Exercise therapy in polycystic ovary syndrome: a systematic review

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Submitted on July 7, 2010; resubmitted on August 5, 2010; accepted on August 11, 2010

TABLE OF CONTENTS

- Introduction
- Methods
  - Study characteristics
  - Study design
  - Search strategy
- Results
  - Identification of articles
  - Data synthesis
  - Description, duration, type and intensity
- Outcomes
  - Cardiovascular risk factors
  - Reproductive function
  - Study quality, heterogeneity and compliance
- Discussion
- Conclusions and recommendations

BACKGROUND: Polycystic ovary syndrome (PCOS) is a common endocrine disorder, affecting 8–12% of women. Lifestyle modification, including increased physical activity, is the first-line approach in managing PCOS. A systematic review was performed to identify and describe the effect of exercise as an independent intervention on clinical outcomes in PCOS.

METHODS: Five databases were searched with no time limit. A pre-specified definition of PCOS was not used. Studies were included if exercise therapy (aerobic and/or resistance) could be evaluated as an independent treatment against a comparison group. Outcomes measured included cardiovascular risk factors [insulin resistance (IR), lipid profiles, blood pressure and weight] and reproductive measures (ovulation, menstrual regularity and fertility outcomes). Quality analysis was performed based on the Cochrane Handbook of Systematic Reviews and the Quality of Reporting of Meta-Analyses checklist.

RESULTS: Eight manuscripts were identified (five randomized controlled trials and three cohort studies). All studies involved moderate intensity physical activity and most were of either 12 or 24 weeks duration with frequency and duration of exercise sessions ranging between studies. The most consistent improvements included improved ovulation, reduced IR (9–30%) and weight loss (4.5–10%). Improvements were not dependant on the type of exercise, frequency or length of exercise sessions.

CONCLUSIONS: Exercise-specific interventions in PCOS are limited. Studies vary considerably in design, intensity and outcome measures; therefore conclusive results remain elusive. Larger, optimally designed studies are needed to both gain insights into the mechanisms of exercise action and to evaluate the public health impact of exercise of PCOS.

Key words: polycystic ovary syndrome / exercise / lifestyle modification / weight / insulin resistance
Introduction

Polycystic ovary syndrome (PCOS) is a complex endocrine disorder affecting 8–12% of reproductive-aged women. Currently recognized as the leading cause of anovulatory infertility, PCOS is the most common endocrinopathy in reproductive-aged women (Carmina and Lobo, 1999; Gambineri et al., 2002; Azziz et al., 2004; March et al., 2010). Diagnosis of PCOS requires at least two of the following characteristics: clinical or biochemical hyperandrogenism, anovulatory menstrual dysfunction and polycystic ovaries on ultrasound, in the absence of other causes including pituitary and adrenal dysfunction (Zawadaki et al., 1992; Chang et al., 2004). Although not included in the diagnostic criteria, insulin resistance (IR), underpinned by insulin signalling pathway defects, is strongly implicated in the aetiology of PCOS (Corbould et al., 2005, 2006; Diamanti-Kandarakis and Papavassiliou, 2006) and is tightly associated with the reproductive (hirsutism, infertility) and cardiometabolic (impaired glucose tolerance and metabolic syndrome) complications of the disorder (Teede et al., 2006, 2007). In comparison with weight-matched controls, women with PCOS are more insulin resistant and have higher rates of Type 2 diabetes (Dunai et al., 1989; Legro et al., 1999; Solomon, 1999; Norman et al., 2001). This impacts on long-term health by increasing the likelihood of risk factors associated with cardiovascular disease (CVD; Meyer et al., 2005) including increased blood pressure, dyslipidaemia and markers of blood vessel function (Wild, 2002). Furthermore, ~40–60% of women with PCOS are either overweight [body mass index (BMI) $\geq 25$ kg/m$^2$] or obese (BMI $> 30$ kg/m$^2$) with increased central adiposity (Kiddy et al., 1990; Balen et al., 1995), which exacerbates underlying IR and IR-associated metabolic and reproductive complications in PCOS (Kiddy et al., 1990; Ehrmann et al., 2006).

PCOS represents a significant burden on the health care system. In the USA, $\$4.36$ billion was spend in 2004 on PCOS and its related complications with over 40% of this cost attributed to treating reproductive dysfunction (infertility, menstrual dysfunction) and a further 40% attributed to PCOS-related diabetes (Azziz et al., 2005). Effective treatments are needed to reduce the economic burden and improve health outcomes for women with PCOS.

Current pharmaceutical interventions for PCOS primarily focus on addressing reproductive dysfunction and IR. Treatments for reproductive dysfunction include the oral contraceptive pill (OCP) which regulates menstrual cycles and reduces hirsutism and acne. However, long-term use of the OCP remains controversial due to potential adverse metabolic and cardiovascular effects (Meyer et al., 2007; Soares et al., 2009; Teede et al., 2010). Fertility treatments for PCOS include ovulation induction agents (e.g. clomiphene citrate) with second-line therapies including exogenous gonadotrophins (Norman, 2004, 2008). Metformin is commonly used to improve IR in Type 2 diabetes and in PCOS also improves IR with clinical benefits; including improved ovulatory function (Harborne et al., 2005; Nestler, 2008; Palomba et al., 2009; Tang et al., 2010). Despite these available treatments there is currently no ideal pharmacological intervention in PCOS and for this reason lifestyle modification, including weight loss, remains first-line management to improve cardiovascular risk factors and reproductive dysfunction (Moran et al., 2006). Lifestyle modification strategies in PCOS include dietary intervention with energy restriction and/or altered diet composition and increased physical activity. Weight loss achieved through energy restriction has improved insulin sensitivity and ovulation rates with or without modifying macronutrient composition (Kiddy et al., 1992; Holte et al., 1995; Hollmann et al., 1996; Moran et al., 2003). Despite this, there is no long-term evidence to suggest that specific dietary compositions (e.g. high protein/low carbohydrate) lead to long-term improvements in health outcomes in women with PCOS (Stamets et al., 2004). Furthermore, following modest weight loss, weight maintenance remains a challenge long-term (Waddan, 1993; Ayyad and Andersen, 2000) and in PCOS, may lead to worsened clinical features if weight is regained.

