Behavioural activation therapies for depression in adults with non-communicable diseases (Protocol)


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ABSTRACT

This is a protocol for a Cochrane Review (Intervention). The objectives are as follows:

The primary objective is to examine the effects of behavioural activation compared to any control group for the treatment of depression in adults with non-communicable diseases (NCDs). The secondary objectives is to examine the effects of behavioural activation compared to each control group separately (no treatment, waiting list, other psychological therapy, pharmacological treatment, or any other type of treatment as usual) for the treatment of depression in adults with NCDs.
**BACKGROUND**

**Description of the condition**

**Depression**

The term ‘depression’ is often used to describe major depressive disorder when diagnosed in a clinical setting. It is characterised by a period of at least two weeks of depressed mood, or a persistent loss of interest or pleasure in activities which were previously considered enjoyable, or both (APA 2013). A range of symptoms may accompany these key features of depression and reduce quality of life. These include weight loss or weight gain, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue, loss of energy, feelings of excessive guilt and worthlessness, diminished concentration, and recurrent thoughts of death (APA 2013).

Depression is the fifth global cause of disease burden in terms of years lived with a disability (YLD), and was ranked in the top 10 of YLD in 191 out of 195 countries worldwide (Vos 2017). In 2014, 7.1% of the population living in the 28 countries of the European Union was estimated to report depression, with higher rates reported by women, older people, and people living in cities (Eurostat 2014). In a national survey conducted in the USA, the 12-month and lifetime prevalence of depression were 10.4% and 20.6%, respectively (Hasin 2018). Similarly, the lifetime prevalence for depression in England was 19% in 2014 (HSE 2014). A meta-analysis of data from 35 countries found a 52% increased risk of mortality in people with depression (Cuijpers 2014). Global estimates of the burden of disease show that 4.4% of people worldwide suffer from depressive disorder. These figures vary considerably depending on geographical regions; for depression, rates vary from 3.6% in the Western Pacific to 5.4% in Africa. More than 80% of people who have mental disorders live in low- and middle-income countries (Rathod 2017).

Depressive disorders can have a long-lasting impact on patients, their families, and wider society. They often co-occur with anxiety disorders (WHO 2017), and are associated with marked personal and societal economic losses due to healthcare costs for mental and comorbid physical healthcare, reduced productivity in the workplace, and years of life lost (Alonso 2011; Greenberg 2015).

**Non-communicable diseases**

Non-communicable diseases (NCDs) are chronic diseases caused by a combination of genetic physiological, environmental and behavioural factors. The four most common physical NCDs are cardiovascular disease (CVD), cancer, chronic respiratory diseases, and type 2 diabetes (WHO 2017). According to the World Health Organization (WHO), 41 million people die annually due to NCDs, corresponding to 71% of all deaths worldwide. NCDs affect people of all age groups, 15 million occurring between the ages of 30 and 69 years (WHO 2017). Despite the resurgence of certain infectious diseases, such as tuberculosis and dengue, the global burden of infectious disease overall is decreasing (or becoming more stagnant in some countries), and being replaced by an increased burden of disease for non-communicable diseases, as well as common mental disorders (Vos 2017). NCDs decrease patients’ health-related quality of life substantially (Dyer 2010; Solli 2010).

**Comorbidity of depressive disorders in patients with non-communicable diseases**

NCDs commonly co-occur with depressive disorders (Patel 2015). There is a complex bidirectional association between depression and NCDs (Ngo 2013). Co-existence of depression with a NCD worsens outcomes for both conditions and is associated with poorer self-management and treatment adherence, reduced treatment response and higher morbidity and mortality for both the mental and the physical disorder (BPS 2010; NICE 2009). NCDs and mental disorders are associated with similar behavioural factors, such as tobacco use, unhealthy diet, physical inactivity, and harmful alcohol use (Stein 2019). Pathophysiological factors, such as increased cytokine levels or other inflammatory markers may increase the risk of developing and worsening depression (Katon 2003).

CVD is the leading cause of death globally (Roth 2017). Comorbid depression is common in CVD patients (approximately 15%), and the prevalence of depression in patients with CVD is higher than in the general population (Hare 2014). Increased levels of depression in postmyocardial infarction patients is associated with a 1.6- to 2.7-fold increased risk of impaired outcomes within 24 months of the event (Meijer 2011). The association between depression and CVD risk factors is bidirectional (Pan 2012). Depression is thought to be a risk factor for CVD through a combination of behavioural (smoking, alcohol intake, physical inactivity, and obesity) and biological components (affecting the nervous system, hormone secretion, immune system, and cardiovascular functions) (Dhar 2016).

Prevalence estimates of major depression (15%), minor depression (20%), and anxiety disorders (10%) in patients treated for cancer are more than double that observed in the general population. Two-thirds of patients with cancer and depression also have clinically significant anxiety symptoms. Figures vary by cancer type and it is suggested that this is due to the differing prognoses, pain levels, and degrees of body image disruption associated with each tumour type, as well as specific tumour-related neuropsychiatric effects and treatment-related neuropsychiatric side effects (Pitman 2018).

Mental health problems are approximately three times more prevalent among people with chronic obstructive pulmonary disease (COPD) than in the general population (NICE 2009). Patients with COPD show increased levels of psychological distress, which in turn leads to increased exacerbation rates. Up to 55% of patients suffering from COPD also suffer from anxiety and depression (Laurin 2012). In the UK, it was reported that mortality rates for people with comorbid asthma and depression were twice the level among those with asthma alone (Walters 2011).

It is estimated that depression occurs in 13% to 18% of diabetic patients, which worsens glycaemic control and is associated with increased complications. Mild depression is thought to often go undiagnosed in diabetic patients because many of the somatic symptoms are similar (Hermans 2013).

**Description of the intervention**

Pharmacological and psychological interventions, alone or in combination, are recommended in clinical guidelines for the treatment of mild to moderate depression. Behavioural activation is one of the recommended therapies (NICE 2009).
Antidepressants are a standard treatment for moderate to severe depression in healthcare settings, whereas for subthreshold depressive symptoms or mild depression, low-intensity psychosocial therapy and psychological therapies are recommended (NICE 2009). Although antidepressants have shown efficacy in the treatment of depression, non-adherence to antidepressant medication is common (Hunot 2007; Ten Doeschate 2009; Van Geraett 2009), and can lead to relapse and recurrence of depression (Gardsarsdottir 2009). Non-adherence is related to a multitude of factors, including concerns about antidepressants relating to side effects, dependence, and experience of withdrawal symptoms (Davies 2018; Hunot 2007; Sansone 2012). Studies of treatment for psychiatric disorders, including depression, consistently report that patients prefer psychological treatment to medication (McHugh 2013).

