PHYSICAL ACTIVITY AND MENTAL HEALTH
A focus on depression

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There is a compelling and extensive body of literature supporting the role of physical activity in enhancing and maintaining physical health. Over the last decade there has been rapid growth in research findings concerning the mental health benefits of physical activity. The journal Mental Health and Physical Activity is dedicated to the topic while 2013 saw the publication of the most comprehensive synthesis to date on the subject (Ekkekakis 2013a). The collective body of evidence presents a strong case that physical activity similarly helps enhance and maintain many dimensions of mental health. As Boreham and Riddoch (2003, p. 24) neatly encapsulated, “from the cradle to the grave, regular physical activity appears to be an essential ingredient for human well-being”.

What is mental health?

The terms mental health and mental illness are commonly differentiated as they are not mutually exclusive. Mental health has been defined as a state of well-being in which the individual realizes his or her own potential, can cope with the normal stresses of life, can work productively and fruitfully, and is able to make a contribution to her or his own community (WHO 2001). In contrast, mental illness is any health condition characterized by alterations in thinking, mood, or behaviour (or some combination thereof) associated with distress and/or impaired functioning (United States Department of Health and Human Services 1999); common examples include depression or anxiety. Referred to as a two continua model of mental illness and mental health, the model holds that both are related, but distinct dimensions: one continuum indicates the presence or absence of mental health, the other the presence or absence of mental illness (Westerhof & Keyes 2010). This has important implications. First, it allows the possibility of being diagnosed as having a mental illness but still having the capacity to achieve positive mental health. As such it justifies the promotion of mental health to individuals with a mental illness rather than just considering treatment or prevention. Second, mental health problems such as subclinical levels of depression or anxiety can affect us all without necessarily becoming a clinical, diagnosed condition. Consequently, mental health promotion has the capacity to improve the quality of life of clinical and non-clinical populations alike (Faulkner, Trinh, & Arbour-Nicitopoulos 2015). Mental health promotion might be seen as a form of inoculation to prevent or alleviate poor mental health.
Physical activity promotion as mental health promotion

Physical activity promotion has a number of positive attributes that make it an attractive approach to promoting mental health. First, it could be a cost-effective alternative for those preferring not to use medication or who cannot access therapy. For example, non-drug treatments such as cognitive behavioural therapy can be costly and difficult to access. Although not without methodological limitations, the Fraser Institute in Canada publishes reports on waiting times for psychiatric treatment. This consists of two components: waiting after being referred by a general practitioner before consultation with a psychiatrist, and then waiting to receive treatment after the first consultation with a psychiatrist. For Canada as a whole, the total waiting time in 2009 was estimated at 16.8 weeks (Esmail 2009). In the UK, the national average waiting time from referral to treatment (psychological therapies) is approximately 5 weeks although there is also considerable national variation with some having median waits of 3 months or longer (Health and Social Care Information Centre 2014).

Second, physical activity is associated with negligible deleterious side-effects. Compare this to concerns about antidepressant medication. A well-publicized systematic review (Sharma et al. 2016) reported that the risk of suicidality and aggression doubled in children and adolescents taking antidepressants (selective serotonin and serotonin-norepinephrine reuptake inhibitors). Third, physical activity is an effective method for improving important aspects of physical health thus the promotion of physical activity for mental health can be seen as a “win-win” situation with both mental and physical health benefits accruing (Faulkner, Hefferon, & Mutrie 2015). Fourth, given the scope of the burden of poor mental health, there is the need for population-based promotion, prevention, and treatment strategies. Attempts are already made to promote physical activity at a population level, for example, through social marketing campaigns developed by public health agencies and the development and dissemination of physical activity guidelines. Arguably, physical activity is a scalable, population level intervention for promoting mental health unlike pharmacotherapy and counselling (Faulkner, Trinh, & Arbour-Nicitopoulos 2015).

