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The influence of patients' attributions of the immediate effects of treatment of depression on long-term effectiveness of behavioural activation and antidepressant medication

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ABSTRACT

Patients' attributions of effects of treatment are important, as these can affect long-term outcome. Most studies so far focused on the influence of attributions to medication for anxiety and depression disorders. We investigated the effects of patients' attributions made after acute treatment on the long-term outcome of antidepressant medication (ADM) and psychological treatment (behavioural activation, BA). Data are based on a randomized trial testing the effectiveness of BA vs. ADM for major depression (MDD) in Iran. Patients with MDD (N = 100) were randomized to BA (N = 50) or ADM (N = 50). Patients' attributions were assessed at post-test (after completion of the treatments). Scores on an attribution questionnaire were factor analysed, and factor scores were retained as predictors of depressive symptoms at 1-year follow-up. Regression analysis was used to test whether attributions predicted depressive symptoms at 1-yr follow-up, controlling for symptom level, condition, and their interaction at post-test. *Belief in coping efficacy* was the only attribution factor significantly predicting 1-year HRSD scores, controlling for condition, post-test HRSD and their interaction. It also mediated the condition differences at follow-up. *Credit to self* was the single attribution factor that predicted BDI follow-up scores, controlling for condition, posttest BDI, and their interaction. It partially mediated the condition differences on the BDI at follow-up. Attribution to increased coping capacities and giving *credit to self* appear essential. In the long-term (at 1 year follow-up), the difference in outcome between BA and ADM (with BA being superior to ADM) is at least partially mediated by attributions.

1. Introduction

Antidepressant medication (ADM) is a standard treatment for depressed patients in current psychiatric guidelines (American Psychiatric Association, 2000; (Frank et al., 1990) and the most recent practice guideline for the initial treatment of patients with mild to moderate major depressive disorder (MDD) is antidepressant medication and depression-focused psychotherapy. For depressed patients with severe MDD with or without psychiatric features however, ADM is the first choice (American Psychiatric Association, 2010). The short-term effectiveness of ADM is well studied and comparable to that of CBT and IPT, although dropout from ADM is higher (Cuijpers, Straten, Oppen, & Andersson, 2008). Much less is known about the long-term effectiveness of ADM, and how it compares to that of psychological treatment. A recent meta-analysis reported a trend towards superiority in relapse prevention of CBT compared to maintenance of ADM over 5 studies (OR = 1.62; p = 0.07). The superiority of CBT over ADM became significant after exclusion of one outlier, OR = 1.77, $p < 0.05$ (Cuijpers et al., 2013). The same meta-analysis reports clear evidence of superiority of CBT over ADM when ADM is discontinued after the acute treatment phase over eight studies, OR = 2.61, $p < 0.001$ (see also Imel et al., 2008). Thus, when patients stop taking antidepressant medication, those who recovered from their depressive episode are at a substantial risk for recurrence, whereas CBT appears to offer a better protection for future relapse. The superior effects of CBT over ADM in relapse prevention seem to hold for both the Beckian approaches

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(Hollon, Thase, & Markowitz, 2002; Hollon et al., 2005; Dobson et al., 2008) and for behavioural activation (BA) (Dobson et al., 2008; Moradveisi, Huibers, Renner, Arasteh, & Arntz, 2013a, 2013b).

The important question then arises: what explains the apparent superior long-term effects of psychological treatment over ADM? It has been argued before that where ADM only alleviates depression symptoms as long as the medication is used, patients in psychotherapy actually learn to get better and stay well (Hollon et al., 2005; Paykel et al., 2005). More specifically, it was found that the skills that patients acquire in CT actually predict the prevention of relapse after treatment (Strunk, DeRubeis, Chiu, & Alvarez, 2007). From a behavioural activation point of view, a likely reason of relapse after discontinuation medication is that patients did not change their coping skills. The lack of reinforcement, patterns of avoidance and rumination might still exist, although antidepressant medication might reduce temporarily their effects on mood. In contrast, those patients treated with behavioural activation have acquired healthy behavioural skills and new coping styles that might reduce relapse (Moradveisi et al., 2013a, 2013b).

Another explanation is that patients' beliefs about why they recovered in therapy (attributions) impact the sustaining of gains. It has been postulated by Brewin and Antaki (1982) that patients who attribute gains to their own efforts are more likely to sustain those gains compared to those who attribute improvement to external causes such as a drug's activity or a therapist's charisma. A study by Basoglu, Marks, Klic, Brewin, and swinson (1994) investigated attributions made by patients with panic disorder and agoraphobia who had participated in an RCT comparing 8 weeks of alprazolam or placebo (medication treatment) plus exposure or relaxation (psychological treatment; relaxation being the "psychological placebo"). At the end of 8 weeks of treatment, 40 patients who much/very much improved assessed how much they attributed their gains to medication or to their own efforts. At the treatment-free follow-up in week 43, those who at week 8 had attributed their gains to medication and felt less confident about coping without medication had more severe withdrawal symptoms and a higher loss of gains in comparison to those who at week 8 had attributed their gains to their own efforts during treatment. Another study by Biondi and Picardi (2003) that investigated panic disorder with agoraphobia reported similar results. They found that 60% of the patients with panic disorder who attributed improvement to medication in a combined medication-psychotherapy treatment relapsed, whilst those who attributed improvement to the self-reported no relapse. Although similar attributional processes have been hypothesized to play a role in the differential long-term effects of CBT vs. ADM in depression treatment, no study so far assessed this to the best of the present authors' knowledge.

Behavioural activation (BA) is a relatively new treatment for patients with major depressive disorder (MDD) (Jacobson et al., 1996). Recent studies have shown that BA is an effective treatment for depression that might even be more effective than cognitive therapy in severely depressed patients (Dimidjian et al., 2006). To date, no study investigated the effects of attribution to medication and attribution to the self on treatment effects of BA in comparison to antidepressant medication (ADM) for participants with MDD. The data presented in this paper are drawn from a randomized controlled trial comparing BA and antidepressant medication (Sertraline) for patients with MDD, in which BA proved to be superior to ADM (Moradveisi et al., 2013a, 2013b). The focus of this paper is on whether depressed patients' attributions of treatment effects (i.e. to the medication or to the self), impact the long-term effects of treatment, assessed after approximately one year. If it is true that CBT has better long-term effects than ADM because of attribution of improvement to controllable factors in the self instead of to external factors such as medication, two predictions follow.

(1) Attribution of treatment effects to the self will predict better long-term effects of treatment, even after controlling for the short-term effects. In contrast, attribution of treatment effects to medication will not be associated, or negatively associated, with long-term treatment effects.
(2) Attribution of treatment effects to the self will mediate the long-term differences between BA and ADM that were observed in our trial.