There is a wide range of psychological therapies available for the treatment of depressive disorders. Psychological therapies may be categorised into four philosophical and theoretical schools of thought, comprising psychoanalytic/dynamic (Freud 1945; Jung 1963; Klein 1960), behavioural (Skinner 1953; Watson 1924; Wolpe 1958), humanistic (Maslow 1943; May 1961; Rogers 1951), and cognitive approaches (Beck 1979; Lazarus 1971). Each school of thought incorporates several different and overlapping psychotherapeutic approaches.

Behavioural activation stems from a behavioural psychotherapy approach first developed in the 1970s by Lewishohn and colleagues (Dimidjian 2011). It is based on the concept that depression results from deprivation of positive reinforcement, and the treatment focuses on identifying and scheduling pleasurable activities, thus increasing contact with sources of positive reinforcement (Kanter 2012).

When cognitive behavioural therapy (CBT) was developed and disseminated, behavioural activation approaches based purely on operant (learning from the consequences of behaviours) and respondent (responsive behaviour as a result of a stimulus) principles were thought insufficient. However, the interest in the feasibility of behavioural treatments for depression has since been renewed (Dimidjian 2011; Ekers 2014; Hopko 2003). Jacobson showed that the behavioural component of CBT was as effective as the full package of CBT, and investigators developed a new and more comprehensive model of behavioural activation that would be amenable to dissemination (Jacobson 1996; Jacobson 2001).

How the intervention might work

Skinner proposed that depression was associated with an interruption in established sequences of healthy behaviour that were previously positively reinforced by the social environment and were based on operant conditioning principles (in which behaviour patterns are learnt, rather than instinctive) (Skinner 1953). In subsequent expansions of this model, reduction of positively reinforced healthy behaviours has also been attributed to a decrease in the number and range of reinforcing stimuli available to the individual, lack of skill in obtaining positive reinforcement (Lewinsohn 1974), increased frequency of punishment, or a combination of two or all of these (Lewinsohn 1984).

Behavioural activation can be defined as a brief psychotherapeutic approach that seeks to change the way a person interacts with their environment, aiming to:

1. increase access to positive reinforcers of healthy behaviours;
2. reduce avoidance behaviours that limit access to positive reinforcement; and
3. understand and address barriers to activation.

Treatments are collaborative and focused on the present. Many differing techniques are incorporated into treatment; however all use self-monitoring of a mood-environment link and scheduling of new or adaptive behaviours to meet targets (Kanter 2012). In doing so, the therapy helps people to make contact with potentially reinforcing experiences (Jacobson 2001).

The original model of behavioural activation, developed by Jacobson, was defined primarily by the elimination of cognitive intervention elements (Dimidjian 2006). On the basis of its original design, behavioural activation model components commonly include developing a shared treatment rationale; increasing access to pleasant events, activities, and consequences; activity scheduling and developing social skills self-monitoring links between behaviour and mood; and activity scheduling to promote contact with sources of positive reinforcement from the person’s environment. In some cases the use of some form of problem solving or functional analysis is added to overcome any potential barriers to the scheduling of activities. No attempt is made to directly restructure cognitions, however the exploration of the consequence of rumination in restricting access to positive reinforcement is a common focus of the approach (Kanter 2012; Veale 2009). The treatment can result in significant neurobiological changes to the brain’s reward circuitry (Kanter 2012).

It is thought that behavioural activation could be effective in the treatment of patients with depression and comorbid NCDs by supporting people to identify activities they would like to engage in and reintroduce valued activities that they have stopped doing. Positive reinforcement from valued activities through self-monitoring, activity scheduling, and functional analysis helps to break the vicious cycle of limiting activities and depressive symptoms.

Why it is important to do this review

According to the clinical guidelines produced by the National Institute for Health and Clinical Excellence (NICE), behavioural activation is one of the recommended treatment options for subthreshold depressive symptoms, mild to moderate depression, and severe depression, along with CBT and interpersonal therapy. However, the guidelines acknowledge that evidence for behavioural activation is currently less robust than for the other recommended therapies (NICE 2009).

Behavioural activation is increasingly receiving attention as a potentially cost-effective intervention for common mental disorders, including for populations with comorbid NCDs, and it may be easier to deliver and implement than other psychological therapy models because it can be delivered in less sessions, over a shorter period of time, and by mental health workers who are not specialists (Richards 2016). Given this resurgence of interest, a comprehensive review of the comparative effectiveness and acceptability of behavioural activation interventions for common mental disorders in this patient group with NCDs is now timely to inform and update clinical practice and future clinical guideline development.

A Cochrane Review on the effectiveness of behavioural activation for the treatment of depression is ongoing (Uphoff 2019). To allow
for meaningful meta-analyses in a relatively homogenous patient population, people with comorbidities were excluded from that review. The current review will fill this gap in the literature by specifically addressing the population of people with depression and co-morbid NCDs.

**OBJECTIVES**

The primary objective is to examine the effects of behavioural activation compared to any control group for the treatment of depression in adults with non-communicable diseases (NCDs). The secondary objectives is to examine the effects of behavioural activation compared to each control group separately (no treatment, waiting list, other psychological therapy, pharmacological treatment, or any other type of treatment as usual) for the treatment of depression in adults with NCDs.

**METHODS**

Criteria for considering studies for this review

**Types of studies**

For consistency and to facilitate interpretation of the results of this review in the wider context of evidence on behavioural activation for depression, we will follow methods described in the published protocol 'Behavioural activation therapies for depression in adults' where possible (Uphoff 2019).

Randomised controlled trials (RCTs) will be eligible for inclusion in this review. We will include trials employing a cross-over design (whilst we acknowledge that this design is rarely used in psychological therapy trials), but we will only use data from the first active treatment phase. Cluster-RCTs are also eligible for inclusion.

Quasi-RCTs, in which treatment assignment is decided through methods such as alternate days of the week, are not eligible for inclusion.

**Types of participants**

**Participant characteristics**

Trials with men and women aged 18 years and over are eligible for inclusion. We will exclude trials that contain participants under 18 years of age. If a trial includes both adults and children, we will contact authors to request data for adult participants only. If these data are not available, we will exclude the trial. Participants must have depression (mild, moderate or severe) with a comorbid non-communicable disease (NCD). NCDs to be included are the four most prevalent NCDs worldwide: cardiovascular disease (CVD), cancer, chronic respiratory disease and type 2 diabetes. Postnatal depression is considered a separate condition with contributing factors distinct from major depressive disorder, and we will therefore exclude patients with this condition. We will also exclude participants with subthreshold depression.

