Prenatal Exercise and Cardiorespiratory Health and Fitness: A Meta-analysis

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ABSTRACT

CAI, C., S.-M. RUCHAT, A. SIVAK, and M. H. DAVENPORT. Prenatal Exercise and Cardiorespiratory Health and Fitness: A Meta-analysis. *Med. Sci. Sports Exerc.*, Vol. 52, No. 7, pp. 1538–1548, 2020. **Purpose:** This study aimed to examine the influence of prenatal exercise on maternal cardiorespiratory health and fitness during pregnancy. **Methods:** Online databases were searched up to February 25, 2019. Studies of randomized controlled trials (RCTs) were eligible, which contained information on the relevant population (pregnant women), intervention (subjective or objective measures of frequency, intensity, duration, volume, or type of exercise), comparator (no exercise intervention), and outcomes (maternal cardiorespiratory fitness, including $\dot{V}O_{2max}$, submaximal $\dot{V}O_2$, $\dot{V}O_2$ at anaerobic threshold, and cardiorespiratory health, including resting heart rate, and resting systolic and diastolic blood pressures during pregnancy). **Results:** From 2699 unique citations, 26 RCTs (N = 2292 women) were included. Of these, one study reported measured $\dot{V}O_{2max}$, seven reported predicted $\dot{V}O_{2max}$, three reported submaximal $\dot{V}O_2$, and two studies reported VO_{2AT}. "Low"- to "high"-certainty evidence revealed that exercise was associated with improved predicted/measured $\dot{V}O_{2max}$ (5 RCTs, n = 430; mean difference [MD], 2.77 $mL \cdot kg^{-1} \cdot min^{-1}$; 95% confidence interval [CI], 0.32 to 5.21 $mL \cdot kg^{-1} \cdot min^{-1}$; I^2 = 69%), reduced resting heart rate (9 RCTs, n = 637; MD, −1.71 bpm; 95% CI, −3.24 to −0.19 bpm; I^2 = 13%), resting systolic blood pressure (16 RCTs, n = 1672; MD, −2.11 mm Hg; 95% CI, −3.71 to −0.51 mm Hg; I^2 = 69%), and diastolic blood pressure (15 RCTs, n = 1624; MD, −1.77 mm Hg; 95% CI, −2.90 to −0.64 mm Hg; I^2 = 60%). **Conclusion:** Prenatal exercise interventions improve maternal predicted/measured $\dot{V}O_{2max}$ and reduce resting heart rate and blood pressure. This review highlights the need for additional high-quality studies of cardiorespiratory fitness (namely, $\dot{V}O_{2max}$ and $\dot{V}O_2$ peak) in pregnancy. PROSPERO registration number: CRD42019131249.

Key Words: PRENATAL EXERCISE, CARDIORESPIRATORY FITNESS, $\dot{V}O_{2max}$, HEART RATE, BLOOD PRESSURE

Cardiorespiratory fitness (CRF) is the ability of the respiratory and cardiovascular systems to provide muscles with oxygen during physical activity and is the most commonly quantified as maximal oxygen uptake ($\dot{V}O_{2max}$) (1). Low $\dot{V}O_{2max}$ is strongly associated with the development of cardiovascular disease and mortality (2). Given this relationship, the American Heart Association now recommends the measurement of CRF in clinical practice (3–6). There are three methods popular for the measurement of CRF. Maximal exercise testing involves measuring ventilatory oxygen ($\dot{V}O_2$) at the maximal level of aerobic exercise ($\dot{V}O_{2max}$) and is the gold standard measure of CRF. However, $\dot{V}O_{2max}$ is often not achieved during pregnancy resulting in a peak exercise test. Submaximal exercise testing involves measuring or imputing $\dot{V}O_2$ at some predetermined target of heart rate or work output, which can be used to estimate or predict $\dot{V}O_{2max}$. When fitness is increased, $\dot{V}O_2$ at this physiological threshold is higher because of the body's increased ability to do work. Aerobic capacity at the anaerobic threshold (VO_{2AT}) involves measuring $\dot{V}O_2$ at the anaerobic threshold, that is, the point at which the body switches from aerobic to anaerobic metabolism, as measured by the inflection point of blood lactate. VO_{2AT} is an important measure of CRF, as it may be a better indicator of endurance exercise performance than $\dot{V}O_{2max}$, and provides a comparator upon which to base changes in fitness at submaximal levels (7).

In nonpregnant populations, interventions prescribing moderate-to-vigorous physical activity are designed to increase CRF through numerous adaptations including increases in skeletal muscle vasodilatory capacity via mediators, such as nitric oxide, elevated cardiac output, reduced resting blood pressure,

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and increased oxygen-carrying capacity of the blood (8–10). In line with the results of the 11 meta-analyses published by the Canadian Guidelines for Physical Activity throughout Pregnancy consensus panel (11–21), exercise interventions in the prenatal period, which were likely to improve CRF, resulted in a ~40% reduction in serious complications of pregnancy (gestational diabetes, preeclampsia, and hypertension in pregnancy) without increasing the risk of having a miscarriage, preterm delivery, or small baby. As a result, we believe that the prenatal period is a critical time to increase CRF. Because of the low CRF of women of childbearing age (22), there are potentially serious detrimental consequences for women who do not increase their CRF during the prenatal period (23,24). Although pregnant women are encouraged to engage in at least 150 min of moderate-to-vigorous physical activity each week to improve maternal and fetal health (3,25), the effect of prenatal exercise on CRF has surprisingly not been systematically examined, and thus, consensus has not been clearly established in this area. Multiple factors complicate the integration and interpretation of data in this field. Although recent guidelines no longer provide an upper limit for exercise intensity (3,26), many historical guidelines have recommended avoiding activities above a moderate intensity (27,28), leading to an increased occurrence of submaximal testing and measurement of $\dot{V}O_2$ at the anaerobic threshold. Furthermore, no submaximal exercise test protocol has been validated for use in pregnancy. However, synthesis of the available research is requisite to highlight this important gap and promote propagation of work in this area.

