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Brief report

Changes in well-being and quality of life in a randomized trial comparing dynamic psychotherapy and pharmacotherapy for major depressive disorder



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ABSTRACT

Background: Major depressive disorder (MDD) is associated with a decrease in quality of life (QOL) and well-being. Therefore, researchers are increasingly complementing traditional symptom measurements with QOL and well-being assessments in order to broaden the evaluation of treatment outcomes. The current prospective study investigated the effectiveness of supportive–expressive therapy (SET), anti-depressant medication (MED) and placebo (PBO) in improving QOL and well-being in patients with MDD. **Methods:** Data from a randomized controlled trial (trial registration: NCT00043550) comparing SET, MED and PBO for the treatment of depression ($N=156$) were analyzed. Outcome measures addressed patients' QOL and physical and mental well-being. Changes in outcomes were assessed across and between treatments using linear mixed models.

Results: Across treatments, patients showed significant improvement in QOL and mental and physical health measures, as well as a reduction in interpersonal distress and depressive and anxiety symptoms ($p \leq .002$ for all measures). Those changes were not only the products of a decrease in depressive symptoms, but also predicted subsequent reduction in symptoms. No significant differences were found between the three treatment conditions.

Limitations: The limitation is the study's moderate sample size.

Conclusions: Current treatments for depression significantly improve patients' QOL and well-being. No significant differences were found between the three conditions examined in this study. The current study highlights the role of well-being in predicting subsequent symptomatic change.

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1. Introduction

Major depressive disorder (MDD) has a lifetime prevalence of approximately 10%, and is currently the 4th leading cause of disability worldwide (Kessler, 2012). Depression is associated with a decrement in health that is significantly greater than that associated with other chronic diseases (Moussavi et al., 2007). More than 60% of patients with MDD have a clinically significant impairment in their quality of life (QOL) (Rapaport et al., 2005, Zeng et al., 2013). Medi-

cation (Fournier et al., 2010), psychodynamic psychotherapy (Barber et al., 2012), and even placebo (Walsh et al., 2002) have been shown to effectively reduce depressive symptoms. However, not to be overlooked is the ability of such treatments to increase QOL and the ability to actively participate in society.

In a recent randomized controlled trial (RCT) (Barber et al., 2012), supportive–expressive therapy (SET), antidepressant medication (MED) and placebo (PBO) were found to be equally effective in reducing depressive symptoms when treating patients with MDD. Taking into account the substantial effect of MDD on quality of life (Rapaport et al., 2005, Zeng et al., 2013), and the growing interest in complementing traditional symptom measures with additional QOL measures when evaluating treatment effectiveness (Ishak et al., 2011), we examined whether SET, MED and PBO in this setting had a significant effect on QOL and mental and physical well-being throughout treatment. In addition, we aimed

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to evaluate whether outcomes differed among the three treatment conditions. We further examined whether these measures are the products or predictors of changes in depressive symptoms.

2. Methods

2.1. Participants

Patients diagnosed with MDD were randomly assigned to one of three treatment conditions: SET, MED, or PBO ($N=156$). Details regarding inclusion criteria and study procedures have been previously reported (Barber et al., 2012). The mean age was 37.5 ($SD=12.2$) and 92 participants (59%) were female. Approximately half (48%) of the patients were Caucasian, 45% were African Americans, and the rest were Latino (5%) or Asian (2%). At intake 84.5% of patients had at least one comorbid Axis I disorder. Comorbidities included anxiety disorders (44.9%) and current substance abuse or past dependence disorder (35.3%). In addition, 46.2% had a comorbid Axis II personality disorder. The study (clinicaltrials.gov identifier: NCT00043550) was approved by the Institutional Review Board, and all patients gave their informed consent in writing prior to screening.

2.2. Treatments

Treatments were provided for a total of 16 weeks. Patients treated with SET ($N=51$) received 20 sessions of manualized psychodynamic therapy for depression (Luborsky et al., 1995) twice weekly during the first 4 weeks of treatment and weekly for the remainder of treatment. Treating psychotherapists had a minimum of 10 years of psychotherapy experience in SET. In the medication (MED; $N=55$) and placebo (PBO; $N=50$) conditions, patients received either Sertraline or placebo delivered by experienced psychopharmacologists using a manualized clinical management model (Fawcett et al., 1987); non-responders were switched to Venlafaxine (MED) or to a second placebo (PBO) after 8 weeks. Patients in the MED and PBO were seen weekly for the first 6 weeks, after which they were seen every 2 weeks at the discretion of the treating psychopharmacotherapist.

