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Review

Lactate-driven equine conditioning programmes

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ABSTRACT

Equine conditioning programmes are rarely driven by science. Indeed, the scientific literature on conditioning responses often refers to conventional technique rather than physiological driving parameters. This, alongside poor classification of conditioning protocols, has reduced the possibility of comparative data analysis. Recent interest into lactate-driven conditioning programmes has driven this review which provides a summary of equine protocols used to date and their responses. Key areas identified for further standardisation and/or investigation include (1) the treadmill acclimation protocol and markers of its efficiency, (2) the design and frequency of standardised exercise tests used, and (3) the interpretation of data for the development of effective and realistic conditioning programmes.

Introduction

Equine conditioning programmes typically favour sport specific conventional techniques (von Wittke et al., 1994; Evans, 2000) with too little influence from scientific knowledge. However, it has been suggested that conventional technique is not always adequate and that the performance of, for example, racehorses (von Wittke et al., 1994) and elite event horses (Serrano et al., 2002) could be improved and injury rates reduced (Naylor, 2009) by the use of more scientific protocols.

Even within the scientific literature, training intensity has frequently been classified by conventional technique, velocity and/or gait (see, for example, Leleu and Cotrel, 2006; Lindner et al., 2006). Programmes defined by a percentage of maximal oxygen uptake (VO_{2max}), a percentage of maximal heart rate (HR_{max}) or a blood lactate concentration are often limited in their practical application due to unrealistic workloads (Naylor, 2009) or poorly defined protocols with respect to work intensity – velocity, distance, exercise duration and treadmill incline in particular (Werkmann et al., 1996). A comparative analysis of the literature, as well as the development of science-based conditioning programmes and their practical application are therefore restricted.

In human endurance sports, VO_{2-La4} (oxygen uptake at a blood lactate concentration of 4 mmol/L) has been considered the best determinant of fitness (Takeshima and Tanaka, 1995), with blood lactate profiles being used as indicators of training intensity (Auvinet, 1996). Indeed, V_{La4} (velocity at a blood lactate concentration of 4 mmol/L) and other velocity–lactate relationships have been successfully used to define human (e.g., Heck et al., 1985) and equine (e.g., Evans et al., 1995) conditioning protocols. Research into the specific effects of exercise intensity, frequency and duration on equine lactate-driven conditioning (LDC) programmes have amplified over the last 15 years (see, Evans et al., 1995; Werkmann et al., 1996; Trilk et al., 2002; Lindner et al., 2009b) and forms the basis of this review.

Blood lactate concentration

During exercise, an exponential relationship occurs between blood lactate concentration and velocity (Seeherman and Morris, 1990) where initial values are relatively low and stable followed by a rapid increase that peaks within 15 min of recovery, with timescale dependent upon workload (Harris and Snow, 1988; Marlin et al., 1990; Lindner et al., 1992).

The deflection of the lactate–velocity curve represents the start of an imbalance between lactate production and removal/metabolism (Donovan and Brooks, 1983; Mazzeo et al., 1986) and so has been referred to as the onset of blood lactate accumulation (OBLA). Less accurately, another term, the anaerobic threshold, representing a switch between predominantly aerobic and anaerobic metabolism has also been used (Wasserman et al., 1973). Although the switch of metabolic preference is a major contributor to the rapid increase in blood lactate levels it is unlikely to be the sole cause. Additional influential factors include an increase in lactate efflux from muscle to blood (Hyyppä and Pösö, 1998), reduced lactate removal via renal and splanchnic blood (Donovan and Brooks, 1983; MacRae et al., 1992) and altered lactate buffering relating to the muscle carnosine content (Sewell et al., 1991).

The deflection within the lactate–velocity curve has been seen to occur in horses (Wilson et al., 1983) and humans (Foster and Cotter, 2006) at a blood lactate concentration of 2–4 mmol/L,

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leading to the frequent use of an arbitrary unit of 4 mmol/L for comparative purposes. However, those values were often determined during short duration standardised exercise tests (SETs) that did not take into account the maximum lactate steady state (MLSS).

The MLSS, represents the highest workload that can be maintained (for 25–40 min at a constant exercise intensity) before lactate accumulation occurs, and has been referred to as the aerobic–anaerobic lactate threshold. In humans MLSS has been considered the optimal workload for improving endurance performance (Mader et al., 1976, cited in Lindner, 2010) but this has yet to be confirmed.

Blood lactate concentrations produced at MLSS do not correlate with values determined at the lactate–velocity deflection point in running or cycling humans (Aunola and Rusko, 1992) and so reliance on 4 mmol/L blood lactate as a marker of the anaerobic threshold is unrealistic in exercise testing (Stegmann et al., 1981; Beneke and von Duvillard, 1996). However, the anaerobic threshold can be identified with good reproducibility (Aunola and Rusko, 1984) and it has been confirmed as a valid estimate of MLSS in cyclists (Aunola and Rusko, 1992).

Due to individual differences in the MLSS value resulting from varying lactate kinetics (Stegmann et al., 1981), the term *individual anaerobic threshold* has been introduced. In humans, more aerobically fit individuals had lower MLSS values (Stegmann et al., 1981) as did those with a higher percentage of highly oxidative muscle fibre types (Aunola and Rusko, 1992). The sporting discipline also had an impact upon MLSS due to the work mode (Krűger et al., 1990) and in particular the driving force of the dominant muscle (Beneke and von Duvillard, 1996) and possibly its mass (Beneke and von Duvillard, 1996; Beneke et al., 2001). Blood lactate concentrations at MLSS of 3.1, 5.4 and 6.6 mmol/L were found for rowing, cycling and speed skating, respectively (Beneke and von Duvillard, 1996).