Pregnancy is associated with a number of profound physiological adaptations to the cardiovascular system including an increase in nitric oxide–mediated dilation, resting heart rate and cardiac output, and a decrease in blood pressure that may influence the normal adaptations to chronic exercise (29). As part of a larger Cochrane review published in 2009, it was implied that regular exercise during pregnancy may improve or maintain physical fitness. However, these data were not synthesized in meta-analyses because of inconsistency in the measures used to quantify physical fitness across studies (30). In the subsequent decade, additional randomized controlled trials (RCT) reporting on CRF during pregnancy have been published. Therefore, this systematic review and meta-analysis sought to assess whether prenatal exercise improves maternal CRF including aerobic capacity ($\dot{V}O_2$), submaximal aerobic capacity, and $\dot{V}O_2$ at anaerobic threshold, or cardiorespiratory health including resting heart rate and blood pressure in pregnant women.

METHODS

Protocol and Registration

This review was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines on systematic reviews and meta-analyses (31). The protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO; Registration No. CRD42019131249).

Information Sources

A structured search of electronic databases (MEDLINE, EMBASE, CINAHL, Scopus, Web of Science, Cochrane Library, Trip, and ProQuest Dissertations & Theses) up to February 25, 2019, was performed by a research librarian (A. S.) and peer reviewed by second research librarian (see Supplemental Digital Content, Appendix, for the online supplement for complete search strategies, http://links.lww.com/MSS/B903). The reference lists of included articles and relevant systematic reviews were checked manually to search for potentially relevant articles. The language of publication was not restricted. Studies published in languages other than English, Spanish, Chinese, or French that were considered to be potentially relevant were translated through Google Translate, and if deemed potentially relevant, they were translated by a native speaker. The complete search strategy is presented in the online supplement (Supplemental Digital Content, Appendix, http://links.lww.com/MSS/B903).

Eligibility Criteria

This study was guided by the participants, interventions, comparisons, outcomes, and study design framework.

Study design. The study design was restricted to RCTs. Case studies, narrative or systematic reviews, letters, commentaries, and editorials were excluded.

Population. The population of interest was pregnant women (at any stage of pregnancy).

Intervention. The intervention was any reported measures of frequency, intensity, duration, volume, or type of prenatal exercise (beginning after conception) lasting at least 1 wk.

Comparison. Eligible comparator was no exercise intervention during pregnancy.

Outcomes. Relevant outcomes were measures of CRF (maximal aerobic capacity ($\dot{V}O_{2max}$), submaximal aerobic capacity (submaximal $\dot{V}O_2$), aerobic capacity at the anaerobic threshold ($\dot{V}O_{2AT}$)) and cardiorespiratory health (resting heart rate, resting systolic blood pressure, and resting diastolic blood pressure).

Study Selection and Data Extraction

Two reviewers (C. C. and M. H. D.) independently assessed the titles and abstracts of articles identified by the search. Studies were selected for full-text review by at least one reviewer. All full-text articles were screened by two independent reviewers for eligibility (C. C. and M. H. D.). In the event of a disagreement, eligibility was determined based on discussion between the two reviewers and by decision of a third reviewer when needed. Two reviewers independently extracted the data in Microsoft Excel. If the study had multiple publications, the most recent or complete publication was selected; however, relevant data from all publications related to each unique study were extracted. Study characteristics (e.g., study period, study design, and country) and population characteristics (e.g., number of participants, age, prepregnancy body mass index (BMI), parity, and pregnancy complications), exposure (e.g., exercise frequency, intensity, time, and type of exercise), and outcomes ($\dot{V}O_{2max}$, submaximal $\dot{V}O_2$, $\dot{V}O_{2AT}$, resting heart rate, resting systolic blood pressure, and resting diastolic blood pressure) were extracted (Supplemental Table 1, Supplemental Digital Content, Appendix, http://links.lww.com/MSS/B903). If data were not available for extraction, the corresponding authors were contacted for additional information. Where data were only presented in figures and authors could not be reached via e-mail, data were extracted using WebPlotDigitizer (Web Plot Digitizer, V.3.11; Ankit Rohatgi, 2017, Austin, TX), an online tool that supports the extraction of numeric data from graphs (32,33).

Quality Assessment and Certainty Assessment

Quality assessment (risk of bias). The risk of bias was evaluated following the *Cochrane Handbook* (34). All RCTs were examined for potential sources of bias (i.e., selection bias, reporting bias, performance bias, detection bias, attrition bias, and "other" sources of bias). Risk of bias across studies was rated as "serious" when studies with the greatest influence on the pooled result (contributing >50% of the weight given in forest plots) presented a "high"-risk of bias. Two researchers (C. C. and M. H. D.) conducted the evaluation process independently. The differences in ratings were resolved through discussion. The quality assessment of each included study is presented in online Supplemental Table 2 (Supplemental Digital Content, Appendix, http://links.lww.com/MSS/B903).

Certainty assessment (Grading of Recommendations Assessment, Development and Evaluation). Two reviewers (C. C. and M. H. D.) independently assessed the certainty of evidence across studies for each outcome using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) tool (35). Evidence from RCTs began with a high certainty of evidence rating and was downgraded if there were concerns of risk of bias, indirectness (e.g., cointerventional study design), inconsistency, imprecision, or risk of publication bias. Inconsistency was considered serious when heterogeneity was high ($I^2 \geq 50\%$) or when only one study was assessed. Imprecision was considered serious when the 95% confidence interval (CI) crossed the line of no effect. Publication bias was assessed via funnel plots when more than 10 studies were included in the forest plot. When there were fewer than 10 studies, publication bias was not downgraded. The GRADE assessment (35) is presented in online Supplemental Table 3 (Supplemental Digital Content, Appendix, http://links.lww.com/MSS/B903).