Incorporating moderate exercise as a treatment for clinical complications in PCOS may be favourable, considering the beneficial effects exercise has in other insulin resistant populations, independent of weight loss (Handy et al., 2001; Bruce et al., 2004). Moderate to higher intensity aerobic exercise can be defined as an intensity between 50 and 85% of maximum oxygen consumption ($\text{VO}_{2\text{max}}$) or 60–90% of maximal heart rate (Pate et al., 1995). A single bout of moderate exercise enhances whole-body glucose disposal and improves insulin sensitivity in skeletal muscle short-term (Richter et al., 1989). Ongoing moderate physical activity at least three to five times per week, has consistently been shown in high-risk groups to reduce Type 2 diabetes risk (Tuomilehto et al., 2001; Knowler et al., 2002) and improve cardiovascular risk factors (i.e. weight, lipid profiles and blood pressure) (Goodyear and Kahn, 1998; Shephard and Balady, 1999). Similarly, resistance or weight-bearing exercise training in combination with aerobic exercise or alone, has also been shown to improve health outcomes in high-risk groups (Maiorana et al., 2002; Cuff et al., 2003; Park et al., 2003; Sigal et al., 2007). When combined with dietary changes, exercise has been shown to potentiate the effects of diet on insulin sensitivity in obese Type 2 diabetic patients (Lampan and Schteingart, 1991). Therefore, moderate physical activity may be effective in PCOS.

Despite the potential beneficial effects of exercise in PCOS, there are no systematic reviews that evaluate the independent effects of exercise on cardiovascular and reproductive outcomes. Two recent non-systematic exercise-specific reviews highlight the gaps in knowledge around exercise type and intensity required to improve outcomes in PCOS (Hoeger, 2008; Thomson et al., 2010). Consideration of the frequency and duration of exercise, as well as an assessment of sustainability, is also needed to determine the role of exercise in PCOS.

Therefore, the aim of this systematic review is to identify and describe the effect of exercise on clinical outcomes as an independent intervention in women with PCOS. Specifically, it explores the type, intensity, frequency and duration of exercise on reproductive and cardiovascular outcomes to inform clinical practice.

Methods

Study characteristics

This systematic review focused on studies involving premenopausal women diagnosed with PCOS. A pre-defined definition of PCOS was not used for the search strategy to include all published studies capturing the full range of diagnostic criteria. Studies that compared exercise therapy (aerobic or resistance exercise or a combination) to a comparison group receiving no therapy, minimal therapy (standard advice only) or another
A systematic review on exercise therapy in PCOS

non-exercise therapy/treatment were considered for review. Outcomes measured included cardiovascular risk factors and reproductive function. These outcomes were considered to be of high clinical relevance to those wishing to make a recommendation on exercise in PCOS.

Study design

Studies considered for review included: (i) randomized control trials (RCTs), (ii) non-RCTs if the outcomes specified were not conclusive or assessed by RCTs, (iii) intervention duration of at least 2 weeks with a fixed follow-up period and (iv) whether, in conjunction with any other intervention (i.e., diet or medical treatment), it could be assessed as an independent factor on outcomes of health. An intervention duration of at least 2 weeks was based on capturing as many potentially effective interventional studies as possible. Interventions were excluded if they involved: (i) no exercise or only an optional exercise component or (ii) if the study was not published in English.

The aim of the search strategy was to identify and evaluate interventions that assessed exercise as a primary treatment in PCOS. The primary outcomes specified for review were weight, IR, blood pressure, lipids and waist-to-hip ratio (WHR). Secondary outcomes included fertility outcomes, menstrual regularity and ovulation patterns.

Search strategy

A systematic review of the literature was conducted in November 2009 across the following databases: MEDLINE, PsyInfo EMB Reviews (including DARE, Cochrane DSR, Clinical trial registry, Cochrane library, CENTRAL, ACP journal club), EMBASE and CINAHL with no time limit applied to any database. The reference lists of review articles and eligible studies were hand searched to identify other potentially eligible studies. Abstracts from conference proceedings were also considered to identify further studies that were not yet published. The search terms, adapted when required for each database, to identify exercise interventions in PCOS are presented in Table I.

Results

Identification of articles

The combined searches identified 199 papers for review. Each citation was cross-checked and duplicates removed. Two investigators (C.L.H. and C.B.L.) independently searched, reviewed and abstracted data. Papers were removed at initial screening based on the title or abstract if it could be determined that the publication did not meet inclusion criteria (Supplementary Data, Figure S1). Of the 199 papers identified, 35 were duplicate publications and 148 did not relate to PCOS or did not involve exercise as a treatment or intervention for PCOS in the title and/or abstract. A further eight were removed as manuscripts were either case studies/series with no comparison group (n = 4), non-exercise PCOS intervention (n = 1), editorial comment only (n = 1) or were not published in English (n = 2). Full text versions of articles were obtained if eligibility could not be determined from reading the abstract.

Data synthesis

From the 199 identified citations, 8 manuscripts were suitable for inclusion (Supplementary Data, Figure S1). Of the manuscripts identified, three were exercise-only RCTs, three were cohort studies and two were RCTs based on a dietary intervention but contained a structured exercise component to allow the comparison of the effect of exercise intervention on diet alone. Selected studies were assessed for methodological quality using standard forms adapted from the Cochrane Handbook of Systematic Reviews (Higgins and Green, 2009) and the Quality of Reporting of Meta-Analyses checklist (Moher et al., 1999). If results reported were ambiguous, authors of relevant manuscripts were contacted for additional information. Owing to the limited number of RCTs and clinical heterogeneity between studies, for reasons including PCOS diagnostic criteria used, intervention type (aerobic only or aerobic plus resistance with or without dietary restriction) and duration and comparison group used, additional meta-analyses were not performed.

