Effects of ‘warm-up’ exercise on energy provision and exercise performance in horses and humans: a comparative review

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Abstract
Equine and human athletic endeavour often requires near-maximal rates of aerobic metabolism. It, therefore, follows that any practical method of increasing the aerobic contribution to exercise should be of benefit to athletic performance. Prior ‘warm-up’ exercise is widely advocated before exercise performance in order to ‘prime’ the physiological mechanisms of power generation and energy supply. In the present review, we examine evidence that prior exercise, in both the horse and the human, results in marked increases in O2 supply and utilization during subsequent intense exercise. Much of this evidence stems from the study of pulmonary oxygen uptake dynamics and the related concepts of oxygen deficit and critical power. We, therefore, also review the effect of prior exercise in light of the exercise intensity domains in which the prior and subsequent exercise performances take place. Recent evidence suggests that both moderate and heavy exercise should improve subsequent severe exercise performance in both species by ~2-3%, although much work remains to be done to establish the ‘optimal’ warm-up regime(s).

Keywords: Oxygen Uptake kinetics; blood lactate; intensity domains; critical power

Introduction

Athletic performance in both the horse and the human is dependent on effective mechanisms of energy supply and utilization. As reviewed recently1, the horse could be said to represent the gold standard for mammalian aerobic power. The trained Thoroughbred or Quarter Horse, for example, can attain peak galloping speeds in excess of 60 km h⁻¹, and can sustain speeds of more than 40 km h⁻¹ for several minutes. Human sprinters, in contrast, can only achieve the latter speed fleetingly. However, humans are noted for their considerable endurance capability, and this has been suggested recently to be critically important in human evolution2. In both species, however, the power output generated by skeletal muscle for locomotion is associated with an instantaneous increase in energy demand that ultimately must be satisfied by aerobic energy transfer if the work rate is to be sustained for longer than a few seconds3. Ideally, the muscle would be able to instantaneously increase and decrease aerobic energy transfer to meet all cellular energy requirements. However, since the conductance of O2 from the atmosphere to the myocytes follows a pathway of ~1-3 m in both species, this is not possible. The solution to this transport problem is described by the concept of an O2 deficit: because the delivery and consumption of O2 cannot instantaneously match the sudden change in energy demand, mammalian muscle contains energy reserves in the form of high-energy phosphate compounds (chiefly phosphocreatine, PCr) that effectively ‘buy time’ for the cardiorespiratory systems to adjust to
exercise. It is important to note that the $O_2$ deficit concept does not necessarily imply an $O_2$ delivery deficit, but rather a deficit between the instantaneous energy demand and energy supply attributable to mitochondrial ATP resynthesis. If the work rate is such that the aerobic system cannot supply energy at a rate to prevent either the continued depletion of these energy stores or the accumulation of metabolites that might interfere with muscle action, fatigue will inevitably occur. It, therefore, follows that if the aerobic system can be 'primed' somehow to increase more rapidly or more completely, exercise tolerance and, therefore, performance will improve.

The supply and utilization of $O_2$ can be improved by chronic endurance training, which has been shown to markedly accelerate the oxygen uptake ($VO_2$) response to exercise and, therefore, reduce the $O_2$ deficit. However, recent evidence in both humans and horses suggests that an acute bout of prior 'warm-up' exercise can 'prime' the aerobic system and improve exercise performance. The purpose of this review is 3-fold: (1) to describe the effect of prior exercise on the VO$_2$ response in horses and humans; (2) to highlight similarities and differences in the responses following an exercise-induced 'priming' in horses and humans; and (3) to review the implications of the above findings for exercise-induced 'priming' in horses and humans; and (3) to review the implications of the above findings for exercise tolerance and, therefore, performance will improve.

The oxygen deficit concept and exercise intensity domains

It should be noted that 'energy provision' during whole-body exercise is an elusive quantity. Energy provision is usually estimated from measurements of power output, estimates of substrate level phosphorylation and glycolytic flux from biopsy samples or nuclear magnetic resonance spectroscopy (MRS) or, most commonly, through indirect calorimetry. In both horses and humans, intrabreath pulmonary gas exchange profiles can be measured to calculate the breath-by-breath time course of the VO$_2$ response to exercise, which provides for data acquisition with a high time resolution when compared to biopsies and MRS. Furthermore, the instantaneous energy demand can be deduced from applying a step exercise test for long enough to attain a steady state in VO$_2$, at which point the energy demand is met almost exclusively by aerobic ATP provision (Fig. 1A), with demand thought to be communicated to the mitochondria from the various ATPases through the phosphorylcreatine (PCr) shuttle. The anaerobic energy contribution can then be calculated by subtraction of the instantaneous VO$_2$ in the transient phase from the energy demand. The 'O$_2$ deficit', therefore, represents the phosphate bond energy supplied by non-oxidative sources during the early phase of exercise (2–3 min).