Given that a comprehensive review of existing research on physical activity and mental health is beyond the scope of a single chapter, the purpose of this chapter is to focus on the case for physical activity as a strategy to prevent and treat depression. A brief overview of potential mechanisms will be provided before a focus on implications for policy and practice of the existing evidence. In examining the case of depression, it is important to acknowledge that physical activity has a vital role to play in promoting positive mental health beyond a focus on mental illness.

A focus on depression

Depression is a common mental illness that is prevalent globally. It is estimated that 350 million people are currently affected by depression and by the year 2020, it is predicted to be the leading cause of disability worldwide (Ferrari et al. 2013; WHO 2012). This projection is concerning given the impact of depression on overall health and well-being. Depression is associated with a range of symptoms including low energy, lack of interest, low self-worth; poor sleeping patterns and appetite; and overwhelming feelings of sadness and anxiety (WHO 2012). Kessler and Bromet (2013) reviewed international data summarizing other psychosocial and secondary disorders linked with depression and these included reduced role functioning (e.g., low marital and work performance quality) and an elevated risk of chronic secondary disorders such as cancer, cardiovascular disease, and diabetes.
The adverse health effects of depression extend beyond the individual. As a result of lost work productivity and health care costs, the incremental economic burden of depression has risen from $83.1 billion to $210.5 billion between the years 2000 and 2010 in the United States (Greenberg et al. 2015). With the high prevalence of depression and its burden on health and the economy, there is an urgent need to halt these growing trends. Hence, the World Health Organization (2012) declared that preventing depression is an area that warrants urgent attention. One modifiable health behavior shown to prevent the onset of depression is physical activity.

Physical activity as prevention

Mammen and Faulkner (2013) reviewed studies with a longitudinal design examining relationships between physical activity and depression over at least two time intervals. A total of 25 of the 30 studies found a significant, inverse relationship between baseline physical activity and follow-up depression, suggesting that physical activity is preventive in the onset of depression. Given the different ways physical activity was measured in the reviewed studies, a clear dose-response relationship between physical activity and reduced depression was not readily apparent. However, there was evidence that any level of physical activity, including low levels, can prevent future depression. Further, everyday activities such as walking appear to confer a benefit. Meeting the recommended levels of physical activity, established for physical health benefits, appears equally appropriate for preventing depression.

Data from the studies also suggest for individuals who are currently active to sustain their physical activity habits and those who are inactive to initiate a physically active lifestyle to help reduce the odds of developing depression.

Such studies involve large numbers of people and measure physical activity status prior to the incidence of depression. In one example, Lucas and colleagues (2011) conducted a prospective analysis involving 49,821 US women from the Nurses’ Health Study who were free from depressive symptoms at baseline (1996). Physical activity was self-reported in 1992, 1994, 1996, 1998, and 2000. Having clinical depression was defined as reporting either a new physician’s diagnosis of depression or beginning regular use of antidepressant medication. After controlling for a range of potential covariates such as socioeconomic status and physical limitations, there was an inverse age-adjusted dose-response relation between duration of physical activity and depression risk. Depression risk decreased with increasing time spent walking daily at an ‘average pace’ (<20 min/day, relative risk (RR) = 0.94; 20–40 min/day, RR = 0.94; ≥40 min/day, RR = 0.80) or brisk/very brisk pace (<20 min/day, RR = 0.95; 20–40 min/day, RR = 0.88; ≥40 min/day, RR = 0.83).

Although these findings are consistent, they cannot rule out the potential for self-selection and other forms of measurement bias. It is possible that individuals who are more physically active represent a selection of people who happened to have greater education, be of higher socioeconomic status, or social support networks that made them less likely to develop depression, irrespective of their participation in physical activity. However, even when these studies take account of a wide range of possible confounding factors in the statistical modelling (e.g., disability, body mass index, smoking, alcohol, and socioeconomic status), the relationship between physical activity and a decreased risk of depression remains. Despite consistency in the literature regarding a protective function of physical activity, some caution is required given that there may be a number of other factors, such as genetic variations (De Moor et al. 2008), that predict both physical activity and depression and these may not have been fully accounted for in the reviewed studies (Mammen & Faulkner 2013).
Exercise as treatment