Description, duration, type and intensity

Table II summarizes the interventions, including PCOS diagnostic criteria, baseline characteristics, target group inclusion criteria, comparison group, intervention intensity, duration and cardiovascular and reproductive outcomes. Baseline results for a total of 421 participants were included across all studies, with the number of participants in a single study ranging from 12 (Bruner et al., 2006) to 124 women (Giallauria et al., 2008). Of the total 421 participants, 223 received exercise intervention. PCOS diagnosis was confirmed in six of the eight studies by the European Society for Human Reproduction and Embryology/American Society of Reproductive Medicine (Rotterdam) criteria (Bruner et al., 2006; Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008; Thomson et al., 2008), in one study by National Institute of Health criteria (Brown et al., 2009), while one study used diagnosis consistent with the Rotterdam criteria but prior to its conception (Randeva et al., 2002). The mean age of participants varied, ranging from 22 to 36 years. All eight studies recruited overweight to moderately obese women with BMI ranging between 26.8 and 37.9 kg/m² at baseline. Four studies aimed to induce weight loss (Bruner et al., 2006; Vigorito et al., 2007; Palomba et al., 2008;
Table II  Summary of study characteristics and cardiovascular and reproductive outcomes.

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Participants</th>
<th>PCOS diagnostic criteria</th>
<th>Inclusion/exclusion</th>
<th>Intervention</th>
<th>Comparison</th>
<th>Cardiovascular outcomes</th>
<th>Reproductive outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stener-Victorin et al. (2009)</td>
<td>RCT</td>
<td>n 20</td>
<td>Rotterdam</td>
<td></td>
<td>Type: unsupervised exercise at least 3 × 30–45 min/week; Intensity: light-moderate; Duration: 16 weeks</td>
<td>1/Treatment: low-frequency Electroacupuncture (14 × 30 min treatments) 2/Control: general diet and exercise advice</td>
<td>Exercise: ↓ BMI</td>
<td>No change</td>
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<tr>
<td>Brown et al. (2009)*</td>
<td>RCT</td>
<td>n 20</td>
<td>NIH⁷</td>
<td>Inclusion: 1/18–50 year/old 2/sedentary 3/maintain current weight &amp; diet</td>
<td>Type: SET calculated min/week (walk, bike, cross-trainer) no diet change Intensity: moderate (50% VO₂max) Duration: 12 weeks + 8–12 week initiation</td>
<td>No exercise/advice</td>
<td>No change</td>
<td>Not measured</td>
</tr>
<tr>
<td>Vigorito et al. (2007)</td>
<td>RCT</td>
<td>n 90</td>
<td>Rotterdam</td>
<td></td>
<td>Type: SET (cycle 3 × 30 min/week) Intensity: moderate (60–70% VO₂max) Duration: 12 weeks Diet: general advice no caloric restriction</td>
<td>General diet and behavioural advice only</td>
<td>Exercise: ↓ BMI, WHR, BP, fasting insulin and AUC insulin</td>
<td>Exercise: ↑ in normal menstrual cycles</td>
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<tr>
<td>Palomba et al. (2008)</td>
<td>Cohort</td>
<td>n 40</td>
<td>Rotterdam</td>
<td>Inclusion: 1/18–35 year/old 2/BMI (35 kg/m²)</td>
<td>Type: SET (cycle 3 × 30 min/week) Intensity: moderate (60–70% VO₂max) Duration: 24 weeks</td>
<td>Diet: hyperproteic (35% protein/45% CHO/20% fat), hypocaloric (800kcal deficit/day) + weekly education classes</td>
<td>Exercise &amp; Diet: ↓ BMI, WHR, fasting insulin and HOMA-IR in responders (ovulators)</td>
<td>Exercise: ↑ ovulation rate and menses frequency compared to diet (P &lt; 0.05)</td>
</tr>
<tr>
<td>Study</td>
<td>Cohort</td>
<td>Rotterdam</td>
<td>Inclusion</td>
<td>Design</td>
<td>Outcome</td>
<td>Notes</td>
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<tr>
<td>Giallauria et al. (2008)</td>
<td>Cohort</td>
<td>n 124</td>
<td>Exercise (n 62)</td>
<td>Age = 22.8 years, BMI = 29.2 kg/m²</td>
<td>Type: SET (cycle 3 × 30 min/week)</td>
<td>No exercise/advice</td>
<td>BMI, WHR, fasting insulin, AUCinsulin.</td>
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<td></td>
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<td></td>
<td>Control (n 62)</td>
<td>Age = 22.6 years, BMI = 29.5 kg/m²</td>
<td>Intensity: moderate (60–70% VO₂max)</td>
<td>Not measured</td>
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<td></td>
<td></td>
<td>124</td>
<td>BMI = 29.5 kg/m²</td>
<td>Duration: 12 weeks</td>
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<tr>
<td>Randeva et al. (2002)</td>
<td>Cohort</td>
<td>n 21</td>
<td>Exercise (n 12)</td>
<td>Age = 29.7 years, BMI = 34.0 kg/m²</td>
<td>Type: unsupervised, walking program 3–7 × 30 min/week</td>
<td>Those who dropped out/did not begin program</td>
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<td>Control (n 9)</td>
<td>Age = 30.1 years, BMI = 37.6 kg/m²</td>
<td>Intensity: light- moderate (brisk walking)</td>
<td>Exercise: ↓ WHR</td>
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<td>21</td>
<td>BMI ≥ 25 kg/m²</td>
<td>Duration: 24 weeks</td>
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<tr>
<td>Bruner et al. (2006)</td>
<td>RCT</td>
<td>n 12</td>
<td>Diet + Exercise (n 7)</td>
<td>Age = 32.3 years, BMI = 36.2 kg/m²</td>
<td>Type: group nutrition sessions (1/week) + 3 sessions/week: aerobic (cycle/walk, 30 min) + Resistance (2–3 × 10–15 reps)</td>
<td>Nutrition sessions only</td>
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<td>Diet (n 5)</td>
<td>Age = 28.4 years, BMI = 37.1 kg/m²</td>
<td>Intensity: 70–85% predicted HRmax</td>
<td>Both groups: ↓ fasting insulin levels &amp; WC</td>
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<td></td>
<td>12</td>
<td>BMI &gt; 27 kg/m²</td>
<td>Duration: 12 weeks</td>
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<tr>
<td>Thomson et al. (2008)</td>
<td>RCT</td>
<td>n 94</td>
<td>Diet (n 30)</td>
<td>Age = 29.3 years, BMI = 36.1 kg/m²</td>
<td>Type: diet + Aerobic (5 × 25–45 min walk/jog per week)</td>
<td>Diet only: 5000–6000 kj/day (30% protein, 40% CHO, 30% fat)</td>
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<td>Diet + Aerobic (n 31)</td>
<td></td>
<td>Intensity: 60–80% HRmax) + / – Resistance (3 × 12 represents 2 times/week)</td>
<td>All groups: ↓ BMI, fasting insulin, HOMA-IR</td>
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<td>Diet + Aerobic &amp; resistance (n 33)</td>
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<td>Duration: 20 weeks</td>
<td>Exercise groups: ↓ FM, cholesterol, LDL, BP (DAR only)</td>
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<td></td>
<td>94</td>
<td>18–41 year/old</td>
<td>↑ ovulation &amp;/or menstrual cyclicity in all groups</td>
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<td>2/BMI 25–55 kg/m²</td>
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</table>

