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Determinants of resting lipid oxidation in response to a prior bout of endurance exercise

Gregory C. Henderson[1,2] and Brandon L. Alderman[1]

[1]*Department of Exercise Science and Sport Studies, Rutgers University, New Brunswick, New Jersey; and* [2]*Rutgers Center for Lipid Research, Rutgers University, New Brunswick, New Jersey*

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Henderson GC, Alderman BL. Determinants of resting lipid oxidation in response to a prior bout of endurance exercise. *J Appl Physiol* 116: 95–103, 2014. First published November 14, 2013; doi:10.1152/japplphysiol.00956.2013.—A single bout of exercise can alter subsequent resting metabolism for many hours and into the next day. However, differences between men and women, effects of nutritional state, and relative effects of resting metabolic rate (RMR) and respiratory exchange ratio (RER) in controlling the increase in lipid oxidation (Lox) after exercise are not yet clear. Effects of aerobic capacity ($\dot{V}O_{2\ peak}$) and exercise bout parameters (intensity and volume) also remain to be clearly elucidated as does the time course of changes after exercise. We performed a meta-analysis to assess these potential moderators of the impact of endurance exercise [effect sizes (ESs)] on subsequent Lox at rest (ES = 0.91; 95% CI: 0.69–1.12), on the day of exercise (ES = 1.22; 95% CI: 0.89–1.55), and on the following day (ES = 0.60; 95% CI: 0.35–0.85). ES for the exercise-related increase in resting Lox was significantly greater in men than women in the postabsorptive state but similar in the postprandial state. The ES for depression of RER after exercise was similar between men and women, while the ES for RMR in the postabsorptive state tended to be higher in men than women. Finally, $\dot{V}O_{2\ peak}$ and exercise energy expenditure (EEE), but not intensity, were predictive of postexercise Lox. The findings indicate importance of EEE and fitness for ability to achieve robust enhancement of Lox after exercise. The results additionally indicate a gender difference in postexercise Lox that is dependent on nutritional state, as the ES for Lox was lower in women only in the postabsorptive state.

meta-analysis; fuel metabolism; substrate oxidation; sex; sex-based differences

MAINTENANCE OF BODY COMPOSITION OVER TIME, or prevention of fat gain, requires that individuals oxidize at least as much fat as they consume. Thus, not surprisingly, a reduced relative contribution of lipid oxidation (Lox) to energy metabolism is associated with increased risk of gaining weight over time (59, 70). Along with the contribution of a positive energy balance, poor ability to oxidize lipids in certain individuals could contribute to their propensity toward gain and retention of body fat. A physically active lifestyle prevents age-related weight gain over time (33), and this effect could be related to energy expenditure and to energy substrate selection.

Even in individuals who successfully meet current physical activity guidelines for total exercise volume (33) and for whom exercise energy expenditure (EEE) thus significantly adds to total energy expenditure (TEE), weight management through exercise alone can still be difficult. The nonsubstantial impact of exercise on body weight may be because most of the TEE is still from the resting metabolic rate (RMR); in individuals characterized as active, likely RMR is in the vicinity of 75% of TEE (33). EEE remains a minor contributor to TEE, and so in attempts to monitor and manipulate energy substrate metabolism, resting metabolism is a logical and important focus. Although energy substrate metabolism is altered during exercise, the majority of the 24-h period in individuals in Western society is spent at rest. Exercise, however, can be a strategy to alter resting metabolism, as the effects of each individual exercise bout on energy substrate utilization continue many hours into subsequent rest, with Lox remaining elevated above that observed under sedentary conditions for many hours (25). That is to say, resting lipid metabolism is altered by a recent exercise bout.

Previous work has modeled the relationship between exercise intensity and fuel partitioning during exercise (7, 9, 10), but to our knowledge no model exists for the postexercise recovery period. Extensive work has led to the conclusion that women rely upon lipid as a fuel to a relatively greater extent than do men during an endurance exercise bout (13, 15, 16, 21, 22, 25, 26, 30, 56, 60, 62), and this conclusion was reinforced by a meta-analysis on the topic (61). Additionally, some initial observations indicate that there may be differences between men and women during the postexercise recovery period (25, 30). As resting metabolism makes up the vast majority of TEE in most people, even in those engaged in formal exercise training programs, we undertook an attempt to further explore the determinants of postexercise Lox, including the effects of gender, physical fitness, nutritional state, timing of assessment (immediately after, next day), exercise intensity, and EEE. Additionally, we explored the components of the enhancement of Lox after exercise which can include an increase in RMR and a relative shift away from carbohydrate and toward lipid for energy [i.e., reduced respiratory exchange ratio (RER)].

A variety of study designs have been used to investigate the effects of exercise on subsequent substrate oxidation at rest. Investigations have been performed on both men and women, employing a variety of exercise volumes and intensities, and subjects of a wide variety of fitness levels have been studied after exercise in the postabsorptive (fasted) and postprandial (fed) states. Each individual study may have some degree of statistical power limitation, and each pool of study participants represent only a subset of variations in phenotype within society. Based on the limited statistical power inherent to any one individual study, the ability to study important moderators of Lox or substrate utilization during the postexercise window is limited. Therefore, we set out to harness these variabilities in study designs and to provide a statistically powerful test beyond that of any individual study by employing meta-

Address for reprint requests and other correspondence: G. C. Henderson, Rutgers Univ., 70 Lipman Drive, Loree Bldg., New Brunswick, NJ 08901 (e-mail: ghender@rci.rutgers.edu).

analytic and meta-regression approaches to identify the determinants of postexercise Lox.

METHODS

Study aim. We performed a systematic quantitative analysis of published results on energy substrate utilization data derived from pulmonary gas exchange. Specifically, we explored the impact of each single endurance exercise bout in humans and the moderating influence of gender, physical fitness, nutritional state, timing of assessment (immediately after, next day), exercise intensity, and EEE.

