The study of negative change has important implications for the selection of clients for treatment, the suitability of specific procedures for some clients, and the selection, training, and monitoring of therapists" (p. 176). In the same study is stated that it is psychotherapists' responsibility "to be sensitive to both the positive and negative effects of therapy and base our treatment efforts on a broad empirical foundation" (p. 182). Compliance with this duty is impossible without further investigations into the nature, extent, and consequences of these negative

In the light of the foregoing the aim of our study was a) to evaluate the clinical and statistical significance of a short-term Cognitive-Behavioral Group Treatment for comorbid anxious-depressive outpatients who were partially responsive to drug treatment, b) to evaluate the Deteriored patients and what variables contributed to the outcome of the clinical deterioration, and c) to test the feasibility of this intervention as complementary treatment for a psychiatric setting.

MATERIAL AND METHODS

Participants

A sample of 33 patients (9 males; 24 females) were consecutively referred to a psychiatric unit in an University Hospital in Milan, Italy.

The patients were all resident in Milan area and had a mean age of 49.50 (± 12.8 ; range = 19.9–69.4); males 50.1 (± 12.3 ; range = 37.0–69.4); female 49.3 (± 13.2 ; range = 19.9–68.11.

The total sample had two subgroups: a first subgroup with main diagnosis of Anxiety disorder (N=13), mean age of 49.3 (\pm 13.3; range = 19.9–68.2); male 48.0

(± 14.7 ; range = 37.7–58.4); female 49.7 (± 13.8 ; range = 19.9–68.2); and a second subgroup (N=20) with a main diagnosis of Depressive disorder, mean age of 49.6 (± 12.7 ; range = 26.4–69.4); male 50.8 (± 12.8 ; range = 38.11–69.4); female 48.9 (± 13.2 ; range = 26.4–68.1).

All psychiatric diagnosis were made through psychiatric interviews conducted by senior Psychiatrists independent from the current study, based on DSM-IV-TR classification, without psychiatric tests. Patients received a diagnosis of either Anxiety disorder (N=12 with Generalized Anxiety Disorder, and N=1 Adjustment Disorder with anxiety symptoms) or Depressive disorder (N=14 with Anxious Depressive Syndrome, N=5 Major Depressive Recurrent and N=1 Dysthymic Disorder).

Of the participants, 27.27% received serotonin–norepinephrine reuptake inhibitors (SRNIs), and 72.73% selective serotonin reuptake inhibitors (SSRIs). All the patients have undergone multi-component group treatment while taking SSRI or SNRI therapy. Patients were treated with conventional doses of antidepressant medication, mainly with which they were recommended to the treatment and there were no major changes in the pharmacotherapy. All patients completed the program.

Inclusion criteria

Age between 18 and 65; established diagnosis of Anxiety and Depression comorbidity, or Depression-Anxiety comorbidity, signed informed consent and partial response to pharmacological treatment (following the guidelines). All patients had received at least two cycles of drug treatment with adequate duration and dosage for each cycle as indicated by the guidelines, for a mean of 3 months prior to their referral to the psychological outpatients Unit. Patients were treated with conventional doses of antidepressant medication, mainly with which they were recommended to the treatment and there were no major changes in the pharmacotherapy. The judgment of partial responsiveness was based on previous guidelines (Lam et al 2009) and consistent with the guidelines of the I.K. National Institute for Health Care Excellence (NICE, 2009; 2011).

Exclusion criteria

Axis II (APA 2000) comorbidities, drug addiction, schizophrenia and/or medical condition.

Standardized measures included:

Symptom Checklist 90 R (SCL-90 R; Derogatis 1992): a 90 items self-report instrument evaluating a range of symptoms of psychopathology. It assesses nine symptom dimensions: Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism. The sum of the 90 items produces the Global Severity Index (GSI), a measure of overall psychological distress. The internal reliability (Cronbach α) of the scales range from 0.74 for hostility to 0.97 for the GSI (Prinz *et al*

2013). However, factor analytic studies of the Italian version have suggested that the GSI is an optimal measure for the assessment of distress symptoms (Prunas *et al* 2012).

Beck Depression Inventory (BDI; Beck et al 1961): a 21 items self-report rating inventory that assesses the clinical symptoms of depression through asking about feelings over the past week. The score is a sum of the positive answers, ranging from 0 to 63, and scores of 10 or greater reflects the presence of some level of depression. The internal reliability (Cronbach α) of the scale is between 0.73 and 0.92, and a concurrent validity between 0.55 and 0.73 for non-psychiatric subjects (Beck et al 1988).