Intervention literature for clinical depression emerged in the late 1970s (e.g., Greist et al. 1979) and 80s (e.g., McCann & Holmes 1984). In the case of treatment the focus has been on the impact of exercise – physical activity that is structured, often supervised, and undertaken with the aim of maintaining or improving physical fitness or health. An influential Cochrane review conducted by Cooney and colleagues (2013) identified 39 randomized controlled trials (RCTs) and a meta-analysis of these studies showed a moderate effect size (−0.62 (95% confidence interval (CI) −0.81 to −0.42)), for exercise versus no treatment control conditions. Pooled data from eight trials (377 participants) reporting long-term follow-up data found a small effect for exercise (standardized mean difference (SMD) −0.33, 95% CI from −0.63 to −0.03). For the six trials considered to be at low risk of methodological bias, a further analysis showed a small clinical effect in favour of exercise, which did not reach statistical significance (SMD −0.18, 95% CI from −0.47 to 0.11). This latter analysis has been critiqued given questionable inclusion and exclusion criteria (Ekkekakis 2015). Three of the six studies did not include adequate comparators (e.g., compared different doses of exercise). With the analysis restricted to three high-quality trials, the pooled SMD was significantly different from zero (SMD −0.33, 95% CI from −0.59 to −0.07).

Seven trials in the Cochrane review compared exercise with psychological therapy (189 participants), and found no significant difference (SMD −0.03, 95% CI from −0.32 to 0.26). Four trials (n = 300) compared exercise with pharmacological treatment and found no significant difference (SMD −0.11, −0.34, 0.12). In the most well-cited example, the effects of aerobic exercise were compared to sertraline (Zoloft) treatment among 156 older adults with depression (Blumenthal et al. 1999). Participants were randomized to either aerobic exercise (three times per week at 70–85% of their heart rate reserve), sertraline (an antidepressant), or combined sertraline and exercise for 16 weeks. After 16 weeks of treatment, patients in all three groups exhibited significant reductions in depressive symptoms. Notably patients responded more quickly in the medication group. After 10 months, remitted participants (those who no longer met diagnostic criteria for depression) in the exercise group had significantly lower relapse rates than participants in the medication group (Babyak et al. 2000).

Remarkably, Cooney et al. (2013), concluded that exercise appears to be no more effective than psychological or pharmacological therapies. A more suitable conclusion is that exercise appears to be as effective as psychological or pharmacological therapies, and is another evidence-based option for patients and treatment providers to consider. Other meta-analyses consistently report similar findings. Silveira et al. (2013) combined data from 10 RCTs and identified a 0.61 (95% CI from −0.88 to −0.33) standard deviation reduction in the exercise intervention group compared to the control group. Danielsson et al. (2013) reported that aerobic exercise had a similar and positive effect to antidepressants in two trials. Josefsson, Lindwall, and Archer (2014) found an overall effect size of 0.77 (n = 13; 95% CI from −1.14, −0.41) in favour of exercise compared to control conditions. Rosenbaum et al. (2014) reported a large effect of physical activity on depressive symptoms (n = 20; SMD 0.80). The effect for trials with higher methodological quality was smaller than that for trials with lower quality (SMD 0.39 vs 1.35) although this difference was not statistically significant. Another recent meta-analysis determined that the control groups used in many exercise and depression RCTs experienced large and significant improvements in depressive symptoms and these improvements were approximately double that reported in antidepressant meta-analyses (Stubbs et al. 2016). Accordingly, demonstrating a “strong” antidepressant effect of exercise has been challenging thus far.
As with any research endeavour questions always remain. There are methodological limitations to the existing research that largely reflect the behavioural nature of exercise interventions. Treatment blinding is impossible and there is likely a self-selection bias where study participants are choosing to participate in an exercise intervention for the treatment of depression. Further research is required as to what conditions and for whom exercise is most likely to help. In terms of dosage, one review (Stanton & Reaburn 2014) examined the dose characteristics of five RCTs that reported a significant treatment effect of exercise in the treatment of depression. They concluded that the exercise dose should likely use supervised aerobic exercise, and occur three times weekly at moderate intensity for a minimum of nine weeks in the treatment of depression. A similar dosage was recommended in an additional systematic review (Nyström et al. 2015).