Identification of data for the analysis. Relevant papers were identified by searching the PubMed database using various combinations of the following terms: prior exercise, postexercise, post-exercise, lipid oxidation, fat oxidation, substrate oxidation, substrate utilization, RER, and RQ. Additional papers that might have been relevant for inclusion were identified from reference lists from all articles identified from the PubMed search. Studies in which endurance exercise was performed at a steady intensity were included (i.e., not interval exercise or resistance exercise). Because energy substrate metabolism changes with the time of day and time elapsed since the prior meal (25, 30, 53), we only included studies that utilized a time-matched sedentary control condition performed on a separate occasion from that of exercise in the same subjects (i.e., cross-over design). In studies where postexercise nutrition was provided, only those that fed the same energy and macronutrient content under both exercise and sedentary conditions were included. There were not a sufficient number of studies to allow for a separate meta-analysis of those in which energy intake was different between exercise and sedentary conditions, but these individual studies are discussed separately from the main findings in this report. The minimum duration of postexercise recovery was chosen to be 2 h in order to observe persistent changes in resting metabolism. Based on knowledge that men and women can respond differently to exercise (25), and to address a primary aim of investigating gender differences in substrate oxidation, we excluded any papers that pooled men and women into one single group. In the one study in which women were studied in both the follicular and luteal phases of the menstrual cycle (20), data from the follicular phase were used in the analysis because this phase is often chosen when standardizing cycle phase in exercise and substrate utilization research. Mean and variance values for postexercise Lox and for Lox in sedentary control trials were obtained from published papers. When data were displayed graphically but not numerically, authors were contacted to request the mean and variance values, and these data were included in the analysis when authors agreed to provide the results. Mean and variance for RMR and RER, because they are the components in derivation of Lox values, were also obtained as available. Data were categorized as those collected on the day of exercise (on the same calendar day) or on the day after exercise (after an overnight period had passed after exercise). Data were also categorized into those collected in the postprandial (a meal taken immediately before indirect calorimetry assessments) or the postabsorptive state.

Calculations. Exercise duration and relative exercise intensity (% $\dot{V}O_{2\,peak}$) were recorded for each study, as well as energy expenditure of exercise (EEE). When EEE was not reported, it was estimated from published oxygen consumption ($\dot{V}O_2$) rate and exercise duration, estimating 5 kcal energy per liter of $\dot{V}O_2$ (8). If exercise $\dot{V}O_2$ was not reported, from the published mechanical power output (watts), the energy requirement of exercise was estimated from a published table to convert power output to metabolic equivalents (METS) (1). As needed, percentage of energy from fat and carbohydrate (assuming negligible protein oxidation) was calculated from published equations (43). When not provided, RMR was calculated using these percentages of fat and carbohydrate, published $\dot{V}O_2$, and the assumption of 5.05 kcal per liter of oxygen consumption for carbohydrate and 4.7 kcal per liter of oxygen consumption for fat (8). When standard error (SE) but not standard deviation (SD) were available, SD was calculated by multiplying SE by the square root of the reported sample size. Effect sizes (ESs) for postexercise Lox were calculated as followed: [(postexercise mean) − (sedentary mean)]/(sedentary SD).

Statistical analysis. Descriptive statistics were generated using SPSS v. 19 and are expressed as mean ± SE. ESs were calculated and meta-analyses were conducted using Comprehensive Meta-Analysis v. 2.0. Cohen's d was used as the measure of the overall effect, and evidence suggests that Type I error rates for tests of heterogeneity are well controlled using this metric (32). Given that sample size impacts the precision of the ES estimate, each ES was weighted by the inverse of its variance prior to conducting further analyses. A random effects model was used to pool effects and compare results across studies to address potential concerns regarding the lack of independence of data points when multiple effects are derived from a single study (41). Overall ESs for Lox, RER, and for RMR were conducted independently to examine subject and exercise characteristics on these outcomes.

Heterogeneity in meta-analysis refers to the variation in outcomes between studies included in the review. The extent of heterogeneity partly determines the difficulty in drawing overall conclusions from the literature. Understanding the cause of heterogeneity, in part through the use of moderating variable analyses, can increase both the scientific and clinical value of the meta-analysis (64). Detecting heterogeneity among ESs supports the necessity of follow-up moderating analyses. Heterogeneity was examined using Cochran's Q and I^2 statistics. The traditional approach for assessing heterogeneity in meta-analyses has been through the use of Cochran's Q statistic (32, 55). Cochran's Q is computed by summing the squared deviation of each study ES from the overall ES estimate and weighting the contribution of each ES by the inverse of its variance. The alpha value for statistical significance for Q was set at $P \leq 0.10$ because this statistic tends to suffer from low power (24, 34). More recently Cochran's Q has been shown to be heavily influenced by the number of studies in a review such that it has low power with a small number of studies and excessive power with a large number of studies (28). Thus the Q index provides a test of the presence of heterogeneity among ESs along with a corresponding P statistic but not the exact extent of heterogeneity within the meta-analysis (28). The I^2 statistic, which expresses the ratio of the observed variance between outcomes to the total observed variance in ESs (32), has been recommended along with Q to describe the extent of between-studies variability, and was calculated as $(Q - df)/Q \times 100$, where df equals the degrees of freedom. The I^2 statistic was interpreted as small (25% to <50%), medium (50% to <75%), or large (≥75%). Simple, random effects meta-regression analyses were also conducted to examine the association between the outcome variables with exercise intensity, EEE, and $\dot{V}O_{2\,peak}$. Tests of the regression model (Q_R) and its residual error (Q_E) are reported. Because regression analyses are robust against alpha inflation due to multiple tests, a two-tailed alpha value of $P \leq 0.05$ for this test was considered as statistically significant.