Hamilton Depression Rating Scale (HAMD; Hamilton 1960) is a 21-item clinician-administered questionnaire used to indicate depression and evaluate recovery in adults. Scores of 8 or higher indicate depression, and a non-clinical Italian sample has been found to have a mean of 3.5 (Scimeca et al 2014). The scale has an internal reliability of between 0.46 and 0.97 (Bagby et al 2004).

Hamilton Anxiety Rating Scale (HAMA; Hamilton, 1960) is a 14-item clinician-administered questionnaire to indicate adult anxiety and recovery. Scores of 8 or higher indicate anxiety (Bjelland *et al* 2002), and a nonclinical Italian sample has been found to have a mean of 3.6 (Scimeca *et al* 2014. The scale has an internal reliability of between .74 and .96 (Bruss *et al* 1994).

Self-rating Anxiety Scale (SAS; Zung 1971): a 20 items self-report scale that assesses the complaints (primarily somatic symptoms) associated with anxiety symptoms. The respondent indicates how often he or she has experienced each symptom on a 4-point Likert scale consisting of "none or a little of the time" (coded as 1), "some of the time" (coded as 2), "good part of the time" (coded as 3), and "most or all of the time" (coded as 4). The raw total score range is 0–80. Zung (1971; 1974) proposed mean z-scores for diagnostic categories (Anxiety Disorders = 58.7; Depression = 50.7) and healthy controls (33.8). In a clinical sample the test-retest reliability range between .81 and .84 over a period of 1 to 16 weeks (Olatunji et al 2006).

<u>Intervention</u>

The program was modeled after, but not affiliated with, the clinical programs of the Benson-Henry Institute for Mind Body Medicine at the Massachusetts General Hospital (Benson 1975; 1993). The training was designed to provide tools for symptom management in outpatients. In the program, patients were taught a variety of techniques aimed at helping them with their psychological symptoms and as a self-regulatory integrated approach to stress reduction and emotion management including: psychoeducation on different topics, from stress to life-style well being; relaxation techniques (Relaxation Response-RR); mindfulness and meditative techniques; cognitive restructuring techniques. The training was

designed to be delivered in 8 sessions of two hours weekly (with a 10 min break in the middle) in a group format, plus one-hour intake assessment individual session and a one-hour post-treatment assessment individual session. Each session was run by two cotherapists: a psychologist and a physician specializing in psychiatry. During Session 1, the participants were taught about the concepts of stress, coping, and the role of breathing in helping reduce stress. After the break, there was a focus on diaphragmatic breathing, and a debriefing about the content of the session. In Session 2, there was a focus on the psychophysiology of stress and relaxation, followed by an introduction to a number of relaxation exercises, and a debriefing about the content of the session. In Session 3, there was an introduction to the psychophysiology of emotions, followed by instruction and training in a mindfulness exercise, and a debriefing about the content of the session. In Session 4, there were life-style and physical activity assessment, followed by the introduction of a meditation exercise, and a debriefing about the content of the session. In Session 5, there were lessons on life style and nutrition, followed by a contemplation exercise, and a debriefing about the content of the session. Session 6 included a stress and cognitive structuring exercises, followed by further instruction and practice of relation exercises, and a debriefing about the content of the session. Session 7 focused on resilience and protective factors related to anxiety and depression, followed by a relaxation exercise, and a debriefing about the content of the session. Finally, Session 8 dealt with resources about relapse prevention and further relaxation exercises.

Procedure

On referral to the Unit the patients were all given information on the program and the current study, been asked to sign the informed consent in accordance with the Ethical hosting Hospital Policy and Approval. During the intake assessment session, the patients were all given a battery of psychological tests, including the SCL-90 R, BDI, HAM-A.HAM-D, and SAS. Patients then participated in the eight-week multi-component program, as detailed above, receiving one 2-hour group-based session a week for 8 weeks. Following completion of the intervention, the patients were again asked to complete the tests during the final assessment session and had been debriefed regarding the study. A three-months follow up data-gathering is currently on going.

Statistical analysis

All statistical analyses were performed with the software OriginPro 8 SR0. The statistics test for dependent data was Wilcoxon Signed Rank Test and Bonferroni adjustment, when appropriate. To compare the two subgroups (Anxiety and Depression) was used the Mann-Whitney test. Effect sizes (d; Cohen 1988) following Richard & Borglin formula (2011) with a con-

servative result. Analyses of clinically significant change were conducted according to Jacobson & Truax (1991). This method evaluates two criteria for each participant. The first criterion or index (Reliable Change Index, RCI) is whether each participant's score improves such that it is unlikely to be due to the inherent unreliability of the measure. A participant is considered to have experienced reliable change if his or her RCI is greater than 1.96 (Jacobson *et al* 1999). By this procedure, we could also assess Deteriored patients. The second criterion (Clinical Significance Index, CSI) evaluated, in those patients with a reliable change, whether their post-treatment symptom level sets within the "normal" range for this measure. Exactly:

BDI: RCI has been assessed. We also compute the CSI on the reliable data (cut-off score in BDI=15; Gibbons *et al* 2010). Because clinically significant change requires an above-cut-off individual-score at pretreatment and a below-cut-off individual-score at post-treatment, (according to Gibbons *et al* 2010), we could only include in this analysis those patients who had a BDI score \geq 15 at pre-treatment.

