Physiological and metabolic responses of late pregnant women to 40 min of steady-state exercise followed by an oral glucose tolerance perturbation

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Mottola MF, Inglis S, Brun CR, Hammond J. Physiological and metabolic responses of late pregnant women to 40 min of steady-state exercise followed by an oral glucose tolerance perturbation. J Appl Physiol 115: 597–604, 2013. First published June 27, 2013; doi:10.1152/japplphysiol.00487.2013.—We examined the physiological and metabolic responses of 24 active late pregnant women to 40 min of vigorous (95% ventilatory threshold) steady-state treadmill exercise followed by a metabolic perturbation [oral glucose tolerance test (OGTT), 75 g] after exercise. Heart rate and respiratory measures were taken throughout exercise, and blood samples were collected during exercise and every 30 min during the 2-h OGTT. Values were compared with those for a group of physically active nonpregnant women (n = 16) in the luteal phase of the menstrual cycle. Although late pregnant women were heavier, they performed the same work rate and relative oxygen consumption, with less carbon dioxide expired, possibly due to pregnancy-related adaptations in heart efficiency. Resting glucose concentrations were the same between groups, but by 40 min of exercise glucose concentrations were diminished in late pregnant women (P ≤ 0.05, respectively). The pregnancy-induced delay of glucose uptake was seen in response to the postexercise OGTT compared with the nonpregnant women, but insulin sensitivity (ISI) remained (7.4 ± 0.9 vs. 9.7 ± 1.4 ISI, P > 0.05, respectively), with the preservation of the sensitivity of lipolysis inhibition of nonesterified free fatty acids to insulin. These adaptations may be fetoprotective, because our research suggests that 40 min of continuous treadmill exercise is well tolerated by physically active pregnant women. No adverse effects on birth outcome (3.53 ± 0.08 kg birth weight; 39.6 ± 0.33 wk gestational age) were observed.

MANY PHYSIOLOGICAL ALTERATIONS occur with endurance training that serve to offset the stress associated with a single bout of exercise. These adaptations are thought to act in a fetoprotective manner (7), which would allow physically conditioned pregnant women to maintain their normal exercise routines without compromising fetal growth and development. Currently, however, it is not clear how training adaptations and adaptations associated with a normal pregnancy may influence one another. A low obstetric risk gestation is known to result in decreased insulin sensitivity in maternal peripheral tissues in response to a glucose load (25), whereas exercise may attenuate the pregnancy-induced development of insulin resistance (11). Gestation is also associated with increases in plasma triglyceride (TG) and total cholesterol (TC) concentrations (16), whereas exercise training has the opposite effect on these factors (3).

Physiological responses to high or vigorous [6.0 metabolic equivalents (METs) or 60–84% of aerobic capacity reserve/heart rate reserve; Ref. 38] levels of exercise during pregnancy have not been assessed adequately (36). Many women of childbearing age are employed in nontraditional lines of work that demand physical strength and stamina (26), and athletic women may continue training in recreational or competitive athletics during gestation to maintain their fitness level (36). In the postpartum period it may be increasingly difficult to return to a pre-pregnancy routine and occupation if women are unable to maintain pre-pregnancy fitness levels during gestation. Although the American College (Congress) of Obstetricians and Gynecologists (ACOG) guidelines (1) suggest that low-risk pregnant women can participate in 30 min or more of moderate exercise a day on most, if not all, days of the week, it is important to gain a better understanding of how women respond to increasing levels of physical activity during pregnancy. This will ultimately lead to refined exercise guidelines that maximize training benefits during pregnancy without placing undue stress on the developing fetus. In Canada, the guidelines endorsed jointly by the Society of Obstetricians and Gynecologists of Canada (SOGC) and the Canadian Society for Exercise Physiology (CSEP) recommend aerobic exercise within specified target heart rate zones (representing 60–80% of maximum aerobic capacity; Ref. 39) for 30 min, 3–4 times per week (9).

Recent research has been limited to mild- and moderate-intensity maternal exercise of various frequencies and durations, initiating sedentary pregnant women to exercise (10, 17, 31). Historically, compared with sedentary pregnant women, women who continue to exercise throughout pregnancy have higher absolute blood volume measures (30), are able to perform more work with a greater stroke volume response to exercise clamped at 140 beats/min (29), and have minimal thermal (5) and endocrine stress (6) associated with endurance exercise of ~60–64% of maximum aerobic capacity. There are very few current studies examining higher intensities of exercise with fit active pregnant women (35, 36), and one small (n = 6) recent study of elite athletes suggested that maternal exercise above 90% of maximum heart rate (duration not specified) may decrease uterine blood flow and compromise fetal wellbeing (35). Others have reported no adverse fetal
effects after strenuous exercise of short duration in both active and inactive pregnant women (36). There are currently no studies that evaluate maternal responses to vigorous steady-state (a constant work rate; Ref. 32) exercise of longer duration in active women.