RESULTS

Eighteen studies representing 199 men and women met all eligibility criteria and were available for pooling and analysis (2, 14, 20, 23, 25, 27, 30, 40, 44–48, 63, 65–67, 69). All studies were published in the English language between 1991 and 2013. A general description of the study characteristics and primary findings related to postexercise Lox are shown in Table 1. Eleven of the 18 studies included only men (2, 23, 27, 45–48, 63, 65, 66, 69), three were limited to women (14, 44, 67), and four additional studies included and reported data separately for both men and women (20, 25, 30, 40). The average age of the samples was reported in all but one of the studies and was 26.8 ± 1.1 years (range = 21.2–51.7 years).

Table 1. *Effects of acute endurance exercise bouts on subsequent resting lipid oxidation*

Study	Participants (age, BMI)	$\dot{V}O_{2peak}$ (ml·kg^{-1}·min^{-1})	Exercise	Timing of Assessment (hours after end of exercise)	Nutritional State	Results (effect of prior exercise on Lox)
Bahr and Sejersted (1991)	6 moderately active m (23 yr; 21.9 kg/m^2)	49.9	80 min cycle erg @ 75% $\dot{V}O_{2peak}$	Total of 2–7 h	Fed/fasted	Significantly increased in fed and fasted states
Davitt et al. (2013)	12 sedentary obese w (23.8 yr; 37.3 kg/m^2)	25.2	60 min treadmill @ 62.5% $\dot{V}O_{2peak}$	Avg ~7 h	Fed	Significantly increased
Folch et al. (2003)	6 m (27.5 yr; 22.6 kg/m^2); 6 w (luteal *and* follicular phases; 27.2 yr; 21.3 kg/m^2)	43.1 m; 34.3 w	90 min cycle erg @ 50% $\dot{V}O_{2peak}$	Avg 10 h	Fed	Significantly increased in m and w
Gill et al. (2001)	11 moderately active m (51.7 yr; 24.2 kg/m^2)	38.9	90 min treadmill @ 64.9% $\dot{V}O_{2peak}$	Next day, total of 8 h	Fed	Significantly increased
Henderson et al. (2007)	10 moderately active m (24.5 yr; 22.9 kg/m^2); 8 moderately active w (follicular phase; 25.4 yr; 22.2 kg/m^2)	56.6 m; 48.9 w	~60 min cycle erg @ ~65% $\dot{V}O_{2max}$; ~90 min cycle erg @ ~45% $\dot{V}O_{2peak}$	Avg 30–180 min, and next day @ 20 h	Fasted	30–180 min: Significantly increased in both sexes but in m more than w. Next day: Significantly increased in m but not w.
Herd et al. (2001)	8 recreationally active m (27 yr; 24.5 kg/m^2)	3.95 l/min	90 min cycle erg @ 62.3% $\dot{V}O_{2peak}$	Next day, total of 6 h	Fed	No significant change, P = 0.09.
Horton et al. (1998)	14 untrained and endurance trained m (26 yr; 22.6 kg/m^2); 13 untrained and endurance trained w (follicular phase; 26 yr; 20.9 kg/m^2)	55.2 m; 45.6 w	120 min cycle erg @ 40% $\dot{V}O_{2peak}$	Total of 2 h	Fasted	Significant increase in m but not w
Kuo et al. (1999)	6 moderately active m (21.2 yr; 21.9 kg/m^2); 6 moderately active w (22.8 yr; 20 kg/m^2)	48.2 m, 50.5 w	60 min cycle erg @ 65% $\dot{V}O_{2peak}$; 89 min cycle erg @ 45% $\dot{V}O_{2peak}$	Total of 3 h	Fasted	Significant increase in m but not w
Magkos et al. (2006)	7 recreationally active m (28 yr; 22 kg/m^2)	46.5	120 min cycle erg @ 61% $\dot{V}O_{2peak}$	Next day @ 13.5–17.5 h (avg)	Fasted	Significant increase
Magkos et al. (2007)	7 recreationally active m (30 yr; 23 kg/m^2)	42.7	60 min cycle erg @ 60% $\dot{V}O_{2peak}$	Next day @ 14–18 h (avg)	Fasted	No significant change
Magkos et al. (2008)	7 recreationally active m (25 yr; 24 kg/m^2)	43	90 min treadmill @ 28% $\dot{V}O_{2peak}$	Next day @ 14–20 h (avg)	Fasted	No significant change
Magkos et al. (2009)	8 sedentary w (28 yr; 27 kg/m^2)	29	60 min cycle erg @ 60% $\dot{V}O_{2peak}$	Next day @ 14–18 h (avg)	Fasted	No significant change
Malatesta et al. (2009)	12 moderately active m (24.2 yr; 23 kg/m^2)	52.3	60 min cycle erg @ 45% $\dot{V}O_{2peak}$	Avg 2–3 h	Fasted	Significant increase
Thomas et al. (1994)	7 moderately active m (25 yr; 16% BF)	42 (cycle erg); 49.2 (treadmill)	60 min cycle erg and treadmill @ 60% $\dot{V}O_{2peak}$ (data pooled)	Avg 1–2 h, and next day @ 24 h	Fasted	No significant increase at these selected time points
Tobin et al. (2008)	8 sedentary overweight T2D m (age unknown; 29 kg/m^2)	24.7	60 min cycle erg @ 61% $\dot{V}O_{2peak}$	3.5 h	Fasted	No significant change, $P <$ 0.1.
Trombold et al. (2013)	6 recreationally active m (25 yr; 25.6 kg/m^2)	55.5	66.5 min cycle erg @ 48.8% $\dot{V}O_{2peak}$	Next day @ 12–18 h (avg)	Fed	Significant increase
Tsetsonis et al. (1997)	13 moderately active w (43.8 yr; 22.9 kg/m^2); 9 endurance trained w (40.4 yr; 22.2 kg/m^2)	31.7 (untrained); 50.3 (trained)	90 min treadmill @ ~62% $\dot{V}O_{2peak}$	Next day @ 16 −22 h (avg)	Fed	Significant increase in both groups
Westrate et al. (2009)	9 moderately active m (22.6 yr; 22.5 kg/m^2)	Unknown	90 min cycle erg @ 30% $\dot{V}O_{2peak}$; 90 min cycle erg @ 65% $\dot{V}O_{2peak}$	Next day @ 10 h	Fasted	Significant increase after 65% but not 30% $\dot{V}O_{2peak}$