BMI, body mass index; DAR, diet plus exercise and resistance group; F-G score, Ferriman–Gallaway score; FM, fat mass; IR, insulin resistance; RCT, randomized controlled trial; SET, structured exercise training; WHR, waist-to-hip ratio.

1Results published in median ± IQR.
1NIH (National Institute of Health) diagnostic criteria include oligo-ovulation and/or biochemical hyperandrogenism.
1Common exclusion criteria included: other causes of hyperandrogenism or menstrual dysfunction (i.e. thyroid, endocrine disorders, Cushing’s syndrome), ongoing medical illness requiring medication, medications affecting hormone levels or metabolism (i.e. antiandrogens, ovulation induction agents, glucocorticoids, antiobesity drugs), renal impairment, hyperglycaemia (>6.9 mmol/l) or Type 1 or 2 diabetes, hormonal contraceptive use, recent breastfeeding (<3 months), pregnancy or planned pregnancy and smoking.
Thomson et al., 2008) and four did not specifically aim to induce weight loss (Randeva et al., 2002; Giallauria et al., 2008; Brown et al., 2009; Stener-Victorin et al., 2009).

Exercise intervention duration varied with the most common duration either 12 (Bruner et al., 2006; Vigorito et al., 2007; Giallauria et al., 2008) or 24 weeks (Randeva et al., 2002; Palomba et al., 2008; Brown et al., 2009), with two studies reporting 16 (Stener-Victorin et al., 2009) and 20 weeks duration (Thomson et al., 2008). The frequency of exercise sessions included three sessions per week in five studies (Bruner et al., 2006; Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008; Stener-Victorin et al., 2009), four in one (Brown et al., 2009) and five sessions per week in another (Thomson et al., 2008). Randeva et al. progressively increased exercise prescription from three to seven sessions per week over 6 months (Randeva et al., 2002). The length of individual exercise sessions ranged from 30 min in six studies (Randeva et al., 2002; Bruner et al., 2006; Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008; Stener-Victorin et al., 2009), and 45 (Thomson et al., 2008) to 50 min (Brown et al., 2009) in two studies. Six out of eight interventions involved aerobic exercise only (Randeva et al., 2002; Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008; Brown et al., 2009; Stener-Victorin et al., 2009), one study included aerobic and progressive whole-body resistance training (Bruner et al., 2006) and one study included both aerobic only and aerobic plus progressive whole-body resistance interventions (Thomson et al., 2008). All studies except two (Randeva et al., 2002; Stener-Victorin et al., 2009) reported a standard adaptation to aerobic and resistance exercise, if applicable. Intensity of exercise was determined primarily using percentage of maximal oxygen consumption (Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008; Brown et al., 2009) or percentage of maximum heart rate (Bruner et al., 2006; Thomson et al., 2008). In six studies aerobic exercise was performed on a stationary cycle (Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008), treadmill (Thomson et al., 2008) or a combination of the two (Bruner et al., 2006; Brown et al., 2009) according to participant preference at moderate intensity, defined as 50–70% of VO2max or 60–80% HRmax. In two studies, unsupervised, self-monitored walking (Randeva et al., 2002) or other similar aerobic activity (Stener-Victorin et al., 2009) was performed outside of the research facility and recorded in an exercise diary (Randeva et al., 2002) or recalled to a researcher (Stener-Victorin et al., 2009).

Outcomes

Cardiovascular risk factors

Weight

Seven studies reported results for change in BMI (Randeva et al., 2002; Bruner et al., 2006; Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008; Brown et al., 2009; Stener-Victorin et al., 2009) and one reported results for weight only (Thomson et al., 2008). Five studies also reported WHR (Randeva et al., 2002; Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008; Stener-Victorin et al., 2009), three reported waist circumference (Bruner et al., 2006; Thomson et al., 2008; Brown et al., 2009), one reported fat mass (FM) assessed by dual-energy X-ray absorptiometry (DEXA) (Thomson et al., 2008) and one reported results for the sum of two skin fold tests as an indicator of FM (Bruner et al., 2006). Of the eight studies, four reported significant improvement in either BMI (Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008) or weight (Thomson et al., 2008) within 12 weeks of exercise. Three studies involving stationary cycling at moderate intensity for 30 min, three times per week without dietary restriction reported a change in BMI of −1.3 kg/m² (−4.45%) (Vigorito et al., 2007; Giallauria et al., 2008) and −10% (Palomba et al., 2008) in addition to a decrease in WHR post intervention (Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008). In two of these three studies BMI change in PCOS was significantly different from the control group (Vigorito et al., 2007; Giallauria et al., 2008), while in one study a larger BMI change was reported in the dietary intervention group in comparison with the exercise group (Palomba et al., 2008). Thomson et al. found an overall weight loss of 9.4% across all study groups with a corresponding reduction in waist circumference following 20 weeks intervention with no difference found between diet alone or in combination with both aerobic exercise or aerobic and resistance exercise (8.6, 10.1 and 8.6 kg reduction, respectively) (Thomson et al., 2008). A mean loss in FM of 3.9, 7.5 and 6.7 kg was reported in the diet alone and in diet with both aerobic exercise and aerobic and resistance exercise groups respectively, post intervention. Both exercise groups showed a significantly greater reduction than the diet-only group (Thomson et al., 2008). One study reported a small significant (−0.4 kg/m²) change in BMI after 16 weeks of un supervised exercise (Stener-Victorin et al., 2009) and two other studies reported a significant reduction in WHR (Randeva et al., 2002) or subcutaneous fat (Bruner et al., 2006) following exercise despite finding no significant change in BMI (Randeva et al., 2002; Bruner et al., 2006).