SAS: RCI in SAS has been computed following the same procedure. Differently, in calculating CSI, we've got different SDs (Standard Deviation) for males and females because of the statistical significant differences in the validation study's normative data (Olatunji *et al* 2006).

Two different cut-off for males and females have been computed following the Wise formula (2004): males ≤ 34.45 ; females ≤ 38.12 .

SCL-90 R: Following Wise (2004), RCI in SCL-90 R had been computed on the GSI data (Global Severity Index); in this case, CSI cut-off ≤0.93 (Wise 2004).

HAM-D: RCI has been computed following Bagby *et al* (2004); CSI cut-off \leq 7 (Zimmerman *et al* 2004): none of the patients was \leq 7 at pretest.

HAM-A: RCI has been computed following Shear *et al* (2011). CSI cut-off \leq 7 (Zimmerman *et al* 2004) none of the patients had been assessed \leq 7 at pre-test.

RESULTS

Table 1 shows the means and standard deviations for overall symptoms (SCL-90 R), depression and anxiety scores for the overall sample pre and post treatment, as well as percentage of improvement (pre vs post), Wilcoxon Signed Ranks Test z-scores with Bonferroni's adjustment (except for BDI and SCL-90 R) with related statistical significance, and the effect size.

For the total sample all the scales show significant statistical differences in pre-post data.

Table 2 show means and standard deviations for overall symptoms (SCL-90 R), depression (BDI and HAMD), and anxiety (SAS and HAMD) for the anxious and depressed subgroups pre and post treatment, as well as Wilcoxon Signed Ranks Test (subgroup pre vs post).

Tab. 1. Mean (standard deviations) for overall symptoms (SCL-90 R), depression (BDI and HAMD), and anxiety (SAS and HAMD) for the sample pre and post treatment, as well as percentage of improvement (pre vs post), Wilcoxon Signed Ranks Test with Bonferroni's adjustment (except for BDI and R-90 R Overall), and the effect size (d).

Scale	Pre	Post	% improv.	Wilcoxon z	d
SCL-90 R Overall	93.15 (34.42)	60.94 (34.97)	34.58	4.30***	0.9
Depression (BDI)	14.48 (6.96)	11.42 (7.77)	21.13	2.69**	0.4
Depression (Hamilton)	18.91 (5.17)	12.61 (5.14)	33.31	4.86***	1.22
Anxiety (SAS)	40.60 (8.98)	37.07 (7.45)	8.69	2.90**	0.5
Anxiety (Hamilton)	17.48 (4.85)	11.48 (5.09)	34.32	4.21***	1.18

^{**}p<0.01; ***p<0.001

No detectable significant difference between the two diagnostic subgroups, Anxiety and Depression, on each scale, both the pretest and post-test.

Table 3 shows instruments (total sample N=33; BDI N=17 because only patients with BDI initial score \geq 15 were included in the analysis), clinical significance levels with number of subjects and respective percentage. Percentage was setup following these criteria: for Improved patients on the total sample; for the Recovered patients on the number of Improved only.

Table 4 shows Scales and Clinical significance levels (number of subjects and percentage) in each subgroup (Anxiety and Depression).

Discussion

The results demonstrated that the treatment had good patient acceptability, with none of the cohort dropping out of the treatment program.

For the total sample there was a reduction in overall levels of symptoms measured by the SCL-90 R, and patients showed improvement in their depression and anxiety. The effect sizes was large for HAMA, HAMD and SCL-90 R; medium for BDI and SAS.

It was not found significant statistical difference between the two diagnostic subgroups: Anxiety and Depression. Taking into account that negative affectivity and psychological and biological vulnerabilities are shared by anxiety and affective disorders (Clark & Watson 1991), then treatments addressing commonalities could be more efficient and effective. The outcomes of this study support the hypothesis that the intervention addressed common transdiagnostic factors of the two patients' subgroups.

Clinical significance outcomes for the total sample are similar or higher than those available in scientific literature, especially on SCL-90-R, HAMD and HAMA.