Results are averages from each study. BMI, body mass index; m, men; w, women; $\dot{V}O_{2peak}$, peak aerobic capacity; next day, measurement time point on the day after the completion of exercise. $\dot{V}O_{2peak}$ units provided in l/min where not available as ml·kg^{-1}·min^{-1}. Fasted, in postabsorptive state at time of measurements; fed, in postprandial state at time of measurements. Percent body fat (%BF) provided when BMI data unavailable.

Subjects were generally considered healthy with body mass index (BMI) values ranging from 20–37.3 kg/m^2 (23.2 ± 0.54); however, it should be noted that only two studies (14, 65) used an overweight subject sample, one with nondiabetic obese women (14) and the other providing data on type 2 diabetic overweight men (65). Scrutiny of the studies resulted in 86 ESs: 29 for RER, 23 for metabolic rate, and 34 for Lox.

The intensity of exercise ranged from 28–75% $\dot{V}O_{2\,peak}$ (55.4 ± 2.0%) and the duration from 60 to 120 min (80.2 ± 3.1 min), resulting in a total EEE of 364.3–1207.8 kcal (687.9 ± 37.8 kcal). For training modality, the overwhelming majority was limited to cycle ergometry, four studies used treadmill running, and one (63) pooled data using both cycle ergometry and treadmill modes. The majority of studies used moderately

or recreationally active subjects or endurance athletes, with four studies providing data for sedentary individuals. In one study (20), the training or activity status of the participants was unspecified. Average $\dot{V}O_{2\,peak}$ for subjects ranged from 1.97–4.52 l/min (3.20 ± 0.13 l/min) and as expressed per bodyweight were 24.7–56.6 ml·kg^{-1}·min^{-1} (45.8 ± 1.6 ml·kg^{-1}·min^{-1}). Time periods categorized as immediately after exercise ranged between 2 to 10 h of duration, and those on the next day up to 22 h after exercise. In studies that contributed data on the postprandial period, the standardized meal in all cases contained ~1000 kcal of energy. All standardized meals contained a significant amount of carbohydrate (~30–80% of energy). Of note, the study of Folch et al. was unique in that the meal provided only a negligible amount of fat (less than 1% of energy) (20). In each study, postprandial indirect calorimetry assessments began immediately following the standardized meal and proceeded for the duration indicated in Table 1.

Primary Outcomes

Lipid oxidation. The overall ES for Lox was 0.91 ± 0.11, which was significantly different from zero. The Q and I^2 statistics revealed significant heterogeneity across effects: Q(33) = 46.83, I^2 = 32, $P < 0.05$. When pooling postprandial and postabsorptive data into a single analysis, there was a significantly greater increase in Lox observed postexercise in men (1.04 ± .14) compared with women (0.67 ± 0.18): Q(1) = 2.73, $P < 0.10$. When pooling men and women into a single analysis, no significant difference in postexercise Lox was observed as a function of postprandial (1.10 ± 0.27) vs. postabsorptive (0.86 ± 0.12) states: Q(1) = 0.69, $P > 0.10$. However, effects of nutritional state depended upon sex as follows. Men and women did not significantly differ in Lox ES in the postprandial state: Q(1) = 0.09, $P > 0.10$. However, importantly, in the postabsorptive state the effects were significantly larger for men: Q(1) = 4.80, $P < 0.05$ (Fig. 1). For men, no significant difference was found for Lox ES in the postabsorptive state (1.02 ± 0.14) than in the postprandial state (1.22 ± 0.49): Q(1) = 0.16, $P > 0.10$. Women in the postabsorptive state exhibited an ES of 0.50 ± 0.20, while in the postprandial state the ES for Lox was 1.04 ± .36, a difference that was not statistically significant: Q(1) = 1.8, $P = 0.18$. Additionally, a general time course was observed in which a greater ES for Lox occurred immediately following exercise (1.22 ± .17) compared with that observed the day following exercise (0.60 ± 0.13): Q(1) = 8.51, $P < 0.05$.

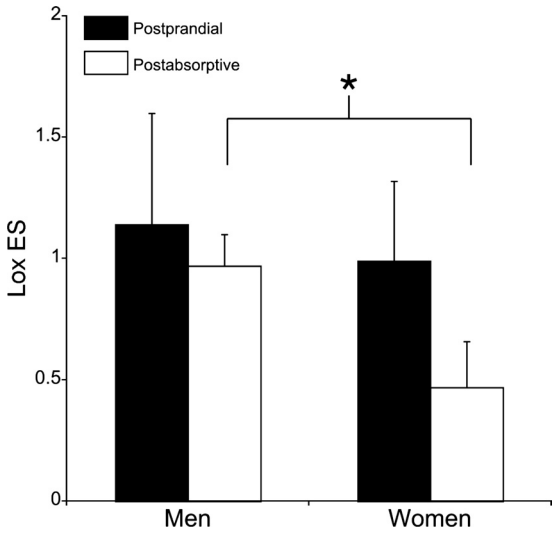

Fig. 1. The impact of a prior endurance exercise bout compared with a sedentary condition on lipid oxidation (Lox) in men and women while in the postprandial (fed) and postabsorptive (fasted) state. Values are means and SE. * Postabsorptive men different from postabsorptive women: $P < 0.05$. ES, effect sizes.