We tested the first prediction by assessing participants' beliefs about factors explaining improvement after treatment, and testing their predictive power in explaining long-term depressive complaints, assessed at 49 weeks, whilst controlling for the level of these complaints as assessed immediately after treatment (week 13). The second prediction was tested by formal mediation tests, investigating whether attributions statistically mediated the difference between conditions in long-term effects, even when controlling for the short-term effects of treatment. Implicated in the attribution mediation hypothesis is that attributions that play a role in explaining the differences between BA and ADM on the long-term effects should differ significantly between conditions; we therefore also tested whether attributions differed between BA and ADM.

2. Methods

Main treatment outcome findings and the sample characteristics of the study have been reported elsewhere (Moradveisi et al., 2013a, 2013b). The original sample consisted of 100 depressed patients from Sanandaj, Iran, between the ages of 18–60 years (mean 31.37, SD 8.97), 85 women, with a primary diagnosis of MDD according to the Diagnostic and Statistical Manual of Mental Disorders DSM-IV-TR (American Psychiatric Association, 2000), confirmed by the Structured Clinical Interview for the DSM-IV-TR Axis-I Disorders Clinical Trials Version (SCID-CT) (First, Williams, Spitzer, & Gibbon, et al., 2007). Participants had to have a score of $\geq$19 on the Beck Depression Inventory, second edition (BDI-II) (Beck, Steer, & Brown, et al., 1996) and a score of $\geq$14 on the 17-item Hamilton Rating Scale for Depression (HRSD) (Hamilton, 1960). The present study reports on the 70 participants with complete data at the 3-months and 1-year follow-up assessments. These were all treatment completers. The 70 participants of the present study and the 30 that had incomplete data differed in two aspects: the sample of the attribution study had relatively less often a comorbid personality disorder (11.4% vs. 40%, $p < 0.01$), and relatively less male participants (8.6% vs. 30%, $p = 0.012$). The study was approved by the local Committee of Medical Ethics, Second Session of Kurdistan University of Medical Sciences. All participants signed written informed consent to participate in the study.

3. Treatments, measures and assessments

Participants were randomized by an independent coordinator. Fifty participants were randomly assigned to each condition, behavioural activation ($N = 50$) and antidepressant medication ($N = 50$). Participants in the BA group received 16 sessions over 12 weeks. For the first 4 weeks they received two sessions per week, and for the following 8 weeks one session per week. No patient in the BA group took medication during the three months of the treatment phase.

Patients in the ADM group received sertraline, which is the usual treatment in Iran for depression. In week 1, participants in the ADM group started with 25 mg/daily of sertraline, and the dosage

in week 2 increased to 50 mg, 75 mg in week 4, and 100 mg in week 6 up to week 12. Psychiatrists could reduce the dosage temporarily in case of side-effects and then increase the dosage to the previous level. The maximum dosage of sertraline was 100 mg per day. After 12 weeks, antidepressant medication use was discontinued.

Depression severity was assessed with the modified 17-item version of the HRSD (Hamilton, 1960) and the BDI-II (Beck et al., 1996). Both measures were administered at baseline, 4, 13 (three months of treatment), and 49 weeks (also referred to as 1 year follow-up). HRSD assessments were done by evaluators blind to treatment conditions. Independent assessors assessed the HRSD for TAU patients and the BDI for BA patients before every treatment session and supplied results to psychiatrists and therapists.

The attribution questionnaire was constructed by two authors (L.M and A.A) on the basis of Basoglu's attribution questionnaire (Basoglu et al., 1994). We modified items to make them applicable to depression treatment, and replaced items 3, 4, 7, 9, and 14 to add more items with explicit attribution to psychological treatment. The questionnaire had 15 items (Table 1). Items were scored on 9-point Likert scales (0 = not at all; 8 = very much so). Participants filled out all items, they were instructed to use their subjective belief if they had no experience with what was asked. Item 14 caused too many interpretation problems, probably because of the double negation, and was left out further analyses. It should be noted that most participants had previous experience with ADM before entering the trial.

4. Statistical analysis

The attribution items were subjected to a Principal Component Analyses, retrieving components with Eigenvalue >1, also using the scree test to decide on the number of components to extract, followed by Oblimin rotation. Attribution scores were based on factor scores from the Principal Component Analysis, which are by definition centred. Regression analyses were used to estimate the influences of attribution on depression severity at week 49, controlling for depression severity at week 13 and the interaction between condition and depression severity at week 13, if significant. We used HRSD-follow up and BDI-follow up as dependent variables, and centred predictors, by using Z-scores of BDI and HRSD at week 13, and treatment condition dummies −0.5 (ADM) and 0.5 (BA). Predictors were centred as main effects cannot be interpreted validly when interactions are included in the model. For each dependent variable, the following predictors were forced into the model: z-score of the dependent variable at week 13, condition, and their interaction (if significant); this was model 1. Next, the 4 factor scores of the belief questionnaire were entered, as well as their interactions with condition. As none of these interactions was significant, we will not report them. Thus, the second model we report had all 4 factor scores as predictors in addition to the predictors of model 1. Next, non-significant predictors involving attribution factors were deleted backwards, using $p < 0.05$ as a criterion, leading to models 3, 4, etc., until only significant attribution predictors remained. The significant attribution factors were then tested as mediators of long-term differences between BA and ADM, controlling for post-test level of the dependent variable (HRSD, respectively BDI) and its interaction with condition (if significant). The mediation tests were executed using Hayes and Preacher (2014; see also Preacher & Hayes, 2004) bootstrap mediation test with the SPSS Macro "Mediate" (http://www.afhayes.com/spss-sas-and-mplus-macros-and-code.html, downloaded, downloaded March 9, 2013), using 50,000 replications. This test allows multiple covariates in the model. We used a high number of replications to get more precise estimates (less

Table 1
Factor loadings of the items of the attribution questionnaire.