The purpose of the present study was to examine the physiological responses to 40 min of vigorous steady-state exercise followed by an oral glucose load (to provide a metabolic perturbation after exercise) in physically active late pregnant women compared with nonpregnant women with similar aerobic capacity who were in the luteal phase of the menstrual cycle. We chose not to include sedentary pregnant women because they would not be able to endure the high-intensity 40-min exercise in late pregnancy. Our study was designed to compare cardiorespiratory responses at rest, during 20, 30, and 40 min of steady-state exercise at 95% ventilatory threshold (VT) between the late pregnant and nonpregnant women, and to examine the interaction of blood glucose, insulin, TG nonesterified free fatty acids (NEFA), and cholesterol during exercise and after ingestion of a 75-g glucose load 15 min postexercise. We hypothesized that physically active late pregnant women would respond differently to 40 min of steady-state exercise followed by the oral glucose load, compared with the nonpregnant women. In addition, to assess birth outcome, we recorded birth weight, length, gestational age, Apgar scores at 1 and 5 min, and any complications associated with birth.

MATERIALS AND METHODS

Participants

Women were recruited from the university and London community fitness facilities. Participants were physically active women between 16 and 20 wk of an uncomplicated pregnancy and nonpregnant women with comparable physical activity backgrounds (based on activity history). All women were required to undergo medical pre-screening and fill out a physical activity readiness questionnaire [PAR-Q (28) for nonpregnant women or the PARmed-X for Pregnancy (40)]. Women were excluded if any contraindications to exercise were identified or if medical approval was not attained for the pregnant women. All nonpregnant women were tested during the luteal phase of the menstrual cycle, as determined by blood progesterone concentrations, and those unable to accurately predict the onset of menses or taking oral contraceptives were excluded. All research procedures were approved by the Human Ethics Research Board at the University of Western Ontario and all women gave written informed consent.

Steady-State Exercise Protocol and Oral Glucose Tolerance Test

Steady-state work rate was determined using data obtained from a peak treadmill exercise test to volitional fatigue (24), conducted between 16 and 20 wk of gestation. Methodology for the peak exercise treadmill test has been reported previously (24) and was performed 2 days before the steady-state exercise test for nonpregnant women.

Respiratory gases were continuously collected using the SensorMedics Vmax 29c breath-by-breath gas analysis unit during the exercise tests (Yorba Linda, CA). Before each exercise test, the gas analyzer was calibrated and the flow sensor meter was calibrated to a 3-liter syringe (SensorMedics), with the acceptable range within ±2% variability (24).

VT was calculated from the peak exercise data using the Y-slope method (2). Work rate at VT was calculated from vertical power using the equation

\[ P = ma[v_1(\sin \theta)] \]

where \( m \) is the mass (kg) of the woman, \( v_1 \) is the treadmill speed (m/s) at VT, \( a \) (9.81) is the gravitational constant, and \( \sin \theta \) represents the treadmill grade at VT (34).

The steady-state treadmill exercise test was performed between 34 and 38 wk of gestation for pregnant women and within the luteal phase of the menstrual cycle for the nonpregnant women (as confirmed by progesterone concentrations from the resting blood sample). One hour before the steady-state exercise test, women were given a standardized meal [1 pouch (38-g serving) of Nestle Carnation Instant Breakfast mixed with 250 ml of reduced fat (2%) milk; 248 kcal, 14.2 g protein, 3.8 g fat, 39.3 g carbohydrate] to ensure that results were not altered by variation in dietary intake (32). In addition, all women avoided caffeine and exercise 12 h before the treadmill test (32). Room temperature was maintained at 20 ± 2°C with 55% humidity.

The 40 min of steady-state exercise were preceded by a 5-min warm-up and followed by a 5-min cool down at 3 mph (4.8 km/h), 0% grade. During the 5-min warm-up, the speed and incline were slowly ramped up to each individual’s prescribed work rate corresponding to 95% of VT based on the specific target heart rate (HR) from their peak test. Oxygen consumption (\( \dot{V}O_2 \)) and carbon dioxide production (\( \dot{V}CO_2 \)) were computed in real time on a breath-by-breath basis, and HR was recorded throughout the test using a four-lead ECG. Respiratory gases and HR were calculated as a 1-min average immediately before the 5-min warm-up (baseline) and just before the 5-min recovery from exercise, while subjects were standing on the treadmill (32). Steady-state physiological cardiorespiratory parameters were defined from the Vmax 29c gas analysis unit (SensorMedics) as less than 10% variation in \( \dot{V}O_2 \), minute ventilation (Vt), and HR during the last 30 s average before each time point (20, 30, and 40 min of exercise).