An important assumption upon which the O$_2$ deficit concept is based is that the pulmonary VO$_2$ response reflects the muscle energetics in both amplitude and time course. Recent experiments by a number of groups have provided convincing evidence that this is indeed the case, for both moderate- and heavy-intensity exercise.
(for excellent reviews, see Behnke et al.8 and Rossiter et al.9). Specifically, Grassi et al.10 demonstrated a close temporal correspondence between the pulmonary and muscle VO2 responses during moderate upright cycle exercise, and Rossiter et al.11,12 have convincingly demonstrated the temporal coupling of VO2 and [PCr] kinetics during prone knee extension exercise in the moderate- and heavy-intensity exercise domains. These latter experiments and the recent work of Kindig et al.13 add further credence to the role of high-energy phosphate turnover, independent of alterations in O2 delivery14 or increased substrate flux through the pyruvate dehydrogenase complex (PDH)15–19. In short, the VO2 response to the ‘normal’ condition (that is upright exercise at sea level, utilizing a large muscle mass) indirectly reflects cellular energetic events with sufficient time resolution and statistical confidence to draw valid inferences regarding the energy provision of exercise in humans, and probably also in horses20,21.

It is important to note that the general scheme described above is valid for work rates that do not engender a sustained elevation in blood [lactate]. Work rates that are associated with elevated blood [lactate] manifest discrete and predictable metabolic and gas exchange responses of the individual to the imposed work rate (Fig. 1B), which can be used as a means of classifying the relative intensity of exercise. Presently, four exercise intensity ‘domains’ have been identified in humans, namely ‘moderate’, ‘heavy’, ‘severe’ and ‘extreme’3,7,22–24. Appreciation of these intensity domains is essential to the interpretation of the effect of warm-up exercise in both horses and humans, because each domain is associated with distinct VO2 kinetic responses and, crucially, fatigue mechanisms.

The moderate exercise intensity domain encompasses all work rates below the lactate threshold (LT) and/or gas exchange threshold as determined from an incremental exercise test. In this domain, there is little or no perturbation of blood acid-base status (i.e. blood [lactate] remains at, or very close to, baseline values) and following phase I, VO2 rises in a monoexponential fashion to attain a steady state (with a ‘gain’, or O2 cost, approximating 10 ml min-1 W-1) within 2–3 min in healthy human subjects (Fig. 1). The rise in VO2 above baseline is composed of an abrupt increase in VO2 that is associated with increased cardiac output25, which has been termed the ‘cardiodynamic phase’ (phase I), followed by an exponential rise to the steady state, referred to as the primary, or fundamental phase (phase II)3. The time course with which a steady state is attained is described using the primary, or phase II time constant (τ). The net increase in VO2 above baseline at the steady state is known as the primary VO2 amplitude (Fig. 1A). The moderate domain is often considered the ‘control’ domain, because it provides the simplest response to characterize mathematically3,7,25. Furthermore, moderate cycling exercise can continue for many hours in motivated human subjects, indicating that the observed responses in this domain are, over the usual period of study (minutes not hours), free from the confounding influence of fatigue.

The heavy exercise domain comprises those work rates between the LT and the so-called ‘critical power’ (CP), which represents the asymptote of the hyperbolic relationship between work rate and time to fatigue26 (Fig. 1B). The CP is functionally, if not exactly27, equivalent to the ‘maximal lactate steady state’ (MLSS) which is itself defined as the highest work rate at which blood [lactate] is elevated above baseline values but stable over time28. Therefore, during heavy exercise, there is a sustained acidosis, which does not become more severe as exercise proceeds. In this domain, the primary gain of exercise is not discernibly different from that observed in the moderate domain (i.e. ~10 ml min-1 W-1), although whether the primary τ is longer in heavy-compared to moderate-intensity exercise is controversial. One important discriminating feature of heavy-compared to moderate-intensity exercise is that at approximately 2–3 min following the onset of exercise, a secondary rise in VO2 becomes evident, which elevates VO2 above the anticipated steady-state value and delays the attainment of this steady state (this might typically require 5–15 min, depending on the proximity of the work rate to the CP; Fig. 1B). As a consequence of this behaviour, this component of the response has been termed the VO2 slow component3. Qualitatively, at least, the characteristic VO2 response to moderate and heavy exercise does not differ appreciably between horses and humans. The works of Langsetmo et al.20 and Langsetmo and Poole21 demonstrate that the primary and slow components of the VO2 response are also evident in the horse. According to the CP concept, heavy exercise could theoretically continue indefinitely. In reality, however, the tolerable duration of heavy exercise is finite29 and appears to be determined by the rate of endogenous fuel stores utilization and/or the attainment of hyperthermia30.

