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Review

Problem solving therapies for depression: A meta-analysis

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Abstract

Purpose. – In the past decades, the effects of problem-solving therapy (PST) for depression have been examined in several randomized controlled studies. However, until now no meta-analysis has tried to integrate the results of these studies.

Methods. – We conducted a systematic literature search and identified 13 randomized studies examining the effects of PST, with a total of 1133 subjects. The quality of studies varied.

Results. – The mean standardized effect size was 0.34 in the fixed effects model and 0.83 in the random effects model, with very high heterogeneity. Subgroup analyses indicated significantly lower effects for individual interventions in studies with subjects who met criteria for major depression, studies in which intention-to-treat analyses were conducted instead of completers-only analyses, and studies with pill placebo and care-as-usual control groups. Heterogeneity was high, and the subgroup analyses did not result in clear indications of what caused this high heterogeneity. This indicates that PST has varying effects on depression, and that it is not known to date what determines whether PST has larger of smaller effects.

Conclusion. – Although there is no doubt that PST can be an effective treatment for depression, more research is needed to ascertain the conditions and subjects in which these positive effects are realized.

Keywords: Problem-solving therapy; Depression; Meta-analysis

1. Introduction

In the past few decades, dozens of psychological treatments have been developed for the treatment of depression [7,9,25]. However, only a limited number of these treatments have been examined in well-designed randomized controlled trials, and only very few therapies have been examined in five or more trials [24]. Most research has been conducted on cognitive behavior therapy and to a lesser extent on interpersonal therapy, couple and marital therapy, and life review therapy for older adults [24,8].

Problem-solving therapy (PST) is another psychological treatment which has been examined in several randomized controlled trials. In PST, the patient systematically identifies his or her problems, generates alternative solutions for each problem, selects the best solution, develops and conducts a plan, and evaluates whether this has solved the problem [20,11].

There are several types of PST for depression. The first type, 'social problem-solving therapy' (SPST) was developed in 1980s [11,22], and is typically conducted in a group format of 10–12 sessions. This treatment does not only focus on the problem-solving skills themselves, but also on changing those attitudes or beliefs that may inhibit or interfere with attempts to engage in the remaining problem-solving tasks. The second type, PST for primary care (PST-PC), was developed in 1990s [20], and is applied individually in six sessions. It focuses on the core elements of problem-solving and can be used by trained nurses. The third type of problem-solving, self-examination therapy (SET) [5,6], is aimed at determining the major goals in their life, investing energy only in those problems that are related to what matters and learning to accept those situations that

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cannot be changed. Problem-solving skills are the core element of this approach. SET is typically used in a guided-self-help format, but can also be applied in group and individual settings.

Early studies on PST in 1980s used volunteer samples recruited from the community [21,23], while more recent studies have examined the effects of PST in primary care [20] and clinical settings [1]. However, until now no systematic review or meta-analysis has attempted to integrate the results of randomized trials of PST. This is especially important because some studies have found strong effects of PST [21], while others found none or only very modest effects [26].

While several trials of PST have reported positive results, we wanted to examine whether these results remain significant in a meta-analytic approach and we decided to conduct a comprehensive meta-analysis of randomized controlled trials of PST.

2. Method

2.1. Identification and selection of studies

Studies were traced by means of several methods. First, we used a large database of 777 papers on the psychological treatment of depression in general. This database was developed through a comprehensive literature search (from 1966 to March 2005) in which we examined 5178 abstracts in Pubmed (1224 abstracts), Psycinfo (1336), Embase (1118) and the Cochrane Central Register of Controlled Trials (1500). We identified these abstracts by combining terms indicative of psychological treatment (psychotherapy, psychological treatment, cognitive therapy, behavior therapy, interpersonal therapy, reminiscence, life review) and depression (both MeSH-terms and textwords). For this database, we also collected the primary studies from 22 meta-analyses of psychological treatment of depression [8]. For the current study, we examined the abstracts of these 777 studies, and selected the ones which focused on PST. In addition, we examined the references of earlier reviews on PST [24,18], and we reviewed the reference lists of retrieved papers.

We included studies in which (1) the effects of PST (2) on adults (3) with a depressive disorder or an elevated level of depressive symptomatology (4), were compared to a control condition or another (psychological or pharmacological) treatment (5) in a randomized controlled trial. No language restrictions were applied.

We defined PST as a psychological intervention in which the following elements had to be included: definition of personal problems, generation of multiple solutions to each problem, selection of the best solution, the working out of a systematic plan for this solution, and evaluation as to whether the solution has resolved the problem.

2.2. Quality assessment

There are at least 25 scales available to assess the validity and quality of randomized controlled trials [14]. There is no evidence, however, that these scales provide more reliable assessments of validity. We prefered therefore to use a simple approach for assessing the validity of the studies, as suggested in the Cochrane Handbook [14]. We assessed the validity of the studies using four basic criteria [14]: allocation to conditions is done by an independent (third) party; adequacy of random allocation concealment to respondents; blinding of assessors of outcomes; and completeness of follow-up data.

2.3. Meta-analysis

We calculated effect sizes (d) by subtracting (at post-test) the average score of the control group (M_e) from the average score of the experimental group (M_c) and dividing the result by the average of the standard deviations of the experimental and control group (SD_{ec}). An effect size of 0.5 thus indicates that the mean of the experimental group is half a standard deviation larger than the mean of the control group. Effect sizes of 0.56−1.2 can be assumed to be large, while effect sizes of 0.33−0.55 are moderate, and effect sizes of 0−0.32 are small [15].

In the calculations of effect sizes we only used those instruments that explicitly measure depression (Table 1), such as the Beck Depression Inventory [4], and the Hamilton Depression Scale [12]. If more than one depression measure was used, the mean of the effect sizes was calculated, so that each study (or contrast group) only had one effect size. In one study more than one experimental condition was compared to a control condition [23]. In this case, the number of subjects in the control condition was evenly divided over the experimental conditions so that each subject was used only once in the meta-analyses.

To calculate pooled mean effect sizes, we used the computer program Comprehensive Meta-analysis (version 2.2.021), developed for support in meta-analysis. As we expected considerable heterogeneity, we decided to calculate mean effect sizes both with the random effects model and the fixed effects model. In the fixed effect model it is assumed that all studies in the meta-analysis are drawn from the same 'population' of studies and all factors which could influence the effect size are the same in all the study populations. In the fixed effects model the observed effect size differs between studies only because of the random error inherent in each study. In the random effects model, on the other hand, it is assumed that the included studies are drawn from 'populations' of studies that differ from each other systematically. In this model, the effect sizes resulting from included studies differ because of the random error within studies (as in the fixed effects model), but also because of true variation in effect size from one study to the next.

In our analyses, we have tested whether there are genuine differences underlying the results of the studies (heterogeneity), or whether the variation in findings is compatible with chance alone (homogeneity) [13]. As indicator of homogeneity, we calculated the Q-statistic. A significant Q rejects the null-hypothesis of homogeneity and indicates that the variability among the effect sizes is greater than what is likely to have resulted from subject-level sampling error alone. We also calculated the I^2-statistic which is an indicator of heterogeneity