IR

All eight studies reported results for IR using a number of different methods. All studies reported fasting insulin, three also reported area under the insulin curve (AUCins), a measure of insulin release over time in response to an oral glucose tolerance test (Vigorito et al., 2007; Giallauria et al., 2008; Brown et al., 2009) and three other studies reported homeostatic model assessment of IR (HOMA-IR) (Palomba et al., 2008; Thomson et al., 2008; Stener-Victorin et al., 2009). One study reported baseline quantitative insulin sensitivity index (QUICKI) (Bruner et al., 2006). Both HOMA-IR and QUICKI are calculated based on fasting insulin and glucose levels. Following exercise, fasting insulin improved significantly by 23–30% in three studies (Bruner et al., 2006; Palomba et al., 2008; Thomson et al., 2008), while two studies reported a 9% improvement (Vigorito et al., 2007; Giallauria et al., 2008). The three remaining studies reported no significant change in IR following exercise intervention (Randeva et al., 2002; Brown et al., 2009; Stener-Victorin et al., 2009). In the five studies reporting reduced IR, the two studies using HOMA-IR (Palomba et al., 2008; Thomson et al., 2008) and two using AUCins also reported improvements in these indices of IR (Vigorito et al., 2007; Giallauria et al., 2008). Of the five studies reporting reduced IR all noted improvements within 12 weeks of exercise, with three studies reporting significant difference from controls (Vigorito et al., 2007; Giallauria et al., 2008) or a dietary- restricted comparison group (Palomba et al., 2008), while two studies reported no difference between exercise and dietary comparison groups (Bruner et al., 2006; Thomson et al., 2008).
Blood lipids

Total cholesterol and triglycerides were reported in seven studies (Randeva et al., 2002; Bruner et al., 2006; Vigorito et al., 2007; Giallauria et al., 2008; Thomson et al., 2008; Brown et al., 2009; Stener-Victorin et al., 2009) with six of these also reporting high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol (Bruner et al., 2006; Vigorito et al., 2007; Giallauria et al., 2008; Thomson et al., 2008; Brown et al., 2009; Stener-Victorin et al., 2009). In five of the seven studies, no significant differences were found in any variable post-intervention (Randeva et al., 2002; Bruner et al., 2006; Vigorito et al., 2007; Giallauria et al., 2008; Stener-Victorin et al., 2009) with four of these studies involving aerobic exercise only with no dietary restriction. Brown et al. (2009) reported a significant decrease in triglycerides following 12 weeks of exercise in a weight maintenance study without dietary restriction (Brown et al., 2009). Thomson et al. reported a significant improvement in cholesterol, HDL and LDL within 10 weeks in all groups including diet only (5000–6000 kJ/day consisting 30% protein, 40% carbohydrate and 30% fat) and in combination with aerobic exercise or aerobic and resistance exercise. By 20 weeks significant improvements in cholesterol (10–11%) and LDL concentrations (13–14%) persisted, but only in the two exercise with dietary restriction groups (Thomson et al., 2008).

Blood pressure

Six studies reported results for blood pressure, all including systolic and diastolic pressure (Randeva et al., 2002; Vigorito et al., 2007; Giallauria et al., 2008; Thomson et al., 2008; Brown et al., 2009; Stener-Victorin et al., 2009). Four studies found no significant change in blood pressure following 12 (Giallauria et al., 2008), 16 (Stener-Victorin et al., 2009) and 24 weeks of exercise (Randeva et al., 2002; Brown et al., 2009). One study reported a significant decrease in resting systolic blood pressure only, from 118 to 114 mmHg following exercise (Vigorito et al., 2007). Another study reported a significant change in both resting systolic (119–110 mmHg) and diastolic (65–60 mmHg) pressure in the diet and aerobic plus resistance exercise group but not in the diet alone or in combination with aerobic exercise groups (Thomson et al., 2008).

Reproductive function

Five of the eight studies reported results for reproductive function (Bruner et al., 2006; Vigorito et al., 2007; Palomba et al., 2008; Thomson et al., 2008; Stener-Victorin et al., 2009), providing a pooled total of 256 participants (141 receiving exercise intervention). Four studies used menstrual diaries to report changes in menstrual duration and frequency (Bruner et al., 2006; Vigorito et al., 2007; Palomba et al., 2008; Thomson et al., 2008) and one study additionally evaluated pregnancy rate (Palomba et al., 2008). Of the five studies reporting menstrual function, three reported improvements in menstrual and/or ovulation frequency following exercise (Vigorito et al., 2007; Palomba et al., 2008; Thomson et al., 2008). Two of the three studies reported significant improvement in menstrual and/or ovulation frequency with exercise intervention when compared with diet (Palomba et al., 2008) or control (Vigorito et al., 2007) groups although one study reported no difference between diet alone or diet and aerobic with or without resistance training groups (Thomson et al., 2008). Improvement in reproductive function was not dependent on length of intervention or the type of exercise performed. Vigorito et al. (2007) reported that 60% (27 of 45) of participants who exercised for 12 weeks reported normal menstrual cycles following intervention, however did not provide details on frequency of menses during the study period or results for the control group (Vigorito et al., 2007). Thomson et al. reported results for menstrual function from available data in 59 out of a total 94 participants following a 20 week intervention. Of the 59 participants, 53 reported menstrual dysfunction at baseline. Following the intervention, 49% reported an overall improvement in ovulation and/or menstrual cyclicity with no difference between diet only and diet plus exercise groups (Thomson et al., 2008). Improvements were defined as a change from non-ovulatory to ovulatory cycles, irregular to regular cycles or improvement in intercycle variation (Thomson et al., 2008). Results from a 24-week study completed by Palomba et al. (2008) reported significantly higher menses frequency and cumulative ovulation rate following exercise (n = 20) in comparison with a low-caloric, high-protein diet alone (n = 20). Menses frequency was defined as percentage of spontaneous menses per number of expected menses during the study period and cumulative ovulation rate was defined as the percentage of ovulatory participants per total number of participants. The pregnancy rate during the intervention in the exercise group was 35%, compared with 10% in the diet group, however, this was not found to be significant (P = 0.058).