How does exercise reduce depression?

As per the Bradford–Hill criteria (Hill 1965) for making a case for causality, it is important to understand potential mechanisms of the antidepressant effect of exercise. Several plausible biological and psychosocial mechanisms have been proposed for the efficacy of exercise in treating depression. The primary biological mechanisms that have been suggested can be broadly categorized as changes in 1) neurochemistry or 2) neuroplasticity. Changes in brain neurochemistry refer to altering the amount of neurotransmitters available in the synapses between neurons, and is the primary target of psychiatric medications. Neuroplasticity on the other hand is the creation of new neurons (neurogenesis) and new connections between neurons (synaptogenesis), and is a relatively novel mechanism that has been proposed.

Much of the understanding behind the biological mechanisms that may improve depression symptoms stems from a knowledge of the pharmacodynamic effects of antidepressant medications (Nutt 2008). The most common types of antidepressants are serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs), which primarily increase the amount of serotonin in the brain, an important chemical for mood regulation. Similar to these medications exercise has also been shown to increase serotonin availability, which may explain its anti-depressant effects. It is well established that in animals, an acute bout of exercise is consistently shown to increase both hippocampal (a key brain region in depression) (Meeusen et al. 1996; Wilson & Marsden 1996) and whole brain serotonin concentration (see Meeusen & De Meirleir 1995). In humans where directly measuring serotonin in the brain is not possible, acute bouts of exercise increase free tryptophan (the amino acid precursor of serotonin) in the blood, which is a marker of serotonin production in the brain (Melancon, Lorrain, & Dionne 2012; Nybo et al. 2003). Additionally, exercise also seems to increase the availability of norepinephrine and dopamine, which are other neurotransmitters that have been implicated in depression (Alsuwaidan et al. 2009). Overall, it seems that exercise, like antidepressants, tends to increase the amount of serotonin available and other mood regulating neurotransmitters, which may be one way that exercise improves depression symptoms.

In addition to changing brain chemistry, antidepressants also increase neuroplasticity in the hippocampus and other mood related areas of the brain (Sahay & Hen 2007). Increases in neurogenesis and synaptogenesis have been identified in animals in response to administering antidepressants (Sahay & Hen 2007; Mahar et al. 2014). At least one study has shown that neurogenesis mediates the improvement of depression-like symptoms in a mouse model (Hill, Sahay, & Hen 2015).

Similarly, imaging studies of humans have shown increases in brain volume in the dentate gyrus and hippocampus (Boldrini et al. 2012; Mahar et al. 2014). Exercise appears to induce
the same proliferation of neurons, at least in animal models (Chen 2013). In humans the results are mixed. Only one study has attempted to examine neural growth among patients with depression in response to exercise, and found no change in any of the markers of growth (Krogh et al. 2014). However, other studies have shown growth is possible in response to exercise (Pajonk et al. 2010; Erickson et al. 2011), and thus it should not be discounted as a potential mechanism given the evidence from antidepressant medication.

Overall, it appears that exercise may induce the same physiological changes as antidepressants, which may explain the benefits of exercise for symptoms of depression. As neurochemistry is constantly changing in response to external stimuli, this may be more responsible for acute effects of exercise on depression. In order to reap long-term benefits via these mechanisms, patients must undertake physical activity on a regular basis, similar to a typical prescription for an anti-depressant. On the other-hand, changes in brain anatomy are the result of a cascade of signals and develops over time. Therefore it is reasonable to hypothesize that this mechanism may be responsible for maintenance of remission and prevention. However, in keeping with the “use it or lose it” principle of neurology (Millington 2012), a regular dose of exercise is likely warranted to maintain any beneficial changes in brain anatomy over time. As a result, regardless of which biological mechanisms may be at work, regular physical activity is advisable.