Lindner (2010) investigated the concept of individual anaerobic thresholds in horses using human guidelines of no more than 1 mmol/L change in blood lactate between the 5th and 25th min of an SET at a constant speed (Heck et al., 1985). Crucially, he found that horses showed a marked difference in values obtained at the anaerobic threshold and at MLSS, as suggested by Bourgela et al. (1991) and identified in humans (e.g., Aunola and Rusko, 1992). Reports in the literature where V_{La4} and MLSS are relatively closely related (such as Heck et al. (1985) for running humans) are most likely due to SET design and/or the individual's lactate kinetics.

A surprisingly low V_{Lax} (velocity at a specific blood lactate concentration of *x* mmol/L) of 1.5 mmol/L (i.e. $V_{La1.5}$) was found to best represent the aerobic–anaerobic lactate threshold of horses (Lindner, 2010), which equated to a mean blood lactate concentration of 4.90 ± 0.14 mmol/L post 40 min constant exercise. These results complement the work of Gondim et al. (2007) who used the lactate minimum speed protocol (adapted from Tegtbur et al. (1993)) and found that $V_{La<2}$ could be maintained for up to 33 min, representing MLSS. The low equine MLSS could be explained from human data where a greater proportion of total skeletal muscle use during exercise resulted in lower MLSS values (Beneke et al., 2001); interestingly, Thoroughbred skeletal muscle mass equates to approximately 55% of total bodyweight (BW), compared to 30–40% in other species (Gunn, 1987).

The design of an SET in itself has been shown to affect blood lactate concentration (Lindner et al., 1993) and VO_2 (Rose et al., 1990) among other parameters, and so the use of a single SET protocol of good repeatability has been suggested for use in exercise prescription (Lindner, 2007). The impact of step duration upon the identification of V_{La4} in particular was highlighted by Köster in 1996 (cited by Lindner (2007)); V_{La4} was identified at 10.5, 8.2 and 7.5 m/s for step durations of 1, 3 and 5 min duration, respectively (Lindner, 2007) during an incremental SET on a 6% treadmill incline, with a starting velocity of 6 m/s and increments of 0.5 m/s until a blood lactate concentration of >4 mmol/L was achieved. Thus SET recommendations for V_{La4} determination include (1) the use of at least four steps (3 pre and 1 post 4 mmol/L), (2) 5 min step durations (so decreasing the maximal speed required during the SET and injury risk), (3) a variable initial velocity and increment magnitude to suit the subjects, physiological measures required and equipment used (Couroucé-Melblanc et al., 2010), and (4) flexibility to allow additional steps later on in the conditioning programme when the fitness levels are improved (Lindner, 2007).

Lactate related measures that could be misconstrued from equine literature of varying SET design include: (1) V_{La4}, which increases with conditioning with the magnitude of change being dependent on the workload intensity, duration and frequency (von Wittke et al., 1994; Eaton et al., 1999); (2) HR_4 (the heart rate at 4 mmol/L blood lactate), which increases as the individual is able to work to a higher intensity aerobically (Castejón et al., 1994); (3) VO_{2-La4}, which increased with endurance running performance (Takeshima and Tanaka, 1995), and (4) cardiac capacity (V_{200}), which increases with conditioning (Milne et al., 1976; Wilson et al., 1983; Couroucé, 1999) and can be seen to occur close to V_{La4}, dependent upon the choice of SET protocol (Persson, 1983; Wilson et al., 1983; Couroucé, 1999).

Equine lactate-driven conditioning protocols

Treadmill acclimation

Energy expenditure during treadmill exercise differs quantitatively from that of field conditions (Persson, 1983; Valette et al., 1992; Barrey et al., 1993a) due to physiological, locomotory and environmental influences. The magnitude of some effects can be decreased during the process of acclimation yet few investigations have analysed the process of acclimation itself (Seeherman, 1991; King et al., 1995). Because of this, standard protocols and markers of acclimation efficiency are not available for use.

The majority of LDC research states that acclimation to the treadmill was completed prior to determination of V_{Lax} or the post exercise blood lactate concentration (La_{post}), yet few authors give specifics (Table 1) and only one mentions other equipment, namely, a respiratory gas mask. Secondly, none of the authors provide details on the effectiveness of the process so horses could only be classified as accepting the equipment used. The impact and extent of acclimation upon physiological variables during the SETs remains unknown. Work efficiency, affected by anticipation, apprehension and excitement (Persson, 1967; Snow et al., 1992), and further influenced by variable pre-conditioning workloads (Table 1) would therefore be expected to impact upon individual V_{Lax} values among other parameters.

Standardised exercise tests

The majority of the LDC programmes have been defined by lactate-guided exercise (Table 2), so incremental SETs were run to identify V_{Lax} from the lactate–velocity relationship using exponential regression equations (Valette et al., 1991; Werkmann et al., 1996). Blood sampling took place during short term rest periods (30–60 s) between each increment and lactate was identified via portable lactate analysers (e.g., Accusport) to ensure the required SET intensity was met. Five minute step durations were used by all authors except D'Angelis et al. (2005), with the treadmill incline, warm up protocol, first step velocity and increment magnitude varying to suit the subject, physiological measures required

Table 1
Pre-conditioning exercise history and protocols of acclimation to the equine treadmill.