Study quality, heterogeneity and compliance

Table III summarizes study quality information across the eight studies (five RCT and three cohort studies), including a brief summary of the strengths and weaknesses of each study. Method of randomization included computer generated sequencing in three RCT studies (Thomson et al., 2008; Brown et al., 2009; Stener-Victorin et al., 2009) and envelope concealment in one study (Bruner et al., 2006). One RCT study reported no randomization methods (Vigorito et al., 2007). In four RCTs, participants were recruited through community advertisement and invitation at local hospitals or clinics (Bruner et al., 2006; Thomson et al., 2008; Brown et al., 2009; Stener-Victorin et al., 2009) or at a clinic attended by a principal investigator (Brown et al., 2009). In two of the three cohort studies, allocation to intervention groups was based on participant preference (Giallauria et al., 2008; Palomba et al., 2008) and in one study the control group was retrospectively defined as those participants who did not start the study or who dropped out within the first 6 weeks of the 24 week intervention (Randeva et al., 2002). In all three cohort studies, recruitment was based on patient referral to a specialist clinic, in which an investigator was based (Randeva et al., 2002; Giallauria et al., 2008; Palomba et al., 2008).

Of the five RCTs, one gave details on exercise supervision, reporting that exercise sessions were monitored by a cardiologist, physiotherapist and graduate nurse (Vigorito et al., 2007). Three studies reported only that exercise was supervised (Bruner et al., 2006), completed in the research facility (Brown et al., 2009) or self-directed (Stener-Victorin et al., 2009) while one study gave no specific detail on exercise supervision (Thomson et al., 2008). Two cohort studies reported that exercise was monitored but gave no further information (Giallauria et al., 2008; Palomba et al., 2008) and one did not supervise exercise (Randeva et al., 2002).
<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Level of evidence</th>
<th>Summary</th>
<th>Compliance</th>
<th>Attendance</th>
<th>Strengths</th>
<th>Weaknesses</th>
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<tbody>
<tr>
<td>Stener-Victorin et al. (2009)</td>
<td>RCT</td>
<td>2</td>
<td>Small subset study from larger cohort. Clinical-based intervention comparing exercise to a novel acupuncture treatment with an untreated control group. Recruitment from 2005 to 2008</td>
<td>100% compliance in subset reported</td>
<td>Mean exercise: 3 ± 0.8 sessions/week. Duration not reported</td>
<td>• RCT&lt;br&gt;• High compliance&lt;br&gt;• No exclusion based on weight</td>
<td>• Small sample size&lt;br&gt;• Unsupervised exercise&lt;br&gt;• Menstrual pattern not recorded during study&lt;br&gt;• Sample size/power calculations not reported&lt;br&gt;• Non-parametric statistical analysis</td>
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<tr>
<td>Brown et al. (2009)</td>
<td>RCT</td>
<td>2</td>
<td>Small, intensive clinical-based intervention. 12–24 weeks duration. Recruitment from 2003 to 2005</td>
<td>Drop-out rate: 45%; n 37 recruited and randomized; results provided for n 20 only</td>
<td>Mean exercise: 204 min/week over 3.6 sessions. 89.8% adherence to prescribed exercise</td>
<td>• RCT&lt;br&gt;• Assessed exercise independent of weight loss&lt;br&gt;• High adherence</td>
<td>• Baseline results for completers only&lt;br&gt;• High drop-out rate&lt;br&gt;• Skewed results</td>
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<tr>
<td>Vigonto et al. (2007)</td>
<td>RCT</td>
<td>2</td>
<td>Intensive, clinical-based intervention. 12 weeks duration. Recruitment date not reported</td>
<td>100% completed</td>
<td>78% adherence (28 ± 2 sessions completed). Average duration 92 ± 28 mins/week</td>
<td>• RCT&lt;br&gt;• High compliance. &lt;br&gt;• No exclusion based on weight</td>
<td>• Randomization methods and sample size/power calculations not reported</td>
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<td>Palomba et al. (2008)</td>
<td>Cohort</td>
<td>3-II</td>
<td>Small, clinical-based intervention. 24 weeks duration. Recruitment from 2004 to 2006</td>
<td>Drop-out rate: 25%; n 30 completed (17 exercise, 13 diet) ITT analysis used</td>
<td>Exercise: 70.6% high adherence (≤2 missed sessions/month)&lt;br&gt;Diet: 76.9% high adherence (at least 4900 kcal/week deficit &amp;/or with 30% protein/45% CHO)&lt;br&gt;Diet comparative group&lt;br&gt;Compliance</td>
<td>• Exercise: 70.6% high adherence (≤2 missed sessions/month)&lt;br&gt;• Diet: 76.9% high adherence (at least 4900 kcal/week deficit &amp;/or with 30% protein/45% CHO)&lt;br&gt;• Diet comparative group&lt;br&gt;• Compliance</td>
<td>• Cohort study&lt;br&gt;• Allocation according to participant preference&lt;br&gt;• ITT analysis to account for drop-out&lt;br&gt;• Under-powered (70%) to detect differences</td>
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<td>Study</td>
<td>Design</td>
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<td>Cohort Description</td>
<td>Exercise Details</td>
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<td>Giallauria et al. (2008)</td>
<td>Cohort</td>
<td>124</td>
<td>Large, intensive, clinical-based intervention. 12 weeks duration; Recruitment from 2004 to 2007</td>
<td>100% completed 80% adherence to exercise (26 ± 2 sessions over 85 ± 4 days)</td>
<td>• High compliance&lt;br&gt; • No exclusion based on weight&lt;br&gt; • Cohort study&lt;br&gt; • Allocation according to participant preference&lt;br&gt; • Randomization methods and sample size/power calculations not reported</td>
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<td>Randeva et al. (2002)</td>
<td>Cohort</td>
<td>21</td>
<td>Retrospective cohort study. Self-directed and monitored study. 24 weeks duration. Recruitment date not reported</td>
<td>Drop-out rate: 43% n 12 completed exercise n 9 did not start/complete program. Used as control</td>
<td>Exercise: 80% adherence to target exercise (23.8 ± 5 mins/day over 24 weeks)&lt;br&gt; • Low-intensity&lt;br&gt; • Low cost&lt;br&gt; • Cohort study&lt;br&gt; • Unsupervised exercise&lt;br&gt; • Control group defined retrospectively&lt;br&gt; • Risk of bias in results</td>
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<td>Bruner et al., (2006)</td>
<td>RCT (pilot)</td>
<td>12</td>
<td>Pilot study with clinical-based setting. 12 weeks duration. Recruitment period not reported</td>
<td>Not reported</td>
<td>Not reported&lt;br&gt; • RCT&lt;br&gt; • Small sample size&lt;br&gt; • Body composition/fat mass assessed with two skin fold tests only</td>
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<td>Thomson et al., (2008)</td>
<td>RCT</td>
<td>94</td>
<td>Large, intensive intervention. Clinical research-based setting. 20 weeks duration. Recruitment period not reported</td>
<td>Drop-out rate: 33% by week 10 45% by week 20 n 52 completed (14 diet 18 diet + aerobic 20 diet + aerobic + resistance)</td>
<td>Not reported&lt;br&gt; • RCT&lt;br&gt; • Resistance training arm&lt;br&gt; • Comprehensive body composition&lt;br&gt; • Intensive with multiple components&lt;br&gt; • High drop-out</td>
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ITT, Intention-to-treat analysis; RCT, randomized controlled trial.