Severe exercise is defined here as the range of work rates at which no steady state can be attained either in blood [lactate] (or pH) or VO2. At work rates above the CP/MLSS, blood [lactate] and VO2 rise and pH falls until the exercise is terminated by fatigue51 (Fig. 1B). Whether or not the VO2max is attained depends principally upon the duration of exercise: supra-CP work rates that are sustained for longer than approximately 2–3 min are likely to enable the attainment of VO2max whereas shorter durations are not24. The tolerable duration of exercise in this domain appears to be related to the rate at which VO2 rises in the slow
phase (and presumably the rate at which muscle pH and [PCr] fall); exercise tolerance is typically limited to less than a further minute once the VO$_{2_{\text{max}}}$ has been attained. In severe exercise, the VO$_2$ slow component is larger than is observed during heavy exercise (it is largest at work rates just above the CP if subjects continue to exhaustion). Indeed, the slow component can reach values $>800$ ml min$^{-1}$, which might take the end-exercise gain (i.e., the primary gain, plus the gain of the VO$_2$ slow component term) to $\sim 15$ ml min$^{-1}$ W$^{-1}$ (Fig. 1B). It is surprising, therefore, that this fundamental concept has yet to be acknowledged in any of the classical textbooks of human exercise physiology. Fatigue during severe-intensity exercise, as noted above, appears to have quite different aetiology to that during heavy exercise. Consistent with the related concepts of a maximal accumulated O$_2$ deficit$^{32}$ and CP$^{31}$, task failure in this domain is associated with the exhaustion of a fixed and finite energy store, which has been termed the ‘anaerobic capacity’. The data presented in Fig. 1B for heavy and severe exercise are noteworthy because the range of work rates that these distinctly different VO$_2$ responses represent is surprisingly small. The subject sustained 250 W for a full 30 min without fatigue (heavy exercise), 280 W for 12 min 3 s and 300 W for 6 min 22 s, which neatly illustrates the hyperbolic nature of the power-duration relationship.

The distinction between the severe- and extreme-intensity domains is not based on a physiological landmark but rather on the observed behaviour of VO$_2$: extreme-intensity exercise represents work rates at which exhaustion occurs before the attainment of VO$_2_{\text{max}}$, despite the fact that these work rates typically demand $>120$% VO$_2_{\text{max}}$. Little work has been conducted on constant work rate exercise in the extreme domain and, therefore, the mechanisms of fatigue are not clear. However, it seems that one difference in the aetiology of fatigue during extreme compared to severe exercise is that severe exercise exhausts the anaerobic capacity, whereas extreme exercise does not$^{32}$.

**Prior exercise in the horse: severe-intensity exercise responses**

The vast majority of the world’s most prestigious horse racing events are contested over distances of less than 5000 m, an obvious exception being the English Grand National (6800 m). The average speed measured during this event is 12.5 m s$^{-1}$ (45 km h$^{-1}$)$^{35}$, meaning that the average performance time is $\sim 9$ min. This event time is not dissimilar to that of human athletes performing the 3000 m steeplechase, itself requiring sustained exercise at speeds eliciting VO$_2_{\text{max}}$. It could, therefore, be argued that, for the most part, horse racing events take place at galloping speeds approaching or (more often) exceeding VO$_2_{\text{max}}$.

Because horse racing is performed in the severe- or extreme-intensity domain, it is not surprising to find that the numbers of studies investigating ‘endurance capacity’ in the horse are considerably smaller than those investigating ‘anaerobic capacity’; in human exercise physiology, studies of endurance capacity outnumber the latter two to one, which reflects the typical duration of most human sporting pastimes ($>5$ min). To date, no scientific study in horses investigating prior exercise has investigated the effect on the physiological response to submaximal moderate or heavy exercise: all have so far chosen to consider the effect on severe-intensity exercise responses.

The response of horses to severe exercise has received much empirical attention$^{34-38}$. The study of Rose et al.$^{34}$ was the first to attempt to measure VO$_2_{\text{max}}$, O$_2$ deficit and ‘O2 debt’ (recovery VO$_2$) in the exercising horse using an open-flow gas collection system. This allowed pulmonary gas exchange to be measured at 15 s intervals during treadmill exercise designed to elicit 120% VO$_2_{\text{max}}$. The more recent work of Hinchcliff et al.$^{38}$ measured the maximal accumulated O$_2$ deficit (MAOD) before and after ten weeks of intense training, and noted values of 60–90 ml kg$^{-1}$, with higher values occurring after the training period. Collectively, the results of these studies show that, like its measurement in humans, the MAOD appears to reflect a quantity that is stable (Eaton et al.$^{35}$ showed no differences in MAOD measured in exhaustive bouts ranging from 105 to 125% VO$_2_{\text{max}}$), trainable$^{38}$ but nevertheless sensitive to experimental protocol$^{34}$. By definition, the MAOD reflects the transfer of energy independent of that derived from O$_2$ transfer from the lung to the blood, and is, therefore, composed of energy derived from high-energy phosphate hydrolysis not compensated by mitochondrial energy transfer, glycolytic flux resulting in lactate production and the consumption of myoglobin-bound O$_2$.$^{3,32}$ Of crucial importance in the present context is that exercise tolerance in the severe-intensity domain appears to depend on the rate at which the O$_2$ deficit is accumulated (and the aforementioned energy stores depleted). Because exhaustion ensues when the MAOD has been incurred, exercise tolerance will be improved if the MAOD is increased (training or nutritional supplementation appear to be the only options available to meet this end) or if the VO$_2$ dynamics are speeded, thus reducing some of the early depletion of the anaerobic capacity$^{1}$.