While it may be argued from a reductionist perspective that psychosocial mechanisms may also fundamentally represent biological changes in neurochemistry and neuroanatomy, these changes are likely to be myriad, difficult to attribute directly to exercise, and spread throughout the brain. Several psychosocial mechanisms have been proposed including improvements in self-evaluations (e.g., self esteem), distraction from negative thoughts, and affect regulation (whereby patients are able to manage their current feeling state by engaging in acute bouts of exercise as they feel they need to) (Craft 2013). Correspondingly, depression is characterized by negative self-evaluation, rumination on negative thoughts, persistent depressed mood, and is often brought on or worsened by stress (American Psychiatric Association 2013). While the distractibility of PA is debatable, and likely highly dependent on context and ability of the participant (Craft 2013), the ability of exercise to improve self-evaluations (Spence, McGannon, & Poon 2005) and improve affect after an acute dose (Ekkekakis, Parfitt, & Petruzzello 2011) is well established. Despite this, relatively little research has examined whether these factors mediate the changes in depression as a result of physical activity.

Notably, one study has examined whether changes in coping self-efficacy – a component of self-evaluation – and distraction were related to changes in depression during an exercise intervention (Craft 2005). After controlling for baseline levels, increases in self-efficacy were correlated with lower depression levels, while distraction was not; ultimately, this led the author to conclude that there was stronger support for self-efficacy as a mechanism for treating depression than distraction.

As well, studies have shown that while acute bouts of exercise can improve affect among people with major depression (Bartholomew, Morrison, & Ciccolo 2005), it would be difficult from a practical perspective to examine whether brief improvements during single bouts of exercise translate into long-term reduction of symptoms. However, a recent self-report cross-sectional study of young adults found that moderate intensity exercise moderated depression symptoms among people with high levels of life stress (Maio, Sabiston, & O’Loughlin 2015), indicating that physical activity may help reduce depression by moderating stress – a negative emotion. Due to the limited research in this area, the psychosocial mechanisms proposed remain plausible factors in the use of exercise to treat depression. Just as there is a wide range of medications available with slightly differing mechanisms of action,
it is reasonable to consider that when physical activity works for depression, it may not be working in the same way for every individual. However, unlike medication, where the pharmacodynamics are well documented, and the mechanism is thereby presumed, the mechanisms behind the benefit of exercise are less definitive. The implications of this for legitimizing the role of exercise as a treatment for depression are unknown.

**Exercise as therapy: Little to argue against it!**

There is a relatively large and consistent body of literature that indicates being physically active prevents depression and that exercise can alleviate depressive symptoms. The effect appears comparable to other therapeutic approaches including medication. Also, there is a range of plausible mechanisms that explain why physical activity may have an antidepressant effect. From a population-health perspective, promoting physical activity may serve as a valuable mental health promotion strategy in reducing the risk of developing depression (Mammen & Faulkner 2013). Exercise also appears, on the surface at least, to be a cheap, safe, and accessible option for treating depression. Yet, how likely is someone seeking help for depression to be prescribed exercise as treatment?

One way to address this question is to consider national guidelines for the treatment of depression. We reviewed evidence based treatment guidelines for clinical depression in Canada, the United Kingdom (UK), the United States (US), and Australia and New Zealand (ANZ) (Malhi et al. 2015; National Collaborating Centre for Mental Health and National Institute for Health and Clinical Excellence, 2010; Ravindran et al. 2009; Work Group On Major Depressive Disorder 2010). Table 12.1 provides an overview of the guidelines. All four guidelines recommend exercise for the treatment of mild to moderate depression, at least as an adjunct, although each guideline varies in the nature of their endorsement of exercise.