**Setting**

Trials could be conducted in a primary, secondary, specialist, or community setting.

We will exclude trials involving inpatients, as these represent settings which differ with regards to the complexity of patients' healthcare needs, the way patients access care, and the way in which interventions are delivered and embedded in clinical practice. The same intervention may therefore lead to different results in inpatient settings compared to other settings, and we would not be able to ascertain whether this would be a result of the type of participants, the delivery of the intervention, or features of the setting itself. If a trial includes both inpatients and outpatient settings, we will contact authors to request data for participants eligible for inclusion in our review only. If these data are not available, we will exclude the trial.

Nursing homes in this review are considered outpatient settings, as they are places of residence. Hospice care is considered specialised medical care and we will therefore exclude studies conducted with participants in a hospice.

We will include trials that focus on specific populations - nurses, care givers, participants at a specific workplace with depression - if all participants meet the criteria for depression.

We will include studies from all countries.

**Diagnosis**

We will include all trials that focus on acute phase treatment of clinically diagnosed depression in patients with comorbid NCDs (CVD, cancer, chronic respiratory disease and type 2 diabetes).

We will include trials adopting any standardised diagnostic criteria to define participants suffering from an acute phase unipolar depressive disorder. Accepted diagnostic criteria include Feighner criteria, Research Diagnostic Criteria and criteria of the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III)*; *APA 1980*, *DSM-III-Revised (APA 1987)*, *DSM-Fourth Edition (DSM-IV; APA 1994)*, *DSM-IV-Text Revision (APA 2000)*, *DSM-Fifth Edition (DSM-5; APA 2013)*, and *International Classification of Diseases, Tenth Edition (ICD-10; WHO 1992)*. Earlier trials may have used *ICD-Ninth Edition (ICD-9; WHO 1978)*, but ICD-9 is not based on operationalised criteria, so we will exclude trials using ICD-9 to diagnose depression.

We include participants diagnosed with anxiety, or with symptoms of anxiety, as long as they are also diagnosed with depression.

**Types of interventions**

**Experimental interventions**

Previously published Cochrane Reviews for treatment of depression provided a framework for psychological therapies including behavioural therapy (Churchill 2013; Hunot 2013; Shinohara 2013). Given recent developments in literature and practice regarding behavioural activation approaches, we consider behavioural activation as part of behavioural therapies, rather than being classified as a ‘third wave’ therapy. In line with the behavioural therapy for depression review (Uphoff 2019), we created the comparator categories of psychological therapies on the basis of both treatment approach (e.g. their theoretical background and the manuals used) and content (e.g. therapeutic techniques employed). See also Appendix 1.

**Behavioural activation**

We will include trials evaluating treatment approaches for depression and anxiety that are either explicitly called ‘behavioural activation’, or treatments that are described using the main elements of behavioural activation for depression, such as pleasant events and activities, activity scheduling, positive reinforcement from the
environment, positive interaction or re-engagement with the environment. This means that we will include behavioural therapies in the treatment group as long as they are described using the main elements of behavioural activation. Interventions that contain some elements of behavioural therapy, such as cognitive behavioural therapy (CBT) or problem solving therapy, are not eligible for inclusion.

**Format of psychological therapies**

Therapies delivered by therapists of all levels are eligible for inclusion. This includes psychologists or psychotherapists accredited by a professional body for psychology or psychotherapy, who completed formal training to deliver psychological therapies, as well as lay counsellors and non-specialist therapists who have been specifically trained to deliver treatment according to a behavioural activation protocol.

We will include computerised and self-help interventions if they were facilitated by a therapist. This means at least some element of interaction with a therapist is required.

Psychological therapies conducted on an individual or group basis are eligible for inclusion.

The number of sessions is not limited, and we accept psychological therapies delivered in only one session.

**Comparators**

All comparators are accepted as long as they are not a type of behavioural activation. We categorise psychological therapies as behavioural therapy, social skills training/assertiveness training, relaxation therapy, CBT, third wave CBT, psychodynamic, humanistic and integrative approaches.

**Behavioural therapy**

If we identify any behavioural therapies that do not contain the main elements of behavioural activation, we will include them as comparators.

**Social skills training/assertiveness training**

The social skills training model proposes that depressed people may have difficulty initiating, maintaining and ending conversations (Jackson 1985). Because of these deficits, the individual is unable to elicit mutually reinforcing behaviour from other people in his or her environment. Social skills training subserves assertive assertion and conversational skills, together with more specialised sub-skills, such as dating and job interview skills. Different social contexts may be targeted, for example interaction with friends, family members, people at school, or at work, and interventions such as instruction, modelling, rehearsal, feedback and reinforcement are used to enable the development of new responses (Jackson 1985). As assertiveness training represents a key component of social skills training, we included it in this category.

**Relaxation therapy**

Relaxation training is a behavioural stress management technique that induces a relaxation response, helping to switch off the fight/flight response and causing levels of stress hormones in the bloodstream to fall. A variety of techniques may be used to induce relaxation, the most common of which is Jacobson’s progressive muscle relaxation training (Bernstein 1973).

**Cognitive behavioural therapies (CBTs)**

In CBT, therapists aim to work collaboratively with patients to understand the link between thoughts, feelings, and behaviours, and to identify and modify unhelpful thinking patterns, underlying assumptions and idiosyncratic cognitive schemata about the self, others and the world (Beck 1979). Cognitive change methods for depression are targeted at the automatic thought level in the first instance and include thought catching, reality testing and task assigning as well as generating alternative strategies (Williams 1997). Behavioural experiments are then used to re-evaluate underlying beliefs and assumptions (Bennett-Levy 2004). We categorised these therapies into six subcategories: cognitive therapy, rational emotive behaviour therapy, problem solving therapy, self-control therapy, a coping with depression course and other CBTs.

