No reserve in isokinetic cycling power at intolerance during ramp incremental exercise in endurance-trained men

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Ferguson C, Wylde LA, Benson AP, Cannon DT, Rossiter HB. No reserve in isokinetic cycling power at intolerance during ramp incremental exercise in endurance-trained men. J Appl Physiol 120: 70–77, 2016. First published November 12, 2015; doi:10.1152/japplphysiol.00662.2015.—During whole body exercise in health, maximal oxygen uptake (V˙O2max) is typically attained at or immediately before the limit of tolerance (LoT). At the V˙O2max and LoT of incremental exercise, a fundamental, but unresolved, question is whether maximal evocable power can be increased above the task requirement, i.e., whether there is a “power reserve” at the LoT. Using an instantaneous switch from cadence-independent (hyperbolic) to isokinetic cycle ergometry, we determined maximal evocable power at the limit of ramp-incremental exercise. We hypothesized that in endurance-trained men at LoT, maximal (4 s) isokinetic power would not differ from the power required by the task. Baseline isokinetic power at 80 rpm (Piso, measured at the pedals) and summed integrated EMG from five leg muscles (ΣiEMG) were measured in 12 endurance-trained men (V˙O2max = 4.2 ± 1.0 l/min). Participants then completed a ramp incremental exercise test (20-25 W/min), with instantaneous measurement of Piso and ΣiEMG at the LoT. Piso decreased from 788 ± 103 W at baseline to 391 ± 72 W at LoT, which was not different from the required ramp-incremental flywheel power (352 ± 58 W; P > 0.05). At LoT, the relative reduction in Piso was greater than the relative reduction in the isokinetic ΣiEMG (50 ± 9% vs. 63 ± 10% of baseline; P < 0.05). During maximal ramp incremental exercise in endurance-trained men, maximum voluntary power is not different from the power required by the task and is consequent to both central and peripheral limitations in evocable power. The absence of a power reserve suggests both the perceptual and physiological limits of maximum voluntary power production are not widely dissociated at LoT in this population.

central fatigue; peripheral fatigue; V˙O2max; electromyography; exercise tolerance

EXERCISE INTOLERANCE IS A key determinant of quality of life and a strong predictor of all-cause mortality (38). The ability to sustain whole body exercise is strongly correlated with aerobic capacity (V˙O2max), typically assessed by cardiopulmonary measurements during a progressive, incremental exercise test to the limit of tolerance (LoT). This symptom-limited test provides a reliable measure of (among others) the capacity for oxygen transport and utilization available to support the muscular energy demands of the exercise task (23, 54). While the attainment of V˙O2max and LoT during incremental exercise are typically closely coincident, it is unclear whether the two processes are linked, either directly or via common mechanisms: each having both “central” and “peripheral” components.

V˙O2max is dependent on O2 delivery (e.g., central cardiac output and peripheral blood flow distribution) and O2 extraction (e.g., peripheral capillary-to-myocyte O2 diffusion and mitochondrial O2 utilization) (46, 53). For sedentary humans exercising at sea level, the predominant limiting factor determining V˙O2max is the peripheral capacity for O2 extraction (45). Endurance-trained individuals, on the other hand, have preferential skeletal muscle adaptations such that a greater muscle oxidative enzyme activity and capillarity push the predominant limiting factor determining V˙O2max towards the center, i.e., limited by cardiac output (35, 44). The point at which V˙O2max is reached is presumed to place increased and intolerable strain on intramuscular metabolism for ATP production. At this point the ability to continue exercise would necessitate either an improvement in exercise economy (unlikely, given that both work efficiency and economy progressively fall during high-intensity exercise; Ref. 43) or a further increased contribution from substrate-level phosphorylation (glycogenolysis to produce lactate or phosphocreatine breakdown), with the consequent accumulation of metabolic by-products that contribute to muscle fatigue (2). Thus one hypothesis is that V˙O2max is achieved at the point at which, or very soon after which, muscular energy provision, or its consequences, limits the ability for muscle power production (10, 11, 26, 39), and hence determines the LoT (37). In this scenario, V˙O2max determines the maximum capacity for sustained power production and the LoT.