Clinical significance appears to be of a greater value for the subgroup of depressed patients, even though it is of notable effects for anxious patients too. The reasons for this pattern of results require further exploration, but may include the social support offered to individuals in group sessions, which is known to help alleviate depressive symptoms (Cohen & Wills 1985).

Outcomes are different on each given scale of the study because scale's specific contents are often not overlapping and different measures of different domains will produce different RCI classification results (Beckstead *et al* 2003). Furthermore, no all scales have the same reliability, affecting the RCI on that scale: the less reliable the instrument, the greater the difference required to achieve a statistically reliable change (Wise 2004).

Deteriored patients' total number was small: nevertheless, the qualitative analysis showed that unwanted effects had been amenable to extra-therapy factors (marital, familiar or job conflicts emerged during the treatment).

Tab. 2. Mean (standard deviations) for overall symptoms (SCL-90 R), depression (BDI and HAMD), and anxiety (SAS and HAMD) for the anxious and depressed sub groups pre and post treatment, as well as Wilcoxon Signed Ranks Test (sub group pre vs post) and effect size (d).

	Depression sub group				Anxiety sub group			
	Pre	Post	Wilcoxon z	d	Pre	Post	Wilcoxon z	d
SCL-90 R Overall	90.25 (33.16)	53.00 (28.25)	3.60***	1.32	97.62 (37.19)	73.15 (41.60)	2.38*	0.6
Depression (BDI)	14.95 (7.84)	13.35 (8.73)	1.47	0.2	13.77 (5.56)	8.46 (4.98)	2.32*	1.07
Depression (Hamilton)	18.75 (5.56)	12.20 (6.04)	3.78***	1.08	19.15 (4.71)	13.23 (3.47)	3.08***	1.71
Anxiety (SAS)	40.82 (7.99)	37.06 (5.83)	2.56**	0.6	40.31 (6.13)	37.08 (6.54)	1.45	0.49
Anxiety (Hamilton)	17.05 (5.65)	10.20 (4.77)	3.63***	1.44	18.15 (3.36)	13.46 (5.09)	2.13*	0.92

^{*}p<0.05; **p<0.01; ***p<0.001

There can be little doubt that traditional RCI recovery rates are extremely conservative psychotherapy outcome measures and that the scores shifted into the functional range stand for "unequivocally treatment (...) success[es]"(Jacobson *et al* 1984; p.498).

In summary, the clinical effect of this group treatment can then be evaluated in positive terms. The multi-component program was further successful with those patients who previously shown little improvement with pharmacological intervention. Positive outcomes are unlikely to be effected by the pharmacological treatments, because the patients have been under the same drug treatment dosage three months previously and during the whole group treatment time. That the training was successful with patients who had previously demonstrated little change in their symptomatology through the use of pharmacological interventions is encouraging, and adds to the number of psychological supports that may be considered for this patient group.

These outcomes support the effectiveness of this brief multi-component intervention. Symptoms of Anxiety and Depression can be modulated and reduced by learning self-management and self-regulation skills. This short-term training offers a cost-effective tool in treating the most common and co-occurrent psychiatric disorders claimed in public health settings.

Study limitation

There are some limitations to the current study that need to be noted. The first limitation is the lack of a control group. However, the fact that patients had no shown improvement before their involvement in the program, suggests that it might be useful to integrate it to the pharmacological approach. Also reassuring is the fact that much evidence (Butler *et al* 2006) indicate the superiority of CBT treatment compared to placebo or waiting-list. However, this study was observational in nature and the gathering of a data group control is a future research goal. The data gathering is still ongo-

ing, so that the longitudinal effectiveness outcomes are going to be available.

Other feature to highlight is that clinician-rated scales' outcomes are higher than self-report outcomes: in the area of depressive disorder the agreement between self-reported and clinician-rated measures of depression severity is far from perfect, even though there is a correlation rated from moderate to strong between clinician-rated scales and self-reported questionnaires (Cameron et al 2011; Cuijpers et al 2010; Domken et al 1994). Uher et al (2012) highlighted that self-report and clinician-rated outcomes are not equivalent, each of the two providing unique information that is relevant to clinical analysis. The most accurate prediction of outcome can be achieved when both, clinician and selfrating assessments, are available. It could be assumed that this is the case for the Anxiety Disorders as well (Sartorius et al 1990).

Finally the sample size when patients are split into two diagnostic classes is small, and so the results should be interpreted with caution.

Tab. 3. Scale (sample size) and clinical significance levels (number of participants and percentage).