**Third wave cognitive and behavioural therapies (third wave CBTs)**

Third wave CBT approaches conceptualise cognitive thought processes as a form of ‘private behaviour’ (Hayes 2006; Hofmann 2008). Third wave CBTs target the individual’s relationship with cognitions and emotions, focusing primarily on the function of cognitions, such as thought suppression or experiential avoidance (an attempt or desire to suppress unwanted internal experiences, such as emotions, thoughts and bodily sensations (Hofmann 2008). A range of strategies, including mindfulness exercises, acceptance of unwanted thoughts and feelings and cognitive diffusion (stepping back and seeing thoughts as just thoughts), are used to bring about change in the thinking process. Drawing from psychodynamic and humanistic principles, third wave CBT approaches often place great emphasis on use of the therapeutic relationship. We categorised these therapies into subcategories: acceptance and commitment therapy, compassionate self mind training, functional analytic psychotherapy, metacognitive therapy, mindfulness-based cognitive therapy, dialectical behaviour therapy and other third wave CBTs.

**Cognitive behavioural therapy bibliotherapy**

When the patient does not have access to a qualified therapist or CBT practitioner they may seek therapy through the use of self-help materials incorporating a CBT approach (Anderson 2005).

**Psychodynamic therapies**

Grounded in psychoanalytic theory (Freud 1949), psychodynamic therapy uses the therapeutic relationship to explore and resolve unconscious conflict through transference and interpretation, with development of insight and circumscribed character change as therapeutic goals, and relief of symptoms as an indirect outcome. Brief therapy models have been devised by Malan 1963, Mann 1973 and Strupp 1984. Psychodynamic therapies include those based on a drive/structural model (Freud 1949), relational model (Luborsky 1988; Strupp 1984), and integrative analytic model (Mann 1973), among others.

**Humanistic therapies**

Contemporary models of humanistic therapies differ from one another somewhat in clinical approach, but all focus attention on the therapeutic relationship (Cain 2002), within which therapist ‘core conditions’ of empathy, genuineness and unconditional positive regard (Rogers 1951), are regarded as cornerstones for facilitating client insight and change. These include the following subcategories: person-centred therapy (Rogerian), gestalt therapy, expe-
Integrative therapies are approaches that combine components of different psychological therapy models. Integrative therapy models include interpersonal therapy (Klerman 1984), cognitive analytic therapy (Ryle 1990), and Hobson’s conversational model (Hobson 1985), manualised as psychodynamic interpersonal therapy (Shapiro 1990). With its focus on the interpersonal context, interpersonal therapy was developed to specify what was thought to be a set of helpful procedures commonly used in psychotherapy for depressed outpatients (Weissman 2007), drawing in part from attachment theory (Bowlby 1980), and CBT within a time-limited framework. Cognitive analytic therapy, also devised as a time-limited psychotherapy, integrates components from cognitive and psychodynamic approaches. The conversational model integrates psychodynamic, interpersonal and person-centred model components.

Counselling interventions traditionally draw from a wide range of psychological therapy models, including person-centred, psychodynamic and cognitive behavioural approaches, applied integratively, according to the theoretical orientation of practitioners (Stiles 2008). Therefore, we will usually include trials of counselling with integrative therapies. However, if the counselling intervention consists of a single discrete psychological therapy approach, we will categorise it as such, even if the intervention is referred to as ‘counselling’. If the intervention is manualised, this will inform our classification.

Motivational interviewing and other forms of integrative therapy approaches are also included in this category.

**Waiting list**

Participants are randomly assigned to the active intervention group or control group, and they will either receive the intervention first or be assigned to a waiting list until all participants in the intervention group have received the intervention. During the course of the trial, people on the waiting list can receive any appropriate medical care.

**Attention placebo**

We define this as a control condition that is regarded as inactive by both researchers and participants in a trial.

**Psychological placebo**

We define this as a control condition in a trial that is regarded by researchers as inactive but is regarded by participants as active (also called placebo therapy or sham treatment).

**Medication**

All evidence-based pharmacotherapy, which will predominantly include antidepressants (e.g. selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, mirtazapine, bupropion, nonselective monoamine oxidase inhibitors); any dose, route of administration, duration, and frequency.

**Medical placebo**

All types of medical placebos or ‘sugar pills’. No treatment

Trial participants not receiving any treatment for depression during the course of the trial.

**Excluded interventions**

We will exclude trials of long-term, continuation or maintenance therapy interventions designed to prevent relapse of depression or to treat chronic depressive disorders from this review. Similarly, we will exclude trials of interventions designed to prevent a future episode of depression.

We will exclude psychological therapy models based on social constructionist principles (that focus on the ways in which individuals and groups participate in the construction of their perceived social reality), including couples therapy (Jacobson 1993), family therapy (Crane 2002), solution-focused therapy (de Shazer 1988), narrative therapy (White 1990), personal construct therapy (Kelly 1955), neurolinguistic programming (Bandler 1982) and brief problem solving (Watzlavick 1974). These therapies work with patterns and dynamics of relating within and between family, social and cultural systems to create a socially constructed framework of ideas (O’Connell 2007), rather than focusing on individuals’ reality. A previously published Cochrane Review on couples therapy for depression has recently been updated (Barbato 2018), and a review of family therapy for depression is to be updated (Henken 2007).

**Types of outcome measures**

**Primary outcomes**

1. Treatment efficacy for depression: the number of participants who responded to treatment, as determined by changes in scores for Beck Depression Inventory (BDI; Beck 1961), Hamilton Rating Scale for Depression (HAMD-HDRS; Hamilton 1960), or Montgomery-Asberg Depression Rating Scale (MADRS; Montgomery 1979), or in scores from any other validated depression scale. We will use HAMD-D, and if this is not available we will use MADRS, and if the latter is not available then we will use BDI. If BDI is not available we will use the measure most frequently used across trials. Many trials define response as 50% or greater reduction on BDI, HAMD-D, etc., with some trials defining response using Jacobson’s Reliable Change Index (Jacobson 1992); we accepted the trial authors’ original definition and preferred Jacobson’s Reliable Change Index if this was used in addition to other response outcomes.

2. Treatment acceptability: the number of participants who dropped out of psychological therapy for any reason at all reported time points.

**Secondary outcomes**

1. Improvement in depression symptoms, based on a continuous outcome of group mean scores at the end of treatment. If multiple measures have been used for this outcome within one trial, we will adopt the same hierarchy used for the primary outcome ‘treatment efficacy for depression’.

2. Quality of life, as assessed with the use of validated measures such as Short Form (SF)-36 (Ware 1993), EQ-5D (EuroQol; Brooks 1995), and World Health Organization Quality of Life (WHOQOL; WHOQOL 1998).