In their study of 13 horses exercising to exhaustion at 115% VO$_2_{\text{max}}$, Tyler et al.$^{36}$ showed that prior exercise (at 50% VO$_2_{\text{max}}$) resulted in a faster increase in VO$_2$ and carbon dioxide output (VCO$_2$), a smaller
MAOD and no change in blood [lactate] during exhaustive exercise. The lactate accumulation measured at 50% VO\(_{2\text{max}}\) in the geldings used in the study of Tyler \textit{et al.}\textsuperscript{36} was 2.8 mM (range 1.0–4.7 mM). Such changes in blood lactate concentration would, in humans at least, reflect exercise performed above the LT (i.e. in the heavy-intensity domain\textsuperscript{3}). Nevertheless, as shown in Fig. 2, the response profiles for VO\(_2\) measured by Tyler \textit{et al.}\textsuperscript{36} demonstrate that prior exercise increased VO\(_2\) at all time points after the transition to severe-intensity exercise. Interestingly, Tyler \textit{et al.}\textsuperscript{36} calculated that prior exercise reduced the magnitude of the oxygen deficit (from 47.3 to 34.7 ml kg\(^{-1}\)), because the tolerable duration of exercise was not altered by prior exercise, despite VO\(_2\) being significantly increased. The results of Tyler \textit{et al.}\textsuperscript{36} are conceptually puzzling, since an intervention that increases VO\(_2\) during exercise performed at the same speed should reduce the rate of anaerobic energy transfer and, therefore, the rate at which these finite energy stores are depleted, but it does not follow that the capacity for anaerobic metabolism (the size of these stores) should also be reduced at the onset of exercise using their warm-up procedures.

In a follow-up to the study of Tyler \textit{et al.}\textsuperscript{36}, McCutcheon \textit{et al.}\textsuperscript{37} investigated the muscle metabolic responses to severe exercise (115% VO\(_{2\text{max}}\)) after ‘light’ and ‘high-intensity’ warm-ups in six Standardbred horses. The warm-up regimes (light, 50% VO\(_{2\text{max}}\) for 10 min; high-intensity, 5 min at 50% VO\(_{2\text{max}}\) followed by 45 s intervals of galloping at 80, 90 and 100% VO\(_{2\text{max}}\), each warm-up followed by 5 min of rest) resulted in a significant increase in pulmonary VO\(_2\) compared to no prior exercise, which was accompanied by improved exercise tolerance in both prior exercise trials (of ~40 and 15% after light and high intensity, respectively). Interestingly, these investigators found no effect of prior exercise on the anaerobic capacity as reflected by the MAOD (values of ~85 ml kg\(^{-1}\)), in stark contrast to the results of Tyler \textit{et al.}\textsuperscript{36}. The extension of run time to fatigue was the most likely cause of the similarities in the O\(_2\) deficit observed by McCutcheon \textit{et al.}\textsuperscript{37}, and these results are consistent with the notion that the tolerance to exercise in the severe-intensity domain is closely related to the depletion of the anaerobic work capacity\textsuperscript{29,39–41} (see below).

The reduction in the blood and muscle lactate responses observed by McCutcheon \textit{et al.}\textsuperscript{37} and the