Pre-conditioning workload	Treadmill acclimation protocol	Authors
8 months paddock rest with 30 days adaptation to handling		D'Angelis et al. (2005)
6 months light work followed by 4 months paddock rest		Eaton et al. (1999)
⩾4 months paddock rest		Evans et al. (1995)
	2 months pre-conditioning	Ferlazzo et al. (1996)
3 months paddock rest		Gottlieb-Vedi et al. (1995)
12 weeks box rest with one 50 m walk each week	Walk, trot and high speed work wearing a respiratory gas mask during the first 2 weeks of box rest, then a single 10 min walk and trot session in the week before conditioning	Hinchcliff et al. (2002)
Traditional race training or ⩽3 months stud break	4–6 weeks of an unknown protocol	Lindner et al. (2009a)
Untrained	3 weeks prior to conditioning	Rivero et al. (2002)
2 months pre-conditioning (unknown intensity)	First month, canter 2–4 min/day every second day	Rivero et al. (2006)
	Second month, ⩽8 m/s, 6% incline, ⩽25 min/day, every second day	Rivero et al. (2007)
No exercise undertaken – unknown duration	Walk and trot 2 weeks prior to conditioning	Trilk et al. (2002)
Untrained with sand paddock access for 3 h/day	3 weeks prior to conditioning	Vervuert et al. (2002a)
	10 min at 2 m/s and 2 min at 4, 6 and 8 m/s three times a week	Vervuert et al. (2002b)
	First month, trained to gallop	Werkmann et al. (1996)
	Second month, ⩽8 m/s, 6% incline, ⩽25 min/day, every second day	

Key; empty cells represent details that were not mentioned by the respective authors.

Table 2
Standardised exercise tests (SETs) used to identify lactate-driven conditioning (LDC) velocities (via V_{Lax} or La_{post}) and/or assess the responses to conditioning.

SET design	Treadmill incline (%)	Warm up (m/s)	1st step velocity (m/s)	Step increments (m/s)	Rest duration mid increments (s)	End of test marker	Authors
Lactate-guided exercise; incremental SET used pre LDC to identify V_{Lax}	–	–	6	0.5	60	La > 4	Ferlazzo et al. (1996)
	–	10 min at 1.5–4					Rivero et al. (2006, 2007)
	6	10 min at 1.5–4					Werkmann et al. (1996)
	17	5 min at 2.2 5 min at 2.8 0% incline	2.8	0.3	60	La > 4	Gansen et al. (1997, 1999) Lindner et al. (1997)
		5 min at 2 10 min at 4	5	1	60	La_{10}	Rivero et al. (2002) Vervuert et al. (2002a) Vervuert et al. (2002b)
			2.8	0.3	60	La > 4	Lindner et al. (2009b)
Lactate-guided exercise; incremental SET used pre and during LDC to identify V_{Lax}	6	5 min at 1.7	4–4.5	0.5	30	La > 4	Trilk et al., 2002
		5 min at 3.4 30 s rest 0% incline					
	6		1 min at 2	2		8 m/s	D'Angelis et al. (2005)
	6	5 min at 1.7 5 min at 4 0% incline	4	0.5	60	La > 4	Lindner et al. (2009a)
La_{post} driven exercise; Intensity altered to meet required La_{post} using data from SET and conditioning bouts	6.25		2 min at 4–5	1	0	HR_{200+} or 8 m/s if wearing gas mask	Gottlieb-Vedi et al. (1995)
	4°		90 s at 4	1 from 7 m/s	0	Vo_2 plateau or fatigue	Hinchcliff et al. (2002)
Lactate-guided exercise and La_{post} driven exercise	–		3 min at 4	2–8 m/s, then 1	–	Fatigue	Eaton et al. (1999)
	10				60		Evans et al. (1995)

Key; empty cells or – represent details that were not provided by the respective authors; V_{Lax}, velocity at a chosen blood lactate concentration; La_{post}, post exercise blood lactate concentration; La > 4, blood lactate concentration of >4 mmol/L; La_{10}, blood lactate concentration at 10 m/s; HR_{200+}, heart rate of >200 bpm; VO_2, oxygen uptake. Unless otherwise stated, warm up was completed at the same treadmill incline as the test and step durations were of 5 min.

and equipment used. Typically one SET was completed at the start of the LDC programme to identify V_{Lax} with only three authors re-identifying V_{Lax} at frequent intervals during the LDC programme (Table 2).

An alternate approach to lactate-guided exercise can be defined as La_{post} – driven exercise (Table 2). This protocol uses La_{post} concentrations, regularly taken from an SET or exercise bout, to fine tune workload to a required La_{post} value. The SET bout durations were shorter than those used for lactate-guided exercise (in order to obtain physiological measures close to fatigue such as VO_2), and La_{post} was assessed as frequently as every second exercise bout in order to meet the required workload.

Evans et al. (1995) and Eaton et al. (1999) used a mixture of both lactate-guided and La_{post} driven exercise to design their LDC protocol. Although their emphasis was more towards lactate-guided exercise they found that the frequency and intensity of SETs affected their results. The impact of SET design and frequency, alongside the use of variable detraining periods (between different

LDC protocols investigated in succession) requires further investigation and must be considered in both data interpretation and exercise prescription.