Alternative proposals to exercise limitation at V˙O2max are broadly encompassed by central, neuromuscular mechanisms. These include the influence of afferent feedback from fatiguing muscles to limit central motor drive and/or increased spinal inhibition of cortical drive (3, 5–7, 51); a “central governor” integrating peripheral signals, particularly cardiac strain, to limit the activity of the motor system (40, 41); and the modulation of muscle activity via the sense of effort (32, 34), i.e., recognizing the related but independent influences of the physiological strain and the perception of the task on the central and peripheral neuromuscular system. In these scenarios, the capacity for sustained muscle activation at the LoT determines V˙O2max.

Despite the significance of the mechanisms limiting exercise tolerance, during whole body exercise at V˙O2max there remains a lack of information on the process that ultimately limits muscle power production. A fundamental question, which remains unresolved, is whether or not the maximum voluntary
power producing capacity at \( V_{\text{O}_2}\text{max} \) and LoT exceeds that required by the exercise task (32). In other words, is there capacity for a brief increase in power output at the point of intolerance, or conversely, is the peak voluntary power at the \( V_{\text{O}_2}\text{max} \) and LoT equal to the power demands of the task? Identifying a power reserve at LoT is consistent with the exercise limitation residing at the capacity for sustained muscle activation, whereas no power reserve is more consistent with muscle metabolism and fatigue ultimately causing exercise intolerance.

During isometric single-muscle group contractions, torque generated during a maximal velocity contraction does not exceed the requirement at the LoT (2, 14, 20). This is unsurprising given the isolated nature of the task, which should limit strain on cardiac output and ventilation for example, thereby isolating the fatiguing processes to be proximal to the local musculature. During dynamic whole body exercise that elicits peak cardiopulmonary strain and \( V_{\text{O}_2}\text{max} \), however, the complexities of instantaneously implementing the isokinetic torque measurements required for precise assessment of power producing capacity at LoT have hampered the ability to address this question. Using a maximum voluntary cycling power test, Marcora and Staiano (32) reported that the power producing capacity exceeded the demands of the task by almost threefold, i.e., a neuromuscular “power reserve” remains at the LoT, and exercise is limited by the perception of effort. However, this study was criticized because it did not appropriately control for muscle contraction velocity, which has a dramatic influence over power production in both fresh and fatigued states (12, 13, 18, 24, 31, 33, 48). We have recently addressed this complexity by implementing an instantaneous switch between standard (hyperbolic) cycle ergometry control and isokinetic cycling, which allows peak isokinetic power (\( P_{\text{iso}} \)) to be assessed at baseline and at the instant of intolerance (22). We found that in a heterogeneous group of participants (aged 29 to 72 yr, and \( V_{\text{O}_2}\text{max} \) 23.5 to 62.5 ml·min\(^{-1}\)·kg\(^{-1}\)), \( P_{\text{iso}} \) was slightly (18%), but significantly, greater at the LoT than the demands of the incremental exercise. However, because of the heterogeneous characteristics of the group, it was unclear whether this small power reserve was related to differences in aerobic capacity, age, habitual physical activity close to \( V_{\text{O}_2}\text{max} \), or some other factor, among the participants.

Thus the aim of this study was to test whether a “power reserve” remains at the LoT in young endurance-trained men. We hypothesized that, at \( V_{\text{O}_2}\text{max} \), \( P_{\text{iso}} \) is related to the reduction in maximal muscle activity and not different from the power requirement of the incremental exercise task at the LoT. We tested this hypothesis in endurance-trained men, who were well motivated and familiarized with the sensations of maximal exercise and more likely to be limited by central \( O_2 \) delivery rather than peripheral \( O_2 \) extraction (53). Therefore, we supposed that endurance-trained individuals would be better able to demonstrate a power reserve should one exist.

**METHODS**

**Participants and ethical approval.** Twelve healthy, endurance-trained men (means ± SD; 22 ± 1 yr; 182 ± 8 cm; 76 ± 8 kg) volunteered and provided written informed consent to participate in the study. Participants were screened with a health and physical activity questionnaire, with individuals identified as having any known disease excluded. All procedures were approved by the Faculty of Biomedical and Life Sciences Ethical Committee for nonclinical research (University of Leeds) and complied with the latest version of the Declaration of Helsinki. Participants visited the temperature-controlled laboratory on a maximum of two separate occasions, with a minimum of 24 h between visits. Before each visit participants abstained from strenuous exercise (previous 24 h), alcohol consumption (24 h), and food and caffeine ingestion (3 h).