3. Social adjustment and social functioning, including Global Assessment of Function scores (Luborsky 1962).
4. Improvement in anxiety symptoms, as measured using a validated continuous scale, either assessor-rated, such as the Hamilton Anxiety Scale (HAM-A; Hamilton 1959), or self-report, including the Trait subscale of the Spielberger State-Trait Anxiety Inventory (STAI-T; Spielberger 1983), and the Beck Anxiety Inventory (BAI; Beck 1988). We will use HAM-A, and if this is not available we will use STAI-T, and if this is not available we will use BAI. If BAI is not available we will use the measure most frequently used across trials.

5. We will collect any data on adverse effects, such as counts of completed suicides, attempted suicides, or worsening of symptoms for each study and summarise the data in narrative form.

Search methods for identification of studies

Electronic searches

An Information Specialist will conduct searches on the following bibliographic databases using relevant subject headings (controlled vocabularies) and search syntax, appropriate to each resource. The search strategies will be designed to identify RCTs of 'behavioural activation', or the main elements of behavioural activation in participants with the four most common physical NCDs (cardiovascular disease (CVD), cancer, chronic respiratory diseases, and type 2 diabetes) who have also been clinically diagnosed with a depressive disorder. We will search the databases below, including global health databases to capture emerging evidence on the effectiveness of behavioural activation in populations with comorbidities in low- and middle-income countries.

1. Cochrane Common Mental Disorders Trials Register (CCMD-CTR), all available years.
2. Cochrane Central Register of Controlled Trials (CENTRAL; current issue).
3. Ovid MEDLINE (1946 onwards) (Appendix 2).
5. Ovid PsycINFO (1806 onwards).
7. Latin American and Caribbean Health Sciences Literature (LILACS), all available years (via WHO Global Health Index Medicus).
8. African Index Medicus, all available years (via WHO Global Health Index Medicus).
9. Index Medicus for the South East Asia Region (ISMEAR), all available years (via WHO Global Health Index Medicus).

We will not apply any restrictions on date, language or publication status to the searches.

We will search international trials registries via the World Health Organization’s trials portal (ICTRP), and ClinicalTrials.gov to identify unpublished or ongoing trials.

We will rerun all searches close to publication if the initial search date is greater than 12 months. We will also search for any relevant retraction statements and errata.

Searching other resources

Grey literature

We will search the following sources of grey literature (primarily for dissertations and theses).

4. ETHOS - the British Libraries e-theses online service (ethos.bl.uk).
5. Open Access Theses and Dissertations (oatd.org).

Reference lists

We will check the reference lists of all included trials and relevant systematic reviews to identify additional trials missed from the original electronic searches (e.g. unpublished or in-press citations).

Personal communication

We will contact trial authors and subject experts for information on unpublished or ongoing trials, or to request additional trial data.

Data collection and analysis

Selection of studies

At least two review authors (EU, MPI) will examine the abstracts of all publications obtained through the search strategy. We will then obtain full articles of all trials identified by any one of the review authors and two review authors (EU, MPI, DB, PM, MPU, RR) will independently assess full-texts according to the criteria relating to characteristics of the studies, participants, and interventions. We will discuss reasons for disagreement with a third review author (EU, RC, NS, or DE) and contact external experts or trial authors if necessary in order to reach agreement. We will record reasons for excluding records at this stage. For all included studies, we will link multiple reports from the same study. We will present a PRISMA flow diagram to show the process of study selection (Moher 2009).

Data extraction and management

At least two review authors (EU, DB, PM, MPU, RR) will independently extract data from each trial. These review authors will discuss any disagreement with an additional review author (EU, RC, NS, or DE) and, when necessary, will contact the authors of the trials for further information.

We will extract and enter into a spreadsheet information regarding the following: trial population, sample size, interventions, comparators, potential biases in the conduct of the trial, source of funding, outcomes (including adverse events, number needed to treat for an additional beneficial outcome (NNTB)), follow-up and methods of statistical analysis.

Management of time points

We plan to summarise and categorise post-treatment outcomes and outcomes at each reported follow-up point as follows: short-term (up to 6 months post-treatment), medium-term (7 to 12 months post-treatment) and long-term (longer than 12 months).

Assessment of risk of bias in included studies

We will assess risk of bias for each included trial using the Cochrane 'Risk of bias’ tool (Higgins 2016), which considers the following domains:

1. Risk of bias arising from the randomisation process, including allocation and randomisation.
2. Risk of bias due to deviations from the intended interventions, including blinding of participants and people delivering the interventions.
3. Missing outcome data.
4. Risk of bias in measurement of the outcome, including blinding of outcome assessors.
5. Selective outcome reporting.
6. Other sources of bias.

For cluster-RCTs and cross-over trials, we will use the templates specifically designed to assess these types of trials, with the same domains.

In the 'Other sources of bias' domain we will consider any additional problems with bias, including the following issues specific to psychological therapy trials.

1. Treatment fidelity: was the therapy monitored against a manual or a scale through audiotapes or videotapes?
2. Researcher allegiance/conflict of interest: did the researcher have a vested interest for or against the therapies under examination?
3. Therapist allegiance/conflict of interest: did the therapist have a vested interest for or against the therapies provided?

We will make a judgement on the risk of bias for each domain within and across trials, and categorise this as low, unclear, or high risk of bias.

Two review authors (EU, DB, PM, MPu, RR) will independently assess the risk of bias in selected trials and discuss any disagreements with a third review author (EU, RC, NS, or DE). Where necessary, we will contact trial authors for further information. We will present all 'Risk of bias' data graphically, and narratively in the text. We will use allocation concealment as a marker of trial quality for the purpose of undertaking sensitivity analyses.

**Measures of treatment effect**

**Continuous outcomes**

Where trials use the same outcome measure for comparison, we will pool data by calculating the mean difference (MD). When trials use different measures to assess the same outcome, we will pool data with standardised mean difference (SMD) and calculate 95% confidence intervals (CIs).

A SMD of zero means that the intervention and control groups have equivalent treatment effects. We anticipate that, for most measures, a lower score will indicate greater improvement. For example, a lower score on depression symptom instruments indicates an improvement in symptoms. In these cases, a SMD less than zero indicates that the intervention has a greater effect than the control. A SMD greater than zero indicates that the intervention has a smaller effect than the control. Interpretation of the SMD is reversed in cases where a greater continuous score indicates greater improvement.

**Dichotomous outcomes**

We will analyse dichotomous outcomes by calculating a pooled risk ratio (RR) and 95% CIs for each comparison.

In addition, we will calculate the number needed to treat to benefit (NNTB) with 95% CIs for all dichotomous outcomes to facilitate interpretation; this is the expected number of people who need to receive the intervention rather than the comparator for one additional person to achieve a beneficial outcome (Schünemann 2017).