While currently being revised, the Canadian Network for Mood and Anxiety Treatments (CANMAT) Clinical guidelines (Ravindran et al. 2009) for the management of major depressive disorder (MDD) in adults considers exercise within the portfolio of complementary and alternative medicine treatments. These include strategies such as light therapy, acupuncture, nutraceutical therapies (e.g., omega-3 fatty acids), and herbal therapies (e.g., St. John’s Wort). Being categorized as such might conceivably signal to clinicians and the general public that exercise is not mainstream and possibly not even medical treatment. In terms of the specific guidelines regarding exercise, it was concluded that there was Level 2 evidence for the benefit of exercise as adjunct to medications in mild to moderate MDD, but not as monotherapy. Level 2 evidence is defined as “at least 1 RCT with adequate sample size and/or meta-analysis with wide confidence intervals” (see Ravindran et al., 2009, p. S55). It is not clear how these conclusions were drawn given the number of appropriately sized RCTs and meta-analyses actually cited by the authors in reaching these conclusions – including two large RCTs demonstrating an antidepressant effect equal to medication. Such a lukewarm endorsement regarding the evidence for exercise in the context of depression suggests broader barriers to the acceptance and promotion of exercise as a credible treatment option for depression.

The US guidelines mention exercise in the “choice of initial treatment modality” subsection dealing with treating the acute phase of depression (Work Group On Major Depressive Disorder 2010). While exercise is recommended both as monotherapy and adjunct for acute treatment, in the guidelines it is simultaneously overlooked as a treatment by not being provided a distinct section like pharmacotherapy, psychotherapy, or
<table>
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<th>Document Details</th>
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<th>Severity: Mild to Moderate</th>
<th>Severity: Severe</th>
<th>Comorbidities</th>
<th>Dosage</th>
<th>How to Discuss or Prescribe</th>
<th>Prevention: Depression in General Population</th>
<th>Prevention: Relapse</th>
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<td>+ *</td>
<td>+ [Adjunct]*</td>
<td>+ General Health &amp; Well-being</td>
<td>National Guidelines(^1,,^2); Most days of the week &gt;150min MPA or &gt;75min VPA /week</td>
<td>Active Encouragement; Motivation more challenging in Severe Depression</td>
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<td>+ Weight &amp; BMI</td>
<td>Based on patient preferences and adherence</td>
<td>Face-to-face education, books, pamphlets, &amp; trusted web sites provided by physician</td>
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Notes: + = Supports, - = Discourages, 0 = No statement. RANZCP = Royal Australia and New Zealand College of Psychiatrists, CANMAT = Canadian Network for Mood and Anxiety Treatments, NICE = National Institute for Health and Care Excellence, APA = American Psychiatric Association, MDD = Major Depressive Disorder, MPA = Moderate Physical Activity, VPA = Vigorous Physical Activity.

* = No explicit recommendation on using physical activity (PA) as monotherapy or adjunct therapy based on severity, however guidelines state “motivating more severely depressed patients to engage [in PA] is a challenge” implying PA likely to be used as an adjunct in severe depression.

\(^1\) (Brown et al. 2012).
\(^2\) (Ministry of Health 2015).
complementary and alternative therapies such as St. John’s Wort or light therapy. Specifically, while these other therapies receive an assessment of the level of evidence similar to the CANMAT guidelines, exercise does not receive a formal review of research quality despite relevant studies being cited throughout the document. Rather the evidence for exercise is briefly reviewed in the subsection: Provide Education to the Patient and the Family stating that “data generally support at least a modest improvement in mood symptoms for patients with major depressive disorder who engage in aerobic exercise . . . or resistance training” (p. 29). There is also brief mention that physical activity may reduce the prevalence of depressive symptoms in the general population. Despite the recommendations that exercise be considered for treatment, and may be useful in prevention, it is not included in a summary figure of treatment modalities on page 31. The wording used to describe exercise as a treatment modality is also notable: “if a patient with mild depression wishes to try exercise alone for several weeks as a first intervention, there is little to argue against it . . . .” (p. 30). So true! Such a statement can be interpreted in a number of ways. One is that exercise is not a credible first choice strategy to be recommended by a mental health professional. These same guidelines also provide dosage recommendations for physicians to provide patients and suggest that the optimal regimen for physical activity is one that patients will follow. Educational brochures and pamphlets are suggested for discussing physical activity with patients.