![Fig. 2 Mean ± SEM oxygen uptake responses to exhaustive exercise requiring 115% VO\(_{2\text{max}}\) with or without prior warm-up exercise in 13 Standardbred geldings. Note the increased aerobic contribution to the exercise after a warm-up, but the attainment of a similar peak VO\(_2\). Data redrawn from Tyler \textit{et al.}\textsuperscript{36}, used with permission](image-url)
demonstration of an increased VO$_2$ response to severe intensity exercise in horses$^{36,37}$ leads to the obvious conclusion that prior exercise somehow elevates the aerobic contribution to subsequent exercise which spares the finite anaerobic energy reserves which are theoretically crucial to sustained high-intensity exercise performance$^{39}$. However, the fidelity of the VO$_2$ data, and in particular the choice of supra-VO$_{2\text{max}}$ work rates, makes the observed changes in the VO$_2$ response very difficult to interpret. Further work by Geor et al.$^{42}$, using an identical experimental protocol to McCutcheon et al.$^{37}$ save for the termination of exercise at 2 min (rather than exhaustion), provided the first direct estimates of VO$_2$ and VCO$_2$ kinetics in the horse following prior moderate- and high-intensity exercise. Although no blood lactate measurements were made, the effects of these protocols on the acid–base status were presumably similar to those of McCutcheon et al.$^{37}$. Geor et al.$^{42}$ observed a significant speeding of VO$_2$ kinetics ($\tau$ reduced from $\sim$23 to 16 s) after both prior moderate- and high-intensity exercise, a speeding that appeared to be correlated with the increase in venous blood temperature. One important issue to address when analysing the VO$_2$ kinetics is to establish whether the alteration in the VO$_2$ responses is related to the changes in the kinetics (i.e., the rate at which the steady state is approached) or to changes in the ‘steady state’ amplitude itself. As shown in Fig. 3, it is difficult, if not impossible, to discriminate between these two possibilities when the VO$_2$ response projects significantly above VO$_{2\text{max}}$$^{43,44}$. At present, all prior exercise studies in the horse share this potential shortcoming. To understand the influence of prior exercise on energy provision further, we must turn our attention to the exercising human subject.

**Effects of prior exercise in humans are intensity dependent**

One notable difference between prior exercise studies in the horse and the human is the exercise domain in which the subsequent (or criterion) bout has taken place. In the horse, exercise has been performed almost exclusively in the upper region of the severe domain (above the VO$_{2\text{max}}$). In humans, the effect of prior exercise has been studied in three of the four intensity domains. The rationale for this ‘broad-brush’ approach is that prior exercise has been used as an intervention to illuminate the issue of whether O$_2$ delivery or O$_2$ utilization determines the VO$_2$ kinetics at the onset of exercise. In the early 1990s, the prevailing view was that O$_2$ delivery might influence the VO$_2$ kinetics above the LT, and the seminal work of Gerbino et al.$^{45}$ was consistent with this view: prior heavy exercise speeded the VO$_2$ kinetics only during sub-
sequent heavy exercise. Neither prior moderate exercise: nor prior heavy exercise altered the moderate exercise on kinetics. Furthermore, prior moderate exercise did not alter the VO$_2$ kinetics during subsequent heavy exercise. In short, exercise that induced a metabolic (lactic) acidosis resulted in an elevated VO$_2$ throughout a subsequent bout of heavy exercise. These responses are shown in Fig. 4A. At the time it was suggested that this reflected an acidosis-mediated improvement in muscle perfusion and, therefore, O$_2$ delivery, which was given strong support when speeded overall VO$_2$ response profiles—during heavy exercise when subjects breathed an O$_2$-enriched inspire—were observed$^{46}$. Further work demonstrated that the speeding of the VO$_2$ kinetics was caused by a reduced VO$_2$ slow component$^{47}$ and not a speeding of the VO$_2$ primary component, since both Gerbino$^{35}$ and MacDonald$^{36}$ interpreted the overall VO$_2$ kinetics without partitioning the VO$_2$ response into its kinetic components.

The effect of including the slow component in the monoexponential characterization of the VO$_2$ response is most clearly seen in Fig. 4B, where the kinetics are markedly speeded (the time constant is smaller) when the fitting window includes the entire exercise responses shown in panel A. When the window is constrained to include only the primary component (first ~2 min of data), there is no difference in the kinetic response (the time constant is unchanged). This has important implications for the interpretation of the VO$_2$ responses in both humans and horses, as both species evidence a VO$_2$ slow component during heavy and severe exercise. Burnley et al.$^{48}$ further demonstrated that, when the influence of an elevated baseline was removed by extending recovery duration from the standard 6 to 12 min, the reduced VO$_2$ slow component was preceded by an increased VO$_2$ primary component amplitude. With two notable exceptions$^{49,50}$, all subsequent studies in humans have shown no change in the primary time constant during heavy exercise preceded by prior heavy exercise$^{41,46,47,51-54}$. As reviewed in Jones et al.$^5$ and Burnley et al.$^6$, these observations have important mechanistic implications. These findings would seem to rule out improved bulk O$_2$ delivery or increased flux through the PDC per se in eliciting the effect of prior exercise, in spite of the fact that both increased O$_2$ delivery$^{55}$ and acetyl group stockpiling$^{56,57}$ have been observed following prior exercise. Crucially, neither increased O$_2$ delivery nor PDC activation have been shown to increase muscle O$_2$ uptake$^{15-19}$.