If one trial uses both continuous and dichotomous variables for the same outcome, we will give preference to the continuous outcome. If different outcomes are used, for example depression score and clinical depression yes/no, we will report both.

**Unit of analysis issues**

**Cluster-randomised trials**

We will include cluster-randomised trials as long as proper adjustment for the intracluster correlation can be conducted in accordance with the Cochrane Handbook for Systematic Reviews of Interventions (Higgins 2011a).

**Cross-over trials**

We will include trials employing a cross-over design in the review, but we will only use data from the first active treatment phase.

**Trials with multiple treatment groups**

Multiple-arm trials (those with more than 2 intervention arms) can pose analytical problems in pair-wise meta-analysis. For trials with more than two relevant active treatment arms, we will manage data in this review as follows.

**Dichotomous data**

We will collapse data from relevant active intervention arms into a single arm for comparison, or we will split data from relevant active treatment arms equally between comparator arms (Higgins 2011b).

**Dealing with missing data**

We will manage missing dichotomous data through intention-to-treat (ITT) analysis, in which we will assume that participants who dropped out after randomisation had a negative outcome. We also plan to conduct best/worse case scenarios for the clinical response outcome, in which we will assume that dropouts in the active treatment group had positive outcomes and those in the control group had negative outcomes (best case scenario), and that dropouts in the active treatment group had negative outcomes and those in the control group had positive outcomes (worst case scenario), thus providing boundaries for the observed treatment effect. If a large amount of information is missing, we will give these best/worst case scenarios greater emphasis in the presentation of results.

We will analyse missing continuous data on an endpoint basis, including only participants with a final assessment, or by using the last observation carried forward (LOCF) to the final assessment, if trial authors report LOCF data. When SDs are missing, we will attempt to obtain these data by contacting trial authors. When SDs are not available from trial authors, we will calculate them from P values, t-values, CIs or standard errors, if these are reported in the articles (Deeks 1997).

If a vast majority of SDs are available and only a minority of SDs are unavailable or unobtainable, we plan to use the method devised by...
Furukawa and colleagues to impute SDs and calculate percentage responders (da Costa 2012; Furukawa 2005; Furukawa 2006). If we use this method, we will interpret data with caution and will take into account the degree of observed heterogeneity. We will also undertake a sensitivity analysis to examine the effect of the decision to use imputed data.

If additional figures are not available or obtainable and it is not deemed appropriate to use the Furukawa method as described above, we will not include the trial data in the comparison of interest.

Assessment of heterogeneity

We will assess statistical heterogeneity using the Chi^2 test, which provides evidence of variation in effect estimates beyond that of chance. Because the Chi^2 test has low power to assess heterogeneity when a small number of participants or trials are included, we will conservatively set the P value at 0.1 (Deeks 2017). We will also quantify heterogeneity using the I^2 statistic, which calculates the percentage of variability due to heterogeneity rather than to chance (Higgins 2003). We consider I^2 statistic values in the range of 50% to 90% to represent substantial statistical heterogeneity and will explore them further. However, the importance of the observed I^2 statistic depends on the magnitude and direction of treatment effects and the strength of evidence for heterogeneity. Forest plots generated in Review Manager 5 (RevMan 5; Review Manager 2014), will provide an estimate of tau^2, the between-trial variance in a random-effects meta-analysis (Deeks 2017). To provide an indication of the spread of true intervention effects, we will also use the tau^2 estimate to determine an approximate range of intervention effects for the primary outcome using the method outlined in the Cochrane Handbook for Systematic Reviews of Interventions (Deeks 2017).

Assessment of reporting biases

As far as possible, we will minimise the impact of reporting biases by undertaking comprehensive searches of multiple sources (including trials registries), to identify unpublished material and including non-English language publications.

We will also try to identify outcome reporting bias in trials by recording all trial outcomes, planned and reported, and noting where outcomes are missing. If we find evidence of missing outcomes, we will attempt to obtain any available data directly from the trial authors.

We plan to construct funnel plots to establish the potential influence of reporting biases and small-trial effects (Sterne 2017).

Data synthesis

We plan to conduct a meta-analysis of included trials. Given the potential heterogeneity of behavioural activation approaches for inclusion, together with the likelihood of differing secondary comorbid mental disorders and different NCDs in the population of interest, we will use a random-effects model in all analyses.

Subgroup analysis and investigation of heterogeneity

Clinical heterogeneity

We plan to conduct the following subgroup analyses for primary outcomes treatment efficacy and treatment acceptability, for the main comparison 'behavioural activation versus any control group'.

1. Country: we plan to conduct subgroup analyses with studies conducted in high-income countries and studies conducted in low- and middle-income countries, as we expect the study setting to influence heterogeneity. Countries are grouped according to the World Bank income classification (The World Bank 2019).

2. Level of therapist: we plan to conduct analyses separately for specialist (qualified or accredited mental health specialist with substantial training), non-specialist (short training, lay workers or primary care workers) therapists, or specialist in training (e.g. several years of training in psychotherapy or mental health nursing). Although psychotherapy has traditionally been delivered by mental health specialists, the effectiveness of behavioural activation delivered by non-specialists is of great interest in low-resource settings such as low- and middle-income countries.

3. Type of NCD: we plan to analyse data for subgroups: CVD, cancer, chronic respiratory disease and type 2 diabetes because we expect that these different NCDs might affect mental health differently, and factors associated with these diseases might influence success of behavioural activation therapy.

Sensitivity analysis

We plan to conduct the following sensitivity analyses for primary outcomes treatment efficacy and treatment acceptability, for the main comparison 'behavioural activation versus any control group'.

1. Trial quality: we will exclude low quality trials in a sensitivity analysis, if we identify a number of higher quality trials. As a marker of quality, we will use the 'allocation concealment' criteria from the 'Risk of bias' assessment.

2. Mode of delivery: we will exclude therapies delivered through computer-based or electronic guidance without a substantial face-to-face component.

3. Group therapy: we will exclude trials of group therapy for behavioural activation as the mode of delivery of psychotherapy could influence effectiveness of the therapy.

'Summary of findings' table

We plan to construct a 'Summary of findings' table to present the main findings of the review. We report the outcomes listed below and present standardised effect size estimates and 95% CIs. Two review authors (EU, MP, DB, RR, PM, MPu, RR) will independently use the GRADE approach to assess the quality of the evidence for each outcome, and agreement will be sought between them, if necessary with help from a third review author (EU, RC, DS, DE) (Schünemann 2017). We will use GRADEproGDT to create our 'Summary of findings' table (GRADEpro), and follow standard methods as described in the Cochrane Handbook for Systematic Reviews of Interventions to prepare our 'Summary of findings' table (Schünemann 2011).