	Deteriorated	No Change	Improved*	Recovered**
SCL-90 R	3	6	24	22
(N=33)	(9.09%)	(18.18%)	(72.73%)	(91.67%)
HAMD	1	11	21	4
(N=33)	(3.03%)	(33.33%)	(63.64%)	(19.08%)
BDI	2	6	9	8
(N=17)	(11.76%)	(35.29%)	(52.94%)	(88.89%)
HAMA	2	13	18	8
(N=33)	(6.06%)	(39.39%)	(54.54%)	(44.44%)
SAS	2	22	9	8
(N=33)	(6.06%)	(66.67%)	(27.27%)	(88.89%)

^{*} Percentage was setup on the total sample

Tab. 4. Scale and clinical significance levels (number of participants and percentage) in each subgroup (Anxiety and Depression).

	Depression sub group (N=20)				Anxiety sub group (N=13)			
	Deterior.	Unchang.	Improve	Recover.	Deterior.	Unchang.	Improve	Recover.
SCL-90 R	1	4	15	14	2	2	9	8
	(5%)	(20%)	(75%)	(93.3%)	(15.4%)	(15.4%)	(69.2%)	(88. 9%)
HAMD	1	4	15	4	0	5	8	1
	(5%)	(20%)	(75%)	(26.7%)	(0%)	(38.5%)	(61.5%)	(12.5%)
BDI *	2	3	6	4	2	0	4	4
	(18.2%)	(27.3%)	(54.5%)	(66.7%)	(33.3%)	(0%)	(66.7%)	(100%)
НАМА	1	6	13	7	2	6	5	1
	(5%)	(30%)	(65%)	(53.8%)	(15.4%)	(46.1%)	(38.5%)	(20%)
SAS	0	15	5	5	2	7	4	4
	(0%)	(75%)	(25%)	(100%)	(15.4%)	(53.8%)	(30.8%)	(100%)

^{*}BDI Depression sub group N=11; BDI Anxiety sub group N=6; because only participants who began treatment with a BDI score of 15 or above were included in this analysis

^{**} Percentage was setup on the number of Improved only

REFERENCES

- American Psychiatric Association (2000). Diagnostic and statistical manual of mental disorders. 4th ed. Washington, DC. Text rev.
- 2 Andrews G, Slade T, Issakidis C (2002). Deconstructing current comorbidity: data from the Australian national survey of mental health and well-being. *Brit J Psychiat.* 181: 306–314.
- 3 Bagby RM, Ryder AG, Schuller DR, Marshall MB (2004). The Hamilton Depression Rating Scale: has the gold standard become a lead weight? Am J Psychiat. 161: 2163–2177.
- 4 Barlow DH, Allen LB, Choate ML (2004). Toward a unified treatment for emotional disorders. Behav Ther. 35: 205–230.
- 5 Beck AT, Steer RA, Garbin MGJ (1988). Psychometric properties of the Beck Depression Inventory Twenty-five years of evaluation. Clin. Psych. Review. 8: 77–100.
- 6 Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J (1961). An inventory for measuring depression. Arch Gen Psychiat. 4: 561–571.
- 7 Beckstead DJ, Hatch AL, Lambert MJ, Eggett DL, Goates MK, Vermeersch DA (2003). Clinical significance of the Outcome Questionnaire (OQ-45.2). Behav Anal Today. 4(2): 79-90.
- 8 Benson H (1975). The relaxation response. New York: Morrow. ISBN: 0688029558, 158 p.
- 9 Benson H & Stuart E (1993). The Wellness Book. New York: Fireside, ISBN 978-0671797508, 512 p.
- 10 Bjelland I, Dahl AA, Haug TT, Neckelmann D (2002). The validity of the Hospital Anxiety and Depression Scale: an updated literature review. J Psychos Res. 52(2): 69–77.
- 11 Borkovec TD (1994). The nature, functions, and origins of worry. In Davey GCL, Tallis F, editors. Worrying: perspectives on theory, assessment and treatment. Wiley series in clinical psychology. Oxford, England: Wiley, p. 5–34.
- 12 Borkovec TD, Abel JL, Newman H (1995). Effects of Psychotherapy on Comorbid Conditions in Generalized Anxiety Disorder. J Consult Clin Psychol. 63(3): 479–483.
- 13 Borkovec TD & Costello E (1993). Efficacy of applied relaxation and cognitive behavioral therapy in the treatment of generalized anxiety disorder. *J Consult Clin Psychol.* **61**(4): 611–619.
- 14 Bright JI & Baker KD (1999). Professional and Paraprofessional Group Treatment for Depression: A Comparison of Cognitive-Behavioral and Mutual Support Interventions. *J Consult Clin Psychol.* **67**(4): 491–501.
- 15 Brown TA & Barlow DH (1992). Comorbidity among anxiety disorders: Implications for treatment and DSM-IV. J Consult Clin Psychol. 60: 835–844.
- 16 Bruss GS, Gruenberg AM, Goldstein RD, Barber JP (1994). Hamilton anxiety rating scale interview guide: joint interview and test-retest methods for interrater reliability. *Psychiat Res.* 53: 191–202.
- 17 Butler AC, Chapman JE, Forman EM, Beck AT (2006). The empirical status of cognitive-behavioral therapy. A review of meta-analyses. Clin Psychol Rev. 26: 17–31.
- 18 Cameron IM, Cardy A, Crawford JR, du Toit SW, Hay S, Lawton K, et al (2011). Measuring depression severity in general practice: discriminatory performance of the PHQ-9, HADS-D, and BDI-II. Br J Gen Pract. 61: 419–426.
- 19 Clark LA & Watson D (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. J Abnorm Psych. 103: 103–116.
- 20 Cohen J (1988). Statistical power analysis for the behavioral sciences (2nd ed). Psychology Press (New York, USA), ISBN 978-0805802832, 590 p.
- 21 Cohen S & Wills TA (1985). Stress, social support, and the buffering hypothesis. *Psychol Bull.* 98(2): 310.
- 22 Cuijpers P, Berking M, Andersson G, Quigley L, Kleiboer A, Dobson KS (2013). A meta-analysis of cognitive-behavioural therapy for adult depression, alone and in comparison with other treatments. Can J Psychiat. 58(7): 376–385.
- 23 Cuijpers P, Li J, Hofmann SG, Andersson G (2010). Self-reported versus clinician-rated symptoms of depression as outcome measures in psychotherapy research on depression: a meta-analysis. Clin Psychol Rev. 30: 768–778.