Item	Factor 1 belief in tablet efficacy	Factor 2 belief in coping efficacy	Factor 3 credit to self	Factor 4 indifference to tablets	BA mean (SD)	ADM mean (SD)
6. I can do things more easily mainly because I feel more secure with my tablets	0.937				0.30 (0.82)	3.67[a] (2.17)
15. I do believe my tablets are real because I can recognize their effect on me	0.932				0.40 (0.90)	3.20[a] (1.88)
1. In the past, carrying tablets with me was helpful even though I did not have to take them most of the time	0.751				0.50 (1.11)	3.23[a] (1.81)
13. My tablets do not calm me down, but I still think they are helpful	0.694			0.335	0.73 (1.89)	3.73[a] (2.03)
3. I can do things more easily because my efforts improved my feelings		0.907			6.88 (2.22)	4.67[a] (2.04)
7. I feel less depressed since I started psychological treatment		0.883			6.90 (2.68)	4.80[a] (2.01)
8. I will not be able to cope all by myself when my treatment ends	0.386	−0.814			0.85 (1.98)	3.17[a] (2.00)
5. I do not feel more confident in coping with my problem now		−0.712			1.73 (2.44)	3.83[a] (1.42)
12. I like myself better for having achieved this improvement so far	0.235		0.880		6.48 (2.06)	5.77 (2.01)
2. I do not deserve any credit for the improvement I have made	0.313		−0.641	−0.372	0.80 (1.34)	2.23[a] (1.87)
11. I have learned things during my treatment that are helping me to cope better	−0.306		0.473		7.83 (0.55)	5.63[a] (2.11)
4. I feel better with having psychological treatment				−0.625	7.35 (1.55)	6.17[a] (1.56)
9. I do not worry about coming off my tablets because my efforts can help me to feel better				0.600	3.78 (3.63)	4.90 (2.11)
10. It does not matter whether my tablets are real or dummy as long as they help me	0.395		−0.324	0.508	0.68 (1.76)	5.00[a] (2.51)

Note. Items listed by factor in order of loading, factor loadings <0.25 are not shown. Loadings on factors 3 and 4 were reversed to facilitate interpretation. Item (14) ("I do not have good feeling about my tablets because I did not play an important role in improving my depression") was excluded from the analyses as responses were inconsistent probably because of the double negation.

[a] Significantly different between conditions ($p < 0.005$). Non-significant differences had a p-level >0.10.

estimation error) as the 95% CI intervals were near zero. Statisticians have criticized the use of relatively small numbers of replications in Monte Carlo tests like the bootstrap, and the underestimation of the error resulting from relatively small numbers of replications (Koehle, Brown, & Haneuse, 2009). The higher the number of replications, the more precise the estimate becomes, and therefore practical aspects (computational time) and not statistical theory usually determines the number of replications. With the mediation test we assessed whether the difference between BA and ADM in depression severity at 1-year follow-up is statistically explained by attributions. The mediation test examines whether the direct effect of condition on 1-year depression severity is explained by an "indirect" effect through attribution, that is whether BA and ADM differ in attribution, and whether this difference accounts for the long-term differences in depression severity. Mediation is inferred when (i) attribution differs between conditions (i.e., condition predicts attribution); (ii) attribution predicts 1-year depression severity; (iii) the effect of condition becomes non-significant after controlling for attribution; (iv) the indirect path from condition to 1-year depression severity through attribution is significant. Partial mediation is concluded when all criteria are fulfilled except (iii), that is the effect of condition remains significant (despite reducing in strength). As the indirect path effect is the product of the effects of condition on the mediator (i.e., attribution) and of the mediator on 1-year depression severity, the distribution of the indirect path effect is usually not normal (but highly skewed). Testing the indirect path is therefore done in the Preacher and Hayes (2004) approach by a bootstrap test; high numbers of samples (with replacement) of the same N as the empirical sample are taken, the indirect path is calculated for every sample, and thereby a simulated distribution of the indirect path is created. From this simulated distribution the 95% confidence interval (CI) is derived and if it does not contain zero, significance at $p < 0.05$ of the indirect path is concluded. The mediation tests were controlled for the level of severity at 3 months as well as for the (significant) interaction of condition by level of severity at 3 months. Figs. 1–4 illustrate the mediation models that were tested.

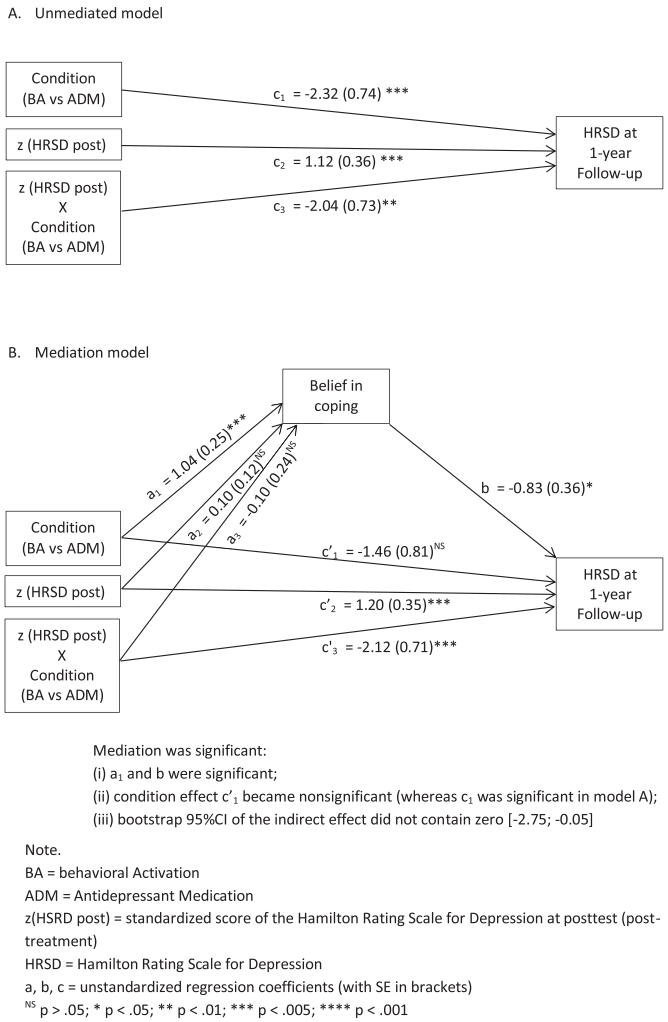


Fig. 1. Mediation of effects of treatment condition on 1-year depression assessed with the HRSD by attribution factor "belief in coping".

5. Results

5.1. Structure of the attribution scale

A Principal Component Analysis indicated 4 components with Eigenvalue >1. The four components explained 72.43% of the variance. After Oblimin rotation three of the four components could be well interpreted: (1) *belief in tablet efficacy*; (2) *belief in coping efficacy*; (3) *credit to self*; whereas the fourth was provisionally labelled (4) *indifference to tablets* (to facilitate interpretation, the original loadings of factors 3 and 4 were reversed). Table 1 presents the item's factor loadings per factor in order of size, omitting loadings with an absolute value <0.25. The factor inter-correlations ranged from −0.37 to 0.22. For further computations, factor scores were retained (reversed for factors 3 and 4), with higher scores representing higher positions on the dimensions as listed above. As might be expected, BA participants scored higher on *belief in coping efficacy*, $t(68) = 4.36$, $p < 0.001$, and *credit to self*, $t(68) = 3.81$, $p < 0.001$, compared to ADM participants. ADM participants scored higher on *belief in tablet efficacy*, $t(68) = 11.24$, $p < 0.001$, and *indifference to tablets*, $t(68) = 3.02$, $p = 0.004$.