*Level of evidence according to National Health & Medical Research Council guidelines for study quality (NH & MRC, 1999).
Compliance varied considerably between studies. The highest drop-out rates reported were 40–45% across three studies (Randeva et al., 2002; Thomson et al., 2008; Brown et al., 2009), all with a longer study duration (20–24 weeks) and more exercise sessions per week (four to seven) when compared with other studies. Two of these had small participant numbers (Randeva et al., 2002; Brown et al., 2009). One study, also of 24 weeks duration, reported a smaller drop-out rate of 25% however only required three 30-min sessions per week (Palomba et al., 2008). The best compliance was 100%, reported in three studies of 12–16 week duration involving three 30-min sessions per week with no dietary component (Vigorito et al., 2007; Giallauria et al., 2008; Stener-Victorin et al., 2009). Two studies involving a dietary comparison group reported better compliance in the exercise group/s, compared with those receiving dietary restriction (Palomba et al., 2008; Thomson et al., 2008). Overall attendance rate at supervised intervention sessions was above 70% in four studies (Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008; Brown et al., 2009), with no information being provided in two studies (Bruner et al., 2006; Thomson et al., 2008).

All studies except two RCT studies (Brown et al., 2009; Stener-Victorin et al., 2009) used parametric statistical analysis. One study used intention-to-treat analysis (Palomba et al., 2008). One cohort study reported that they were underpowered to detect a significant difference in their primary end-point; cumulative pregnancy rate (Palomba et al., 2008), while all other studies provided no power calculation or detailed methods for determining sample size (Randeva et al., 2002; Bruner et al., 2006; Vigorito et al., 2007; Giallauria et al., 2008; Thomson et al., 2008; Brown et al., 2009; Stener-Victorin et al., 2009).

**Discussion**

This is the first systematic review to evaluate the effectiveness of exercise intervention in PCOS. We evaluated the type, intensity, frequency and duration of exercise and the impact on reproductive and cardiovascular health outcomes in women with PCOS. Lifestyle intervention, including exercise, is first-line therapy in the majority of women with PCOS (Moran et al., 2009b). Despite this, only eight PCOS exercise intervention studies were identified, with only five of these being RCTs. With limited gold standard RCTs, cohort studies were also captured. Across studies, there was considerable variation in study design, including type, frequency and supervision of exercise. Control interventions varied with some involving a dietary comparison group and others having non-therapy controls. Also, the differences in primary and secondary end-points between studies meant that the reproductive and cardiovascular outcomes evaluated in this review were not assessed in all studies. When successful studies, defined as those that showed improvement in at least two primary health outcomes specified for review were assessed, all included supervision of moderate intensity (60–70% VO2max; equivalent to 75–80% HRmax) exercise and reported improvements within 12 weeks of exercise, regardless of study duration. The majority involved at least three 30-min aerobic sessions per week (i.e. 90 min per week) without dietary restriction or resistance exercise (Vigorito et al., 2007; Giallauria et al., 2008; Palomba et al., 2008), although two studies reporting improvements included these components (Bruner et al., 2006; Thomson et al., 2008). The most consistent improvements reported were IR, weight loss including an associated decrease in WHR and reproductive function, although the latter was not widely assessed.

Weight loss occurred in all successful interventions with the exception of one (Bruner et al., 2006). Percentage weight loss varied with the largest changes seen in studies of longer duration, rather than being determined by the amount of exercise performed per week. Decreases in weight, although modest, were associated with changes in WHR, improved ovulation and cardiovascular features, including fasting insulin. The majority of studies reported weight change, but not body composition change, with the exception of one study (Thomson et al., 2008). Assessment using direct methods, such as DEXA would provide valuable information on changes in fat distribution and body composition with exercise. In particular, subcutaneous abdominal adipose tissue and visceral fat are both tightly associated with IR (Holte et al., 1995; Despres et al., 2008) and evaluating changes in these measures following exercise may provide insight into mechanisms of IR in PCOS. The weight change reported across studies is similar to previous non-exercise, dietary interventions in PCOS (weight loss between 5 and 15%) (Moran et al., 2009b). Modest weight loss of ~5% has previously been shown to improve CVD risk factors in overweight PCOS women (Pasquali et al., 1994; Jakubowicz and Nestler, 1997) and in other IR populations (Knowler et al., 2002).