2.3. Measures

Secondary outcomes included anxiety and depressive symptoms as well as well-being status, quality of life and interpersonal distress. Anxiety severity was measured with the structured interview guide for the Hamilton Anxiety Rating Scale (HARS; Hamilton, 1959) and with the Beck Anxiety Inventory 21-item self-report measure (BAI; Beck et al., 1988a). Severity of depressive symptoms was measured with the Beck Depression Inventory, a 21-item self-report measure (BDI; Beck et al., 1988b). Mental and physical health was measured with the Medical Outcomes Study 36-item Health Survey (Ware, Sherbourne 1992), which yields two summary scores developed from the original measure: the general mental health component score (MCS) and the physical health component score (PCS). Interpersonal distress was measured with the 64-item version of the Inventory of Interpersonal Problems (Horowitz et al., 2000), with the mean of all items indicating the general level of interpersonal distress. Quality of life was measured with the 93-item Quality of Life Enjoyment and Satisfaction Questionnaire (QLESQ; Endicott et al., 1993), which yields indices for various areas of functioning (i.e., physical health, mood, interpersonal relationships, household activities, and ability to complete work/hobbies). In addition, the 17-item version of the clinician-administered Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967) was used to examine the temporal

relationship between the HRSD that serves as the primary outcome measure in treatments for depression and our secondary outcomes. All outcome measures were evaluated at six time points (week 0, 4, 6, 8, 12 and 16), excluding interpersonal distress, which was examined at three time points (week 0, 8 and 16).

2.4. Statistical analyses

All analyses were carried out on an intention-to-treat basis, which included all participants randomized for the trial. Baseline differences in demographic and clinical characteristics between conditions were investigated using a one-way analysis of variance (ANOVA) for continuous variables and χ^2 tests of independence/Fisher exact test for categorical variables.

Outcomes were analyzed using linear mixed models (SAS PROC MIXED), (Littell et al., 2006), which take into account the unbalanced effect caused by missing observations. The analysis of each outcome consisted of two longitudinal models. The first model examined linear trends in the outcome variables over time for the entire sample (SET, MED, and PBO). The second model examined differences in this trend over time between type of treatments in predicting outcomes and included a variable for time, type of treatment (a three-category variable of condition), and the treatment \times time interaction. Significant interactions were further investigated for specificity. We assumed random effects for both subject intercept and time effect. Because gender and minority status were found to be related to Hamilton Rating Scale for Depression symptom scores in the main outcome study, we repeated the data analyses controlling for gender and minority status. The sample enabled the detection of a medium effect size (.48) with a power $>80\%$ when comparing MED or SET to PBO over the longitudinal period (Diggle et al., 1994). Within- and between-group effect sizes were computed as d , the standardized mean difference, and defined as small ($d=.20$), medium ($d=.50$), and large ($d=.80$; Cohen, 1988). Pattern-mixture models (Hedeker and Gibbons, 1997) were implemented to assess whether estimates per the linear mixed models were dependent on missing data patterns (for more details, see Gallop and Tasca, 2009).

Finally, to examine the bidirectional association between HRSD and the secondary outcome measures over time, we employed autoregressive cross-lagged modeling (ARCL; e.g., Collins and Sayer, 2001; Zilcha-Mano et al., in press). In this ARCL model, HRSD and each one of the secondary outcomes were introduced in Level 1 of the models (within-subject) along with the dependent variable at the subsequent time (either HRSD or the secondary outcome, depending on the model).

3. Results

A total of 156 patients were included in the outcomes analyses. No differences between conditions were found for baseline demographics or clinical characteristics. Table 1 describes the multilevel models for changes in outcomes throughout time, showing improvement in all areas examined. Specifically, patients showed improvement in quality of life (across all the seven subscales, $p<.0001$), mental health ($p<.0001$) and physical health ($p<.002$), as well as reductions in interpersonal distress ($p<.0001$), depressive symptoms ($p<.0001$) and anxiety symptoms (the latter were examined using both self-report and clinician-rated measures, $p<.0001$ for both measures).

Similar to the main outcome study, significant treatment differences in the time trends failed to be found for interpersonal distress, depressive and anxiety symptoms, and mental and physical health (all $ps \geq .09$, see Table 2). For quality of life, significant differences were only found for the Work subscale, in

Table 1
Changes in Outcome Measures across Treatments.