The observation of speeded primary VO$_2$ kinetics in some$^{49,50}$ but not other$^{51-54}$ studies is difficult to reconcile, although differences in the severity of the prior exercise (Tordi et al.$^{50}$ performed three sprint bouts before heavy exercise, whereas Burnley et al.$^{57}$ performed 6 min of heavy exercise), or the conditioning of the subjects (highly versus. recreationally trained) may have been contributory. In an effort to resolve the issue, Wilkerson et al.$^{41}$ conducted a study that repeated the methods of Tordi et al.$^{50}$, with the exception that the criterion bout of exercise was performed at 105% VO$_{2\text{max}}$. As a result, the data of Wilkerson et al.$^{41}$ were the first to be performed in the same region of the severe domain as those studies performed in the horse, a region in which the authors anticipated an O$_2$ delivery limitation would be present. Wilkerson et al.$^{41}$ demonstrated that the primary VO$_2$ kinetics were unaltered by prior exercise that induced a lactacidosis (baseline [lactate] of ~7.7 mM), but the primary VO$_2$ amplitude was elevated in the second exercise bout, a finding consistent with the work of Burnley et al.$^{37,48,58}$. Interestingly, Wilkerson et al.$^{41}$

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Fig. 4 Oxygen uptake response to two bouts of exercise at 70% of the difference between the LT and VO$_{2\text{max}}$ (severe exercise). Panel A shows the superimposition of the two exercise bouts; panel B shows the estimated time constant from a monoexponential fitting for various fitting windows. Note that the time constants for each bout only begin to differ significantly (i.e., the confidence intervals no longer overlap) when the fitting window is >180 s, indicating that the primary (phase II) τ is not different.
found no change in the calculated O₂ deficit after prior severe exercise, a finding entirely consistent with studies of severe exercise in the horse\textsuperscript{37-39}. Therefore, prior heavy and severe exercise appears to increase the aerobic contribution to subsequent exercise without altering the amount of work that can be performed anaerobically. The demonstration by Burnley \textit{et al.}\textsuperscript{58} that prior heavy exercise also increases the integrated electromyogram in the first 2 min of exercise suggests that increased motor unit recruitment and not, as originally proposed, enhanced O₂ delivery may be primarily responsible for the effect of prior exercise.

**Human and equine responses to prior exercise: implications for exercise tolerance and performance**

In both the horse and the human, it appears that prior exercise results in an enhanced-aerobic contribution to subsequent heavy or severe exercise. Though the data are sparse in horses, in humans it appears that these effects are most likely demonstrated following an exercise-induced acidosis. The mechanism that underpins this effect has been argued to be related to an improved O₂\textsuperscript{50} or metabolic substrate delivery\textsuperscript{56,57}, an increase in motor unit recruitment\textsuperscript{58} or a combination of these effects\textsuperscript{36,41}. Improvements in substrate delivery (O₂ and acetyl groups) undoubtedly occur, but the effect of prior exercise on VO₂ kinetics seems to be an issue of augmented substrate demand (consequent to increased motor unit recruitment) rather than the correction of an initially inadequate substrate supply. The most recent work on this issue is consistent with this proposal: Sahlin \textit{et al.}\textsuperscript{59} demonstrated that although muscle [acetyl carnitine] was 6-fold higher after prior repeated sprint exercise, the primary time constant was unaltered and there was an increased O₂ and PCR cost in the first 3 min of exercise at ~75% VO₂\textsubscript{max}. It is difficult to envisage a mechanism other than enhanced motor unit recruitment explaining results such as these, especially in light of other similar data showing increased O₂ delivery after prior severe exercise\textsuperscript{55}. It is interesting to note that the increase in the primary VO₂ amplitude caused by priming exercise is often of a similar magnitude to the reduction in the VO₂ slow component. This may imply that in the first bout of heavy exercise the initial recruitment of motor units is inadequate to maintain the imposed power output for more than 1-2 min, resulting in additional motor unit recruitment with time and, therefore, a VO₂ slow component develops as these new fibres increase their O₂ consumption. In contrast, at the onset of the second heavy bout a signal or set of signals are present that provide a better indication of the requirements for prolonged work, resulting in greater initial motor unit recruitment and reducing the requirement for further motor unit recruitment with time. As a result, the primary VO₂ amplitude would be increased and the slow component reduced by a like amount. A more detailed exposition of this hypothetical model is presented in Burnley \textit{et al.}\textsuperscript{60}. Irrespective of the precise mechanism, the increased aerobic contribution to high-intensity exercise consequent to prior exercise may be of great benefit to both the equine and human athlete, on whom great demands for O₂ transport and utilization are placed during performance.