Data Synthesis

Review Manager v5.3 (Cochrane Collaboration, Copenhagen, Denmark) was used to conduct the statistical analyses. Postintervention mean values and their SDs were used in the meta-analyses. When the studies reported SE, SD values were obtained from the SEM by multiplying by the square root of the sample size (36). Significance was set at $P < 0.05$. Inverse-variance weighting was applied to obtain change scores using a random-effects model. *A priori*–determined subgroup analyses were conducted when possible for the following subgroups: 1) women diagnosed with diabetes (gestational (GDM), type 1 or type 2) compared with women without diabetes; 2) women with prepregnancy BMI ≥25.0 $kg \cdot m^{-2}$ compared with women with prepregnancy BMI <25.0 $kg \cdot m^{-2}$; 3) women who were previously inactive compared with those who were previously active; 4) duration, frequency, intensity, volume, or start of exercises; and 5) type of test used to measure $\dot{V}O_2$ (e.g., predicted or direct measurement of $\dot{V}O_{2max}$, submaximal $\dot{V}O_2$, and $\dot{V}O_{2AT}$). If a study did not provide sufficient detail to allow it to be grouped into the *a priori* subgroups, then a third group called "unspecified" was created.

When 10 or more studies were included (36), meta-regression analyses were also performed using the metareg command in STATA 15.0 (37) to examine a linear association between cardiorespiratory health and fitness and intensity, frequency, duration, and volume of exercise, using a random-effects model and illustrating the regression line. I^2 statistic was used to assess the heterogeneity between the studies. In the case of $I^2 \geq 50\%$, heterogeneity was explored further with sensitivity analyses. If data were not suitable for meta-analysis, authors were contacted to obtain additional information. Data were synthesized narratively if authors were unable to provide additional numerical data.

RESULTS

Study Selection

The literature search identified 2699 unique citations, with 26 RCTs ($n = 2292$) from 11 countries included in this systematic review. A Preferred Reporting Items for Systematic Reviews and Meta-Analyses diagram of the search and study selection results is shown in Figure 1. A complete list of excluded studies with reasons is presented at the end of the online supplement (Supplemental Digital Content, Appendix, http://links.lww.com/MSS/B903). Six corresponding authors were sent letters requesting additional information or clarification of data from six studies (38–43). Two authors responded and one author provided additional information for the meta-analysis (see Supplemental Digital Content, Appendix, for the detailed list, http://links.lww.com/MSS/B903.)

Study Characteristics

Individual study characteristics are presented in online Supplemental Table 1 (Supplemental Digital Content, Appendix, http://links.lww.com/MSS/B903). Among the included interventions, the frequency of exercise ranged from 2 to 7 $d \cdot wk^{-1}$, at low to vigorous intensity ranging from 15 to 60 min per session. Exercise interventions were initiated at >6 wk of pregnancy, ranging in duration from 6 to 32 wk. The exercise modalities included walking, stationary cycling, muscle workout, treadmill walking, stair stepping, aerobics classes, and a combination of various types of exercise. $\dot{V}O_{2max}$ was assessed using the Danish Step test (predicted) (44), a multistage treadmill

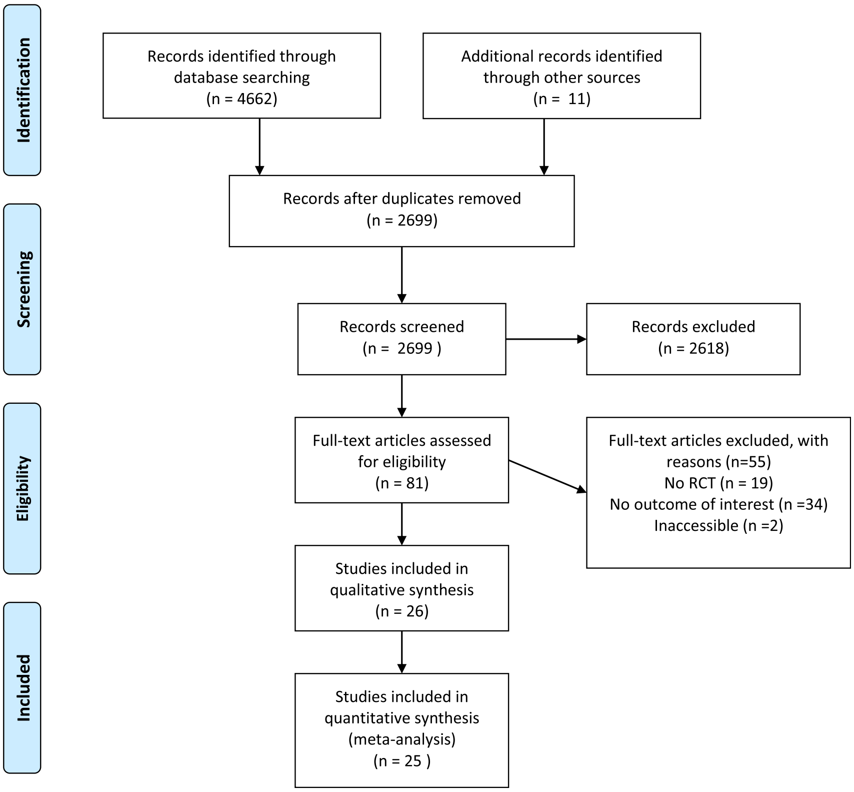

FIGURE 1—Study flow diagram.

test (predicted) (40,45), the Schwinn cycle ergometer test (predicted) (46), and a maximal progressive exercise test on a cycle ergometer (directly measured) (41). Submaximal $\dot{V}O_2$ was assessed using a walking test (47), stationary cycle test (48), and 6-min walk test (49). $\dot{V}O_{2AT}$ was assessed using a treadmill test (50,51).

Details of compliance were unavailable for 12 of 26 studies (40,41,45,46,51–58). Reported compliance (i.e., percent of exercise sessions attended by study participants) was >80% in seven studies (38,42,50,59–62), around 50%–79% in six studies (47–49,63–65), and 33% in another study (66). Protocol compliance was calculated based on the number of participants who were not lost to follow-up or excluded because of medical complications.