Respiratory exchange ratio. The overall ES for RER was 0.82 ± 0.10, which was significantly different from zero. The Q and I^2 statistics revealed a lack of heterogeneity across effects: Q(27) = 23.96, I^2 = 0, $P > 0.10$. Despite this lack of heterogeneity among the ESs, we examined potential moderators based on a priori study hypotheses and to determine the relative role of RER in postexercise substrate oxidation. No significant difference in RER was observed between men (ES = 0.83 ± 0.12) and women (ES = 0.80 ± 0.16). There was also no significant difference in ESs as a function of postprandial/postabsorptive state of the sample. RER effects for men did not differ from women in either the postabsorptive (ES = 0.84 ± .13 vs. 0.82 ± 0.21) or postprandial (ES = 0.81 ± 0.43 vs. 0.85 ± 0.35) state, respectively. Notably, postprandial state RER data were only attained for men (20, 66) and for women (14, 20) in two studies each, whereas a far more extensive RER data set was analyzed for the postabsorptive state. A trend emerged indicating larger RER effects in the immediate recovery period following exercise (ES = 0.99 ± 0.15) relative to those observed the next day (ES = 0.65 ± 0.15), Q(1) = 2.54, $P = 0.11$.

Resting metabolic rate. ESs for metabolic rate were derived from 12 studies. The overall observed ES was 0.26 ± 0.10, which was significantly different from zero. The Q and I^2 statistics revealed a lack of heterogeneity across effects: Q(22) = 12.13, I^2 = 0, $P > 0.10$. However, similar to RER, follow-up tests were conducted to assess the relative role of RMR in postexercise substrate utilization. Women had less accentuation of metabolic rate postexercise (ES = 0.06 ± 0.16) relative to men (ES = 0.38 ± 0.13), and this trend approached but did not reach statistical significance: Q(1) = 2.51, $P = 0.11$. In terms of the effects of feeding, overall average ES for the postabsorptive state was 0.29 ± 0.11, while in the postprandial state the impact of exercise on RMR was smaller with an ES of 0.05 ± 0.26: Q(1) = 0.73, $P > 0.10$. In the postabsorptive state, men exhibited a significantly larger ES for RMR (ES = 0.51 ± 0.15) than women (ES = 0.05 ± 0.17): Q(1) = 3.94, $P < 0.10$. No significant sex difference was found in the postprandial state: Q(1) = 0.02, $P > 0.10$. However, only three effects emerged from samples in the postprandial state with one of the observations taken immediately after exercise in overweight women (14) while the other two were assessed on the following day in men (23, 66). When analyzing RMR data with postprandial and postabsorptive states pooled together immediately postexercise, men expressed a larger effect for metabolic rate (0.59 ± 0.19) than women (0.11 ± 0.19): Q(1) = 3.0, $P < 0.10$. This effect was not statistically different the following day, although the trend remained with men (0.29 ± 0.20) retaining higher rates than women (−0.07 ± 0.29): Q(1) = 1.0, $P > 0.10$. No statistically significant effects were observed for exercise on RMR during the immediate postexercise recovery period (ES = 0.39 ± 0.15) relative to values

measured the following day (ES = 0.17 ± 0.16): Q(1) = 0.96, $P > 0.10$.

Regression Analyses

Separate regression analyses were conducted using Lox ESs as the dependent variable with exercise intensity (% $\dot{V}O_{2\ peak}$) and EEE (kcal) as predictors in meta-regression analyses to determine the impact of these exercise characteristics on postexercise Lox. No evidence of ES moderation was found for exercise intensity ($Q_R = 0.79$; $P > 0.05$; $R^2 = 0.05$; $Q_E = 33.47$; $P > 0.05$). However, the overall meta-regression model was found to be significant for EEE ($Q_R = 8.89$; $P < 0.01$; $R^2 = 0.18$; $Q_E = 32.34$; $P > 0.05$). Thus overall EEE ($\beta = 0.42$; $z = 2.98$; $P < 0.01$) but not exercise intensity ($\beta = 0.22$; $z = 0.89$; $P > 0.05$) accounted for significant variation in the overall effect of endurance exercise on subsequent Lox. We also ran these meta-regression analyses by gender to determine whether this moderation of EEE on postexercise Lox exists for both men and women. EEE was a significant predictor of postexercise Lox for men ($Q_R = 6.20$; $P < 0.01$; $R^2 = 0.24$; $Q_E = 19.20$; $P > 0.05$) but not for women ($Q_R = 0.32$; $P > 0.05$; $R^2 = 0.001$; $Q_E = 16.12$; $P > 0.05$). Similar to the overall model, exercise intensity did not significantly moderate postexercise Lox in men ($Q_R = 1.51$; $P > 0.05$; $R^2 = 0.14$; $Q_E = 19.82$; $P > 0.05$) or women ($Q_R = 0.01$; $P > 0.05$; $R^2 = 0.01$; $Q_E = 11.38$; $P > 0.05$). However, these gender-specific analyses were underpowered and thus results should be interpreted with caution.

Separate meta-regression analyses were conducted using Lox ESs as the dependent variable with $\dot{V}O_{2\ peak}$ as a predictor to determine whether postexercise Lox is greater among more aerobically fit subjects. For total $\dot{V}O_{2\ peak}$, expressed in l/min, the test for linear regression with Lox was significant, $Q_R = 3.86$; $P < 0.05$, indicating that aerobic fitness was a significant predictor of postexercise Lox. Examination of the beta weight ($\beta = 0.27$, $z = 1.96$, $P < 0.05$) indicated that aerobic fitness was positively predictive of postexercise Lox. A similar trend was observed when $\dot{V}O_{2\ peak}$ was normalized to bodyweight ($Q_R = 3.57$; $P = .058$). When running the meta-regression analysis by gender, statistical significance was lost, likely indicating that sample size or power became insufficient when splitting the data into two sets.