When using prior exercise to attempt to improve performance, one must also consider the duration of recovery between prior and subsequent exercise, because prior exercise has the capacity to induce a fatigu ing or potentiating effect on muscle function. Whether fatigue or performance enhancement occurs is dependent on both the character of the prior exercise (intensity and duration) and the recovery duration between the first bout and the second\textsuperscript{60}. However, the effect of recovery duration has not been addressed in the horse, and [only] to a limited extent in the human. One key question, so far unanswered in either species, is how long does the effect of the prior exercise outlined above last? It is clear that the answer is, so far in humans, ‘at least 15 minutes’\textsuperscript{41}, but we can say no more than that at present. Practically and mechanistically, this question is an important one. The observation that the effect of prior exercise persists considerably beyond the time required for VO₂ to return to its baseline may be pivotal in uncovering the mechanism responsible for the effect. In short, any candidate mechanism or agent proposed to be involved in the effect must demonstrate a similarly tardy recovery time course to that of the effect. Furthermore, the possibility that horses recover at a different rate to humans (which seems plausible given the demonstration of faster on- and off-transient VO₂ kinetics) should be investigated, since the practical recommendations for pre-competition ‘warm-up’ may not be transferable between species.

It is likely that exercise tolerance is improved by prior heavy exercise, because the increased primary amplitude has been suggested to be related to an increase in motor unit recruitment at the onset of exercise (see Burnley \textit{et al.}\textsuperscript{60} for review). This is consistent with the earlier work of Astrand and Saltin\textsuperscript{61}, which presents a convincing case for the role of muscle mass recruitment in exercise tolerance: one subject performed cycle exercise to exhaustion at 343 W, demanding a VO₂ of 4.70 L min\textsuperscript{-1}; the actual VO₂ achieved was 4.05 L min\textsuperscript{-1} and the subject reached volitional exhaustion in ~3 min. In a subsequent test, the transfer of 100 W from the legs to the arms led to the attainment of the same absolute VO₂ but the subject sustained exercise for ~6 min (Fig. 5).
These findings suggest that if an intervention results in increasing the muscle mass recruited during heavy and severe exercise, the tolerable duration of the task is likely to be improved. We contend that the findings of an increased muscle electromyogram coupled with an elevated primary VO₂ amplitude, in humans at least, is evidence that prior heavy exercise increases the volume of muscle recruited during subsequent exercise. Below, we review the evidence for the ergogenic effect of warm-up exercise.

Effects of prior exercise on equine and human performance

Depending on both its ‘dose’ and the recovery period following it, prior exercise can be used to cause muscle fatigue or potentiate subsequent muscular performance60. This most probably accounts for the equivocal findings in the literature examining the effect of warm-up exercise on exercise tolerance. Studies have shown improvements37,62, no change36 and decrements41 in time to fatigue in both horses and humans. The CP concept introduced above can be used to understand the findings of the above studies, since time to fatigue occurred in less than 10 min in all cases. If several different constant work rates above the CP are performed to fatigue and plotted as power vs. time to fatigue, a characteristic curve appears (Fig. 6A). This curve has been shown to occur in both humans31 and horses63, and provides a clear framework with which to establish the effects of prior exercise on exercise tolerance, since prior exercise could affect the capacity to perform exercise aerobically (given by the CP) or anaerobically (given by the inherent degree of curvature). These relationships are expanded upon below.

Figure 6 presents two possible effects of prior heavy exercise on the tolerable duration of severe intensity work, using the above CP concept and the data of Jones et al.62. If prior exercise increased the time to exhaustion as a consequence of an increase in the

![Fig. 6 Schematic illustration of the CP concept for human cycle ergometry. In panel A, eight exhaustive exercise bouts are performed to yield the hyperbolic power-duration curve (T_{lim} = AWC/(P² CP)), where T_{lim}, AWC, P and CP are time to exhaustion, anaerobic work capacity, test power output and critical power, respectively. CP represents the asymptote of the power duration curve, and AWC represents its degree of curvature and is synonymous with the maximal O₂ deficit. Panels B and C show the theoretical effect of increases in AWC and CP on the power-duration curve, respectively. The second vertical line in panel C shows the 294 W CP asymptote. See text for further details](image-url)
anaerobic work capacity by ~2 kJ (middle panel) without a change in the CP, then the tolerable duration of the work would be consistently extended at all imposed power outputs by ~13%. In contrast, an increase in the CP of 10 W would result in an increase in time to exhaustion amounting to 167% at 300 W, 32% at 325 W and only 9% at 400 W. At ~370 W, the benefit of increasing CP is identical to the increase in anaerobic work capacity and is smaller at all power outputs thereafter. Importantly, the effect of prior exercise on the power-duration curve for high-intensity exercise has not been rigorously established in horses or humans. Jones et al. found only limited evidence of a change in either parameter of the curve after prior heavy exercise (the 2.7 kJ increase in the anaerobic work capacity did not attain statistical significance). No data are currently available on this issue in horses, and no study has found evidence of an increase in the accumulated O2 deficit after priming exercise; which suggests that no change in the anaerobic work capacity should be expected. At the three work rates studied by Jones et al. (100, 110 and 120% VO2 peak), time to exhaustion was extended by 59, 30 and 29%, respectively. That the relative extension of time to exhaustion was almost twice as large at the lowest work rate studied is actually more consistent with an experimentally induced change in the CP, not the anaerobic work capacity. Nevertheless, consistent with previous work, Jones et al. demonstrated an increased aerobic contribution to the subsequent work after prior heavy exercise. Therefore, prior warm-up exercise appears to increase the aerobic contribution and extend the tolerable duration of subsequent exercise in the severe domain (the very domain characteristic of most equine and many human athletic endeavours).