Conditioning programmes

LDC protocols are typically performed on a treadmill at a predetermined V_{Lax} (Table 3) with exercise bouts 3–5 times a week for a total of 3–12 weeks. A treadmill incline of 17% was used with Haflingers (Gansen et al., 1999; Lindner et al., 2009b) due to a low treadmill maximal speed. Most other protocols used Thoroughbreds or Standardbreds with an incline of between 0% and 6%, with only the older Thoroughbreds (5.8 ± 0.2 years as opposed to a mean of 2 years) using a 10% incline (Evans et al., 1995). Indeed, lactate responses to exercise (reflecting energy expenditure) have been shown to differ with incline (Valette et al., 1992; Barrey et al., 1993b; Galloux et al., 1993; Couroucé et al., 2000) and breed (Castejón et al., 1994).

Details of velocity, distance and duration of exercise bouts were often incomplete (Table 3) hence simple use of the equation velocity (m/s) = distance (m)/time (s) provided further clarity, with most exercise bouts lasting ⩽25 min. Typical velocities of between 3 and 8 m/s were used, unless short term fast bouts were integrated for which a maximum velocity of 11.5 m/s was achieved. It must not, however, be ignored that LDC exercise bouts are normally completed on a treadmill with inclines of up to 17% in some instances. Thus, for practical application in the field, required velocities would differ significantly, possibly making the workload unrealistic (Rivero et al., 2007; Naylor, 2009).

Equine lactate-driven conditioning responses

Aerobic/anaerobic capacity

At race speeds, approximately 70% of a horse's immediate energy needs are met via aerobic metabolism (Eaton et al., 1995) with the contribution increasing further with longer duration bouts. The aerobic capacity of an individual can be measured during an SET, determining VO_{2max}, however anaerobic metabolism can only be estimated in vivo from the maximal accumulated oxygen deficit (Eaton et al., 1995); expressed as oxygen equivalents.

Table 3
Conditioning protocols used to meet the required blood lactate concentrations (V_{LAx}).

V_{Lax}	Velocity (m/s)	Distance (m)	Time (min)	Treadmill incline (%)	Frequency	Duration (weeks)	Authors
1.5	3.1	8370	45	17	EOD	6	Gansen et al. (1997, 1999) Lindner et al. (2009b)
<2	4.0 ± 0.7	1600 week 1–2, 2400 week 3–5, 3200 week 6–7, 4000 week 8–9	6.7, 10, 13.3, 16.7		5/week	9	Eaton et al. (1999)[a]
2	5.1–5.7	230–257	45	6	EOD	6	Trilk et al. (2002)[a]
2.5	6.5 ± 0.28	1950, 5850, 9750	5, 15 or 25	6	EOD	3	Ferlazzo et al. (1996)
			5, 15 or 25	6	EOD	3	Rivero et al. (2006, 2007) Werkmann et al. (1996)
	3.4	9180	45	17	EOD	6	Gansen et al. (1997, 1999) Lindner et al. (2009b)
2–4	3.8 ± 0.7	1600 week 1–2, 2400 week 3–4, SET week 5, 3000 week 6–7, 3600 week 8–9	7, 10.5 – 13.2, 15.8	10	5/week	9	Evans et al. (1995)[a]
80% of 4	3.3 4.2 4.2	10000 week 1–4 15000 week 5–8 20000 week 9–12	50 60 80	6	EOD	12	D'Angelis et al. (2005)
4	8.0 ± 0.48	2400, 7200, 12000	5, 15 or 25	6	EOD	3	Ferlazzo et al. (1996)
	4 (slow) 7–11.5 (fast)	6 km week 1, 10 km week 12	2 2	0 week1–4 1–5° week 5–12	EOD	12	Gottlieb-Vedi et al. (1995)[a]
			Intervals 5, 15 or 25	6	EOD	3	Rivero et al. (2006, 2007) Werkmann et al. (1996)
	3.8	5700	25	17	EOD	6	Gansen et al. (1997, 1999) Lindner et al. (2009b)
4–8	8.0 ± 0.6	1600 week 1–2, 2400 week 3–4 SET week 5 3000 week 6–7, 3600 week 8–9	3.3,5 6.25, 7.5	10	5/week	9	Evans et al. (1995)[a]
	8.0 ± 0.6	1600 week 1–2, 2400 week 3–5 3200 week6–7, 4000 week 8–9	3.3, 5 6.7, 8.3		5/week	9	Eaton et al. (1999)[a]
⩾7	8.7 ± 0.2 (peak)	1044 × 2 bouts	1.99 ± 0.17		4–5/week	10	Hinchcliff et al. (2002)[a]
10	7.9 ± 0.4	2370	5	6	EOD	6	Lindner et al. (2009a)
2.5 and 4+	4.7–5.6 (slow) 6.2–7.2 → 10.3–11.4 (fast)	27810	60–90 (slow) 15 (fast)	– 0	EOD and alternate fast–slow	5 6	Rivero et al. (2002) Vervuert et al. (2002a)
				0		6	Vervuert et al. (2002b)

Key; SET, standardised exercise test; V_{Lax}, velocity at a chosen blood lactate concentration; italics, data calculated (speed = distance/time); empty cells, details that were not provided by the respective authors; →, increasing speed during exercise bout; EOD, every other day.

[a] Authors that reassessed V_{Lax} during the conditioning programme.

Typically, 10–23% increases in VO_{2max} have been reported within 6–9 weeks of conditioning in horses (Evans and Rose, 1988; Knight et al., 1991), within which the LDC increases of 17–20% fall. Consistent improvements in aerobic capacity occurred despite variable protocols, such as V_{La4-8} (Evans et al., 1995) or $V_{La>8}$ (Eaton et al., 1999), or V_{La2-4} and $V_{La>7}$ (Hinchcliff et al., 2002). Related aerobic variables were rarely stated. In one report, VO_{2peak} increased (11%), yet blood lactate at VO_{2peak} was unchanged post LDC at $V_{La\geqslant 7}$ for 10 weeks (Hinchcliff et al., 2002). In another, VO_{2-La4} increased (27%) with V_{La2-4} or V_{La4-8} LDC for 9 weeks (Evans et al., 1995).