DISCUSSION

On the basis of an analysis of findings from across 18 studies incorporating a variety of study designs, we report that both men and women exhibit a significant increase in Lox after a bout of endurance exercise, but that nutritional state modulates the magnitude of this response in women. Women show similarly robust increases in Lox after exercise while in the postprandial state but less accentuation of Lox than men when in the postabsorptive (fasted) state. We also report that RER is significantly depressed after exercise in both sexes and not different between men and women. Effects of exercise on subsequent metabolic rate are modest compared with RER but may account for gender differences in postabsorptive Lox with greater increases in RMR after exercise in men than women. These meta-analytic findings depict fundamental and novel aspects of sexual dimorphism in energy substrate metabolism. We have also characterized moderating influences of exercise bout parameters and physical fitness on the responses of Lox to prior exercise which further characterizes the control of energy metabolism in humans. Below we discuss the findings in the context of the general effects of physical fitness and exercise volume, potential mechanisms controlling postexercise Lox, sex differences in metabolism, research study design issues, and future directions for research in this area.

The enhancement of Lox by recent exercise, though more robust on the day of exercise, was significant in all conditions (men, women, postprandial, postabsorptive) even on the day after exercise. Thus while it was shown previously that chronic exercise training increases resting Lox (58), it appears that the impact is likely due to a sustained effect of the most recent exercise bout that continues to alter substrate use for a day or longer. However, though changes in resting Lox are a response to the most recent bout, there may be a role for chronic exercise training to enhance this response, as we observed a significant correlation between fitness ($\dot{V}O_{2\ peak}$) and Lox ESs. Also, EEE correlated with Lox ESs, and chronic training improves one's ability to achieve a high EEE, so physically fit individuals may experience greater increases in Lox after each exercise bout as a result of achieving greater EEE.

Next, it should then be considered what the underlying mechanisms could be for the elevation of fat oxidation rates after a single bout of endurance exercise in various circumstances. The primary regulation could either be at the site of fat oxidation (mitochondria), or fat oxidation could be simply driven by free fatty acid (FFA) supply. If FFA supply is of greatest importance, then regulation of lipolysis and blood flow to the lipolytic tissues would be critically important. Particularly in the early postexercise recovery period when FFA mobilization can exceed the apparent rate of lipolysis (12, 25, 29), adipose blood flow is likely a predominant factor in regulation of FFA supply to peripheral tissues (29), and its contribution may continue to some extent for an extended period. If regulation were in mitochondria within the tissue(s) responsible for increased Lox, then it would be important to ask which tissue(s) increase their Lox after exercise. It is typically assumed that the exercised skeletal muscle displays elevated fat oxidation rates after exercise. However, it can be inferred that both during (3, 68) and after (68) exercise, nonmuscle tissues display substantial rates of lipid oxidation as evidenced by the whole body RER being lower than the working limb respiratory quotient. So, we propose that both muscle and nonmuscle tissues are likely involved in elevation of Lox after exercise. As plasma FFA concentration was reported only in a small subset of the studies that met our inclusion criteria, there were insufficient data to include plasma FFA concentration into our analysis. Nonetheless, generally a rise in Lox is associated with a rise in plasma FFA concentration in the postabsorptive state (25, 30, 47), so FFA supply may at least in part contribute to the control of postexercise Lox in various tissues. However, recently it was reported that the substantial rise in postexercise Lox in the postprandial state in obese women is not accompanied by a paralleled rise in plasma [FFA] (14), and so FFA supply (concentration of FFA in plasma) is not tightly coupled with Lox in all circumstances. It is well established that carbohydrate is the major fuel for exercise (7, 9, 10), and lipid is an alternative fuel that can spare carbohydrate, so it is intuitive that lipid oxidation would be elevated during the postexercise recovery period and that the

degree of change in Lox would be related to the amount of carbohydrate use during exercise. As discussed below in the context of sexual dimorphism in postexercise Lox, glycogen depletion is not sufficient to entirely explain the variations between conditions and groups for postexercise Lox, but indeed it could play some role in the coarse control of Lox. The lower ESs for Lox on the day after exercise than day of exercise are consistent with glycogen stores controlling Lox, because the carbohydrate sparing (i.e., reduced RER) during the recovery period would allow for movement back toward carbohydrate balance over time even despite any continuation of the negative energy balance caused by exercise. Additionally, the reduction in postexercise RMR over time would play a role in resumption of Lox back to baseline rates. In summary, postexercise Lox may be regulated both by FFA supply in certain instances as well as by mitochondrial respiration and fuel selection in various tissues. In addition to control of fatty acid oxidation, it may be of additional interest in future work to explore the basis for the apparent sex difference in accentuation of RMR after exercise which could be related to uncoupling or metabolic burden of processes such as lipolysis and gluconeogenesis. Mitochondrial biogenesis in muscle and other tissues such as liver, as modulators of postexercise Lox, may also be an important area for future work.