Quality Assessment and Certainty Assessment

Common sources of bias included unreported outcome blinding, poor or unreported compliance, and inappropriate treatment of missing data when attrition occurred (Supplemental Table 2, Supplemental Digital Content, Appendix, http://links.lww.com/MSS/B903). Overall, the certainty of evidence ranged from "low" to "high" (Supplemental Table 3, Supplemental Digital Content, Appendix, http://links.lww.com/MSS/B903). The most common reasons for downgrading the certainty of evidence were serious risk of bias, inconsistency, indirectness, and imprecision. No evidence of publication bias was observed (Supplemental Figs. 41–42, Supplemental Digital Content, Appendix, http://links.lww.com/MSS/B903).

Synthesis of Data

$\dot{V}O_{2max}$. All but one study utilized protocols that predicted, rather than measured, $\dot{V}O_{2max}$. Overall, there was low-certainty evidence from five RCTs (n = 430 women) (40,41,44,45,58), indicating that prenatal exercise interventions were associated with an increase in $\dot{V}O_{2max}$ after intervention compared with no exercise (mean difference [MD], 2.77 $mL\cdot kg^{-1}\cdot min^{-1}$; 95% CI, 0.32–5.21 $mL\cdot kg^{-1}\cdot min^{-1}$; $I^2 = 69\%$; Fig. 2). The certainty of evidence was downgraded from high to low because of indirectness and inconsistency.

There was high-certainty evidence from three RCTs (n = 77 women) (38,42,46), indicating that prenatal exercise interventions were associated with a small increase in absolute $\dot{V}O_{2max}$ (in liters per minute) compared with no exercise (MD, 0.25 $L\cdot min^{-1}$; 95% CI, 0.11–0.39 $L\cdot min^{-1}$; $I^2 = 0\%$; Fig. 3A). One study (66) that could not be included in the meta-analysis ($\dot{V}O_{2max}$ was reported in changes from baseline) indicated that the prenatal exercise intervention was not associated with $\dot{V}O_{2max}$ changes from baseline compared with no exercise (n = 74 women; exercise group: mean ± SD, 0.24 ± 2.1 $mL\cdot kg^{-1}\cdot min^{-1}$; control group: mean ± SD, −0.71 ± 2.6 $mL\cdot kg^{-1}\cdot min^{-1}$; "moderate" certainty, downgraded because of inconsistency).

$\dot{V}O_2$ at the anaerobic threshold. Overall, there was low-certainty evidence from two RCTs (n = 116 women)

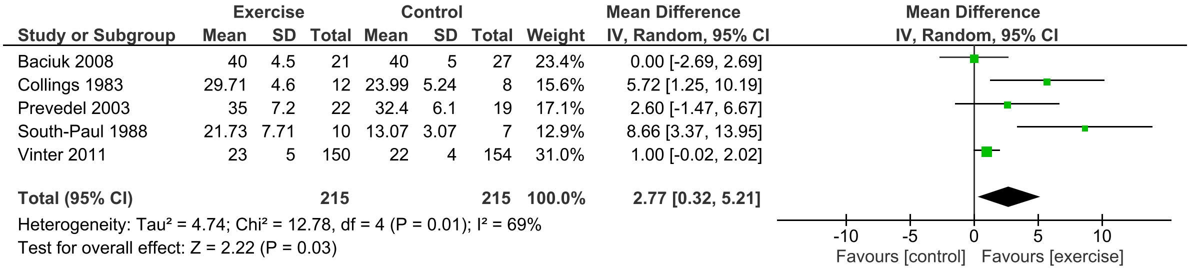

FIGURE 2—Effects of prenatal exercise intervention compared with control on $\dot{V}O_{2max}$ after intervention (predicted or measured; in milliliters per kilogram per minute). Analyses conducted with a random-effects model. MD values are in milliliters per kilogram per minute. *df*, degrees of freedom; IV, inverse variance.

(50,51) showing that prenatal exercise interventions did not influence $\dot{V}O_{2AT}$ compared with no exercise (MD, 1.22 mL·kg^{-1}·min^{-1}; 95% CI, −0.83 to 3.28 mL·kg^{-1}·min^{-1}; I^2 = 82%; Fig. 3B). The certainty of evidence was downgraded because of inconsistency and imprecision.

Submaximal $\dot{V}O_2$. There was high-certainty evidence from three RCTs (*n* = 177 women) (47–49), indicating that women who participated in a prenatal exercise intervention had a small increase in $\dot{V}O_2$ at submaximal exercise intensities after the intervention compared with those who did not receive an exercise intervention (MD, 0.61 mL·kg^{-1}·min^{-1}; 95% CI, 0.17–1.04 mL·kg^{-1}·min^{-1}; I^2 = 0%; Fig. 3C). One study (45) that could not be included in the meta-analysis ($\dot{V}O_2$ was reported in liters per minute) indicated that a prenatal exercise intervention was associated with a small increase in submaximal $\dot{V}O_2$ compared with no exercise (*n* = 169 women; exercise group: mean ± SD, 1.65 ± 0.38 L·min^{-1}; control group: mean ± SD, 1.52 ± 0.24 L·min^{-1}; moderate certainty, downgraded because of inconsistency). Another study that could not be included in the meta-analysis (SD was not provided) (58) indicated that a prenatal exercise intervention was not associated with postintervention submaximal $\dot{V}O_2$ compared with no exercise (*n* = 13 women; exercise group: mean, 20.13 mL·kg^{-1}·min^{-1}; control group: mean, 19.01 mL·kg^{-1}·min^{-1}; low certainty, downgraded because of serious risk of bias and inconsistency).