In its stepped-care approach, the UK guidelines explicitly include physical activity as one of three low intensity psychosocial treatment options for people with persistent subthreshold depressive symptoms or mild to moderate depression (National Collaborating Centre for Mental Health and National Institute for Health and Clinical Excellence, 2010). These include individual guided self-help based on the principles of cognitive behavioural therapy (CBT), computerized cognitive behavioural therapy (CCBT), or a structured group physical activity programme. Such programmes should be delivered in groups with support from a competent practitioner and consist typically of three sessions per week of moderate duration (45 minutes to 1 hour) over 10 to 14 weeks (average 12 weeks). There is no mention of physical activity protecting against depression in the general population.

The most recent guidelines from Australia and New Zealand (Malhi et al. 2015) classify physical activity as a healthy lifestyle intervention along with diet and smoking cessation. Like the US guidelines, the new ANZ guidelines make recommendations for physical activity in each treatment category identified in Table 12.1. Specifically, recommendations include that physical activity can be used to prevent worsening of symptoms in sub-threshold depression and that physical activity has demonstrated to be an effective treatment, at least as an adjunct, for all levels of depression. There is, however, no explicit statement about when it would be appropriate to consider physical activity as a monotherapy, just that it may be more difficult to motivate individuals with more severe depression. Taken together the guidelines advocate for promoting physical activity for all patients with depression or depressive symptoms. Furthermore, the guidelines advocate a physical activity dose consistent with public health guidelines to promote overall health and well-being.

In addition to recognizing the efficacy in treating depression, the ANZ guidelines recommend physical activity for both preventing depression in the general population, citing the Mammen and Faulkner (2013) review, as well as preventing relapse for patients in the maintenance phase of treatment. As can be expected from the most recently released guidelines, the ANZ guidelines represent the most up-to-date collection of research on the use of physical activity to treat depression, and, much like the UK guidelines, have explicitly identified exercise as a therapeutic option.
Overall, Canadian, UK, US, and ANZ guidelines for treating depression all mention a role for exercise although the North American guidelines are more cautious. Importantly, only the UK guidelines suggest that exercise programmes be undertaken with the support of a trained professional. However, no guideline provides any information as to whom patients could or should be referred to in order to receive adequate support for engaging in exercise (e.g., registered kinesiologists or occupational therapists). Any elaboration of the potential role of physical activity protecting against depression is scant although this is not necessarily surprising given the treatment focus of the guidelines.

Implications for future physical activity policy and practice

A range of factors may speculatively explain why there is such caution in recommending the consideration of exercise as a treatment option. Little focused attention has been given to this potential gap between evidence and practice. One qualitative study explored perceptions of twenty-one course directors of clinical psychology programmes in the United Kingdom (Faulkner & Biddle 2001). While participants were broadly positive regarding physical activity there was little, if any, mention of its role as a treatment strategy within curricula. At that time at least, mental health professionals were likely not receiving any exposure to the research evidence concerning exercise and little training for implementation. It is also likely that a process for knowledge exchange between researchers conducting exercise and depression research and practitioners was, and still remains, not well established. As a result, exercise as a treatment may be peripheral to how mental health professionals are being trained to treat individuals with depression. An incompatibility of exercise with traditional models of understanding and treating depression may still remain for many (Faulkner & Biddle 2001). Disciplinary territorialism also likely plays a role in that “exercise” is considered the mandate of “other” professionals. As commented by one director (Faulkner & Biddle 2001, p. 441), “exercise does not appear as a ‘terribly glamorous solution’, neither ‘clever enough’ nor ‘psychologically based enough’”. As another continued,

I think there’s an issue almost of legitimacy, like you’ve done all this training with quite sophisticated models and interventions and psychological work, and you’re asking people to go out for a run. It’s almost too simple.

