COCHRANE CORNER

In this new section in *Advances* we will highlight recent Cochrane Systematic Reviews of interest to psychiatrists.

¹This review is a an abridged version of a Cochrane Review previously published in the *Cochrane Database* of Systematic Reviews, 2013, issue 9, doi: 10.1002/14651858.CD004366. pub6 (see www.thecochranelibrary. com for information). Cochrane Reviews are regularly updated as new evidence emerges and in response to feedback, and the Cochrane Database of Systematic Reviews should be consulted for the most recent version of the review.

We thank the Cochrane Review Group for their support in publishing these reviews.

Exercise for depression[†]

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Background

Depression is a common and important cause of morbidity and mortality worldwide. It is commonly treated with antidepressants and/or psychological therapy, but some people prefer alternative approaches such as exercise. There are a number of theoretical reasons why exercise may improve depression. This is an update of a review first published in 2009.

Objectives

To determine the effectiveness of exercise in the treatment of depression in adults compared with no treatment or a comparator intervention.

Search strategy

We searched the Cochrane Depression, Anxiety and Neurosis Review Group's Controlled Trials Register (CCDANCTR) to 13 July 2012. This register includes relevant randomised controlled trials from the following bibliographic databases: The Cochrane Library (all years); MEDLINE (1950 to date); Embase (1974 to date) and PsycINFO (1967 to date). We also searched www.controlledtrials.com, ClinicalTrials.gov and the WHO International Clinical Trials Registry Platform. No date or language restrictions were applied to the search.

We conducted an additional search of the CCDANCTR up to 1 March 2013 and any potentially eligible trials not already included are listed as 'awaiting classification'.

Selection criteria

Randomised controlled trials in which exercise (defined according to American College of Sports Medicine criteria) was compared with standard treatment, no treatment or a placebo treatment, pharmacological treatment, psychological treatment or other active treatment in adults (aged 18 and over) with depression, as defined by trial authors. We included cluster trials and those that randomised individuals. We excluded trials of postnatal depression.

Data collection and analysis

We extracted data on primary and secondary outcomes at the end of the trial and end of follow-up (if available). We calculated effect sizes for each trial using Hedges' *g* method and a standardised mean difference (SMD) for the overall pooled effect, using a random-effects model risk ratio for dichotomous data. Where trials used a number of different tools to assess depression, we included the main outcome measure only in the meta-analysis. Where trials provided several 'doses' of exercise, we used data from the biggest 'dose', and performed sensitivity analyses using the lower 'dose'. We performed subgroup analyses to explore the influence of method of diagnosis of depression (diagnostic interview or cut-off point on scale), intensity of exercise and the number of sessions of exercise on effect sizes. We also performed the 'risk of bias' assessments. Our sensitivity analyses explored the influence of study quality on outcome.

Main results

Thirty-nine trials (2326 participants) fulfilled our inclusion criteria, of which 37 provided data for meta-analyses. There were multiple sources of bias in many of the trials. Randomisation was adequately concealed in 14 studies, 15 used intention-to-treat analyses and 12 used masked ('blinded') outcome assessors.

For the 35 trials (1356 participants) comparing exercise with no treatment or a control intervention, the pooled SMD for the primary outcome of depression at the end of treatment was -0.62 (95% Cl -0.81 to -0.42), indicating a moderate clinical effect. There was moderate heterogeneity ($I^2 = 63\%$).

When we included only the six trials (464 participants) with adequate allocation concealment, intention-to-treat analysis and masked outcome assessment, the pooled SMD for this outcome was not statistically significant (-0.18, 95% Cl -0.47 to -0.11). Pooled data from the eight trials (377 participants) providing long-term follow-up data on mood found a small effect in favour of exercise (SMD = -0.33, 95% Cl -0.63 to -0.03).

Twenty-nine trials reported acceptability of treatment, three reported quality of life, none reported cost, and six reported adverse events. For acceptability of treatment (assessed by the number who dropped out during the intervention), the risk ratio was 1.00 (95% Cl 0.97 to 1.04).

Seven trials compared exercise with psychological therapy (189 participants), and found no significant difference (SMD = -0.03, 95% Cl -0.32 to 0.26). Four trials (n=300) compared exercise with pharmacological treatment and found no significant difference (SMD = -0.11, 95% Cl -0.34 to 0.12). One trial (n=18) reported that exercise was more effective than bright light therapy (SMD=-6.40, 95% Cl -10.20 to -2.60).

For each trial that was included, we independently assessed for sources of bias in accordance with the Cochrane Collaboration 'risk of bias' tool. In exercise trials, there are inherent difficulties in masking both those receiving the intervention and those delivering the intervention. Many trials used participant selfreport rating scales as a method for post-intervention analysis, which also has the potential to bias findings.

Authors' conclusions

Exercise is moderately more effective than a control intervention for reducing symptoms of depression, but analysis of methodologically robust trials only shows a smaller effect in favour of exercise. When compared with psychological or pharmacological therapies, exercise appears to be no more effective, though this conclusion is based on a few small trials.

Assessed as up to date: July 13, 2012 See more at: http://summaries.cochrane.org/CD004366/exercise-fordepression#sthash.bWhY63KK.dpuf

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