The present results add to the growing understanding of metabolic differences between men and women. As previously reviewed (61), women exhibit relatively higher rates of fat oxidation (lower RER) than men during exercise, and it has been reported that in the postabsorptive state men exhibit a relatively greater increase in lipid oxidation after exercise than women (25). Previously, conclusions were that while women are better able to oxidize fat during exercise, men are better able to do so afterwards. However, the current findings of our meta-analysis indicate that this postexercise sex difference is only present when subjects are in the postabsorptive state during resting metabolism measurements. When in the postprandial state, in the hours soon after a standardized meal, our systematic analysis of published literature indicated that men and women experience a similar postexercise increase in Lox. Thus nutrition needs to be considered in considering gender differences in exercise-related resting metabolism. Despite differences in Lox ESs, there were no significant sex differences in postexercise RER, indicating that metabolic rate may play a role in determining the sex difference in Lox. In agreement with this inference, ES for RMR after exercise was higher in men than women in the postabsorptive state. This difference for RMR, along with no difference in RER, indicates that the gender difference in Lox in the postabsorptive state may be driven by RMR rather than percent contribution of fat to energy use. Often this increase in metabolic rate after exercise is assessed through a calculation of excess postexercise oxygen consumption (EPOC), but it should be considered that the caloric equivalent of VO_2 depends upon the RER, and so assessments of EPOC are flawed unless a true metabolic rate is calculated based upon the relative contribution of fat and carbohydrate to the total oxygen consumption. For this reason, we analyzed metabolic rate (i.e., RMR) rather than $\dot{V}O_2$ when assessing effects of prior exercise on this contributor to total Lox. The effects of exercise on subsequent RMR are quite modest compared with effects on RER, but we have discovered that the RMR, nonetheless, may play a role in the sex difference in postexercise Lox. As discussed above, glycogen depletion by exercise can potentially play a role in determining postexercise Lox, so it is worth considering if indeed sex differences in substrate selection during exercise correspond to the sex difference in recovery that are reported here. Men rely relatively more heavily upon carbohydrate during exercise than do women (13, 15, 16, 21, 22, 25, 26, 30, 56, 60, 62), so this could lead to higher postexercise Lox by directing glucose to glycogen and away from oxidation during the postexercise recovery period. However, this interpretation is problematic, as the sex difference in postexercise Lox that we describe in the present report was only apparent in the postabsorptive state, but when a meal was taken after exercise (postprandial state), the sex difference was essentially abolished. The postprandial period is the time when net glycogen synthesis would occur to an appreciable extent (which spares carbohydrate from oxidation); the sex difference in Lox could be expected to be accentuated (not blunted) in the postprandial state if it were simply driven by prior glycogen depletion. Other work has supported this inference that glycogen depletion is not sufficient to explain postexercise Lox; when studying the postabsorptive state in which only limited glycogen resynthesis is generally expected, it was shown that glycogen depletion was not the determinant of postexercise fatty acid oxidation but rather energy balance per se played a greater role (54). Lastly, as the present data indicated, the RMR was a major contributor to the sex difference in postexercise Lox, so in considering possible mechanisms, rather than relative fuel partitioning, higher postexercise metabolic efficiency in women than men should be considered as a trait that may be independent of carbohydrate balance alone.

In our analysis we included papers in which exercise was allowed to cause a negative energy balance. When designing a study of the postexercise recovery period, indeed it is a challenge to decide if diet should be adjusted to accommodate for the EEE. Numerous investigative teams have conducted postexercise lipid metabolism studies in which no extra energy was provided (such as those listed in Table 1) such that exercise could manipulate energy balance which may be one of the major metabolic benefits of exercise, even when the alteration is only temporary. Other studies of postexercise metabolism have included a different dietary intake in the exercise condition than in the control condition, increasing energy intake to compensate for the EEE (4, 17, 57). In some studies even further increases to dietary energy intake, beyond that to cover EEE, were provided to compensate additionally for an expected increase in postexercise RMR (49, 51, 52). Generally, feeding extra energy would be expected to blunt postexercise Lox, so it is critical to consider what an appropriate postexercise energy intake would be in comparison to sedentary control conditions. Our goal in the laboratory (and in this report) is to identify physiological changes that represent those occurring in the real world under free-living conditions, and the common finding has been that an acute bout of exercise does not increase subsequent hunger or ad libitum energy intake (5, 6, 11, 31, 35–39, 42). At this point there is little evidence that ad libitum postexercise energy intake is spontaneously increased after exercise, and, though surprising, a significant increase in energy intake during chronic exercise training is also not observed (18, 19). To describe these phenomena, the association between energy expenditure and energy intake has been

described as being only a loose coupling (5). We only included studies in our analysis in which the same energy intakes were provided in the control and exercise trials, and we consider this study design to be consistent with the published literature on dietary responses to exercise. We excluded studies in which a higher caloric content of the standardized diets was provided in exercise trials compared with the sedentary trials, because substrate oxidation findings are expected to be altered by the added energy intake, and so it would be inappropriate to pool these studies together with the others in our meta-analysis. There was not sufficient literature to allow for a separate meta-analysis on studies with higher energy intake in exercise trials than sedentary trials; however, maintaining energy balance through dietary alteration after exercise is an interesting study maneuver, so we discuss the results from these studies here briefly. Furthermore, because of the interindividual variation in the behavioral response to exercise, there are likely certain individuals who do increase their energy intake appreciably in response to exercise (50), so this type of study design would derive data that are directly relevant to a subset of the population. Of the published studies compensating for the EEE with extra energy intake, or even further increasing intake to account for expected increases in RMR after exercise, those that employed a randomized order of trials are discussed here (17, 51, 52, 57). In a study of men in which energy intake on the day of exercise compensated for the EEE, no increase in Lox was observed immediately after exercise (actually tended to be lower in the exercise trial than sedentary trial: ES = −0.30). However, on the day after exercise, after additional time had passed following the extra energy intake, the Lox then tended to be higher in the exercise trial than control trial (ES = 1.3), though not statistically significant in their analysis (17). Their results suggest a possibility that postexercise Lox is suppressed transiently when diet compensates for EEE (17). Alternatively, in another study with the same type of dietary control (higher energy intake in exercise trial than the sedentary trial), Lox was increased after exercise if the exercise intensity was sufficiently high (57). When 24-h Lox was assessed in men in which extra energy was consumed in the endurance exercise trial to compensate both for the EEE and RMR elevation afterward, there was no significant effect of exercise on Lox, and fat balance actually tended to be slightly more positive in the exercise trial than sedentary trial (ES = 0.30) because of the fat ingestion associated with the additional food intake (51). Additional work from this group, using the same type of dietary control, led to similar findings (both in men and women) that modest increases in postexercise Lox did not reach statistical significance and would have been largely outweighed by the increase in fat intake in the exercise trials (52). In conclusion, though in some cases there may still be an increase in Lox after exercise when participants consume extra energy after exercise, this is not a consistent finding and any increase in Lox may be unable to favorably alter fat balance because of the increased fat intake when diet is purposefully manipulated to compensate for the energy expenditure associated with exercise. Additional work in this area may clarify the degree to which Lox can increase after exercise when food intake compensates for energy expenditure, but it appears that for optimal impact of exercise on Lox and fat balance, ideally dietary energy intake would be unchanged in response to exercise.