In line with our first objective, the comparison included in the 'Summary of findings' table will be behavioural activation versus any control group.

We will include the following outcomes (measured up to 24 months) in the 'Summary of findings' table.

1. Treatment efficacy (number of participants responding to treatment).
2. Treatment acceptability (number of participants who dropped out).
3. Improvement in depression outcomes as a continuous score.
4. Quality of life.
5. Social adjustment/functioning score.
6. Improvement in anxiety symptoms as a continuous score.

We will create the ‘Summary of findings’ table before writing our discussion, abstract, and conclusions, so that the review authors can jointly consider the potential impact of the study quality for each outcome on the mean treatment effect and our confidence in these findings. Our confidence in the mean treatment effects based on the GRADE assessments will then be reflected in the interpretation of the results, which informs the abstract, lay summary, and discussion sections of the review.

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Disclaimer: the views and opinions expressed therein are those of the review authors and do not necessarily reflect those of the NIHR, the National Health Service (NHS) or the Department of Health and Social Care.
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Additional references

Alonso 2011

Anderson 2005

APA 1980

APA 1987

APA 1994

APA 2000

APA 2013

Bandler 1982

Barbato 2018

Beck 1961

Beck 1979

Beck 1988

Bennett-Levy 2004

Bernstein 1973

Bowby 1980

BPS 2010

Brooks 1995

Cain 2002

Churchill 2013

Crane 2002

Cuijpers 2014

da Costa 2012
Davies 2018

de Shazer 1988

Deeks 1997

Deeks 2017

Dhar 2018

Dimidjian 2006

Dimidjian 2011

Dyer 2010

Ekers 2014

Eurostat 2014

Freud 1949

Furukawa 2005

Furukawa 2006

Gardardstottir 2009

GRADEpro [Computer program]
McMaster University (developed by Evidence Prime), GRADEpro GDT. Version accessed 2 October 2019. Hamilton (ON): McMaster University (developed by Evidence Prime).

Greenberg 2015

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Hamilton 1960

Hare 2014

Hasin 2018

Hayes 2006

Henken 2007

Hermanns 2013
Hermanns N, Caputo S, Dzida G, Khunti K, Meneghini LF, Snoek F. Screening, evaluation and management of depression

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Higgins 2011a

Higgins 2011b

Higgins 2016
Laurin 2012

Lazarus 1971

Lewinsohn 1974

Lewinsohn 1984

Luborsky 1998

Malan 1963

Mann 1973

Maslow 1943

May 1961

McHugh 2013

Meijer 2011

Moher 2009

Montgomery 1979

Ngo 2013

NICE 2009

O’Connell 2007

Pan 2012

Patel 2015

Pitman 2018

Rathod 2017

Review Manager 2014 [Computer program]

Richards 2016

Rogers 1951

Roth 2017

**Ryle 1990**


**Sansone 2012**


**Schünemann 2011**


**Schünemann 2017**


**Shapiro 1990**


**Shinohara 2013**


**Skinner 1953**


**Solli 2010**


**Spielberger 1983**


**Stein 2019**


**Sterne 2017**


**Stiles 2008**


**Strupp 1984**


**Ten Doesschate 2009**


**The World Bank 2019**


**Uphoff 2019**


**Van Geffen 2009**


**Veale 2008**


**Vos 2017**


**Walters 2011**


**Watson 1924**

**Watzlawick 1974**

**Weissman 2007**

**White 1990**

**WHO 1978**

**WHO 1992**

**WHO 2017**

**Williams 1997**

**Wolpe 1958**

**APPENDICES**

**Appendix 1. Categories of psychological therapies**

<table>
<thead>
<tr>
<th>Categories</th>
<th>Abbreviation</th>
<th>Subcategories</th>
<th>Abbreviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Behavioural therapies</td>
<td>BT</td>
<td>Behavioural therapy (Lewinsohn)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Behavioural activation (original model) (Jacobson)</td>
<td>BA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Social skills training/assertiveness training</td>
<td>SST/assertion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Relaxation therapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other behavioural therapies</td>
<td></td>
</tr>
<tr>
<td>2. Cognitive-behavioural therapies</td>
<td>CBT</td>
<td>Cognitive therapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rational emotive behaviour therapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Problem solving therapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Self-control therapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Coping with depression course</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other cognitive behavioural therapies</td>
<td></td>
</tr>
<tr>
<td>3. Mindfulness-based ‘third wave’ cognitive and</td>
<td>Third wave CBT</td>
<td>Acceptance and commitment therapy</td>
<td>ACT</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Compassionate mind training</td>
<td></td>
</tr>
</tbody>
</table>

Behavioural activation therapies for depression in adults with non-communicable diseases (Protocol)
### Functional analytic psychotherapy
- Extended behavioural activation (eBA)
- Metacognitive therapy
- Mindfulness-based cognitive therapy
- Dialectical behaviour therapy
- Other third wave cognitive and behavioural therapies (other third wave CBT)

### 4. Psychodynamic therapies
- Drive/structural model (Freud)
- Relational model (Strupp, Luborsky)
- Integrative analytic model (Mann)
- Other psychodynamic therapies

### 5. Humanistic therapies
- Person-centred therapy (Rogerian)
- Gestalt therapy
- Experiential therapies
- Transactional analysis
- Existential therapy
- Non-directive/supportive therapies
- Other humanistic therapies

### 6. Interpersonal, cognitive analytic and other integrative therapies
- Interpersonal therapy (IPT)
- Cognitive analytic therapy (CAT)
- Psychodynamic interpersonal therapy
- Cognitive behavioural analysis system of psychotherapy
- Counselling
- Motivational interviewing
- Other integrative therapy approaches

---

**Appendix 2. MEDLINE search strategy**

Ovid MEDLINE(R) and Epub Ahead of Print, In-Process & Other Non-Indexed Citations and Daily <1946 onwards>