Vertical power was again calculated for the steady-state exercise test.

An indwelling catheter was placed in an antecubital vein. Blood samples were drawn (15 ml each time) just before subjects stood on the treadmill after sitting for 20 min (rest), at 20 and 40 min of steady-state exercise, and finally at 15 min after exercise (ExRec).

After the 15-min postexercise blood draw (ExRec), which represented the sample before the glucose load, each woman was given 10 min to consume an orange-flavored drink containing 75 g of glucose (296 ml; WVR Canlab, Mississauga, Canada). Blood was then drawn at 30, 60, 90, and 120 min post-glucose ingestion while the woman rested in a recliner.

Blood Sample Analyses

Blood samples were centrifuged for 10 min at 3,000 rpm and 4°C. Plasma was stored at −80°C until analysis. Variability was calculated as <5% for each of the following assay analyses.

Glucose. Plasma glucose concentrations were determined using the YSI 2300Stat plus dual analyzer (Interscience), and each sample was run in duplicate. When variability was >0.1 mmol/l, samples were run in triplicate and the average of the two closest means was taken.

Insulin. A standard insulin radioimmunoassay kit (Coat-A-Count; Intermec) was used; 200 μl of plasma were pipetted into a polypropylene tube coated with anti-insulin antibody, and 1 ml of 125I-labeled insulin solution was added. Gamma radiation was measured in counts per minute (Gamma 5500 counting system; Beckman Instruments, Fullerton, CA) as a measure of the radioactive decay of bound 125I-insulin and inversely proportional to plasma insulin concentrations and compared with a standard curve generated from control samples of known concentrations. Values are reported in microinternational units per milliliter (μIU/ml).

Area under the curve. Area under the curve (AUC) was calculated for both glucose and insulin concentrations for the oral glucose tolerance test (OGTT) after exercise using the following formula:
AUC = \left[ 0.5(x_0 + x_1) \right] / 2 + \left[ 0.5(x_2) + 0.5(x_3) + 0.5(x_4) \right],
\]
where \( x_0 \) represents plasma concentrations at ExRec, and \( x_2, x_3, x_4, \) and \( x_5 \) represent plasma concentrations at 30, 60, 90, and 120 min post-glucose ingestion, respectively (4). Values are expressed as millimoles per liter per 2 h for glucose and microinternational units per milliter per 2 h for insulin.

**Insulin sensitivity index.** Insulin sensitivity during the OGTT performed immediately after exercise was calculated using the whole body insulin sensitivity index [ISI (21)]. ISI was calculated as follows:

\[
\text{ISI} = \frac{10,000}{(\text{FGP} \cdot \text{FPI} \cdot \text{OGTT}_{\text{gluc}} \cdot \text{OGTT}_{\text{ins}})^{0.5}},
\]
where FGP represents fasted plasma glucose, FPI represents fasted plasma insulin, OGTT\text{gluc} represents the mean plasma glucose values from the OGTT, and OGTT\text{ins} represents the mean plasma insulin values from the OGTT. We substituted recovery values from steady state exercise for the fasted values to evaluate the effects of the steady-state exercise.

**NEFA.** NEFA was analyzed spectrophotometrically (Turner model 340; Barnstead/Thermolyne, Dubuque, IA) using a standardized enzymatic assay kit (NEFA C; Wako Chemicals, Richmond, CA) with a wavelength of 550 nm. Values are expressed as milliequivalents per liter.

**Triglyceride and cholesterol.** Plasma TG and TC concentrations were determined using automated spectrophotometric analysis (Johnson & Johnson Vitros DT60 II chemistry system; Clinical Diagnostics, Mississauga, Canada). High-density lipoprotein cholesterol (HDL-C) analysis used 50 \( \mu \)l of each sample treated with dextran sulfate (0.9 g/l) and magnesium chloride (45 mmol/l). After centrifugation, 10 \( \mu \)l of each aliquot was used for HDL-C analyses.

Low-density lipoprotein (LDL) was calculated using the following formula (13):

\[
\text{LDL} = \text{TC} - \text{HDL-C} - \text{VLDL} \quad \text{(VLDL} = \text{TG} / 2.2),
\]
where VLDL is very-low-density lipoprotein. All lipid values are expressed in millimoles per liter.