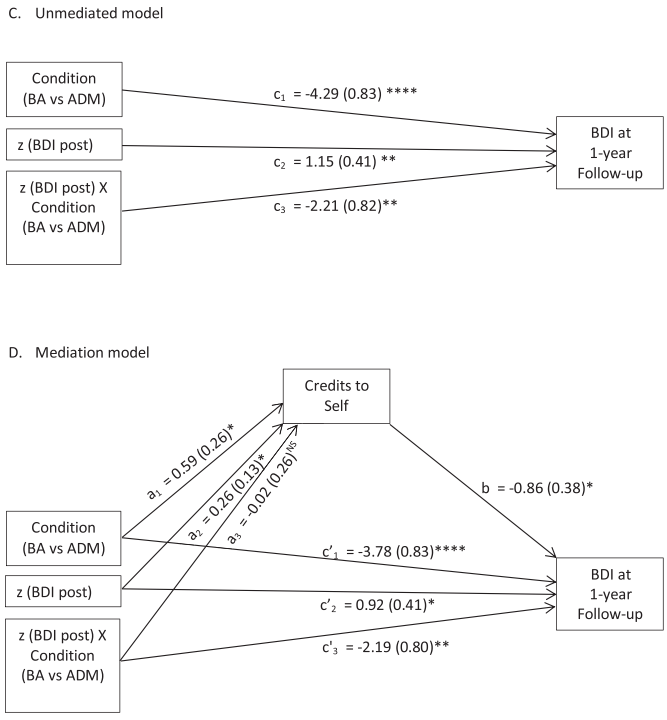
6. Effects of attribution on BDI and HRSD

Tables 2 and 3 present the results of the regression analyses of the full model for HRSD and BDI, respectively.

6.1. HRSD

The two-way condition × zHRSD post interaction was significant for the HRSD in all models and therefore included in all (Table 2). The interaction reflected a positive association between HRSD at 3 months and 1 year in ADM ($r = 0.65$, $p < 0.001$) and an absence of an association in BA ($r = 0.03$, n.s.). The condition was significant in model 1, before entering the attribution factors, reflecting lower HRSD scores at 1-year follow-up in BA than in ADM. After backward deletion of the non-significant attribution factors, *belief in coping efficacy* remained as the single significant attribution factor predicting 1-year HRSD scores, with higher scores predicting lower HRSD scores (Table 2, model 5). In model 5, condition became non-significant ($p = 0.074$; Table 2), suggesting that the condition effect at 1-year follow-up was mediated to a large extent by the *belief in coping efficacy* attribution factor.

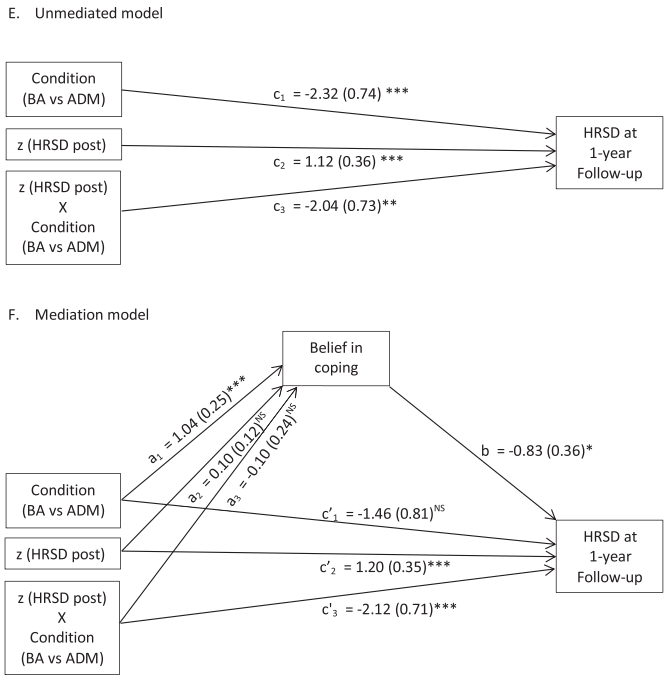
The formal test of mediation with Hayes and Preacher (2014) bootstrap mediation test yielded positive evidence for mediation of the group effect on follow-up HRSD, as the direct effect of group on the mediator (*belief in coping efficacy*) was significant when controlling for posttest HRSD and group by posttest HRSD as covariates (Beta = 1.04, se = 0.25, $t(66) = 4.24$, $p = 0.0001$), and the bootstrap 95% confidence interval of the indirect effect of condition through the mediator on 1-year HRSD (−0.86; SE 0.63) did not



Partial mediation was significant:
(i) a_1 and b were significant;
(ii) condition effect c'_1 remained significant (c_1 was significant in model A, but reduced in strength);
(iii) bootstrap 95%CI of the indirect effect did not contain zero [-1.52; -0.08]

Note.
BA = behavioral Activation
ADM = Antidepressant Medication
z (BDI post) = standardized score of the Beck Depression Inventory at posttest (post-treatment)
BDI = Beck Depression Inventory
a, b, c = unstandardized regression coefficients (with SE in brackets)
NS p > .05; * p < .05; ** p < .01; *** p < .005; **** p < .001

Fig. 2. Partial mediation of effects of treatment condition on 1-year depression assessed with the BDI by attribution factor "credits to self".



Mediation was significant:
(i) a_1 and b were significant;
(ii) condition effect c'_1 became nonsignificant in model B (whereas c_1 was significant in model A);
(iii) bootstrap 95%CI of the indirect effect (-0.86) did not contain zero [-2.77; -0.007]

Note.
BA = behavioral Activation
ADM = Antidepressant Medication
z(HSRD post) = standardized score of the Hamilton Rating Scale for Depression at posttest (post-treatment)
HRSD = Hamilton Rating Scale for Depression
a, b, c = unstandardized regression coefficients (with SE in brackets)
NS p > .05; * p < .05; ** p < .01; *** p < .005; **** p < .001

Fig. 3. Mediation of effects of treatment condition on 1-year depression assessed with the HRSD by attribution factor "lack of belief in coping" from the reduced attribution questionnaire (additional analyses).

contain zero, 95% CI [−2.75; −0.01] (Fig. 1).