**Equipment and measures.** The exercise protocol was performed on a computer-controlled electromagnetically braked cycle ergometer, which allowed for instantaneous switching between cadence-independent (hyperbolic) and isokinetic modes (Excalibur Sport PF; Lode, Groningen, The Netherlands). Power was measured at the bottom bracket of the crank every 2° of angular rotation, where crank power is the product of torque and instantaneous angular velocity (21, 22). During the exercise, surface electromyography (EMG; Telemyo 2400T G2; Noraxon, Scottsdale, AZ) was measured in five muscles (vastus lateralis, rectus femoris, vastus medialis, biceps femoris, and lateral gastrocnemius) of the right leg at 1.500 Hz. Electrode placements followed Surface Electromyography for the Non-Invasive Assessment of Muscle (SENIAM) guidelines. Throughout the exercise protocol expired gases (mass spectrometer) and inspired and expired volumes (turbine; Interface Associates, Laguna Niguel, CA) were sampled and digitized at 50 Hz for breath-by-breath measurement of pulmonary gas exchange and ventilatory variables (MSX; nSpire Health, Hertford, UK). Before each test the mass spectrometer was calibrated using two precision-analyzed gases, and the stability of the mass spectrometer calibration was confirmed by resampling these gases immediately after each exercise test. The turbine was calibrated before each test using a 3-L syringe over a range of different flow profiles. Heart rate and arterial \( O_2 \) saturation were measured throughout the protocol using the R-R interval of a 12-lead ECG (Quest; Burdick, Washington, DC) and earlobe pulse oximetry (Biox 3745; Ohmeda, Louisville, KY), respectively.

**Exercise protocol.** Following a minimum of 1-min seated rest on the cycle ergometer, and 4 min at 20 W, participants completed 6-min cycling at 50 W (below the lactate threshold for all participants), immediately after which peak isokinetic power was measured at baseline (baseline \( P_{\text{iso}} \), in unfatigued state). \( P_{\text{iso}} \) was measured from the mean crank power across five maximal effort crank revolutions at a pedaling cadence of 80 rpm. The ability of the ergometer to maintain the target isokinetic cadence throughout this 3.75 s effort was confirmed post hoc. Subsequently, and after a minimum of 6 min of recovery at 20 W (to allow for stabilization of \( CO_2 \) stores; Ref. 42), each participant performed a symptom limited ramp incremental exercise test (20–25 W/min). The LoT was defined as the point at which the participant was unable to maintain a cadence >55 rpm despite strong verbal encouragement. The final five pedal strokes in this incremental phase were analyzed to determine the crank power at the LoT with the pedal cadence unconstrained, i.e., with the ergometer in the hyperbolic mode (LoT \( P_{\text{hyp}} \)). The cycle ergometer was then instantaneously switched to isokinetic mode and the participant performed a final 5 isokinetic crank revolutions at maximal effort (LoT \( P_{\text{iso}} \), Fig. 1). The participant was then monitored in recovery at 20 W for at least 4 min. Seven of the 12 participants repeated this incremental-\( P_{\text{iso}} \) test on a different day. The mean coefficient of variation for \( P_{\text{iso}} \) at LoT (3%) was similar to our previous report (22). As there was no difference between LoT \( P_{\text{iso}} \) in repeated tests, only the results from the initial test are reported here.

Throughout the protocol, EMG signals from the five leg muscles were recorded in 10-s bins. Specifically, EMG recordings were captured at the following time points: at the end of baseline cycling at 20 W; during the baseline \( P_{\text{iso}} \) measurement; immediately before the onset of the ramp incremental phase; for the final 10 s of each minute during the ramp incremental phase; during the LoT \( P_{\text{hyp}} \) phase; during the LoT \( P_{\text{iso}} \), and in the final 20 s of recovery.