Resting heart rate. Overall, there was high-certainty evidence from nine RCTs (*n* = 637 women) (38,40,49,51,52,56,57,66,67) showing that resting heart rate after intervention was

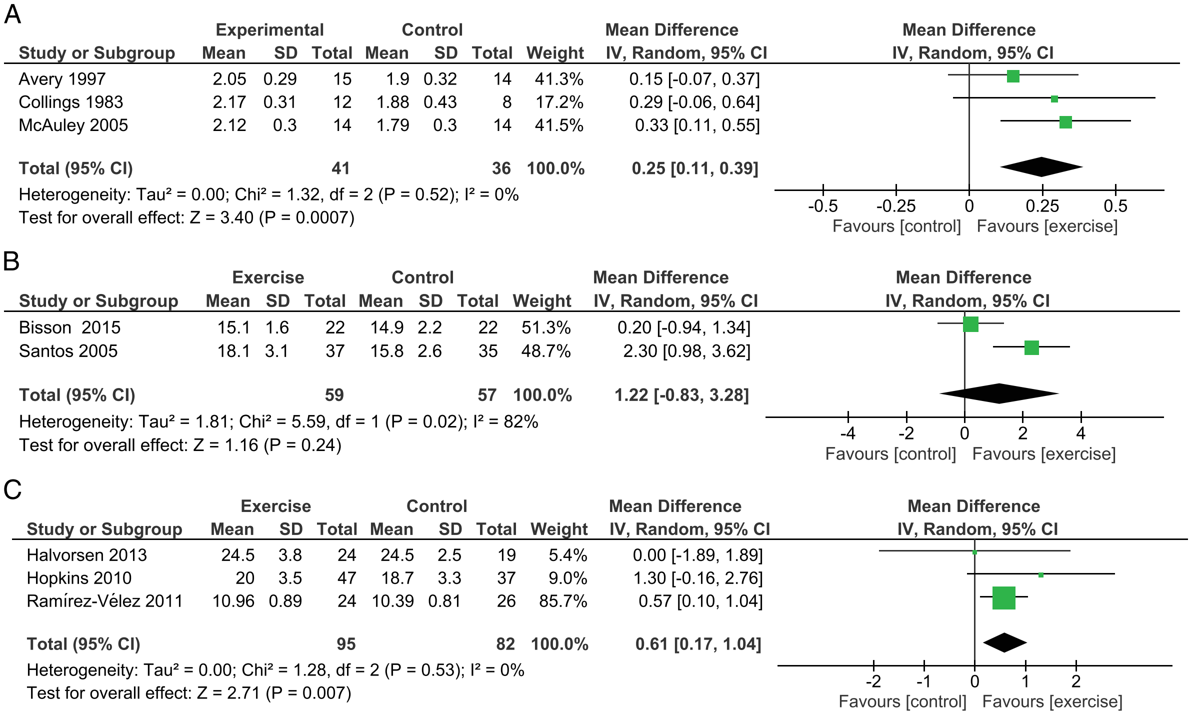

FIGURE 3—Effects of prenatal exercise intervention compared with control on absolute $\dot{V}O_{2max}$, $\dot{V}O_2$ at anaerobic threshold, and submaximal $\dot{V}O_2$. A, Effects of prenatal exercise intervention compared with control on absolute $\dot{V}O_{2max}$ (in liters per minute). MD values are in liters per minute. B, Effects of prenatal exercise intervention compared with control on $\dot{V}O_2$ at the anaerobic threshold. MD values are in milliliters per kilogram per minute. C, Effects of prenatal exercise intervention compared with control on submaximal $\dot{V}O_2$. MD values are in milliliters per kilogram per minute. Analyses conducted with a random-effects model. *df*, degrees of freedom; IV, inverse variance.

associated with a decrease in resting heart rate compared with no exercise (MD, −1.71 bpm; 95% CI, −3.24 to −0.19 bpm; I^2 = 13%; Fig. 4).

Resting systolic blood pressure. Overall, there was low-certainty evidence from 16 RCTs (*n* = 1672 women) (40,44,49,52,54–57,59–64,66,67), indicating that prenatal exercise interventions were associated with a small decrease in resting systolic blood pressure compared with no exercise (MD, −2.11 mm Hg; 95% CI −3.71 to −0.51 mm Hg; I^2 = 69%; Fig. 5). The certainty of evidence was downgraded because of inconsistency and indirectness.

Resting diastolic blood pressure. Overall, there was low-certainty evidence from 15 RCTs (*n* = 1624 women) (44,49,52,54–57,59–64,66,67), indicating that prenatal exercise interventions were associated with a small decrease in resting diastolic blood pressure compared with no exercise (MD, −1.77 mm Hg; 95% CI, −2.90 to −0.64 mm Hg; I^2 = 60%; Fig. 6). The certainty of evidence was downgraded because of inconsistency and indirectness. One study that could not be included in the meta-analysis (SD was not provided) (40) indicated that prenatal exercise intervention was not associated with postintervention resting diastolic blood pressure compared with no exercise (*n* = 48 women; exercise group: mean, 68.91 mm Hg; control group: mean, 65.58 mm Hg; low certainty, downgraded because of serious risk of bias, and inconsistency).

Subgroup analysis. Subgroup analyses of women diagnosed with GDM, a prepregnancy BMI ≥25.0 kg·m^{-2}, previously sedentary women, or metrics of the exercise prescription (frequency, intensity, duration, or volume of exercise) were not significantly different between groups. However, when stratifying by duration of the intervention, studies with a duration less than 20 wk induced a greater reduction in resting diastolic blood pressure compared with those lasting longer than 20 wk (−2.92 vs −0.89 mm Hg; $P < 0.05$ for subgroup differences). Moreover, women who initiated the exercise intervention before 16-wk gestational age had a smaller reduction in resting diastolic blood pressure after the intervention compared with women who began the intervention between 16- and 20-wk gestational ages, and more than 20-wk gestational age (−0.93, −2.75, and −3.91 mm Hg, respectively; $P < 0.05$ for subgroup differences). All other subgroup analyses were not significantly different (online Supplemental Figs. 1–26, http://links.lww.com/MSS/B903).

Meta-regression. Meta-regression analysis using linear regression were conducted when at least 10 studies with sufficient data were available (36). Thus, meta-regression analyses were performed for systolic blood pressure and diastolic blood pressure. Meta-regression analyses did not identify a dose–response relationship between frequency, intensity, duration, volume, or start of exercise and the reduction of systolic or diastolic blood pressure (Supplemental Figs. 27–40, Supplemental Digital Content, Appendix, http://links.lww.com/MSS/B903).