**Progesterone.** Preexercise blood samples were analyzed at a commercial laboratory (Gamma Dynacare Medical Laboratory, London, ON, Canada). Values are expressed in millimoles per liter. Progesterone concentrations >3 mmol/l confirmed the luteal phase of the menstrual cycle in nonpregnant women (20).

**Newborn Assessment**

As a means of assessing gestational outcome, newborn measurements were recorded within 6–18 h following delivery. Gestational age, birth weight, length, Apgar scores at 1 and 5 min, and any complications due to delivery were recorded. These recorded values were compared with normal expected ranges for a Canadian population (8).

**Statistical Analysis**

All statistical analyses were performed with the SPSS 19.0 statistical software package. Independent samples \( t \) tests were performed to compare average values between pregnant and nonpregnant women.

<table>
<thead>
<tr>
<th>Group</th>
<th>Body mass, kg</th>
<th>Time to reach VT, min</th>
<th>Work Rate, W</th>
<th>HR, beats/min</th>
<th>( V_i ), l/min</th>
<th>( V_{O_2} ), ml·kg(^{-1} )·min(^{-1} )</th>
<th>( V_{CO_2} ), ml·kg(^{-1} )·min(^{-1} )</th>
<th>Oxygen Pulse, ml·beat ( ^{-1} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnant</td>
<td>67.7 ± 2.1</td>
<td>14.8 ± 1.4</td>
<td>192.0 ± 4.4</td>
<td>146.0 ± 3.1</td>
<td>43.5 ± 2.1</td>
<td>21.6* ± 0.8</td>
<td>22.5* ± 0.8</td>
<td>9.9 ± 0.3</td>
</tr>
<tr>
<td>Nonpregnant</td>
<td>66.8 ± 2.5</td>
<td>17.9 ± 0.9</td>
<td>205.4 ± 5.9</td>
<td>149.4 ± 2.5</td>
<td>43.5 ± 3.6</td>
<td>24.3 ± 0.9</td>
<td>25.1 ± 0.8</td>
<td>10.9 ± 0.7</td>
</tr>
</tbody>
</table>

Values are means ± SE of measures at 16–20 wk of gestation for the pregnant group and during the luteal phase for the nonpregnant group at ventilatory threshold (VT) during the peak exercise test. Work rate at VT was calculated from vertical power using the equation \( P = mL(v_i \sin \theta) \), where \( m \) is the mass of the woman, \( v_i \) is the treadmill speed at VT, \( \alpha \) is the gravitational constant, and \( \sin \theta \) represents the VT treadmill grade (34). HR, heart rate; \( V_i \), minute ventilation; \( V_{O_2} \), relative oxygen consumption; \( V_{CO_2} \), carbon dioxide output. *\( P \) = 0.05, difference between groups.

**RESULTS**

**Participant Characteristics**

A total of 39 pregnant and 29 nonpregnant women were recruited. Fifteen pregnant and 13 nonpregnant women were excluded from the final analysis due to noncompliance (too busy to commit) or incomplete data sets (unable to obtain blood samples). Thus, for the pregnant group, 24 physically active women between 16 and 20 wk of an uncomplicated pregnancy continued to exercise 3–4 times per week (confirmed by exercise logs and heart rate monitor) at the appropriate target HR intensity (95% of VT from their peak test). The pregnant women completed the steady-state exercise test followed by the glucose load at 34–38 wk of gestation. The nonpregnant group was composed of active women with comparable aerobic capacity (\( n = 16 \)) as determined by the peak assessment. Table 1 presents the measurements taken at VT during the peak exercise test. Although time to reach VT, work rate, HR, \( V_i \), and oxygen pulse at the time of VT was not different between the groups, relative \( V_{O_2} \) (21.6 ± 0.8 vs. 24.3 ± 0.9 ml·kg\(^{-1} \)·min\(^{-1} \) – 1) and \( V_{CO_2} \) (22.5 ± 0.8 vs. 25.1 ± 0.8 ml·kg\(^{-1} \)·min\(^{-1} \) – 1) were significantly less in the pregnant women at 16–20 wk gestation than in the nonpregnant women in the luteal phase of the menstrual cycle (\( P \leq 0.05 \)). At the time of the peak test, body mass index was not different between the groups (pregnant, 23.4 ± 0.6 kg/m\(^2\); nonpregnant, 24.0 ± 0.6 kg/m\(^2\)).