**Data analyses.** Breath-by-breath responses were edited in the \( V_{\text{O}_2} \) domain to exclude any occasional erroneous breaths (greater than

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Power, cadence, and EMG measurements. Power and cadence traces during baseline $P_{\text{iso}}$, LoT $P_{\text{hyp}}$, and LoT $P_{\text{iso}}$ are displayed in Fig. 2 for a representative subject. The instantaneous pedaling cadence and the mean required flywheel power at the LoT in the ramp incremental exercise test are shown. Also displayed are the rectified, filtered EMG signals for the vastus lateralis, vastus medialis, and rectus femoris during the 3.75 s of maximal effort crank revolutions.

Mean baseline $P_{\text{iso}}$ was 788 ± 103 W, attained with a measured isokinetic cadence of 80.5 ± 0.1 rpm. The $P_{\text{hyp}}$ measured at the crank at the LoT, was 310 ± 58 W and occurred at 55 ± 4 rpm ($P < 0.05$ vs. both $P_{\text{iso}}$ conditions). The LoT $P_{\text{iso}}$ was 391 ± 72 W at a measured isokinetic cadence of 80.3 ± 0.2 rpm, which was not different from the cadence during the baseline $P_{\text{iso}}$ measurement ($P = 1.000$). LoT $P_{\text{hyp}}$ and LoT $P_{\text{iso}}$ decreased to 39 ± 5 and 50 ± 9% of baseline $P_{\text{iso}}$, respectively.

Neither LoT $P_{\text{hyp}}$ (310 ± 58 W) nor LoT $P_{\text{iso}}$ (391 ± 72 W), both measured at the crank, were different from the flywheel power required at the point intolerance in the incremental test (352 ± 58 W; LoT $P_{\text{iso}}$ vs. flywheel comparison, $P = 0.116$, ES = 0.81, $\beta = 0.73$; $P_{\text{hyp}}$ vs. flywheel comparison, $P = 0.093$, ES = 1.64, $\beta = 0.90$; Fig. 3). The absence of a difference between the LoT $P_{\text{hyp}}$ and the required flywheel power at the LoT validated the assumption that participants were driving the pedals maximally in the final stages of the ramp incremental test to the point of intolerance. The absence of a difference between LoT flywheel power and $P_{\text{iso}}$ (mean: 39 ± 48 W; range: −12–105 W; equivalent to 96–135% of the power required to maintain the incremental exercise task at LoT) demonstrated that there was no “power reserve” at the limit of ramp incremental cycle ergometry (Fig. 3).

There was no difference in $\Sigma iEMG$ at 20 W between baseline (10 ± 3%) and recovery (10 ± 6%; $P = 0.813$), relative to baseline $P_{\text{iso}}$ $\Sigma iEMG$ (100%). The relationship between the $\Sigma iEMG$ and $P_{\text{iso}}$ at the LoT (isokinetic at 80 rpm; $\Sigma iEMG$ and $P_{\text{iso}}$ both normalized to the baseline $P_{\text{iso}}$ test) is presented in Fig. 4. Overall, the percentage reduction in $P_{\text{iso}}$ was greater than the percentage reduction in $\Sigma iEMG$ (50 ± 9 vs. 63 ± 10% of baseline; $P < 0.004$). However, there were two distinct groups: in some participants the reduction in $P_{\text{iso}}$ was closely proportioned to the reduction in muscle activity (i.e., within 10%; $n = 6$; Fig. 5A), while in others the reduction is $P_{\text{iso}}$ was greater than the reduction in muscle activity (i.e., >10%; $n = 6$; Fig. 5B). Nevertheless, despite the heterogeneity in maximal evocable muscle activity, isokinetic power production was not different from the required flywheel power at LoT (Fig. 3). There was a weak relationship between $V_{O2\max}$ and the relative reduction in $P_{\text{iso}}$ at the limit of tolerance ($r^2 = 0.348$; $P = 0.057$; Fig. 5C).