DISCUSSION

In this systematic review and meta-analysis of 26 RCTs (*n* = 2292), there was low- to high-certainty evidence demonstrating that relative, submaximal, and absolute $\dot{V}O_{2max}$ were increased, whereas while resting heart rate, systolic blood pressure, and diastolic blood pressure were reduced after the exercise intervention. Dose–response analyses did not identify a relationship between the frequency, intensity, duration, volume, or timing of the initiation of exercise with any markers of cardiorespiratory health.

The current meta-analysis demonstrated an 8.6% improvement in $\dot{V}O_{2max}$. A 1-metabolic-equivalent (3.5 mL·kg^{-1}·min^{-1}) increase in $\dot{V}O_{2max}$ is associated with a 12% reduction in mortality (68). However, other studies in clinical populations have suggested that even a 6% increase is clinically meaningful and associated with a reduction in hospitalization for cardiovascular events, cardiovascular mortality, and all-cause mortality (69). Although a threshold for clinically significant improvements in $\dot{V}O_{2max}$ has not been developed for pregnancy, improvements of a similar magnitude have been associated with increased placental growth and functional capacity supporting efficient transfer of oxygen to the fetus to enhance growth and development (70,71). Studies in pregnant populations have linked low-pregnancy $\dot{V}O_{2max}$ with adverse maternal

	Exercise			Control				Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Random, 95% CI
Baciuk 2008	99.28	13.01	21	96.39	13.02	27	4.0%	2.89 [-4.53, 10.31]
Erkkola 1976	77.8	8.5	30	79.7	8.2	32	11.6%	-1.90 [-6.06, 2.26]
Guelfi 2016	84	10	84	85	9	85	21.3%	-1.00 [-3.87, 1.87]
McAuley 2005	96	11.23	14	93	11.23	14	3.2%	3.00 [-5.32, 11.32]
Ramírez-Vélez 2011	77.84	12.8	24	86.28	13.24	26	4.2%	-8.44 [-15.66, -1.22]
Santos 2005	89.5	10.7	37	88.6	11.7	35	7.9%	0.90 [-4.29, 6.09]
Seneviratne 2016	82	8.4	37	85	11.3	37	10.0%	-3.00 [-7.54, 1.54]
Vladimirov 2015	71.27	5.61	50	74.36	5.61	38	28.1%	-3.09 [-5.46, -0.72]
Vázquez-Lara 2018	82.4	4.8	18	82.8	11	28	9.6%	-0.40 [-5.04, 4.24]
Total (95% CI)			**315**			**322**	**100.0%**	**-1.71 [-3.24, -0.19]**

Heterogeneity: Tau² = 0.69; Chi² = 9.16, df = 8 (P = 0.33); I² = 13%
Test for overall effect: Z = 2.21 (P = 0.03)

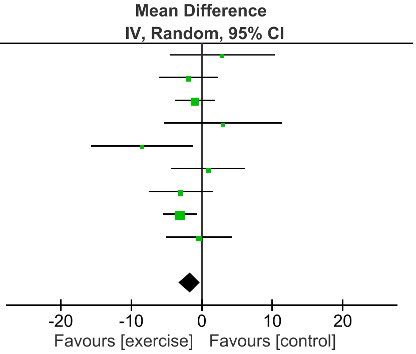

FIGURE 4—Effects of prenatal exercise compared with control on resting heart rate. Analyses conducted with a random-effects model. MD values are in beats per minute. *df*, degrees of freedom; IV, inverse variance.

Study or Subgroup	Exercise Mean	Exercise SD	Exercise Total	Control Mean	Control SD	Control Total	Weight	Mean Difference IV, Random, 95% CI
Baciuk 2008	108.98	13.83	21	104.26	10.19	27	3.7%	4.72 [-2.33, 11.77]
Barakat 2011	114.4	9.1	34	113.7	9.6	33	6.5%	0.70 [-3.78, 5.18]
Barakat 2012	113.8	15.1	138	115.6	11.7	152	8.8%	-1.80 [-4.93, 1.33]
Barakat 2014	114.45	10.43	107	113.33	11.37	93	9.0%	1.12 [-1.92, 4.16]
Erkkola 1976	123.4	14	30	129.5	9.9	32	4.6%	-6.10 [-12.17, -0.03]
Garnæs 2016	120.4	12.4	38	128.1	12.6	36	5.0%	-7.70 [-13.40, -2.00]
Guelfi 2016	103	12	84	104	12	85	7.9%	-1.00 [-4.62, 2.62]
Haakstad 2016	112	8	35	119	14	26	4.6%	-7.00 [-13.00, -1.00]
Halse 2015	110	13	20	109	9	20	3.8%	1.00 [-5.93, 7.93]
Perales 2012	105.3	19.6	35	108.3	9.6	9	2.5%	-3.00 [-12.03, 6.03]
Ramírez-Vélez 2011	109.36	8.74	24	103.31	12.55	26	4.7%	6.05 [0.09, 12.01]
Rodríguez 2012	115	14	25	113	9	30	4.3%	2.00 [-4.36, 8.36]
Seneviratne 2016	113.2	12.2	37	118.5	9.8	37	5.7%	-5.30 [-10.34, -0.26]
Vinter 2011	122	3	150	124	3	154	13.1%	-2.00 [-2.67, -1.33]
Vladimirov 2015	130.32	4	50	135.09	2.79	38	12.1%	-4.77 [-6.19, -3.35]
Vázquez-Lara 2018	105.8	12	18	117	13	28	3.5%	-11.20 [-18.54, -3.86]
Total (95% CI)			**846**			**826**	**100.0%**	**-2.11 [-3.71, -0.51]**

Heterogeneity: Tau² = 4.97; Chi² = 47.87, df = 15 (P < 0.0001); I² = 69%

Test for overall effect: Z = 2.58 (P = 0.010)