- 24 Deacon BJ & Abramowitz JS (2004). Cognitive and Behavioral Treatments for Anxiety Disorders: A Review of Meta-analytic Findings. J Clin Psychol. 60: 429–441.
- 25 Dell'Osso B, Buoli M, Baldwin DS, Altamura AC (2010). Serotonin norepinephrine reuptake inhibitors (SNRIs) in anxiety disorders: a comprehensive review of their clinical efficacy. *Hum Psycho-pharmacol.* 25(1):17–29.
- 26 Derogatis LR (1992). SCL-90-R, administration, scoring and procedures manual-II for the Revised version and other instruments of the Psychopathology Rating Scale Series. Townson: Clinical Psychometric Research.
- 27 DiNardo PA & Barlow DH (1990). Syndrome and symptom comorbidity in the anxiety disorders. In Maser JD, Cloniger CR, editors. Comorbidity in anxiety and mood disorders Washington, DC: American Psychiatric Press, p. 205–230.
- 28 Domken M, Scott J, Kelly P (1994). What factors predict discrepancies between self and observer ratings of depression? J Affect Disord. 31: 253–259.
- 29 Erickson DH (2003). Group cognitive behavioural therapy for heterogeneous anxiety disorders. Cogn Behav Ther. 32: 179–186.
- 30 Erickson DH, Janeck A, Tallman K (2007). Group cognitive-behavioral group for patients with various anxiety disorders. *Psychiat Serv.* 58: 1205–1211.
- 31 Gibbons CJ, Fournier JC, Stirman SW, DeRubeis RJ, Crits-Christoph P, Beck AT (2010). The Clinical Effectiveness of Cognitive Therapy for Depression in an Outpatient Clinic. *J Affect Disord.* **125**:169–176.
- 32 Gunter RW & Whittal ML (2010). Dissemination of cognitivebehavioral treatments for anxiety disorders: Overcoming barriers and improving patient access. Clin Psychol Rev. 30: 194–202.
- 33 Hamilton M (1960). A rating scale for depression. J Neurol Neurosur Ps. 23: 56–62.
- 34 Hamilton M (1969). Diagnosis and rating of anxiety. *Br J Psychiat*. **3**: 76–79.
- 35 Hansen NB, Lambert MJ, Forman EM (2002). The Psychotherapy Dose-Response Effect and Its Implications for Treatment Delivery Services. Clin Psychol Sci Prac. 9: 329–343.
- 36 Hollingworth SA, Burgess PM, Whiteford HA (2010). Affective and anxiety disorders: prevalence, treatment and antidepressant medication use. Aust NZ J Psychiat. 44: 513–519.
- 37 Jacobson NS, Follette WC, Revenstorf D, Baucom DH, Hahlweg K, Margolin G (1984). Variability in outcome and clinical significance of behavioral marital therapy: A re-analysis of outcome. J Consult Clin Psychol. 52: 497–504.
- 38 Jacobson NS, Roberts LJ, Berns SB, McGlinchey JB (1999). Methods for determining the clinical significance of treatment effects: Description, application and alternatives. J Consult Clin Psychol. 67: 300–307.
- 39 Jacobson NS & Truax P (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. J Consult Clin Psychol. 59: 12–19.
- 40 Jorm AF, Morgan AJ, Hetrick SE (2008). Relaxation for depression. Cochrane Database Sys Rev. 4: CD007142.
- 41 Kamaradova D, Prasko J, Sandoval A, Latalova K (2014). Therapeutic response to complex cognitive-behavioral and pharmacological treatment in patients with social phobia. *Act Nerv Super Rediviva*. **56**(3–4): 91–99.
- 42 Kaufman J & Charney D (2000). Comorbidity of mood and anxiety disorders, *Depress Anxiety*. **12**(1): 69–76.
- 43 Kessler RC, Chiu WT, Demler O, Walters EE (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiat*. **62**(6): 617–627.
- 44 Kessler RC, McGonagle KA, Zhao S, Nelson CB, Hughes M, Eshleman S, et al (1994). Lifetime and 12-Month Prevalence of DSM-III-R Psychiatric Disorders in the United States. Results From the National Comorbidity Survey. Arch Gen Psychiat. **51**: 8–19.
- 45 Kessler RC, Ruscio AM, Shear K, Wittchen HU (2010). Epidemiology of anxiety disorders. Curr Top Behav Neurosci. 2: 21–35.