**Steady-State Exercise**

Participant characteristics at the time of the steady-state exercise test are presented in Table 2. Body mass was significantly higher in the late pregnant women. HR and respiratory measures at baseline (standing on the treadmill), 20, 30, and 40 min of steady-state treadmill exercise, followed by recovery (5 min after exercise), are presented in Table 3. Late pregnant women had higher resting HR (88.1 ± 2.6 vs. 73.2 ± 2.6 beats/min; \( P \leq 0.05 \)) compared with the nonpregnant women. This was also seen during recovery, where HR values in late pregnant women were again significantly higher than those seen in nonpregnant women (118.2 ± 2.6 vs. 102.7 ± 3.6 beats/min, respectively). Although the calculated work rate at 40 min was the same in both groups (182 vs. 208 W; Table 3), HR, relative \( V_{O_2} \), and \( V_{CO_2} \) were consistently lower in the late pregnant women compared with the nonpregnant women (\( P \leq 0.05 \)), although oxygen pulse (reflecting the amount of oxygen...
taken up by the pulmonary circulation in a heartbeat as a measure of aerobic capacity; Ref. 32) was not different between groups throughout the test.

### Glucose

Plasma glucose concentrations in late pregnant and nonpregnant women were similar at rest (Fig. 1A) and decreased at similar rates in both groups, showing no difference at 20 min of exercise (3.7 ± 0.1 vs. 4.1 ± 0.2 mmol/l, respectively). At 40 min of exercise, however, glucose concentrations increased in nonpregnant women (4.6 ± 0.1 mmol/l) but not in pregnant women (3.8 ± 0.1 mmol/l; P ≤ 0.05). This difference remained at 15 min of exercise recovery (pregnant, 4.3 ± 0.2 vs. nonpregnant, 5.0 ± 0.1 mmol/l; P ≤ 0.05). After exercise, both groups demonstrated a comparable increase in plasma glucose concentrations following oral glucose intake. At 60 min after glucose ingestion, however, plasma glucose concentrations continued to rise in pregnant women (8.0 ± 0.4 mmol/l) but started to decline in nonpregnant women (6.8 ± 0.4 mmol/l), resulting in a significant difference between the two groups (P ≤ 0.05). This difference remained at both 90 (pregnant, 7.4 ± 0.3 vs. nonpregnant, 6.1 ± 0.3 mmol/l; P ≤ 0.05) and 120 min following the glucose load (pregnant, 6.3 ± 0.3 vs. nonpregnant, 4.7 ± 0.4 mmol/l; P ≤ 0.05). As a result, glucose concentrations for the duration of the OGTT, as measured by calculating AUC (Fig. 1B), were significantly different between the two groups (pregnant, 14.1 ± 0.5 vs. nonpregnant, 12.2 ± 0.5 mmol·l⁻¹·h⁻¹; P ≤ 0.05).

### Insulin

Plasma insulin concentrations are depicted in Fig. 2A. Insulin values were on average almost twice as high in pregnant women at rest (37.8 ± 6.5 vs. 21.7 ± 6.3 μIU/ml), but because of the high variability between participants, this value did not reach significance. In both groups, plasma insulin decreased at the 20-min mark of steady-state exercise, with values remaining twice as high in pregnant compared with nonpregnant women (10.2 ± 1.4 vs. 4.8 ± 0.8 μIU/ml; P ≤ 0.05). Insulin concentrations continued to decrease in pregnant women, approaching the values seen in nonpregnant women at 40 min of exercise. Values remained similarly depressed in both groups at exercise recovery. At 30 min following the oral glucose load, plasma insulin concentrations had risen to similar values in both pregnant (63.6 ± 8.3 μIU/ml) and nonpregnant women (42.1 ± 10.3 μIU/ml) and remained elevated in both groups at the 60-min time point. At 90 min, insulin concentrations began to decline in nonpregnant women (35.6 ± 7.1 μIU/ml) but remained elevated in the late pregnant women (55.6 ± 5.6 μIU/ml; P ≤ 0.05). Values declined for both groups between 90 and 120 min but remained significantly higher in the late pregnant women (37.4 ± 8.0 vs. 18.2 ± 3.7 μIU/ml; P ≤ 0.05). This delay in insulin decline in pregnant women during the later time points resulted in a significantly higher AUC for insulin concentrations during the 2-h OGTT (Fig. 2B).

Conversely, the calculated ISI for the OGTT after exercise was not different between the late pregnant women (7.4 ± 0.9 ISI) and the nonpregnant women (9.7 ± 1.4 ISI; P > 0.05).