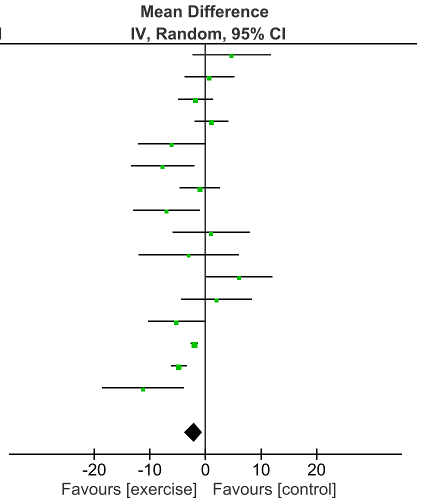

FIGURE 5—Effects of prenatal exercise intervention compared with control on resting systolic blood pressure. Analyses conducted with a random-effects model. MD values are in millimeters of mercury. *df*, degrees of freedom; IV, inverse variance.

and infant outcomes, such as elevated maternal blood pressure, low neonatal Apgar score, and long duration of labor (23,24). As an independent predictor of several metabolic risk factors (e.g., waist circumference, visceral adipose tissue, blood pressure, lipid concentrations, insulin resistance, and systemic inflammation) (9,72), low CRF may be also associated with adverse cardiometabolic disturbances during pregnancy (e.g., GDM, gestational hypertension, and preeclampsia), which are the leading cause of maternal and fetal morbidity and mortality (73,74). Several large longitudinal studies have shown that prepregnancy CRF was inversely associated with the risk of GDM, preterm birth, and small-for-gestational age birth (75,76). Theoretically, increasing $\dot{V}O_{2\max}$ could improve outcomes either directly or via associated improvements in oxygen-carrying capacity, muscle oxygen extraction, and cardiac output; however, this is beyond the scope of this review to comment (77).

Prenatal exercise was associated with a reduction in resting heart rate. Although clinically meaningful changes in resting heart rate have not been established, the American Heart Association suggests that within the normal range (60–100 bpm), the lower resting heart rate is better, as it is inversely related to life expectancy and positively related to reductions in cardiovascular and all-cause mortality (78). Clinically meaningful improvements in blood pressure (>2 mm Hg) were also observed in women who exercised during pregnancy. In nonpregnant populations, a reduction in blood pressure of this magnitude translates a significant reduction in the incidence of cardiovascular disease in both hypertensive and normotensive individuals if achieved at a population level (79,80). A recent meta-analysis showed that prenatal exercise intervention was associated with a significant relative reduction in the odds of gestational hypertension (39%) and preeclampsia (41%) (14). Our analysis further supported the benefits of prenatal exercise in controlling

Study or Subgroup	Exercise Mean	Exercise SD	Exercise Total	Control Mean	Control SD	Control Total	Weight	Mean Difference IV, Random, 95% CI
Barakat 2011	65.9	7.2	34	66.4	8.4	33	5.7%	-0.50 [-4.25, 3.25]
Barakat 2012	69.6	9.6	138	70.8	8.5	152	10.1%	-1.20 [-3.30, 0.90]
Barakat 2014	67.99	9	107	67.6	6.89	93	9.7%	0.39 [-1.82, 2.60]
Erkkola 1976	73.1	10.3	30	77.3	8.4	32	4.2%	-4.20 [-8.90, 0.50]
Garnæs 2016	76.6	8.6	38	80.2	8.7	36	5.4%	-3.60 [-7.54, 0.34]
Guelfi 2016	60	8	84	61	8	85	9.1%	-1.00 [-3.41, 1.41]
Haakstad 2016	71	9	35	76	8	26	4.8%	-5.00 [-9.28, -0.72]
Halse 2015	69	9	20	69	9	20	3.2%	0.00 [-5.58, 5.58]
Perales 2012	62.9	5.8	35	65	8.6	9	2.9%	-2.10 [-8.04, 3.84]
Ramírez-Vélez 2011	66.68	6.98	24	66.46	10.12	26	4.1%	0.22 [-4.57, 5.01]
Rodríguez 2012	69	10	25	66	8	30	4.0%	3.00 [-1.85, 7.85]
Seneviratne 2016	67.8	8.3	37	70	8.7	37	5.5%	-2.20 [-6.07, 1.67]
Vinter 2011	82	3	150	83	3	154	14.7%	-1.00 [-1.67, -0.33]
Vladimirov 2015	77	2.71	50	81.18	3.1	38	13.1%	-4.18 [-5.42, -2.94]
Vázquez-Lara 2018	66.1	8.8	18	72.2	9.7	28	3.4%	-6.10 [-11.53, -0.67]
Total (95% CI)			**825**			**799**	**100.0%**	**-1.77 [-2.90, -0.64]**

Heterogeneity: Tau² = 2.15; Chi² = 35.19, df = 14 (P = 0.001); I² = 60%

Test for overall effect: Z = 3.06 (P = 0.002)

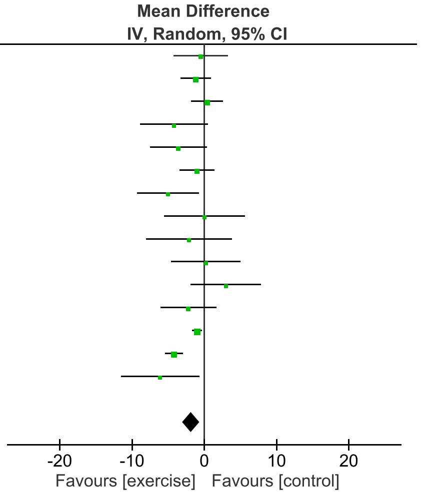

FIGURE 6—Effects of prenatal exercise intervention compared with control on resting diastolic blood pressure. Analyses conducted with a random-effects model. MD values are in millimeters of mercury. *df*, degrees of freedom; IV, inverse variance.

blood pressure. Although the mechanisms underlying the association between prenatal exercise and blood pressure control are unclear, the potential reasons could be the beneficial effects on exercise in the prevention of excessive gestational weight gain (19), the reduction in oxidative stress and inflammation, and associated improvement in endothelial function (81). Engaging in exercise may be particularly important in pregnancy health, as elevated blood pressure remains the leading cause of maternal, fetal, and neonatal morbidity and mortality (82).