Only Hinchcliff et al. (2002) investigated the impact of LDC ($V_{La\geqslant 7}$ for 10 weeks) on anaerobic capacity, where maximal accumulated oxygen deficit was found to increase by 27% (post LDC oxygen demand of 386 ± 42 mL/kg and oxygen uptake of 304 ± 37 mL/kg).

Blood lactate

LDC was found to increase V_{La4} by 2–35% (Table 4) where integration of at least some high intensity exercise (V_{La4-8} and $V_{La\geqslant 7}$) and generally shorter bout durations (<20 min) produced the greatest adaptation, with the exception of Lindner et al. (2009a). Interestingly, the programmes that produced an increase of >30% were the only programmes that exercised the horses 4–5 times a week instead of every other day. The larger changes could also be related to the total LDC duration where greater adaptations were often seen following the longer trials. Again Lindner et al. (2009a) was the exception to this trend; two 5 min bouts at V_{La10} every other day for 6 weeks did not significantly alter V_{La4} (2% increase, Table 4). Over-reaching, however, may have contributed to this contradictory response as post exercise creatine kinase increases were slightly greater following LDC, as were post exercise plasma urea concentrations, indicative of catabolism (Lindner et al., 2009a).

Trilk et al. (2002), assessed the effect of LDC upon V_{La2}, a work intensity that Gondim et al. (2007) and Lindner (2010) indicate is close to MLSS in horses. A 12% increase in V_{La2} occurred post 6 weeks LDC at V_{La2}, slightly less than the 17% increase in V_{La4} (Trilk et al., 2002). While Gottlieb-Vedi et al. (1995) found blood lactate concentrations at a heart rate of 200 beats per min (bpm) (V_{200}) decreased by 28%, post 12 weeks LDC at V_{La4}.

Alternative lactate measures, La_x, representing blood lactate concentrations at a chosen velocity were also investigated (Table 4). Blood lactate concentrations at 8, 9 or 10 m/s typically decreased with LDC by 16–50%, with the greatest response being seen by Evans et al. (1995) reporting an LDC at V_{La2-4} or V_{La4-8} for 9 weeks.

The La_{post} typically peaks within 0–15 min (Harris and Snow, 1988; Marlin et al., 1990; Lindner et al., 1992) with an immediate decline occurring after lower intensity work bouts. LDC decreased La_{post} by 3–30% (Table 4) with the greatest response identified 30 min post $V_{La\geqslant 7}$ exercise (Hinchcliff et al., 2002). Not all authors stated the exact time scale for blood lactate determination, so restricting comparative analysis; nevertheless a number of observations are worth noting.

Data from Vervuert et al. (2002b) suggested that MLSS was probably achieved as blood lactate concentrations remained low (<4 mmol/L) with 60–90 min exercise at V_{La2}. This was unaffected by LDC which suggested that maintenance rather than adaptation may have occurred. Pre-conditioning data from Werkmann et al. (1996) suggested that the horses may have been exercised above their MLSS (La_{post} > 4 mmol/L) while equivalent data from Lindner et al. (2009b) initially looked closer to MLSS. La_{post} values ranged from 1.60 to 6.70, 1.35 to 6.85 and 2.15 to 8.45 mmol/L following work of 45 min duration at V_{Lax} of 1.5 and 2.5 mmol/L or 25 min duration at 4 mmol/L, respectively (Lindner et al., 2009b). This highlights the extent of individual differences and stresses the need for considering SET design in practical exercise prescription. Consideration of the individual's diet and the glycaemic index in particular (Lacombe et al., 2006) are also paramount in data interpretation.

As is seen in humans (Gaesser and Poole, 1988), La_{post} decreased rapidly at the start of conditioning (V_{2-4} or V_{4-8}) after which the rate of change decreased (Evans et al., 1995). This supports the need for periodical blood lactate testing during recovery to

Table 4
Effects of lactate-driven conditioning programmes upon blood lactate related measures.