6.2. BDI

The two-way condition × zBDI post interaction was significant in all analyses, and therefore retained in all models (Table 3). The interaction reflected a positive association between BDI at 3 months and 1 year in ADM (r = 0.58, p = 0.001) and an absence of an association in BA (r = 0.01, ns). The condition was significant, reflecting superior effects of BA above ADM on BDI-scores at 1-year follow-up. After backward deletion of the non-significant attribution factors, *credit to self* was found to be the single significant attribution factor predicting 1-year BDI scores, controlling for condition, posttest BDI and their interaction (model 5, Table 3). *Credit to self* predicted lower BDI scores at 1-year follow-up. In model 5, condition remained significant, though there was a shrinkage in explanatory power, suggesting partial mediation of the condition effect by the *credit to self* factor.

The formal test of mediation with Hayes and Preacher (2014) bootstrap mediation test yielded positive evidence for (partial) mediation of the group effect on follow-up BDI, as the effect of group on the mediator (*credit to self*) was significant when controlling for posttest BDI and group by posttest BDI as covariates (Beta = −0.59, se = 0.26, t(66) = −2.26.24, p = 0.027), and the bootstrap 95% confidence interval of the indirect effect of condition through the mediator on 1-year BDI (−0.51; SE 0.32) did not contain zero, 95% CI [−1.52; −0.08] (Fig. 2).

7. Additional analyses

The analyses so far may be criticized as the attribution questionnaire contained items that might have been difficult to rate for participants not receiving the type of treatment the items refer to. We therefore checked attributional effects within each condition by construing ad-hoc attribution scales from item subsets that did not refer to the other treatment. For ADM, items 1,2,3,5,6,8,9,10,11,12,13 and 15 were considered as none referred to psychological treatment (reversed scoring when indicated). Based on a reliability analysis, items 5, 8, and 13 were deleted. The subscale had (within the ADM subgroup) an internal consistency of 0.53 (Cronbach's alpha) and correlated significantly with change from 3-month to 1-year follow-up changes in HRSD (r = 0.37, p = 0.046) and BDI (r = 0.36, p = 0.049), indicating that higher attributions to medication and lower attributions to skills and the self predicted increases in depression severity from 3-months to one year. Similarly,

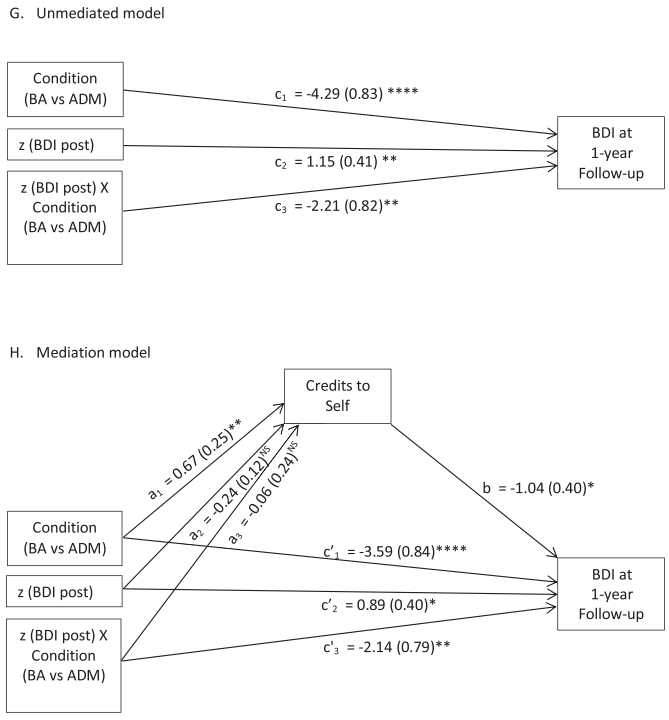


Fig. 4. Partial mediation of effects of treatment condition on 1-year depression assessed with the BDI by attribution factor "credits to self" from the reduced attribution questionnaire (additional analyses).

Table 2
Results of regression analyses testing effects of attributions after 13 weeks of treatment on 49 weeks HRSD scores.

Predictor	Unstandardized coefficients		Standardized beta	t-value	p-value
	B	S.E			
Model 1 (R2 = 0.45)					
Constant	7.90	0.37		21.43	**<0.001**
Condition	−2.32	0.74	−0.33	−3.15	**0.002**
z (HRSD Post)	1.12	0.36	0.33	3.07	**0.003**
Condition* z (HRSD Post)	−2.04	0.73	−0.27	−2.81	**0.007**
Model 2 (R2 = 0.51)					
Constant	7.81	0.38		20.70	**<0.001**
Condition	−1.56	1.40	−0.22	−1.11	0.27
z (HRSD Post)	1.10	0.38	0.32	2.85	**0.006**
Condition* z (HRSD Post)	−2.17	0.76	−0.28	−2.87	**0.006**
Belief in tablet efficacy	0.04	0.64	0.01	0.06	0.95
Belief in coping efficacy	−0.84	0.36	−0.24	−2.30	**0.025**
Credit to self	−0.27	0.37	−0.08	−0.74	0.46
Indifference to tablets	−0.43	0.35	−0.12	−1.25	0.22
Model 3 (R2 = 0.51)					
Constant	7.82	0.36		21.88	**<0.001**
Condition	−1.62	0.85	−0.23	−1.90	0.062
z (HRSD Post)	1.09	0.37	0.32	2.92	**0.005**
Condition* z (HRSD Post)	−2.15	0.71	−0.28	−3.06	**0.003**
Belief in coping efficacy	−0.84	0.36	−0.24	−2.33	**0.023**
Credit to self	−0.27	0.36	−0.08	−0.75	0.46
Indifference to tablets	−0.44	0.34	−0.12	−1.27	0.21
Model 4 (R2 = 0.51)					
Constant	7.83	0.36		22.04	**<0.001**
Condition	−1.77	0.83	−0.25	−2.15	**0.035**
z (HRSD Post)	1.17	0.35	0.35	3.34	**0.001**
Condition* z (HRSD Post)	−2.19	0.70	−0.29	−3.13	**0.003**
Belief in coping efficacy	−0.88	0.36	−0.25	−2.47	**0.016**
Indifference to tablets	−0.49	0.33	−0.14	−1.48	0.14
Model 5 (R2 = 0.49)					
Constant	7.83	0.36		21.82	**<0.001**
Condition	−1.46	0.81	−0.21	−1.82	0.074
z (HRSD Post)	1.20	0.35	0.35	3.38	**0.001**
Condition* z (HRSD Post)	−2.12	0.71	−0.28	−3.01	**0.004**
Belief in coping efficacy	−0.83	0.36	−0.23	−2.31	**0.024**

Note. Condition was centred with BA = 0.5 and TAU (ADM) = −0.5. z (HRSD Post) = standardized HRSD score at post-test (13 weeks). The four factor scores (by definition centred) of the attribution questionnaire taken at post-test were labelled: (1) Belief in tablet efficacy; (2) Belief in coping efficacy; (3) Credit to self; and (4) Indifference to tablets. Significant p-levels are printed bold.

we considered items 2,3,4,5,7,8,11 and 12 for the BA condition, as none referred to medication. Based on a reliability analysis items 3,4,5,7 and 8 were retained (Cronbach alpha = 0.91). Correlations with changes from 3-month to 1-year follow-up failed to reach significance for HRSD (r = −0.22, p = 0.17) and BDI (r = −0.002, p = 0.99).