Interventions prescribing physical activity in nonpregnant populations are consistently associated with increases in CRF. Recent meta-analyses in adults demonstrate an increase in relative $\dot{V}O_{2max}$ (weighted MD, 3.90 mL·kg^{-1}·min^{-1}; 95% CI, 3.45 to 4.35 mL·kg^{-1}·min^{-1}) (9) and a reduction in resting blood pressure (systolic blood pressure: MD, −3.84 mm Hg; 95% CI, −4.97 to −2.72 mm Hg; diastolic blood pressure: MD, −2.58 mm Hg; 95% CI, −3.35 to −1.81 mm Hg) (83) and heart rate (standardized MD, −0.29 bpm; 95% CI, −0.3 to −0.24 bpm) (84) after aerobic exercise interventions. Our results are in accordance with these estimates but with a smaller magnitude. The reasons for the discrepancies in magnitude between pregnant and nonpregnant population are not immediately clear. However, we suggest four possibilities. First, studies in nonpregnant populations have shown that vigorous-intensity exercise is more effective than low or moderate intensities in improving $\dot{V}O_{2max}$ and in reducing resting heart rate and blood pressure (85–87). Twenty-four of the 26 exercise interventions included in the current review prescribed low- to moderate-intensity exercise, which may limit improvements in CRF. Historical attitudes toward activity in pregnancy have led to a lack of information on the potential benefits or harms of vigorous-intensity exercise in pregnancy, which is an important field of inquiry demanding high-quality investigation. The dose–response analyses conducted in the systematic reviews associated with the 2019 Canadian Guidelines for Physical Activity throughout Pregnancy indicated that increasing frequency, intensity, and volume of exercise were associated with a greater reduction in the risk of developing pregnancy complications; no upper limit was identified (14). Furthermore, the systematic review examining the effect of prenatal exercise on fetal heart rate responses did not identify an adverse effect of exercise in the vigorous range (20). As such, the 2019 Canadian Guideline did not provide an upper limit for intensity. Previous global guidelines share mixed views on whether vigorous-intensity exercise is appropriate during pregnancy for all women without contraindication (4,88–90). Second, all postintervention $\dot{V}O_{2max}$ tests were measured in the third trimester of pregnancy. Previous work has demonstrated that physical activity is perceived to be more difficult during late pregnancy for a given $\dot{V}O_2$ compared with earlier in pregnancy (91). As such, tests that measure peak $\dot{V}O_2$ or submaximal tests that predict $\dot{V}O_{2max}$ may underestimate the effect of exercise interventions. Some, but not all, current guidelines recommend the use of $\dot{V}O_{2max}$ testing during pregnancy in a research setting (3–5,26,28) and are required to clarify this relationship. Third, adherence to exercise is critical to induce improvements in cardiorespiratory health and fitness. The studies included in the current review generally had low adherence, with only 27% of studies reporting adherence of >80% to the prescribed exercise program. Finally, although speculative, the normal physiological adaptations to pregnancy may mask some of the cardiovascular adaptations typically observed in nonpregnant populations. During pregnancy, the heart is physiologically dilated and myocardial contractility is increased. Heart rate rises with gestation to support blood volume expansion. The increase in cardiac output early in gestation is primarily mediated by increases in stroke volume, whereas the increase in later gestation is due to increased heart rate. Although stroke volume remains constant in late gestation, increases in maternal heart rate allow for a further increase in cardiac output (92). It is possible that regular physical activity cannot decrease resting heart rate to a great extent, which might be protective biologically/physiologically. Methodologically rigorous interventions including high adherence and retention are required.

The present meta-analysis is the first to examine the relationship between prenatal exercise interventions and metrics of maternal cardiorespiratory health and fitness. Rigorous methodological standards (following GRADE guidelines; [35]) were used to assess the certainty of the evidence; we also examined gray literature and did not limit our search to a single language. A series of meta-regression analyses were performed to examine the dose–response relationship between cardiovascular health and fitness and intensity, frequency, duration, and volume of exercise. However, several considerations should be noted. First, the measurement methods for $\dot{V}O_{2max}$ were different among the five included studies. All studies but one used prediction protocols, which do not have a high accuracy in predicting $\dot{V}O_{2max}$ in pregnant women (93). Another potential limitation is that most of the prenatal interventions included in the meta-analyses were based on low- to moderate-intensity exercise, which restricted our exploration of the effects of vigorous-intensity exercise on our outcomes of interest. There is a clear need for studies examining the effects of prenatal exercise at higher exercise intensities to determine if similar magnitude improvement in CRF or cardiorespiratory health can be reached in pregnant women when compared with nonpregnant populations. Furthermore, some studies were limited to a certain population (e.g., women with GDM, women with overweight, or women who were previously inactive), limiting the generalizability of the findings. However, we attempted to minimize this variation by conducting various subgroup analyses. In the examined studies, adherence was reported in only 14 of 26 studies. Furthermore, adherence was less than 80% in half of the studies that reported adherence. Low adherence in these studies is likely to attenuate the health effects of prenatal exercise interventions. This study calls attention to the significant lack of information on exercise (especially vigorous-intensity exercise) and important measures of CRF ($\dot{V}O_{2max}$ and $\dot{V}O_2$ peak) in pregnancy due to persistent historical opinions regarding exercise and exercise testing in pregnancy. To move forward, we must pursue research in these areas to champion the health of all women and their children.

CONCLUSIONS

Overall, our meta-analysis provides evidence that prenatal exercise interventions have a significant and clinically meaningful effect in improving $\dot{V}O_{2max,}$ and reducing heart rate, systolic blood pressure, and diastolic blood pressure. Physical activity should be considered a critical component of lifestyle modification to improve the cardiovascular health of pregnant women.

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