Variable	Conditioning V_{Lax} (mmol/L)	Exercise duration (min)	Pre-conditioning value	Post conditioning value	% change with (*x* weeks) conditioning	Authors
V_{La2}	2	45	5.1 ± 0.3 m/s	5.7 ± 0.3 m/s	12% ↑ (6)	Trilk et al. (2002)
V_{La4}	<2 or 4–8	7–17 or 3–8	7.0 ± 0.5 m/s	9.2 ± 0.2 m/s	31% ↑ (9)	Eaton et al. (1999)
	2–4 or 4–8	7–16 or 3–8	7.0 ± 0.5 m/s	9.2 ± 0.2 m/s	31% ↑ (9)	Evans et al. (1995)
	4	Repeats of 2	6.6 ± 0.3 m/s	7.8 ± 0.3 m/s	18% ↑ (12)	Gottlieb-Vedi et al. (1995)
	⩾7	Repeats of 2	6.3 ± 0.4 m/s	8.5 ± 0.2 m/s	35% ↑ (10)	Hinchcliff et al. (2002)
	10	2 × 5	6.23 ± 0.41 m/s	6.34 ± 0.53 m/s	2% ↑ (6)	Lindner et al. (2009a)
	1.5	45	–	–	7% ↑	Lindner et al. (2009b)
	2.5	45	–	–	5% ↑ (6)	
	4	25	–	–	2% ↑	
	2	45	5.8 ± 0.3 m/s	6.8 ± 0.4 m/s	17% ↑ (6)	Trilk et al. (2002)
	2.5–4	5–25	7.8 ± 0.1 m/s	7.7 ± 0.2 m/s	–	Werkmann et al. (1996)
La_8	4	Repeats of 2	6.7 ± 0.8 mmol/L	4.4 ± 0.7 mmol/L	32% ↓ (12)	Gottlieb-Vedi et al. (1995)
La_9	2–4 or 4–8	7–16 or 3–8	8.0 ± 1.0 mmol/L	3.9 ± 0.3 mmol/L	50% ↓ (9)	Evans et al. (1995)
La_{10}	Alt. 2.5 and 4+	60–90 and 15	2.2 ± 0.2 mmol/L	2.2 ± 0.4 mmol/L	–	Vervuert et al. (2002b)
			12.3 ± 1.5 mmol/L	10.3 ± 1.7 mmol/L	16% ↓ (6)	
La_{200}	4	Repeats of 2	4.6 ± 0.4 mmol/L	3.3 ± 0.4 mmol/L	28% ↓ (12)	Gottlieb-Vedi et al. (1995)
La_{post}	2–4	7–16	–	0.3–1.9 mmol/L	–	Evans et al. (1995)
	1.5	45	3.32 ± 1.03 mmol/L	3.34 ± 1.82 mmol/L	–	Lindner et al. (2009b)
	2.5	45	4.63 ± 2.12 mmol/L	4.04 ± 0.93 mmol/L	13% ↓ (6)	
	4	25	4.93 ± 1.24 mmol/L	4.77 ± 2.49 mmol/L	3% ↓	
	Alt. 2.5 and 4+	60–90 and 15	2.2 ± 0.2 mmol/L	2.2 ± 0.4 mmol/L	–	Vervuert et al. (2002b)
			12.3 ± 1.5 mmol/L	10.3 ± 1.7 mmol/L	16% ↓ (6)	
	4	25	5.7 mmol/L	4.2 mmol/L	25% ↓ (3)	Werkmann et al. (1996)
$La_{30minpost}$	⩾7	Repeats of 2	17.5 mmol/L	12.5 mmol/L	30% ↓ (10)	Hinchcliff et al. (2002)

Key; –, details that were not provided by the respective authors or no change; V_{Lax}, velocity at a chosen blood lactate concentration; La_x, blood lactate concentration at a chosen velocity (m/s); La_{post}, post exercise blood lactate concentration; Alt., alternate; ↑, increase; ↓, decrease.

facilitate individual comparisons and confirms the need for exercise prescription relative to the individual.

Muscle composition

Skeletal muscle fibre types differ in their contractile properties with relative proportions varying both within and between muscles (Snow and Valberg, 1994). Typically highly oxidative and glycolytic fibres predominate within the deep and superficial aspects of locomotory muscles, respectively (Rivero et al., 1993); type I fibres are solely oxidative, IIA and IIAX act as intermediates (providing energy via both means) and IIX are solely glycolytic (Quiroz-Rothe and Rivero, 2001).

Gluteus medius muscle fibre types, identified via myosin heavy chain (MHC) expression, were affected by LDC of $V_{La2.5–4}$, for 25 min, every other day, for 3 weeks (Rivero et al., 2006). Superficially, the percentage of IIA fibres and the IIA:IIX ratio increased (11% and 21%, respectively) with conditioning at V_{La4} for 25 min whilst deep muscle increased in IIA (12–15%), decreased in IIX (14–16%) and increased in the IIA:IIX ratio (35–37%) following $V_{La2.5}$ or V_{La4} conditioning for 25 min (Rivero et al., 2006). Work of shorter durations, 5 or 15 min, failed to match those changes.

Rivero et al. (2007) found no difference between superficial and deep muscle fibre adaptations ($V_{2.5–4}$ 5–25 min, every other day, for 3 weeks). LDC at V_{La4}, for 25 min produced the greatest response in MHC expression with an increase in IIA fibres (12%) and a decrease in IIX (12%). The IIA:IIX ratio increased with conditioning intensity and duration. Muscle fibre types identified via immunocytochemistry supported the responses of type IIX fibres and IIA:IIX ratio, but not IIA fibres which were unaffected by LDC (Rivero et al., 2007). Cross sectional fibre area (CSA) was also identified by Rivero et al. (2007) with the greatest changes following 15 min LDC at V_{La4}; IIAX increased by 32%, IIX by 19%. Differences were also seen with exercise for 5 or 25 min at this intensity whilst work at $V_{La2.5}$ failed to show a response.

Longer duration conditioning bouts of 50–80 min (increasing over 90 days of LDC) at 80% of V_{La4}, every other day (with one day a week speed play), increased the relative cross sectional area of type I fibres whilst the percentage of IIX fibres decreased (D'Angelis et al., 2005) – a response that was not enhanced by the supplementation of creatine monohydrate (75 g/day).