Lastly, the main analyses were repeated using only items of the attribution questionnaire that did not refer to medication or psychological treatment, that is items 2,3,5,8,11 and 12. A principal component analyses yielded two components with eigenvalue >1 (total variance explained 71.5%), and the scree plot also supported a two-factor solution. After Oblimin rotation, the two factors were interpreted as "*belief in coping efficacy*" and "*credit to self*", see Table 4 for factor loadings. The factor intercorrelation was 0.31. Factor scores were retained for further computations. The conditions differed significantly in mean factor scores; on *belief in coping efficacy* means (SD) were for ADM −0.63 (0.65) vs. for BA 0.47 (0.96), t(68) = 5.10, p < 0.001; and on *credit to self* for ADM −0.53 (1.02) vs. for BA 0.40 (0.78), t(68) = 4.34, p < 0.001. Using the same regression procedures as above, factor 1 scores significantly added to the prediction of HRSD scores at 1-year follow-up, controlling for condition, 3-months HRSD and their interaction (Table 5). The prediction by condition became non-significant, indicating mediation of the condition differences on HRSD at 1-year follow-up *by belief in coping efficacy*. This was confirmed by a formal mediation test, see Figs. 3 and 4. In short, condition significantly predicted the mediator, the mediator significantly predicted the HRSD at follow-up, the direct effect of condition on 1-year follow-up HRSD became non-significant after controlling for the mediator, and the 95% CI of the indirect effect of condition (beta = −1.08) through the mediator did not contain zero (−3.02; −0.022).

Similar findings as from the primary analyses were found for the prediction of 1-year BDI by the revised attribution questionnaire's factors. As is shown in Table 6, factor 2 (*credit to self*), but not factor 1, predicted 1-year BDI controlling for condition, 3-months BDI, and their interaction. Similarly as in the primary analysis, adding factor 2 reduced the contribution of condition, but did not make it non-significant. A formal mediation analysis demonstrated that the (partial) mediation of the condition effect by factor 2 was significant, as condition significantly predicted factor 2, and the indirect effect of condition through the mediator (beta = −0.70) was significant (the 95% CI did not contain zero: (−1.73; −0.16)).

Table 3
Results of regression analyses testing the effects of attributions after 13 weeks of treatment on 49 weeks BDI scores.

Predictor	Unstandardized coefficients		Standardized beta	t-value	p-value
	B	S.E			
Model 1 (R2 = 0.56)					
Constant	9.76	0.42		23.54	**<0.001**
Condition	−4.29	0.83	−0.49	−5.17	**<0.001**
z (BDI Post)	1.15	0.41	0.27	2.79	**0.007**
Condition* z (BDI Post)	−2.21	0.82	−0.23	−2.69	**0.009**
Model 2 (R2 = 0.62)					
Constant	9.79	0.43		22.91	**<0.001**
Condition	−4.73	1.50	−0.54	−3.15	**0.003**
z (BDI Post)	0.96	0.41	0.23	2.36	**0.021**
Condition* z (BDI Post)	−1.97	0.87	−0.20	−2.27	**0.027**
Belief in tablet efficacy	−0.72	0.69	−0.16	−1.04	0.30
Belief in coping efficacy	−0.69	0.39	−0.16	−1.76	0.083
Credit to self	−0.65	0.39	−0.16	−1.66	0.10
Indifference to tablets	−0.50	0.38	−0.12	−1.32	0.19
Model 3 (R2 = 0.61)					
Constant	9.64	0.40		23.96	**<0.001**
Condition	−3.50	0.93	−0.40	−3.75	**<0.001**
z (BDI Post)	0.98	0.41	0.23	2.41	**0.019**
Condition* z (BDI Post)	−2.34	0.79	−0.24	−2.95	**0.004**
Belief in coping efficacy	−0.67	0.39	−0.16	−1.72	0.091
Credit to self	−0.71	0.39	−0.17	−1.85	0.069
Indifference to tablets	−0.43	0.37	−0.10	−1.17	0.25
Model 4 (R2 = 0.60)					
Constant	9.60	0.40		23.88	**<0.001**
Condition	−3.20	0.90	−0.37	−3.56	**<0.001**
z (BDI Post)	0.93	0.40	0.22	2.30	**0.025**
Condition* z (BDI Post)	−2.37	0.80	−0.25	−2.98	**0.004**
Belief in coping efficacy	−0.62	0.39	−0.14	−1.59	0.12
Credit to Self	−0.81	0.38	−0.20	−2.17	**0.034**
Model 5 (R2 = 0.59)					
Constant	9.69	0.40		24.02	**<0.001**
Condition	−3.78	0.83	−0.43	−4.52	**<0.001**
z (BDI Post)	0.92	0.41	0.22	2.24	**0.029**
Condition* z (BDI Post)	−2.19	0.80	−0.23	−2.75	**0.008**
Credit to Self	−0.86	0.38	−0.21	−2.28	**0.026**

Note. Condition was centred with BA = 0.5 and TAU (ADM) = −0.5. z (BDI Post) = standardized BDI score at post-test (13 weeks). The four factor scores (by definition centred) of the attribution questionnaire taken at post-test were labelled: (1) Belief in tablet efficacy; (2) Belief in coping efficacy; (3) Credit to self; and (4) Indifference to tablets. Significant p-levels are printed bold.

8. Discussion

We used a self-report instrument to assess patients' treatment attribution towards either psychotherapy or pharmacological treatment for depression. The instrument was a modification of the instrument constructed by Basoglu et al. (1994), adapted to depression and to represent not only medication but also psychological treatment. The four components of the instrument explained 72.43% of the variance, and were labelled: (1) *belief in tablet efficacy*; (2) *belief in coping efficacy*; (3) *credit to self*; and (4) *indifference to tablets*.