### Lipid and Cholesterol Profiles

At rest, plasma NEFA concentrations were significantly elevated in late pregnant women compared with nonpregnant women (0.312 ± 0.035 vs. 0.195 ± 0.038 meq/l; P ≤ 0.05; Fig. 3). This difference disappeared with exercise, since concentrations rose to comparable levels in both late pregnant (0.331 ± 0.031 meq/l) and nonpregnant women (0.286 ± 0.061 meq/l) at 20 min of exercise and were elevated further at the 40-min mark of exercise (pregnant, 0.433 ± 0.047 vs. nonpregnant, 0.429 ± 0.066 meq/l). Plasma NEFA concentrations peaked in both groups at recovery (pregnant, 0.719 ± 0.052 vs. nonpregnant, 0.672 ± 0.124 meq/l) and decreased

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**Table 2. Participant characteristics at time of steady-state exercise test**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Women</th>
<th>Age, yr</th>
<th>Gestational Age, wk</th>
<th>Body Mass, kg</th>
<th>Height, cm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnant</td>
<td>24</td>
<td>33.5 ± 0.7</td>
<td>35.7 ± 0.4</td>
<td>75.0 ± 1.8*</td>
<td>167.8 ± 1.2</td>
</tr>
<tr>
<td>Nonpregnant</td>
<td>16</td>
<td>33.0 ± 2.1</td>
<td></td>
<td>68.6 ± 2.4</td>
<td>169.0 ± 5.5</td>
</tr>
</tbody>
</table>

Values are means ± SE of characteristics of late pregnant and nonpregnant women at the time of the steady-state exercise test followed by the oral glucose tolerance test. *P ≤ 0.05, difference between groups.

**Table 3. HR, \( \dot{V}O_2 \), \( \dot{V}CO_2 \), and oxygen pulse for late pregnant and nonpregnant women (luteal phase) during steady-state exercise protocol**

<table>
<thead>
<tr>
<th>Time</th>
<th>Group</th>
<th>HR, beats/min</th>
<th>( \dot{V}O_2 ), ml·kg⁻¹·min⁻¹</th>
<th>( \dot{V}CO_2 ), ml·kg⁻¹·min⁻¹</th>
<th>Oxygen Pulse, ml·beat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>P</td>
<td>88.1 ± 2.5*</td>
<td>4.3 ± 0.1</td>
<td>4.1 ± 0.1</td>
<td>3.6 ± 0.1</td>
</tr>
<tr>
<td></td>
<td>NP</td>
<td>73.2 ± 2.5</td>
<td>4.4 ± 0.2</td>
<td>4.1 ± 0.2</td>
<td>4.1 ± 0.2</td>
</tr>
<tr>
<td>20 min</td>
<td>P</td>
<td>140.9 ± 2.2*</td>
<td>18.5 ± 0.6*</td>
<td>18.8 ± 0.6*</td>
<td>9.7 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>NP</td>
<td>149.3 ± 2.3</td>
<td>23.0 ± 1.0</td>
<td>23.8 ± 1.1</td>
<td>10.7 ± 0.7</td>
</tr>
<tr>
<td>30 min</td>
<td>P</td>
<td>142.8 ± 2.2</td>
<td>18.7 ± 0.6*</td>
<td>19.2 ± 0.7*</td>
<td>9.7 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>NP</td>
<td>149.5 ± 2.8</td>
<td>23.0 ± 0.9</td>
<td>23.8 ± 1.0</td>
<td>10.7 ± 0.7</td>
</tr>
<tr>
<td>40 min</td>
<td>P</td>
<td>147.7 ± 2.8*</td>
<td>18.4 ± 0.6*</td>
<td>18.8 ± 0.7*</td>
<td>9.2 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>NP</td>
<td>155.5 ± 2.4</td>
<td>23.4 ± 0.9</td>
<td>23.3 ± 1.2</td>
<td>10.5 ± 0.7</td>
</tr>
<tr>
<td>Recovery</td>
<td>P</td>
<td>118.2 ± 2.6*</td>
<td>4.7 ± 0.2</td>
<td>4.8 ± 0.2</td>
<td>3.0 ± 0.1</td>
</tr>
<tr>
<td></td>
<td>NP</td>
<td>102.7 ± 3.6</td>
<td>5.6 ± 0.6</td>
<td>5.4 ± 0.6</td>
<td>3.9 ± 0.5</td>
</tr>
</tbody>
</table>

Values are means ± SE. Work rate at 40 min of steady-state exercise was calculated as 182.0 ± 14 W for the late pregnant women and 208 ± 14 W for the nonpregnant women (P > 0.05). Work rate was calculated from vertical power as described in Table 1. There was no difference within each group for each variable over time (20 vs. 30 vs. 40 min) during the steady-state exercise test. *P ≤ 0.05, difference between groups.
through the OGTT after exercise, with no differences in NEFA concentrations between groups.