Interval style LDC at V_{La4}, every other day for 12 weeks, had no effect upon fibre type proportions (Gottlieb-Vedi et al., 1995) as identified by myosin ATPase activity staining, and both muscle fibre types (determined by immunohistochemical and electrophoretic methods), and CSA were unaffected by 5 weeks of alternate $V_{La2.5}$ and V_{La4+} LDC every other day (Rivero et al., 2002). However, with concomitant L-carnitine supplementation (known to enhance fatty acid oxidation (Chilibeck et al., 1998), decrease glycogen depletion rates (Lancha et al., 1995) and reduce plasma lactate accumulation (Vecchiet et al., 1990)), IIA fibres increased in number (35%) while type I fibres decreased in both number and CSA by 23% and 24%, respectively (Rivero et al., 2002); thus development of IIA at the expense of type I occurred which is typical of high intensity conditioning (Lovell and Rose, 1991). Importantly, all LDC responses were partially or fully reversed within 5 weeks of de-conditioning (Rivero et al., 2002).

The number of capillaries in contact with fibres (capillary:fibre ratio) increased with V_{La4}, 25 min LDC, in all fibre types (Rivero et al., 2007) whilst 5 min conditioning at V_{La4} or $V_{La2.5}$ increased capillaries surrounding IIAX fibres, and 25 min $V_{La2.5}$ increased those around IIX fibres. In contrast, the capillary:fibre ratio was unaffected by 5 weeks alternate $V_{La2.5}$ and V_{La4+} LDC (Rivero et al., 2002) despite an increase in capillary density (27%) while concomitant L-carnitine supplementation increased the capillary:fibre ratio and capillary density by 40% and 35%, respectively.

Such changes in muscular composition support the use of long duration low intensity exercise for muscular improvements in aerobic capacity (Miyata et al., 1999) providing the additional benefit of reduced musculoskeletal injury risk; alternate $V_{La2.5}$ and V_{La4+} LDC, every other day for 6 weeks, altered bone turnover (formation). Osteocalcin, a marker of bone formation, decreased at rest and increased post low but not high intensity exercise (Vervuert et al., 2002a) whilst the cross-linked C-telopeptide of type I collagen (ICTP), a marker of bone matrix degradation, was unaffected. Exercise induced changes in calcium homeostasis (increased intact parathyroid hormone, decreased ionised calcium and increased ionised phosphorous) were unaffected by LDC (Vervuert et al., 2002b).

Metabolic fuels

Equine skeletal muscle has a greater capacity for glycogen storage than many other species (Snow and Valberg, 1994), contributing to their exceptional exercise capacity. Glycogen content within the superficial/glycolytic region of the gluteus medius muscle was unaffected by 6 weeks conditioning at $V_{La1.5}$–V_{La4} (Gansen et al., 1997, 1999) whilst glycogen content within the deep/oxidative region adapted. Responses were only seen during de-conditioning; a 47% and 48% increase post $V_{La1.5}$ and $V_{La2.5}$ (45 min) LDC, respectively. This response was first seen 9 days post conditioning and was maintained for 5 weeks. V_{La4} for 25 min failed to adapt muscle glycogen at this depth (Gansen et al., 1999).

Rivero et al. (2002) found that glycogen content within individual myofibres (identified via periodic acid-Schiff staining) was unaffected by alternate $V_{La2.5}$ and V_{La4+} LDC over 5 weeks whereas in the presence of L-carnitine an increase of 11% was seen. High intensity LDC ($V_{La\geqslant 7}$) for 10 weeks increased resting and post exercise muscle glycogen concentrations by 17% and 22%, respectively (Hinchcliff et al., 2002), whilst the rate and amount of glycogen use during exercise was unaffected. Correspondingly, the change in muscle lactate concentration with exercise decreased by 59% and the rate of lactate accumulation decreased by 68% (Hinchcliff et al., 2002), whilst La_{peak} was unaffected, as reported previously in humans (Medbø and Burgers, 1990) and horses (Evans et al., 1995). This suggested that LDC enhances aerobic metabolism of carbohydrates and/or lactate clearance/metabolism, although whole muscle buffering capacity was unaltered over the duration of the trial. The impact upon specific fibre types remains to be investigated.

Fuels suspended within blood plasma have also been investigated. Glucose levels decreased during each warm-up, increased during each SET increment and yet were unaffected by V_{La2} LDC for 45 min over 6 weeks (Trilk et al., 2002). Free fatty acid (ffa) concentrations at rest, and the percentage increase of ffa and alanine with exercise were also unaffected by conditioning at $V_{La1.5}$, $V_{La2.5}$ or V_{La4}, for 25–45 min every other day for 6 weeks (Fuhrmann et al., 1997; Lindner et al., 2009b). Resting and post exercise plasma protein concentrations, however, decreased following $V_{La\geqslant 7}$ LDC (Hinchcliff et al., 2002) with only resting values decreasing (7%) with alternate $V_{La2.5}$ and V_{La4} LDC (Vervuert et al., 2002a). Those results may partially be explained via the lack of an impact of LDC at $V_{La2.5}$ or V_{La4} (5, 15 or 25 min, every other day, for 3 weeks) upon total and free plasma iodothyronines (Ferlazzo et al., 1996), known stimulators of carbohydrate and lipid metabolism alongside protein synthesis.

Metabolic enzymes

Enzymatic markers of oxidative metabolism include citrate synthase (CS), succinic dehydrogenase (SDH) and 3-hydroxyacyl CoA dehydrogenase (HAD). Citrate synthase is involved in the entry of

acetyl co A into the citric acid cycle and provides a marker of oxidative capacity and the presence of intact mitochondria. Succinic dehydrogenase facilitates the production of fumarate within the citric acid cycle and plays a further role as complex II within the electron transfer chain, whilst HAD acts as an oxidoreductase within β-oxidation (Stryer, 1995).