IR underpins many of the clinical features and is a treatment target in PCOS (Teede et al., 2006). IR in PCOS is unique, being intrinsically present in the majority of lean PCOS women and further exacerbated by extrinsic obesity-related IR, however the mechanisms underlying IR are yet to be fully elucidated (Dunaif et al., 1989; Teede et al., 2007). Studies have reported abnormalities within the skeletal muscle insulin signalling pathways (Dunaif et al., 2001; Corbould et al., 2005, 2006) and mitochondria (Skov et al., 2008) in PCOS, ultimately reducing skeletal muscle responsiveness to glucose. Fasting insulin was assessed in all studies as a measure of IR, with most studies reporting improvements following intervention, consistent with enhanced insulin sensitivity within skeletal muscle following exercise (Luciano et al., 2002). However, fasting insulin is an insensitive measure of IR (Carmina and Lobo, 2004). Some studies also reported results for HOMA-IR and QUICKI which correlate more closely with gold standard clamp studies. Previous studies report that sensitivity to detect IR is increased 20% with these methods, in comparison with fasting insulin alone (Carmina and Lobo, 2004). No studies to date have used the most accurate insulin clamp techniques to measure IR. In addition, changes within skeletal muscle following exercise, including mitochondrial function and insulin signalling, are yet to be assessed and may provide insights into mechanisms of IR in PCOS. Despite methodological limitations in available research, this systematic review indicates that moderate exercise improves IR in PCOS when fasting insulin or the indices, HOMA-IR and AUCins, are used as an indicator of IR.

IR is tightly associated with reproductive function in PCOS with previous lifestyle interventions showing improvements in ovulation and menstrual frequency with improved IR (Huber-Buchholz et al., 1999; Moran et al., 2003). Results from this review suggest that exercise improves ovulation rates and is potentially more beneficial than dietary restriction in restoring reproductive function. Enhanced insulin sensitivity underpins restoration of reproductive function through hormonal improvements, including reduced androgens. This
improves the ovarian hormonal environment allowing maturation of follicles thereby restoring ovulation. Most studies assessing reproductive function were 5–6 months in duration and relied on self-reported diaries to document change in ovulation. One study described a 60% improvement in ovulation over 3 months, which is of limited usefulness considering the study duration only covered a few ovulatory cycles. Studies of longer duration assessing ovulatory function with accurate measures, including biochemical markers of ovulation (i.e. pregnanediol) are needed.

Results on other cardiovascular outcomes, including blood pressure and lipid profile, were conflicting across studies owing perhaps to the recruitment of a generally younger population of women who were normotensive and had normal blood lipid levels at baseline. Further, as there is substantial clinical heterogeneity of metabolic risk in PCOS, higher risk PCOS phenotypic groups may not have been captured across studies (Barber et al., 2007; Moran and Teede, 2009a). Improvements in blood pressure have consistently been reported following moderate exercise in hypertensive populations (Kokkinos and Papademetriou, 2000) and in some other studies with normotensive participants (Whelton et al., 2002). Change in lipid profile following exercise intervention is less consistently reported (Leon and Sanchez, 2001; Duncan et al., 2003) and may be more likely to improve through dietary intervention. More research is needed to determine the effects of exercise on these cardiovascular risk factors in PCOS women with varying degrees of metabolic characteristics as results from this review are inconsistent. Future interventions may be improved by targeting specific PCOS populations for intervention, including those that are hypertensive, dyslipidemic or with worsened IR.

This review also highlighted other limitations in existing literature. To date, exercise intervention studies in women with PCOS have generally been small with significant participant drop-out rates over a short study duration. Only five randomized studies assessed the effects of exercise in women with PCOS, hence cohort studies were included, increasing the risk of bias in results interpretation. Comprehensive end-point measures were generally not used and drawing useful conclusions remains challenging. Furthermore, all studies identified included young women who were predominantly obese. In order to evaluate exercise as an independent therapy in PCOS, exercise needs to be assessed in a broad range of women across age and BMI ranges with varying phenotypic characteristics of PCOS, including those that are ovulatory/anoovulatory and hyperandrogenic/non-hyperandrogenic (Moran and Teede, 2009a). There are also a lack of studies that assess changes in CVD risk factors and reproductive outcomes with exercise in lean women (BMI <25 kg/m²) with PCOS which also represent a clinically relevant population. To assess the efficacy of exercise therapy independent of weight loss, more weight maintenance studies would be beneficial to assess cardiometabolic and reproductive improvements in PCOS in the absence of weight loss. Improvements in end-points with exercise in PCOS should also be compared with those in non-PCOS controls as this has not been studied to date and may facilitate insights into mechanisms of IR in PCOS. Furthermore, in all studies, moderate-intensity exercise over relatively short duration produced favourable outcomes. However a gap remains as to the short- and long-term effects of other exercise intensities on health parameters in PCOS, including low- (<50% VO2max) or very high-intensity exercise (>85% VO2max). Studies of longer duration would also provide more insight into the sustainability and tolerance of frequent exercise long-term as some studies were high intensity, involving frequent contact which may have attributed to high drop-out rates.

Conclusions and recommendations

PCOS is a prevalent disorder which is underpinned by weight-independent IR and associated with reproductive and cardiovascular complications in women. Lifestyle modification, including exercise, is the first-line therapeutic approach in improving health outcomes in PCOS. Despite this, the present review identified only eight suitable studies, five of these RCTs, incorporating exercise intervention in PCOS. This highlights the need for more comprehensive RCTs. Results from this review suggest that improvements in outcomes assessed were not dependant on length of exercise intervention or the type and frequency of exercise performed as significant clinical benefits were observed in more sustainable, less intensive studies, of shorter duration. Regular, moderate-intensity aerobic exercise over a short period improves reproductive outcomes including ovulation and menstrual cycle regulation in addition to reducing weight and IR in young, overweight women with PCOS. Future studies would benefit by using gold standard, comprehensive end-points to provide mechanistic insights into PCOS particularly around IR, a central feature of this endocrinopathy. Based on the results from this review, women with PCOS should be advised to engage in at least 90 min of aerobic activity per week at moderate intensity (60–70% VO2max) to achieve improved reproductive and cardiometabolic outcomes.

Supplementary data

Supplementary data are available at http://humupd.oxfordjournals.org/.

Funding

H.J.T. and L. J.M. are NH & MRC research fellows and C.L.H. is a NH & MRC funded Ph.D. Scholar.

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