Plasma concentrations of TG and cholesterols were analyzed at rest, at 20 and 40 min of exercise, at recovery, and at the 4 time points of the OGTT. Since no change occurred in any of the values over the exercise time, during recovery, or in response to the OGTT (data not shown), Table 4 presents the lipid profiles for the late pregnant and nonpregnant women at rest only before the exercise test. Plasma TG, TC, LDL, and VLDL concentrations were consistently two to three times higher in late pregnant women compared with nonpregnant women (P < 0.05). Interestingly, no difference in HDL-C concentrations was observed between the two groups.

Plasma progesterone concentrations were more than 30 times greater in the late pregnant (394.2 ± 47.2 mmol/l) compared with the nonpregnant women (4.5 ± 0.5 mmol/l; P < 0.05). All nonpregnant women had concentrations representative of the luteal phase of the menstrual cycle.

Birth Measurements

Average newborn weight was 3.53 ± 0.08 kg and average gestational age was 39.6 ± 0.33 wk. Average Apgar scores were 8.2 ± 1.9 at 1 min, with a minimal value of 7, and 9.1 ± 0.1 at 5 min, with a minimum value of 8. No differences were found in our group compared with the normal expected ranges for a Canadian population (8). No complications were recorded for any of the births.

DISCUSSION

The present study examined the physiological responses to vigorous steady-state exercise in active medically prescreened late pregnant women. Although the current exercise guidelines endorsed by the ACOG (1) offer no limitations to frequency, intensity, or duration of exercise during pregnancy, few controlled studies have analyzed maternal responses to exercise bouts of greater than 30-min duration. For the current study, women remained active throughout pregnancy for 40 min per session, 3–4 times per week, at a vigorous intensity [95% VT ≤ 70% heart rate reserve (32), which is similar to the Canadian guidelines but 10 min longer in duration than currently recommended (9)]. Active low-risk late pregnant women showed no adverse responses to a 40-min bout of treadmill exercise at a vigorous intensity. After delivery, all newborn assessment values were within normal ranges. Thus we suggest that...
regular exercise training at 95% VT (at an average HR of 148 beats/min; range 131–165 beats/min) for 40-min duration, 3–4 times per week, in previously active low-risk women is not detrimental to maternal health or birth outcome. To examine the physiological responses to 40 min of vigorous steady-state exercise followed by an oral glucose load (to provide a metabolic perturbation after exercise) in physically active late pregnant women, we compared glucose tolerance responses of nonpregnant women of similar fitness levels in the luteal phase of the menstrual cycle. This phase corresponds to the highest concentrations of circulating progesterone, thus minimizing differences associated with hormonal changes during pregnancy, as well as diminishing menstrual cycle variation between women in the nonpregnant group. Although the late pregnant women weighed significantly more than the nonpregnant women, the work rate (as equated through vertical power) and the oxygen pulse for the 40-min exercise session was represented by 10.2 ± 0.3 vs. 6.2 ± 3.4, respectively; means ± SD) and our ISI values higher (7.4 ± 3.4 vs. 6.2 ± 3.4, respectively; means ± SD) than those women given an OGTT from the fasted state at 30 wk of gestation (no exercise information given; Ref. 14). Although not giving the OGTT in the fasted state is a limitation of our study, it would appear these results further support the importance of exercise training throughout pregnancy in preventing gestational diabetes. More research is necessary to examine the interplay between maternal exercise and glucose metabolism in late pregnancy.

Table 4. Lipid profiles for late pregnant and nonpregnant women (luteal phase) at rest before steady-state exercise test

<table>
<thead>
<tr>
<th>Lipids</th>
<th>Group</th>
<th>Plasma Concentration, mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglycerides</td>
<td>P</td>
<td>2.57 ± 0.8*</td>
</tr>
<tr>
<td></td>
<td>NP</td>
<td>1.06 ± 0.9</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>P</td>
<td>6.0 ± 0.4*</td>
</tr>
<tr>
<td></td>
<td>NP</td>
<td>3.8 ± 0.2</td>
</tr>
<tr>
<td>HDL-C</td>
<td>P</td>
<td>1.72 ± 0.13</td>
</tr>
<tr>
<td></td>
<td>NP</td>
<td>1.53 ± 0.06</td>
</tr>
<tr>
<td>LDL</td>
<td>P</td>
<td>3.07 ± 0.4*</td>
</tr>
<tr>
<td></td>
<td>NP</td>
<td>1.06 ± 0.06</td>
</tr>
<tr>
<td>VLDL</td>
<td>P</td>
<td>1.17 ± 0.10*</td>
</tr>
<tr>
<td></td>
<td>NP</td>
<td>0.48 ± 0.06</td>
</tr>
</tbody>
</table>