DISCUSSION

The aim of this study was to test whether or not a power reserve remains at the LoT during incremental exercise in young endurance-trained men. The major finding was that during a <4 s maximal isokinetic cycling effort at the point of intolerance, young endurance-trained men were unable to voluntarily increase power output applied to the crank above the level required by the task. Thus the absence of a power reserve suggests that the maximum voluntary power producing capa-

Fig. 1. Schematic of the experimental protocol. Following a period of rest and 20-W baseline pedaling, constant power exercise was performed at 50 W for 6 min, immediately followed by 5 maximal effort isokinetic crank revolutions at 80 rpm [c; baseline isokinetic power ($P_{\text{iso}}$)]. Following a period of recovery at 20 W to allow for the stabilization of body CO2 stores (at least 6 min), a ramp incremental test was performed to the limit of tolerance (LoT) with summed integrated EMG ($\Sigma iEMG$), with this immediately followed by 5 maximal effort isokinetic crank revolutions at 80 rpm (c; LoT $P_{\text{iso}}$). The time points of the $\Sigma iEMG$ measures are also shown (○).

99% of the local mean prediction limits; Ref. 30), which were considered to be uncharacteristic of the underlying physiological response. Lactate threshold was estimated using the V-slope relationship, the end-tidal fractions of O2 and CO2, and the ventilatory equivalents for O2 and CO2 (55). $V_{O2\max}$ was calculated as the mean $V_{O2}$ for an integral number of breaths over the last ~20 s before the LoT.

Baseline $P_{\text{iso}}$, LoT $P_{\text{hyp}}$, and LoT $P_{\text{iso}}$ were each calculated as the mean over five pedal strokes (1.800° rotation), with left and right crank power measurements summed. In addition, the mean required flywheel power at the LoT (i.e., the power determined by the programmed ramp incremental protocol) was calculated from the ramp duration and the power incrementation rate.

For each of the five muscles, the EMG signals were band-pass filtered (20–500 Hz) and rectified, with the signals from five pedal strokes in each time period for each muscle integrated and then summed ($\Sigma iEMG$). The muscle selection was made to reflect the maximal overall muscle activation (maximal motor activity).

Statistical analysis. Power, cadence, and $\Sigma iEMG$ signals were compared using a one-way repeated measures ANOVA, with Bonferroni (cadence and $\Sigma iEMG$), or Dunnett (power; with flywheel power set as the reference variable for comparisons) post hoc analyses performed where appropriate. Statistical significance was set at $P < 0.05$. Effect size (ES; Cohen’s $d$) and statistical power ($\beta$) were also calculated for the comparisons among cycling power measurements at the LoT. All values are reported as means ± SD.
ity at LoT is not different from the demands of the task, such that task failure in whole body exercise at \( V_{O2max} \) is limited by the capacity to produce the required power. We also found that, on average, the reduction in muscle power production during a maximal isokinetic effort was greater than the reduction in muscle activity (from \( \%EMG \)) implicating an important role for muscle fatigue in limiting exercise tolerance at \( V_{O2max} \) in young endurance-trained men.

**Maximum voluntary power at \( V_{O2max} \)** Central to developing an understanding of the relationships among \( V_{O2max} \), LoT, and fatigue is validating the assumption that maximum evocable power is limiting at the point of intolerance. This assumption has been challenged by evidence that at the LoT of constant power exercise at 80% \( V_{O2max} \), participants could generate, on average, \( \sim 500 \) W during a brief “Wingate” style sprint: this was \( \sim 300\% \) more power than that required by the exercise task (32). However, in isolated muscles or muscle groups, the relationship between power output and muscle shortening velocity is parabolic: this relationship is maintained during complex movements such as cycling (12, 13, 48). Therefore, appropriate assessment of whether voluntary maximal power is limiting, or not, requires task and velocity-specific measurements (13, 18, 22, 24, 31) to be made instantaneously at the point of intolerance (1, 22, 47). We overcame this complexity using an instantaneous switch between hyperbolic and isokinetic cycling, allowing us to compare maximal voluntary power production at a fixed velocity. We found that at the point of ramp incremental exercise intolerance, cycling power increased from 310 ± 58 W (LoT \( P_{hyp} \)) to 391 ± 72 W (LoT \( P_{iso} \)) when cadence was increased from \( \sim 55 \) rpm (LoT \( P_{hyp} \); middle column), and at intolerance with isokinetic cadence at 80 rpm (LoT \( P_{iso} \); right).