We have analyzed results for acute bouts of continuous endurance exercise because there are sufficient data in the literature on this topic to allow such an analysis, and because this is a commonly employed exercise approach for improving health. However, future directions could include additional work on high-intensity interval exercise and resistance exercise, and eventually these data can be analyzed through similar meta-analytic and meta-regression techniques to model the impact of sex, fitness, and exercise bout characteristics on postexercise Lox for these alternative exercise modalities. Another consideration that we hope will receive additional attention in future studies is obesity. Presently there are very limited data in the literature on the response of energy substrate metabolism in overweight and obese individuals to prior exercise bouts, and only two of such studies met the criteria for inclusion in the present analysis (14, 65), so more work is needed in this population. To assure that the two studies that used an overweight subject sample (14, 65) did not disproportionately influence the overall findings for Lox, we conducted the analysis both with and without these two studies. The impact on the overall ES was negligible (0.91 vs. 0.90) when these two studies were removed. Moreover, we were unable to examine the correlation between body composition and postexercise Lox due to the restricted range of BMI values across studies. Future work in this area is warranted. Other aspects of postexercise substrate selection that likely deserve attention in the future are the cumulative effects of multiple exercise bouts (i.e., short-term training studies) in which multiple acute responses may sum together into magnified changes in Lox, as well as how the composition of and energy content in postexercise nutrition alters the duration of the responses. To identify determinants of the postexercise response of substrate utilization, we have compared groups of individuals who completed specific types of exercise bouts. There was sufficient variation between study designs to allow us to make discoveries about characteristics of individuals and of exercise bouts that appear to determine the response of postexercise Lox. However, it is worth noting that, in addition to the between-study differences, within each study cohort there would be interindividual variations in how individuals respond to exercise, so the biological complexity controlling the response of energy metabolism to recent exercise is expected to be even greater than that which we were able to identify in the present analysis. Finally, our results for sex-specific metabolic responses to a postexercise meal indicate the possibility of developing sex-specific nutritional strategies in conjunction with exercise training in order to optimize the metabolic response. Specifically, the robustness of the increases in Lox after exercise were similar between men and women in the postprandial state but lower in women than men in the postabsorptive state. So, it appears that Lox tends to be unaffected in men by meal ingestion but enhanced in women by postexercise nutrition. Specifically, women might benefit from the development of ideal meal timing strategies after exercise to optimize the response of Lox. For management of adiposity, in this scenario, it would not be advisable to consume an extra meal, but it might be that moving a meal from another time of day into the early phase of postexercise recovery would assist with achievement of negative fat balance in women. However, we note that caution would be prudent in an attempt to use the study results to support ideas about sex-specific meal timing. Though generally supported by the

trends in the data and the statistically significant sex difference that is dependent on nutritional state, when directly comparing Lox ES from the postprandial and postabsorptive states in women, the difference was not statistically significant. This area of exercise nutrition could be further addressed by additional preclinical work using indirect calorimetry in women and men to further strengthen conclusions, and ultimately ideas about meal timing can be addressed in clinical trials.

In conclusion, there are numerous reports on the impact of acute endurance exercise bouts on subsequent energy metabolism, and we assessed the magnitude of the impact of exercise on resting Lox and related parameters by considering all these studies in aggregate and evaluating ESs and moderating factors. By systematically analyzing these results, we have discovered a role for gender in determining the response of Lox after exercise and identified the relative importance of metabolic rate vs. relative substrate partitioning. We further discovered impacts of physical fitness and exercise volume on the response of Lox to each recent exercise bout. These results add to the growing appreciation that exercise can alter metabolism, not only during exercise, but quite robustly after exercise for hours or even days. The results indicate that when designing research studies in which postexercise substrate utilization is to be assessed, careful consideration should be given to gender, nutritional state, physical fitness, and exercise volume. Practical strategies to increase Lox throughout the day in physically active individuals would potentially include improving physical fitness through chronic exercise training that is sufficiently challenging to raise aerobic capacity, as well as particularly focusing upon exercise volume (EEE) when designing endurance exercise sessions. Ultimately, the changes in substrate utilization after each exercise bout may have implications for use of exercise in managing body composition and other lipid-related aspects of health.

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AUTHOR CONTRIBUTIONS

Author contributions: G.C.H. and B.L.A. conception and design of research; G.C.H. and B.L.A. performed experiments; G.C.H. and B.L.A. interpreted results of experiments; G.C.H. and B.L.A. drafted manuscript; G.C.H. and B.L.A. edited and revised manuscript; G.C.H. and B.L.A. approved final version of manuscript; B.L.A. analyzed data; B.L.A. prepared figures.

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