- 46 Lam RW, Kennedy SH, Grigoriadis S, McIntyre RS, Milev R, Ramasubbu R, et al (2009). Canadian Network for Mood and Anxiety Treatments (CANMAT) clinical guidelines for the management of major depressive disorder in adults. III. Pharmacotherapy. J Affect Disord, 117 (Suppl 1): S26–S43.
- 47 Lambert MJ & Bergin AE (1994). The effectiveness of psychotherapy. In Bergin AE, Garfield SL, editors. Handbook of psychotherapy and behavior change (4th ed.). New York: Wiley, p. 143–189
- 48 Lecrubier Y (1998a). The impact of comorbidity on the treatment of panic disorder. *J Clin Psychiat*. **59**(8): 11–14.
- 49 Lecrubier Y(1998b). Comorbidity in social anxiety disorder: impact on disease burden and management. *J Clin Psychiat*. **59**(Suppl 17): 33–37.
- 50 Lieberman MA, Yalom ID, Miles MB (1973). Encounter groups: First facts. New York: Basic Books, ISBN 978-0465019687, 496 p.
- 51 Linden M & Schermuly-Haupt M (2014). Definition, assessment and rate of psychotherapy side effects. *World Psychiat.* **13**(3): 306–309.
- 52 Lunnen KM & Ogles BM (1998). A Multiperspective, Multivariable Evaluation of Reliable Change. J Consult Clin Psychol. 66(2): 400–410.
- 53 Mathers CD & Loncar D (2006). Projections of global mortality and burden of disease from 2002 to 2030. Plos Med. 3(11): 2011–2031.
- 54 McEvoy PM & Nathan P (2007). Effectiveness of Cognitive Behavior Therapy for Diagnostically Heterogeneous Groups: A Benchmarking Study. J Consult Clin Psychol. 75: 344–350.
- 55 Moses EB & Barlow DH (2006). A new unified treatment approach for emotional disorders based on emotion science. Curr Dir Psychol Sci. 15: 146–150.
- 56 NICE clinical guideline 90 (2009). Depression in adults. www. nice.org.uk/cg90
- 57 NICE clinical guideline 113 (2011). Generalised anxiety disorder and panic disorder (with or without agoraphobia) in adults. www.nice.org.uk/cg113
- 58 Norton PJ & Hope DA (2005). Preliminary evaluation of a broadspectrum cognitive– behavioral group therapy for anxiety. J Behav Ther Exp Psychiatry. 36:79–97.
- 59 Norton PJ & Price EP (2007). A meta-analytic review of cognitivebehavioral treatment outcome across the anxiety disorders. J Nerv Ment Dis. 195: 521–531.
- 60 Norton PJ (2006). Toward a clinically-oriented model of anxiety disorders. *Cogn Behav Ther.* **35**: 88–105.
- 61 Norton PJ (2008). An open trial of a transdiagnostic cognitive-behavioral group therapy for anxiety disorder. *Behav Ther.* **39**: 242–250.
- 62 Norton PJ & Philipp LM (2008). Transdiagnostic approaches to the treatment of anxiety disorders: A metaanalytic review. *Psychot: Theor, Res, Pract, and Train.* **45**: 214–226.
- 63 Ogles BM, Lambert MJ, Sawyer JD (1995). Clinical significance of the National Institute of Mental Health treatment of depression collaborative research program data. J Consult Clin Psycho. 63: 321–326.
- 64 Olatunji BO, Deacon BJ, Jonathan S, Abramowitz JS, Tolin DF (2006). Dimensionality of somatic complaints: Factor structure and psychometric properties of the Self-Rating Anxiety Scale. *J Anxiety Disord.* **20**: 543–561.
- 65 Ormel J, Vonkorff M, Ustun TB, Pini S, Korten A, Oldehinkel T (1994). Common mental disorders and disability across cultures. Results from the WHO Collaborative Study on Psychological Problems in General Health Care. JAMA. 272(22): 1741–1748.