The results of the study indicated that BA and ADM treatment conditions differentially influenced the attribution types. ADM treatment led to relatively stronger beliefs in medication and to stronger *indifference to tablets* compared to BA, whereas BA conduced to relatively stronger beliefs in own coping capabilities and to stronger crediting the self for improvement. It is likely that increased belief in medication was due to improvement of depression symptoms in the ADM condition attributed to the medication participants took, but the higher scores on factor 4 (labelled "*indifference to tablets*") are more difficult to interpret. In any case, this attribution factor did not appear to be important as a mediator, and can be ignored in that sense. Stronger beliefs in coping and stronger crediting the self for improvement in BA than in ADM is probably directly related to learning new strategies and skills in treatment to cope with problems, and the direct experience that one's own actions lead to overcoming problems and improvement in mood. However, we did not assess the degree of skill acquisition and actual application, so we cannot test this interpretation.

Long-term effects were predicted by attributional factors. Attribution of effects to medication does not seem to play a role, but attribution to increased coping capacities and giving credit to the self appear essential. The difference between BA and ADM treatment (with BA > ADM) in the long-term (at 1 year follow-up) is at least partially mediated by attributions. Thus, we have evidence that the superior long-term effects of BA above ADM are at least partially related to self-attributions, which are higher in BA.

Table 5
Results of regression analyses testing effects of attributions from the reduced attribution questionnaire after 13 weeks of treatment on 49 weeks HRSD scores.

Predictor	Unstandardized coefficients		Standardized beta	t-value	p-value
	B	S.E			
Model 1 (R2 = 0.45)					
Constant	7.90	0.37		21.43	**<0.001**
Condition	−2.32	0.74	−0.33	−3.15	**0.002**
z (HRSD Post)	1.12	0.36	0.33	3.07	**0.003**
Condition* z (HRSD Post)	−2.04	0.73	−0.27	−2.81	**0.007**
Model 2 (R^2 = 0.51)					
Constant	7.79	0.36			
Condition	−0.97	0.86	−0.14	−1.13	0.26
z (HRSD Post)	1.03	0.37	0.30	2.79	**0.007**
Condition* z (HRSD Post)	−2.12	0.70	−0.28	−3.03	**0.004**
Belief in coping efficacy	−0.81	0.38	−0.23	−2.13	**0.037**
Credit to self	−0.58	0.38	−1.54	−1.54	0.13
Model 3 (R^2 = 0.49)					
Constant	7.79	0.36		21.62	**<0.001**
Condition	−1.24	0.85	−0.18	−1.46	0.15
z (HRSD Post)	1.21	0.35	0.36	3.42	**0.001**
Condition* z (HRSD Post)	−2.20	0.71	−0.29	−3.11	**0.003**
Belief in coping efficacy	−0.89	0.38	−0.25	−2.34	**0.023**

Note. Condition was centred with BA = 0.5 and TAU (ADM) = −0.5. z (HRSD Post) = standardized HRSD score at post-test (13 weeks). The two factor scores (by definition centred) of the reduced attribution questionnaire (only items that did not refer to medication or psychological treatment) taken at post-test were labelled: (1) Belief in coping efficacy; and (2) Credit to self. Significant p-levels are printed bold.

Table 4
Factor loadings of the 6 items of the reduced attribution questionnaire.

Item	Factor 1 belief in coping efficacy	Factor 2 credit to self
8. I will not be able to cope all by myself when my treatment ends	−0.947	
3. I can do things more easily because my efforts improved my feelings	0.926	
5. I do not feel more confident in coping with my problem now	−0.726	−0.312
2. I do not deserve any credit for the improvement I have made		−0.846
12. I like myself better for having achieved this improvement so far		0.807
11. I have learned things during my treatment that are helping me to cope better		0.627

Note. Factor loadings with absolute value <0.25 are not displayed.

Table 6
Results of regression analyses testing effects of attributions from the reduced attribution questionnaire after 13 weeks of treatment on 49 weeks BDI scores.

Predictor	Unstandardized coefficients		Standardized beta	t-value	p-value
	B	S.E			
Model 1 ($R^2 = 0.56$)					
Constant	9.76	0.42		23.54	**<0.001**
Condition	−4.29	0.83	−0.49	−5.17	**<0.001**
z (BDI Post)	1.15	0.41	0.27	2.79	**0.007**
Condition* z (BDI Post)	−2.21	0.82	−0.23	−2.69	**0.009**
Model 2 ($R^2 = 0.61$)					
Constant	9.64	0.40		23.98	**<0.001**
Condition	−3.00	0.95	−0.35	−3.17	**0.002**
z (BDI Post)	0.92	0.40	0.22	2.27	**0.026**
Condition* z (BDI Post)	−2.34	0.80	−0.24	−2.93	**0.005**
Belief in coping efficacy	−0.54	0.42	−0.23	−1.30	0.20
Credit to self	−0.99	0.40	−0.23	−2.48	**0.016**
Model 3 ($R^2 = 0.60$)					
Constant	9.73	0.40		24.46	**<0.001**
Condition	−3.59	0.84	−0.41	−4.27	**<0.001**
z (BDI Post)	0.89	0.41	0.21	2.21	**0.031**
Condition* z (BDI Post)	−2.14	0.79	−0.22	−2.72	**0.008**
Credit to self	−1.04	0.40	−0.24	−2.61	**0.011**

Note. Condition was centred with BA = 0.5 and TAU (ADM) = −0.5. z (HRSD Post) = standardized HRSD score at post-test (13 weeks). The two factor scores (by definition centred) of the reduced attribution questionnaire (only items that did not refer to medication or psychological treatment) taken at post-test were labelled: (1) Lack of Belief in coping efficacy; and (2) Credit to self. Significant p-levels are printed bold.

The attribution questionnaire we used contained items that referred to ADM or to psychological treatment, and therefore might have been difficult to rate by participants that did not receive, or had previously received, the treatment referred to. We therefore redid the analyses after all items that either referred to ADM or psychological treatment were deleted. Although now only two factors were found, they were similar in content to the two of the four factors of the primary analysis that turned out to be mediators, and they mediated condition differences in the same way as in the primary analysis. That is, the *belief in coping efficacy* factor mediated the condition differences at 1-year on the HRSD, with no mediating role for the other factor. The *credit to self* factor partially mediated the condition differences on the BDI, with no role for the *belief in coping efficacy* factor. This indicates that the main findings of the study are not caused by items that were in content condition-specific. We also explored whether a scale constructed from items that do not refer to psychological treatment related to changes from 3 months to 1-year within the ADM condition, and found evidence for a negative relationship, reflecting that stronger attributions to ADM and weaker attributions to the self and to improved coping are related to poorer long-term effects in the ADM condition, replicating similar associations in the Basoglu et al. (1994) study. Remarkably, a scale constructed from items that do not refer to ADM did not significantly correlate to changes from 3 months to 1 year in the BA condition. This might reflect a restriction of range effect in BA caused by many participants in the BA condition having relatively strong beliefs in their own coping and strong attributions to the self, while at the same time having good immediate and follow-up effects. This indicates that belief in one's own coping and crediting the self for improving are factors that explain differences in long-term effects *between* AMD and BA, but not so much *within* BA.