Outcome	Mean Pre-treatment (SD)			Mean Post-treatment (SD)			F	Df	P	Estimate (S.E.)	Pre-post effect size		
	MED	SET	PBO	MED	SET	PBO					MED	SET	PBO
MOS													
Physical component score	70.52 (18.70)	56.28 (21.25)	62.64 (21.80)	66.59 (18.70)	71.03 (21.25)	70.70 (21.80)	9.50	1.130	<i>P</i> = .002	.40 (.13)	-.21	.69	.36
Mental component score	23.91 (13.04)	26.37 (15.15)	26.77 (13.58)	50.54 (13.04)	62.05 (15.15)	53.80 (13.58)	80.1	1.130	<i>P</i> < .0001	1.72 (.19)	2.04	2.35	1.99
IIP	92.15 (30.78)	107.98 (35.99)	82.16 (36.65)	87.30 (30.78)	73.72 (35.99)	70.44 (36.65)	25.91	1.95	<i>P</i> < .0001	-.97 (.19)	-.15	-.95	-.31
Q-les-Q													
Physical health	.43 (.13)	.35 (.17)	.38 (.16)	.53 (.13)	.58 (.17)	.50 (.16)	43.94	1.89	<i>P</i> < .0001	.009 (.001)	.69	1.33	.75
Feeling	.42 (.10)	.38 (.14)	.39 (.16)	.56 (.10)	.62 (.14)	.55 (.16)	70.84	1.89	<i>P</i> < .0001	.01 (.001)	1.25	1.65	.98
Work	.49 (.19)	.44 (.22)	.47 (.16)	.71 (.19)	.71 (.22)	.58 (.16)	39.42	1.68	<i>P</i> < .0001	.01 (.001)	1.12	1.18	.66
Household duties	.47 (.17)	.45 (.25)	.41 (.19)	.56 (.17)	.63 (.25)	.59 (.19)	55.40	1.74	<i>P</i> < .0001	.01 (.001)	.55	.70	.96
Leisure	.43 (.18)	.37 (.22)	.37 (.22)	.59 (.18)	.62 (.22)	.52 (.22)	35.35	1.89	<i>P</i> < .0001	.01 (.001)	.84	1.11	.62
Social relationships	.44 (.13)	.40 (.16)	.44 (.18)	.53 (.13)	.62 (.16)	.54 (.18)	36.76	1.89	<i>P</i> < .0001	.008 (.001)	.67	1.40	.54
General activities	.39 (.11)	.34 (.14)	.36 (.14)	.53 (.11)	.59 (.14)	.52 (.14)	84.11	1.88	<i>P</i> < .0001	.01 (.001)	1.25	1.71	1.09
HRSA	15.88 (4.74)	17.66 (5.36)	16.96 (5.06)	10.06 (4.74)	10.10 (5.36)	9.52 (5.06)	73.16	1.111	<i>P</i> < .0001	-.39 (.04)	-1.22	-1.40	-1.47
BAI	9.42 (6.25)	9.9 (6.66)	9.08 (5.34)	4.22 (6.25)	2.87 (6.66)	2.75 (5.34)	91.14	1.131	<i>P</i> < .0001	-.33 (.03)	-.83	-1.06	-1.18
BDI	32.09 (9.38)	32.19 (9.86)	29.11 (11.00)	14.91 (9.38)	9.68 (9.86)	12.64 (11.00)	165.29	1.139	<i>P</i> < .0001	-1.00 (.07)	-1.82	-2.27	-1.49

Note. Df = Degrees of Freedom; S.E. = Standard Error. Analyses were performed using PROC MIXED. For MOS and Q-les-Q, higher scores represent improvement in mental and physical health and in QOL, respectively. For IIP, HRSA, BAI and BDI lower negative scores represents a greater reduction in interpersonal distress and symptoms, respectively. Means at post-treatment as well as pre-post effect sizes were calculated based on the intent-to-treat data with post-treatment scores from the linear mixed model analyses.

Table 2
Changes in Outcome Measures Comparing MED and SET to PBO.

Outcome	F	Df	p	Estimate		S.E.		Between-conditions effect size	
				MED	SET	MED	SET	MED	SET
MOS									
Physical component score	.75	2.371	<i>P</i> =.47	-.24	.14	.31	.31	-.57	.33
Mental component score	1.89	2.371	<i>P</i> =.15	.70	-.16	.47	.46	.05	.36
IIP	.70	2.69	<i>P</i> =.49	-.48	.46	-.47	.46	.16	-.64
Q-les-Q									
Physical health	.48	2.76	<i>P</i> =.61	.001	.003	.003	.003	-.06	.58
Feeling	.19	2.75	<i>P</i> =.82	.001	.003	.001	.003	.27	.67
Work	3.32	2.53	<i>P</i> =.04	.006	.004*	.01	.004	.46	.52
Household duties	1.11	2.56	<i>P</i> =.33	-.001	-.004	.003	.003	-.41	-.26
Leisure	.60	2.75	<i>P</i> =.55	.002	.004	.004	.004	.22	.49
Social relationships	1.89	2.75	<i>P</i> =.15	.005	.001	.003	.003	.13	.86
General activities	.65	75.2	<i>P</i> =.52	.003	.001	.002	.002	.16	.62
HRSA	.62	90.2	<i>P</i> =.53	.10	-.001	.10	.11	.25	.07
BAI	.23	2.529	<i>P</i> =.79	-.03	.02	.08	.08	.35	.12
BDI	2.35	2.1639	<i>P</i> =.09	-.35	-.008	.19	.18	-.33	-.78