![Graph showing instantaneous power measured in the right (solid) and left (dashed) cranks during 5 maximum effort cycling pedal strokes (3.75 s; row 1). Also displayed are the measured instantaneous cadence (horizontal solid line) and required crank power at the limit of tolerance of the ramp incremental exercise test (LoT; horizontal dotted line). Bottom: rectified, filtered, EMG signals from the vastus lateralis (row 2), vastus medialis (row 3), and rectus femoris (row 4) during 5 maximum effort cycling pedal strokes. Data are shown in the baseline, unfatigued, condition (baseline \( P_{iso} \); left) at intolerance with unrestrained cadence at \( \sim 55 \) rpm (LoT \( P_{hyp} \); middle column), and at intolerance with isokinetic cadence at 80 rpm (LoT \( P_{iso} \); right).]
and isokinetic power at the limit of tolerance (LoT $P_{iso}$; 80 rpm) relative to the variables such as O$_2$ extraction and muscle oxidative ATP “central” factors such as O$_2$ delivery, rather than “peripheral” exercise is terminated at the point of interception between $P_{iso}$ producing capacity equals the exercise task. In other words, performance fatigue increases until the power progressively, allowing incremental exercise to continue until a greater proportion of baseline $P_{iso}$ has been accessed. While speculative, this protection from performance fatigue may be a consequence of a reduced requirement for intramuscular substrate-level phosphorylation at any given power, slowing the rate of accumulation of metabolites associated with peripheral fatigue development (such as inorganic phosphate; Refs. 2, 36), and/or a greater proportion of fatigue resistant muscle fibers with high oxidative capacity engaged in the exercise (8, 9).

The absence of a power reserve is consistent with the suggestion that during dynamic whole body exercise in endurance-trained individuals, in which rates of O$_2$ delivery and utilization are at, or near, maximum, the perceptual and physiological limits of the exercise are not significantly dissociated at intolerance (cf. Ref. 32). Whether this assertion holds true for different whole body exercise protocols (e.g., short vs. sustained tasks) or in different participant populations (e.g., patients with chronic cardiovascular or pulmonary disease) remains to be determined.

Mechanisms of exercise intolerance in whole body exercise. The reduction in maximal voluntary muscle activation (ΣiEMG) confirms the important role for reductions in central motor drive (i.e., central fatigue) and/or spinal inhibition of cortical drive in performance fatigue (3, 5–7, 20, 22, 29, 51). The approach used in this study is unable to isolate whether this reduction in motor activity occurs at the level of cortical

A schematic of this notion is shown in Fig. 6. The dynamics of performance fatigue are typically shown to be approximately linear during isolated muscle tasks (14, 20) or curvilinear (rapid at the onset and slower as time progresses) for constant power tasks using isolated muscle groups (27, 29), but the dynamics of performance fatigue for whole body incremental exercise are less well known. Using interleaved maximal isokinetic efforts, Cannon et al. (21), demonstrated that exercise below the lactate threshold was not associated with a reduction in peak power but exercise above the lactate threshold was associated with fatigue. Therefore, the schematic in Fig. 6 assumes that performance fatigue develops only once the lactate threshold is exceeded (21). As whole body exercise progresses, performance fatigue increases until the power producing capacity equals the exercise task. In other words, exercise is terminated at the point of interception between $P_{iso}$ (Fig. 6, dashed line) and the required flywheel power (Fig. 6, solid line), with no power reserve (cf. Ref. 2).

We combined the data from this study with those of Coelho et al. (22) to demonstrate that across a wide range of aerobic function (i.e., untrained participants in Coelho et al. and endurance-trained participants in this study) there is a weak significant relationship between V$_{O2max}$ and performance fatigue (the relative reduction in $P_{iso}$; $r^2 = 0.210$; $P = 0.021$). This is consistent with the suggestion that aerobic capacity protects against fatigue-induced reductions in power output, allowing incremental exercise to continue until a greater proportion of baseline $P_{iso}$ has been accessed. While speculative, this protection from performance fatigue may be a consequence of a reduced requirement for intramuscular substrate-level phosphorylation at any given power, slowing the rate of accumulation of metabolites associated with peripheral fatigue development (such as inorganic phosphate; Refs. 2, 36), and/or a greater proportion of fatigue resistant muscle fibers with high oxidative capacity engaged in the exercise (8, 9).