Values are means ± SE of plasma lipid concentrations in pregnant (P) and nonpregnant (NP) women. HDL-C, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; VLDL, very-low-density lipoprotein cholesterol. *P ≤ 0.05, difference between groups.
Late pregnancy is associated with elevations in TC as a result of increases in LDL and VLDL concentrations, which are important for fetal cell membrane development, steroid synthesis, and cell proliferation and differentiation (27). Increases in maternal TG provide NEFA for fetal growth (15), and the rise in maternal NEFA is consistent with the metabolic shift toward augmented mobilization of lipid stores in late gestation (17). In the present study, physically active late pregnant women had consistently higher concentrations of TG and TC in the postabsorptive state, compared with nonpregnant women. Plasma NEFA was elevated in our late pregnant women preexercise, but the difference between groups absorbed in response to the exercise with a similar response between groups to the glucose load after exercise. NEFA concentrations rose as insulin values decreased in response to the exercise test and were highest when insulin was lowest at recovery. As insulin values increased in response to the oral glucose load, NEFA values dropped to below resting values in both groups. Insulin is known to suppress lipolysis (18), and the similarity of the NEFA response to exercise and the glucose load after exercise in the present study between late pregnant and nonpregnant women is intriguing. NEFA values as a result of an OGTT have been found to be negatively correlated with insulin sensitivity, suggesting a direct connection between NEFA dynamics and insulin resistance (37). This was confirmed in women with former gestational diabetes, because despite hyperinsulinemia, higher NEFA concentrations occurred in response to an OGTT, suggesting a reduced sensitivity of lipolysis inhibition to insulin (37). Taken together, the results of the present study suggest that exercise training maintains insulin sensitivity and may have a protective effect as the sensitivity of lipolysis inhibition to insulin may be preserved in the late pregnant women despite the hyperinsulinemia of pregnancy. Further research is necessary to examine the mechanisms for these metabolic changes in fit late pregnant women.

Exercise training has been shown to elevate HDL-C concentrations (41). Because the HDL-C values in late pregnancy are similar to those of the nonpregnant women in the present study, being active throughout pregnancy may, again, offer protection to counter the elevations in VLDL, LDL cholesterol, and TG concentrations seen at rest in late pregnant women. This is relevant because dyslipidemia results in an increased risk for preeclampsia (12) and preterm delivery (27), which are associated with maternal and infant mortality and morbidity. Exercise may therefore be an effective intervention for pregnant women at risk of developing preeclampsia and other lipid-associated diseases.

Limitations of the present study included the timing of the peak exercise test, which occurred at 16–20 wk of gestation rather than late pregnancy, and the small sample size. After the peak test, the pregnant women continued an exercise regimen of 40 min, 3–4 times per week, at 95% VT throughout pregnancy with the steady-state exercise test scheduled for 34–38 wk of gestation. A sedentary late pregnant group was not included because of the intensity and duration of the steady-state exercise protocol. We were concerned that nonexercising late pregnant women would not be able to tolerate this vigorous intensity for 40 min. For birth outcomes we included comparisons with our previously published normative data for our area and found no difference. Although our lipid values were measured in the postabsorptive state, all women were given the same standardized meal 1 h before the exercise test, since we chose not to exercise the late pregnant women in the fasted state. Nutrition was not assessed, but all pregnant women were normal weight and gained within recommended guidelines (Ref. 19; data not shown).

In conclusion, active late pregnant women responded differently to 40 min of steady-state exercise followed by an oral glucose load, compared with nonpregnant women (in the luteal phase) with similar aerobic fitness. Although the late pregnant women were heavier, they performed the same work rate, with the same oxygen pulse, but responded to the exercise with a blunted heart rate and relative oxygen consumption, with less carbon dioxide expired, possibly due to pregnancy-related adaptations in heart efficiency. Metabolically, however, although the late pregnant women had the same resting glucose concentrations, by 40 min of vigorous exercise and into 15 min of recovery after exercise, glucose concentrations were diminished compared with the nonpregnant women. In response to the glucose load after exercise, compared with nonpregnant women, late pregnant women exhibited the pregnancy-induced delay of glucose uptake, but insulin sensitivity remained, with the preservation of the sensitivity of lipolysis inhibition to insulin. More research is needed to investigate the mechanisms responsible for the metabolic and cardiovascular responses to exercise in active low-risk late pregnant women, although the intensity and duration examined in the present study appears safe for both mother and baby.

REFERENCES