Human and equine athletic events are won by an individual with the highest average speed over the set course. As a result, it could be argued that experiments investigating the time to exhaustion after prior exercise lack ecological validity. When time to exhaustion tests are compared to time trials, or to constant duration tests where the mean power output is measured (the latter being more representative of athletic performance), the effect of an intervention on mean power output is typically ~16-fold smaller than the effect on time to exhaustion at intensities approximating VO2max. Thus, although Jones et al. found the time to exhaustion after prior heavy exercise was extended by an impressive 30–60%, in reality the effect on performance is likely to be substantially less than this, perhaps ~2–4%. In agreement with this notion, Burnley et al. observed an increase in mean power output during a constant duration cycling test of ~3% after heavy exercise, which elevated blood [lactate] to ~3 mM at the onset of the performance trial. Interestingly, prior moderate exercise (in which the same amount of external work was completed) had an identical effect on performance, but prior sprint work (elevating baseline blood [lactate] to ~6 mM) did not significantly reduce exercise performance, in contrast to what had been expected. At present, therefore, it appears that both prior moderate and prior heavy exercise can improve exercise tolerance and performance in horses and humans.

**Practical recommendations for ‘priming exercise’**

The recent demonstration of improved exercise tolerance following warm-up exercise will naturally lead practitioners to question the best or ‘optimal’ method of priming exercise. Notwithstanding the obvious influence of psychological and biomechanical factors in any warm-up regime in humans, the work so far conducted in both species can be used to make reasonable recommendations for priming the aerobic system at the onset of intense exercise. In human subjects, prior heavy exercise has been shown to be the most consistent means of measurably priming the aerobic system. This effect has been shown to last for at least 12–15 min, and has been shown to result in an extended time to exhaustion following 10 min of recovery. Although prior heavy exercise has been shown to yield a consistent physiological response to subsequent exercise, equal performance enhancements (increased mean power output) can be gained by performing isocaloric moderate or heavy prior exercise 10 min before the criterion task, with the latter ‘warm-up’ regime inducing a mild lactacidosis at the onset of exercise.

In the exercising horse, there is evidence that the priming effect can be also be achieved with both moderate- and heavy-intensity priming exercise. The work of McCutcheon et al. and Geor et al. suggests that provided the duration of prior exercise is sufficient to elevate muscle and core temperature, the horse will gain a physiological and performance benefit. Whether an additional benefit would be gained from sustained heavy exercise leading to marked differences in ‘primed’ red cell concentrations (and thus O2 transport) is not clear. Since all of the aforementioned studies used a 5 min recovery period between the priming and the performance, little can be said of the optimal recovery duration in the horse. In a practical sense, the method of priming may very much depend on the subsequent activity to be performed. For example, the trainers and jockeys of racing Thoroughbreds would be well advised to consider establishing the most effective means of priming given the need to parade and then gallop to the starting point of the race. A reasonable question...
Effects of ‘warm-up’ exercise in horses and humans would be: can the canter/gallop to the start be used as a high-intensity priming bout, given the short (and presumably variable) recovery duration under these conditions?

Summary

Most major equine sporting events and many prestigious human athletic events demand near-maximal rates of aerobic metabolism. The balance between oxidative and non-oxidative mechanisms of energy transfer can be markedly influenced by the performance of prior exercise in both horses and humans. In both species, the aerobic contribution to subsequent exercise is increased by prior exercise: in humans, this increase is mediated by an increased primary VO$_2$ amplitude (without a change in the rapidity with which it is reached) and a reduced VO$_2$ slow component in response to heavy or severe exercise; in horses the kinetics of the response to severe exercise are speeded, and these alterations occur whether the intensity of the prior work is in the moderate or heavy domain. In contrast, the VO$_2$ response to heavy or severe exercise in humans appears to be altered only following an exercise-induced acidosis. Of greatest practical interest is that work rate tolerance (and by extension exercise performance) in the severe intensity domain is enhanced in both species following prior exercise. Improvements in exercise tolerance and performance have been observed in horses and humans, suggesting that optimizing the warm-up regime in both species could significantly improve subsequent performance.

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References


Effects of 'warm-up' exercise in horses and humans