1 ((behavio* adj1 activat*) or BATD).tw,kf.
2 behavio*.mp. and (self adj (evaluat* or monitor*)).tw,kf.
3 (behavio* adj2 (contracting or modification or modify*)).tw,kf.
4 reinforc*.ti,kf.
5 (reinforce or reinforce or reinforcement or reinforcements or re-inforcement or re-inforcements).ab./freq=2
6 (reinforc* adj3 (behavior* or environment* or experience*)).tw,kf.
7 (reinforc* adj1 (positive or contingent)).tw,kf.
8 (activ* adj2 schedule*).tw,kf.
9 (pleas* or enjoy* or reward*).adj4 (activit* or event?).tw,kf.
10 (operant or instrumental)adj (conditioning or learning)).tw,kf.
11 (positive interaction* or avoid* coping or environmental contingenc* or contingency management).tw,kf.
12 functional analysis.tw,kf.
13 (gain? or reapprais*) adj2 focus*.tw,kf.
14 (psychoeducat* or psycho-educat*) and (behavior* or coping or self management!).ti,ab,kf.
15 or/1-14 [Behavioural Activation]
16 Depression/
17 exp depressive disorder/
18 (depress* or depressed).tw,kf.
19 dysthym*.tw,kf.
20 distress*.tw,kf.
21 (mood? or mental health or ((emotion* or psychological) adj trauma*)).tw,kf.
22 "common mental disorder*".tw,kf.
23 or/16-22 [Depression - Cochrane terms]
24 15 and 23 [BA and Depression]
25 Behavior Therapy/
26 (behavior* adj (counsel* or intervention or train* or treatment or therapy or psychotherapy)).tw,kf.
27 25 or 26 [Behaviour therapy]
28 23 and 27 [Behaviour Therapy and Depression]
29 24 or 28 [BA or Behaviour Therapy AND Depression]
30 Pulmonary disease, chronic obstructive/
31 Bronchitis, chronic/ or Pulmonary emphysema/
32 Lung diseases, obstructive/
33 exp Asthma/
34 exp Respiratory Hypersensitivity/
35 Hypertension, Pulmonary/
36 asthma*.tw,kf.
37 (long-term or longterm or chronic*) adj5 (bronchitis or respirat*).tw,kf.
38 emphysema*.tw,kf.
39 (hyper responsiveness or hyper-responsiveness or allergy or allergi* or hypersensitiv*).adj5 (airway? or respirat*).tw,kf.
41 (long-term or longter or Chronic* or occupational) adj2 lung* adj5 (condition* or disease* or symptom* or problem* or failure*).tw,kf.
42 (respirat* adj2 (condition* or disease* or symptom* or problem*)).tw,kf.
43 pulmonary hypertension.tw,kf.
44 (COPD or COAD or COBD or AECB).tw,kf.
45 or/30-44 [Chronic Respiratory Diseases]
46 exp Diabetes mellitus/
47 Glucose Tolerance Test/
48 Glycated Hemoglobin A/
49 diabet*.tw,kf.(601034)
50 (noninsulin* depend* or non-insulin*depend* or noninsulin*depend* or non-insulin* depend*).tw,kf.
51 (fasting glucose or plasma glucose or glucose tolerance test* or (glycemic adj2 control*)).tw,kf.
52 (HbA1c or A1C or A1c or HB1c or [(glycated or glycosylated) adj ?hemoglobin?]).tw,kf.(50626)
53 (NIDDM or T2D or T2DM).tw,kf.
54 or/46-53
55 exp Diabetes Insipidus/
56 diabet* insipidus.tw,kf.
57 55 or 56
58 54 not 57 [Diabetes]
59 exp Cardiovascular Diseases/
60 (cardio* or cardia* or CVD).tw,kf.
61 (heart* or coronary*).tw,kf.
62 (angina* or ventric*).tw,kf.
63 (myocard* or pericard*).tw,kf.
64 (isch?em* or cerebrovasc*).tw,kf.
65 exp Stroke/
66 (stroke or strokes or poststroke).tw,kf.
67 apoplexy.tw,kf.
68 (brain adj2 accident*).tw,kf.
69 ((brain* or cerebral or lacunar) adj2 infarct*).tw,kf.
70 exp Hypertension/
71 [hypertensi* or hyperlip*].tw,kf.
72 (hypercholester* or hypertriglycerid*).tw,kf.
73 exp Arteriosclerosis/
74 exp Cholesterol/
75 (cholesterol or arterioscler* or atheroscler* or peripheral arter* disease*).tw,kf.
76 Blood Pressure/
77 blood pressure.tw,kf.
78 (emboli* or arrhythmi*).tw,kf.
79 (thrombo* or "atrial fibrillat*").tw,kf.
80 (tachycardi* or endocard* or "sick sinus").tw,kf.
81 or/59-80 [CVD]
82 exp Neoplasms/
83 (cancer* or neoplas* or tumo* or carcinoma* or hodgkin* or nonhodgkin* or adenocarcinoma* or leuk?emia* or metasta* or malignan* or lymphoma* or sarcoma* or melanoma* or myeloma* or oncolog* or psychooncology or psycho-oncology).tw,kf.
84 82 or 83 [Cancer]
85 45 or 58 or 81 or 84 [COPD Diabetes CVD or Cancer]
86 randomized controlled trial.pt.
87 controlled clinical trial.pt.
88 randomized.ab.
89 placebo.ab.
90 clinical trials as topic.sh.
91 randomly.ab.
92 trial.ti.
93 86 or 87 or 88 or 89 or 90 or 91 or 92
94 exp animals/ not humans.sh.
95 93 not 94
96 29 and 85 and 95 [BA or Behaviour therapy and CMDs and COPD Diabetes CVD Cancer and RCTs]
97 29 and 85
98 limit 97 to "systematic review"
99 96 or 98 [BA or Behaviour Therapy and CMDs and COPD Diabetes CVD Cancer and RCTs OR systematic reviews]
100 (exp Child/ or Adolescent/ or exp Infant/) not exp Adult/
101 99 not 100

******************************************************************************

CONTRIBUTIONS OF AUTHORS

EU and NS conceived the idea for this review. Malini Pires led the adaption of this protocol from the protocol 'Behavioural activation therapies for depression in adults' (Uphoff 2019), and all review authors contributed to the writing.

DECLARATIONS OF INTEREST

EU: no conflicts of interest

MP: no conflicts of interest

CB: no conflicts of interest

DB: no conflicts of interest

RC: leads and has responsibility for Cochrane Common Mental Disorders, which has supported parts of the review process and is largely funded by a grant from the National Institute of Health and Research (NIHR) in the UK.

DE: in his role of Chief Investigator, is responsible for the conduct of the ongoing CHEMIST and MODS trials in which behavioural activation therapies are evaluated. He is the author of several publications reporting on trials of behavioural activation.

EF: no conflicts of interest

PM: no conflicts of interest
MP: no conflicts of interest
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