Such simplicity needs to be contextualized in light of the difficulty for many, not just individuals with depression, in initiating and sustaining physical activity participation.

It is likely that such perspectives may have modified since the time of publication. With increasing acknowledgement of the physical health consequences of mental illnesses such as schizophrenia and depression, either directly or associated with medication side-effects, there has been growing recognition of the urgent need to address the physical health needs of individuals with mental illness (Ward, White, & Druss 2015). Yet again such recognition may see exercise considered as one component of a healthy lifestyle to be supported rather than a treatment in and of itself. More concerted knowledge translation efforts may be needed to integrate evidence regarding exercise into training curricula for mental health professionals. A final concern is likely a pragmatic one.

Specifically, is there a structure in place for referring individuals to supervised exercise programmes that includes exercise counselling by qualified practitioners? As highlighted by the UK NICE guidelines, it is recommended that individuals participate in structured group
programmes supervised by a competent support practitioner. Recent meta-analyses also consistently demonstrate that interventions that are structured and supervised demonstrate stronger effects, while dropout is minimized in studies delivered by health care professionals with specific training in exercise prescription (Stubbs et al. 2016). Interventions are currently less successful when less structure and supervision is provided.

One well-publicized RCT in the UK reported that the addition of a facilitated physical activity intervention to usual care did not improve depression outcome or reduce use of antidepressants compared with usual care alone (Chalder et al. 2012). This was an ambitious trial examining whether physical activity counselling, rather than a supervised and structured exercise intervention, could alleviate depression. The intervention programme comprised an initial hour long face to face assessment session followed by two short telephone contacts, then a further face-to-face meeting for half an hour. Over the course of six to eight months, the physical activity facilitator offered up to eight further telephone contacts and one more face-to-face half hour meeting. Ekkekakis (2013b) has presented an extensive critique of this study. Two points warrant attention. First, there was no evidence that the differences in physical activity between the groups changed over the duration of the study. Interpreting the results to suggest that physical activity did not improve depression outcomes is incorrect – the intervention was unsuccessful in increasing physical activity to a level significantly greater than in the control condition. Presumably this must be a necessary precursor to showing a treatment effect. Second, the intervention was delivered by non-specialists with minimal training, and was based on a physical activity counselling model. The pragmatic nature of the trial is to be applauded but it may be that unsupervised, physical activity interventions are likely not sufficient to support behaviour change for many individuals seeking help for treatment.

Given this possibility and likely cost there are clear policy implications of this possibility. Both physical activity and mental health professionals have important roles to play in establishing inter-professional dialogue and collaborating in developing structures of referral to supervised and structured exercise interventions. For example, qualified exercise professionals should be incorporated into mental health care treatment teams. The recent Blueprint for an Active Britain (2015, p. 66) highlights one relevant policy recommendation in this regard:

Every person who has a mental health diagnosis should have access to a named physical activity intervention in line with NICE guidance, based upon proven evidence-based behavioural interventions such as motivational interviewing.

It is likely that policies will need to be established that make this a reality and that provide support and guidance for mental health professionals in applying the evidence concerning exercise and depression. Without policies facilitating the creation of such structures it will not be possible to make exercise, an evidence-based treatment option, more accessible to more individuals seeking help for depression. In tandem with this will be the need for more research focusing on how to integrate exercise treatment within existing systems of care, and assessing the cost-effectiveness of exercise provision in real world settings.

Given the significant burden of depression on the individual, family, and health care system, more attention is needed in examining how depression might be prevented and how more individuals with depression can access structured and supervised exercise interventions. From a policy perspective, greater recognition should be given to the mental health benefits of physical activity within both physical activity and mental health initiatives. From a mental
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health promotion perspective specifically, evidence suggests that promoting any level of physical activity could be an important strategy for the prevention of future depression in addition to an already impressive list of physical and mental health benefits accrued through a physically active lifestyle. The full potential of physical activity as a mental health promotion strategy at a population level has yet to be determined.

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