The results of the study showed that clinician-rated depressive symptoms (HRSD) are predicted by *belief in coping efficacy*, i.e. belief in the effectiveness of specific behaviours and one's capacities to use these behaviours. On the other hand, self-reported depressive symptoms (BDI) were predicted by feeling good about progress and improvement made and attributing this to the self (*credit to self*). There are indications that interviews more validly assess objectifiable symptom manifestations and self-reports better capture symptom experience (Hopwood et al., 2008), and factors like severity of depression and neuroticism appear to play a role in discrepancies between the two (Carter, Frampton, Mulder, Luty, & Joyce, 2009; Enns, Larsen, & Cox, 2000). For example, it has been suggested that more severely depressed patients have difficulties in appropriately rating their symptoms (Enns et al., 2000). It is unclear what underlies the specific relationships between attribution type and method of depression severity assessment. Speculations might consider the possibility that the relationships are meaningful, in the sense that attribution to a concrete cause (i.e. skills) relates to more objectifiable depressive symptoms (those that can be rated by an assessor) whereas attribution to a subjective feeling (i.e. crediting oneself and liking oneself better for accomplishing improvement) is related to a more subjective experience of depressive symptoms. Possibly, behavioural skills specifically reduce objectifiable depression symptoms, whereas feeling emotionally good reduces specifically subjective depression symptoms. Clearly, more research is needed to disentangle the associations if they prove to be replicable.

Our results on the influence of attribution to the self and belief in own coping is consistent with Brewin and Antaki (1982) proposition that patients who attribute their improvement to their own efforts will maintain those improvements better than those who attribute gains to external attribution such as medication. Our findings are also in line with the Basoglu et al. (1994) study, in that patients who attributed their improvement to medication and felt less confident in coping without medication had more severe withdrawal symptoms and more loss of gains than those who attributed their improvement to their own efforts during treatment. Biondi and Picardi (2003) findings are also consistent with our results. They found that panic patients with agoraphobia who attributed their improvement to the self showed no relapse, whereas 60% of those who attributed their improvement to the medication relapsed.

One can speculate about the relationship between attributing progress to the self and one's own coping, and DeRubeis et al.'s (1990) explanation for the superior long-term effects of CBT over ADM treatment. They speculated that in CBT patients learn skills and strategies in coping with life problems which are not learned in ADM. These skills and new strategies may act directly to prevent relapse, but may also lead to increased attributions to own efforts and belief in coping efficacy, which might directly guard patients against relapse. To what degree attributions or skills, or both, explain the superior long-term effects of CBT over (discontinued) ADM is an important topic for further research.

A number of limitations should be mentioned with respect to the data presented in this study. Since most patients in Iran have only access to ADM, offering a relatively new psychological treatment for depression could attract especially those who prefer this new treatment available. Most participants in our sample with recurrent MDD had previously received medication treatment, due to accessibility of medication treatment for psychological disorder in Iran. Moreover, only for the first three months ADM participants were offered medication for free, and many stopped taking medication after that period as they had to pay for it. Thus, for many participants the period in which they took medication was limited which might have influenced the results. Another limitation is that we used a modified version of the instrument constructed by Basoglu et al. (1994), adapted to depression and to represent not only medication but also psychological treatment, which was not validated in a previous study. Moreover, this questionnaire, and therefore also our variation, includes items that do not directly

represent attributions, but beliefs, attitudes and expectations of the treatments. An (tacit) assumption was that these all reflect attributional processes, but strictly speaking this is an empirical issue that needs further study. Another problem with our approach was that not all items referred to experiences that participants had in their treatment condition. Although the instruction to participants to respond in such cases by rating what they believed did not seem to have reduced reliability, and an additional analysis based on items that did not suffer from this limitation yielded the same results, future research should improve on this. Moreover, there might be other attribution dimensions that are important, but are not represented in our questionnaire. Further improving the scale is also important given that the *belief in tablets efficacy* factor had rather low internal consistency in the ADM subgroup (Cronbach alpha = 0.60, based on items 1,6,13,15) despite having a good reliability in the whole sample (Cronbach alpha = 0.88). Thus, the pragmatic approach we choose by modifying Basoglu's questionnaire had its limitations. Given the positive evidence we found for attributions playing a role in long-term effects of BA vs. ADM future studies might consider to improve the assessment of attributions. Still another limitation is that we restricted the analyses to participants with complete data (who happened to be all treatment completers), and dropouts were not included. We are not aware of bootstrap mediation tests for approaches that can handle dropouts with missing data (like mixed regression), but the exclusion of dropouts limits the results to those that complete treatment, whereas attributional processes might also play a role in those who dropout from treatment and/or a treatment study. Importantly, in the sample of which complete data were available for the attribution study there were less comorbid personality disorders and less male participants than in the sample with incomplete data. Treatment dropout was an important characteristic of those that had incomplete data (20 of the 30) and a previous study already reported that comorbid personality disorder was predictive of treatment dropout (Moradveisi et al., 2013a, 2013b). Lastly, although we speculated that the new skills acquired in BA may lead to relatively stronger beliefs in skills and stronger crediting the self for improvement, we could not test this possibility. Also, whether or not attributions are essential for the long-term effects of BA, or just a reflection (or even an “epiphenomenon”) of increased and effective skill use after BA could not be tested with our data.

In conclusion, our study found that long-term effects are predicted by attributional factors. Attribution of effects to medication does not seem to play a role, but attribution to increased coping capacities and giving credit to oneself appear essential. In the long-term (at 1 year follow-up), the difference between BA and ADM (with BA > ADM) is at least partially mediated by attributions. Moreover, we have evidence that the superior long-term effects of BA over ADM at least related to self-attributions, which are higher in BA. One interpretation waiting for further study is that offering BA to depressed patients helps them learn new skills and strategies in coping with problematic life events, which leads to increased attribution to acquired coping skills and crediting oneself, which in turn guards them against a relapse when faced with a difficult condition in their life. Future studies should investigate the effects of attributions for BA and ADM in other clinical settings and cultures.

Contributorship statement

LM, AA, and MH contributed to the design of the study. LM executed this study. LM, and AA conducted the data analyses and the interpretation of the results. LM, AA, and MH drafted the manuscript. LM had full access to the data in the study and had final responsibility for the decision to submit for publication.

Conflict of interest

None.

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