Note. *F*=represent the omnibus tests for the interactions with treatment condition. *Df*=Degrees of Freedom; *S.E.*=Standard Error. Analyses were performed using PROC MIXED. All effects of MED and SET are in comparison to PBO. For MOS and Q-les-Q, higher scores represent improvement in mental and physical health and in QOL, respectively. For IIP, HRSA, BAI and BDI lower negative scores represents a greater reduction in interpersonal distress and symptoms, respectively. Pre-post effect sizes were calculated based on the intent-to-treat data with post-treatment scores from the linear mixed model analyses.

**p* < 0.05.

which patients receiving SET showed greater improvement than those in the PBO condition (*p*=.01). This finding (*d*=.52 while comparing SET to PBO) was no longer significant after applying the Bonferroni correction. Results were unchanged when controlling for gender and minority status.

In order to determine whether the lack of significant differences between treatments was driven by missing data, we classified two patterns of patients' available data (i.e., patients with and without data at week 16) and assessed the interaction of pattern, time, and treatment conditions using a linear mixed models analysis for each of the outcome measures. The pattern-mixture results were non-significant (*ps* > .14).

Finally, we employed autoregressive cross-lagged modeling (ARCL) on those measures that had sufficient validated data (MOS: PCS and MCS, BDI, BAI), in order to determine the temporal relationship between HRSD and secondary outcome measures. Findings show that for all the measures (MOS: PCS and MCS, BDI, BAI), higher well-being and quality of life at a given time point predicted a lower HRSD level at the subsequent time point, while controlling for previous levels of HRSD (all *ps* ≤ .03). Additionally, for half of the measures (BAI, MCS), lower HRSD at a given time point predicted higher well-being and QOL at the subsequent time point, while controlling for previous levels of well-being and quality of life (*ps* ≤ 03).

4. Discussion

Depression is associated with a substantial decrease in QOL, and is a major contributor to decrement in general health (Rapaport et al., 2005, Moussavi et al., 2007). Medication, psychodynamic psychotherapy, and even placebo have been shown to effectively reduce observer-rated depressive symptoms of patients with MDD when compared in a recent RCT (Barber et al., 2012). However, whether these treatments lead to improvement in other areas of functioning in patients with MDD throughout and between treatments has not been previously evaluated. The results of this study showed that patients experienced not only a significant reduction in self-reported depressive symptoms and observer-rated and self-

reported anxiety symptoms, but also a significant improvement in many other aspects of life, including work productivity, social relationships and vitality.

Although the three treatments presumably have different underlying mechanisms and distinct procedures, they showed equivalent efficacy not only in reducing symptoms but also in other more broad parameters of clinical progress. Importantly, the changes through time in well-being found in this study cannot be interpreted just as the consequences of symptomatic change (reduction in depressive symptoms predicting changes in other life domains), since well-being was found to predict symptomatic change and was not merely the products of such a change. Therefore, other explanations should be raised, such as the common factors explanation which suggests common mechanisms of change between different treatments (e.g., Kazdin, 2007). Specifically, all patients met a professional (either a psychopharmacologist in clinical management or psychotherapist in SET) who expressed interest in their well-being. The treatment provider may have also encouraged optimism about the possibility of positive change as well as a plausible rationale for the patient's symptoms (either related to medical or interpersonal causes).

Although the analyses suggest that missing data did not contribute to our findings, methodological shortcomings such as sample size may have contributed to our failure to find differences between treatments. For instance, although our sample was larger than many other studies and enabled us to detect large to medium effects between the conditions, it did not allow for the detection of small effects.

In sum, the possibility that different but equally effective treatments in reducing symptoms will yield different outcomes when broadening the scope to well-being and QOL was not supported. Rather, consistent with other reports in the literature (e.g., Cuijpers et al., 2013), it provided evidence that distinct treatments for depression works equally well regardless of the specific outcome being measured. The current study also shows rare evidence that the changes in well-being were not just the "byproduct" of reduction in depressive symptoms, but were also the predictors of symptomatic change—and therefore are important in the assessment of the effects of treatments for depression.

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Conflict of interest

Dr. Zilcha-Mano has received funding from the Fulbright Program. Dr. Barber has received funding from the National Institute of Mental Health (NIMH), and the National Institute on Drug Abuse (NIDA); authors' fees from Guilford Press, Basic Books, and Cambridge University Press; and honoraria from Lundbeck. The other authors declare no conflict of interest.

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