The LDC at $V_{La<2}$ or $V_{La4–8}$, 5 days a week, for 9 weeks, had no effect upon HAD activity, but did increase CS activity (Eaton et al., 1999), to a slightly greater extent at the higher intensities. SDH was unaffected by alternate $V_{La2.5}$ and V_{La4+} conditioning over 5 weeks (Rivero et al., 2002) whilst 25 min of V_{La4} conditioning, every other day for 3 weeks, increased SDH activity in type I (11%), IIA (12%) and IIAX (20%) fibres and $V_{La2.5}$ LDC for 25 min increased SDH activity in IIA fibres by 14% (Rivero et al., 2007).

Markers of glycolytic metabolism include glycerol-3-phosphate dehydrogenase (GPDH), an indirect marker of glycolytic potential (Peter et al., 1972), which correlated well to further glycolytic enzymes, namely phosphofructokinase, enolase and lactate dehydrogenase (LDH), and played a role within the NADH shuttle.

GPDH activity was unaffected by alternate $V_{La2.5}$ and V_{La4+} LDC over 5 weeks (Rivero et al., 2002) unless horses were supplemented with L-carnitine, which increased activity by 34% – a response that was reversed by 5 weeks de-conditioning. Activity was also found to increase within IIX fibres by 14% following LDC at V_{La4}, for 25 min (Rivero et al., 2007), while interval style LDC at V_{La4}, every other day for 12 weeks, had no effect upon the activity of various oxidative, glycolytic or ATPase regenerating enzymes (Gottlieb-Vedi et al., 1995), namely, CS, HAD, LDH, hexokinase, myokinase or creatine phosphokinase (CPK).

Creatine phosphokinase is involved in the production of phosphocreatine and adenosine diphosphate, helps restore the energy charge of a cell, yet levels were unaffected by exercise or LDC at V_{La2} for 45 min, three times a week for 6 weeks (Trilk et al., 2002). CPK has also been used as a marker of over-training in horses (Bruin et al., 1994) which was thought to occur following two 5 min bouts of V_{La10} exercise every other day for 6 weeks (Lindner et al., 2009a).

Finally, indicators of work efficiency, power output (at 4 mmol/L or a heart rate of 200 bpm; W_{La4}, W_{200}), and stride length (at 8 m/s or 200 bpm; SL_8, SL_{200}) were investigated by Gottlieb-Vedi et al. (1995). Interval style LDC at V_{La4} every other day for 12 weeks increased W_{La4} (15%) but did not change W_{200}. Similarly, SL_8 was unaffected by LDC whilst SL_{200} increased (8%). Responses however, could partially have been due to continued acclimation to the equine treadmill during the LDC trial.

Conclusions

Research to date on LDC has failed to identify responses in blood glucose, ffa or albumin, HAD, iodothyronines, intact parathyroid hormone, blood calcium, phosphorous, pH and ICTP. Capillary density, blood protein and osteocalcin levels, however, have been shown to adapt in those few studies that examined these variables. LDC can produce contradicting responses depending upon the LDC protocol (Table 5), thus highlighting the complexity of conditioning programme design. The design of both the SET and the conditioning programme are paramount. In an attempt to standardise scientific protocol for data comparison and practical application, the use of mandatory variables has been suggested for the classification of workload and conditioning protocols, namely, work mode, speed (and method of determination), duration/distance, frequency, repetition number of exercise steps, ground surface, HR (and equipment), % VO_{2max} and method of determination, VO_2 (and measurement protocol), lactate concentration, blood acid–base balance, PCV (and method of determination) (Rogers et al., 2007).

Further consideration of the relationship between SET design/intensity and that of the conditioning programme is needed; in particular we need to know the impact of (1) variable treadmill acclimation protocols and their efficiency, (2) SET design (and the variable starting velocity, increment magnitude and treadmill incline in particular), (3) the short and long term effects of SET frequency and intensity upon conditioning responses and injury risk, and (4) the extent to which treadmill trials can be applied

Table 5
Visual representation of variable lactate-driven conditioning responses.

Authors	V_{Lax}	Min	A	B	C	D	E	F	G	H	I
Rivero et al. (2006)	2.5	5									
	2.5	15									
	2.5	25									
	4	5									
	4	15									
	4	25									
Rivero et al. (2007)	2.5	5									
	2.5	15									
	2.5	25									
	4	5									
	4	15									
	4	25									
Gottlieb-Vedi et al. (1995)	4	2									
Rivero et al. (2002)	2.5/4+	60–90/15									
	With L-carnitine										
D'Angelis et al. (2005)	80% of 4	50–80									
	With creatine										
Trilk et al. (2002)	2	45									
	4	45									
Lindner et al. (2009a)	10	2 × 5									
Gansen et al. (1999)	1.5	45									
	2.5	45									
	4	25									
Hinchcliff et al. (2002)	⩾7	2									
Eaton et al. (1999)	<2	6–18									
	4–8	3–8									

Key; V_{Lax}, velocity for a chosen blood lactate concentration; min, exercise bout duration in minutes; black cell, conditioning response; grey cell, no conditioning response; A, fibre types; B, fibre cross sectional area; C, capillary:fibre ratio; D, muscle glycogen; E, succinic dehydrogenase; F, citrate synthase; G, glycerol-3-phosphate dehydrogenase; H, creatine phosphokinase; I, blood volumes including plasma volume, packed cell volume and red blood cell volume.

effectively to the practical application of conditioning programmes in field conditions.

Conflict of interest statement

The author of this paper has no financial or personal relationship with other people or organisations that could inappropriately influence and bias the content of the paper.

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