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output (central motor drive), spinal inhibition, or excitation-contracting coupling. However, it is of relevance that Coelho et al. (22) recently demonstrated the relationship between muscle activity and power production during isokinetic cycling is linear, allowing us to make some inferences based on changes in the $\Sigma iEMG$-$P_{iso}$ relationship between baseline and intolerance. We found, overall, that the relative reduction in $P_{iso}$ was greater than the relative reduction in $iEMG$ (Figs. 4 and 5, A and B), suggesting that muscle fatigue and reduced muscle activity combine to reduce maximal evocable power. Afferent feedback from fatiguing muscle is known to reduce spinal excitability and inhibit motor activation (6, 7, 29, 49, 50). While half ($n = 6$) of our participants showed an $\Sigma iEMG$-$P_{iso}$ relationship consistent with a large contribution of muscle fatigue to performance fatigue (similar to Ref. 22), the LoT in the other six participants was strongly associated with the reduction in $iEMG$ alone. The reasons for this variability among individual responses are currently unclear, but these data emphasize how little is understood about the interactions between overt muscle fatigue and the reduction in muscle activation that combine to bring about intolerance during whole body exercise.

It is uncommon for muscle fatigue (e.g., assessed by twitch force measurement) to exceed a given threshold within any individual and task (3, 4, 7, 20, cf. 6, 28), despite interventions that alter the rate at which fatigue-related metabolites are accumulated, such as the magnitude of the external power above the critical power asymptote (19, 52), arterial oxygen concentration (4, 52), preexisting muscle fatigue (3, 26), or manipulations of blood flow (16, cf. 15, 17). The implication of this “fixed threshold” of muscle fatigue is that the absence of a power reserve at LoT requires that central fatigue must be variable during protocols that differ in duration (20) or during exercise in hypoxia (4, 52). This seems at odds with our observation that at LoT some participants show features of the $\Sigma iEMG$-$P_{iso}$ relationship that are consistent with a large component of muscle fatigue, and others with a large component of central fatigue, but all reach the point where external power can no longer meet the demand. Amplification of the central fatigue component for a given level of muscle fatigue may occur during prolonged exercise (20, 49); however, in our study the ramp duration was similar for all participants. Thus, while it seems there was a variable combination of central and peripheral mechanisms that limited whole body exercise in this study, this was neither associated with a peripheral fatigue “threshold” nor a difference among individuals in the physiological limits to performance and the perceptual limits of the task during incremental exercise to $VO_{2\text{max}}$ in endurance-trained young men.

**Limitations.** A potential limitation of this study is the low number of participants, which could limit the ability to detect a power reserve should one exist. The ES (0.81) and moderate $\beta$ (0.73) for the comparison between flywheel power and $P_{iso}$ at the LoT provide some potential for a type II error in the interpretation that there was no power reserve at the LoT. However, an important consideration is the physiological importance of any potential difference in $P_{iso}$ and flywheel power. In an attempt to contextualize the ES we measured the 95% confidence interval of mean fluctuations in crank power during the entire ramp incremental protocol ($\pm 21.5\%$ of the flywheel power), i.e., brief fluctuations in power production are observed in “normal” cycling and vary by $\sim 20\%$. Therefore, we suggest that any power reserve would need to exceed this ‘normal’ fluctuation.
(~20%) to be considered physiologically important. However, our participants could only produce a 12% increase in power between flywheel demand and \( P_{\text{iso}} \) at LoT. Therefore, we believe that the observation in this study is a valid reflection of the absence of a power reserve at LoT.

**Conclusion.** In endurance-trained participants at the \( \dot{V}O_{\text{max}} \) and LoT of ramp incremental exercise, maximum voluntary isokinetic cycling power is reduced to a value not different from the flywheel power required by the task. The absence of a power reserve is consistent with the assertion that the perceptual and physiological limits of maximum voluntary power production are not widely dissociated at the LoT in this population.

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