- 66 Öst L & Breitholtz E (2000). Applied relaxation vs. cognitive therapy in the treatment of generalized anxiety disorder. *Behav Res Ther.* **38**: 777–790.
- 67 Pigott HE, Leventhal AM, Alter G, Boren J (2010). Efficacy and effectiveness of antidepressants: current status of research. *Psychother Psychosom.* **79**(5): 267–279.
- 68 Prinz U, Nutzinger DO, Schulz H, Petermann F, Braukhaus C, Andreas S (2013). Comparative psychometric analyses of the SCL-90-R and its short versions in patients with affective disorders. BMC Psychiatry. 13: 104.
- 69 Prunas A, Sarno I, Preti E, Madeddu F, Perugini M (2012). Psychometric properties of the Italian version of the SCL-90-R: A study on a large community sample. Eur Psychiat. 27: 591–597.
- 70 Richards DA & Borglin G (2011). Implementation of psychological therapies for anxiety and depression in routine practice: Two year prospective cohort study. *J Affect Disord.* **133**: 51–60.
- 71 Sanderson WC & Barlow DH (1990). A description of patients diagnosed with *DSM-III-R* generalized anxiety disorder. *J Nerv Ment Dis.* **178**: 588–591.
- 72 Sartorius N, Andreoli VM, Cassano G, Eisemberg L, Kielholz P (1990). Anxiety. Psychobiological & Clinical Perspectives. London: Taylor & Francis, ISBN 978-1560320647, 300 p.
- 73 Scimeca G, Bruno A, Cava L, Pandolfo G, Muscatello MRA, Zoccali R (2014). The Relationship between Alexithymia, Anxiety, Depression, and Internet Addiction Severity in a Sample of Italian High School Students. Sci World J. 2014: 1–8.
- 74 Shear MK, Vander Bilt J, Rucci P, Endicott J, Lydiard B, Otto MW, et al (2011). Reliability and Validity of a Structured interview Guide for Hamilton Anxiety Rating Scale (Sigh-A). Depress Anxiety. 13(4): 166–178.
- 75 Strupp, HH, Fox R, Lessler K (1969). Patients view their own psychotherapy. Baltimore, MD: Johns Hopkins University Press.
- 76 Tyrer PJ, Seivewright N, Murphys S, Ferguson B, Kingdon D, Barczak B, et al (1988). The Nottingham study of neurotic disorder: Comparison of drug and psychological treatments. Lancet. 2: 235–240.
- 77 Uher R, Perlis RH, Placentino A, Dernovšek MZ, Henigsberg N, Mors O, et al (2012). Self-report and clinician-rated measures of depression severity: can one replace the other? *Depress Anxiety*. **29**(12): 1043–1049.
- 78 Weisberg RB, Beard C, Dyck I, Keller MB (2012). The Harvard/ Brown Anxiety Research Project-Phase II (HARP-II): Rationale, methods, and features of the sample at intake. J Anxiety Disord. 26(4): 532–543.
- 79 Wise EA (2004). Methods for Analyzing Psychotherapy Outcomes: A Review of Clinical Significance, Reliable Change, and Recommendations for Future Directions. *J Pers Assess*, **82**(1): 50–59
- 80 Zimmerman M, Chelminski I, Posternak M (2004). A Review of Studies of the Hamilton Depression Rating Scale in Healthy Controls. Implications for the Definition of Remission in Treatment Studies of Depression. *J Nerv Ment Dis.* **192**: 595–601.
- 81 Zung WWK (1971). A rating instrument for anxiety disorders. *Psychosomatics*. **12**: 371–379.
- 82 Zung WWK (1974). The measurement of affect: Depression and anxiety. In Pichot P, Olivier-Martin R, editors. Psychological Measurement in Psychopharmacology, vol.7. Basel: Karger, p. 170–188.