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REPORT DOCUMENTATION PAGE

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1. REPORT NUMBER: M-3/82	2. GOVT ACCESSION NO.: AD-A106 542
3. RECIPIENT'S CATALOG NUMBER	
4. TITLE (and Subtitle): Lactate accumulation in muscle and blood during submaximal exercise	5. TYPE OF REPORT & PERIOD COVERED
	6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s): P.A. Tesch, W.L. Daniels and D.S. Sharp	8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS: US Army Research Institute of Environmental Medicine, Natick, MA 01760	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
11. CONTROLLING OFFICE NAME AND ADDRESS: US Army Medical Research and Development Command, Ft. Detrick, Frederick, MD 21701	12. REPORT DATE: 21 September 1981
	13. NUMBER OF PAGES: 22 pages
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office)	15. SECURITY CLASS. (of this report)
	15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

DISTRIBUTION OF THIS DOCUMENT IS UNLIMITED

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

NA

NOV 2 1981

A

18. SUPPLEMENTARY NOTES

To be published in Acta Physiol. Scand.

4 mmol/l

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

capillary density and frequency, cycle exercise, fast and slow twitch fibers

(OBLA sub W)

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

Muscle and blood lactate concentration was studied in 10 healthy males during cycling exercise. For each subject the exercise intensity corresponding to a blood lactate concentration of 4 mmol·l^{-1} ($OBLA_W$) was assessed by a step-wise increased exercise intensity protocol. In a second series of experiments the same protocol was performed but exercise was terminated at $OBLA_W$ and a muscle biopsy for subsequent analysis of lactate concentration was obtained from m.vastus lateralis. Biopsies were also taken at rest for histochemical determination of fiber type composition and capillary supply.

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The exercise intensity, which corresponded to OBLA, averaged 159 (117-216) W, equal to 65 (range 55-84) % of VO_2 max, and was found to be correlated to the capillary frequency of the exercising muscle ($r=0.83$, $p < 0.01$). Muscle lactate concentration averaged 6.9 (range 2.1-12.6) mmol/kg w.w. The muscle to blood lactate gradient as well as the change in blood lactate concentration (prior to and 1 min post exercise) were correlated to muscle lactate concentration ($r=0.89$, $p < 0.001$ and $r=0.71$, $p < 0.05$). It is concluded that great individual variations in the muscle/blood lactate gradient do exist during submaximal steady-state exercise, performed at a certain blood lactate level.

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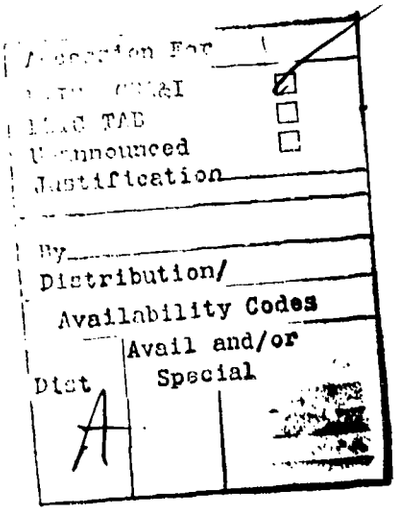

Lactate accumulation in muscle and blood during submaximal exercise

Key words: capillary density and frequency, cycle exercise, fast and slow twitch fibers

Short title: Lactate in muscle and blood

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Acknowledgement

The technical assistance of Robert P. Mello, Elizabeth Heath and Nancy L. Seaver is gratefully acknowledged. The study was partly supported by grants from Karolinska Institute's Research Funds. The views and opinions contained in this report are those of the authors and are not to be construed as official or as reflecting the views of the Department of the US Army.

TESCH, P.A., DANIELS, W.L. & SHARP, D.S.: Lactate accumulation in muscle and blood during submaximal exercise. Acta Physiol Scand , . Received . ISSN - . Exercise Physiology Division, U.S. Army Research Institute of Environmental Medicine, Natick, MA 01760, USA.

Muscle and blood lactate concentration was studied in 10 healthy males during cycling exercise. For each subject the exercise intensity corresponding to a blood lactate concentration of 4 mmol·l^{-1} ($OBLA_W$) was assessed by a step-wise increased exercise intensity protocol. In a second series of experiments the same protocol was performed but exercise was terminated at $OBLA_W$ and a muscle biopsy for subsequent analysis of lactate concentration was obtained from m.vastus lateralis. Biopsies were also taken at rest for histochemical determination of fiber type composition and capillary supply.

The exercise intensity, which corresponded to $OBLA_W$, averaged 159 (117-216)W, equal to 65 (range 55-84)% of $\dot{V}_{O2max}$, and was found to be correlated to the capillary frequency of the exercising muscle ($r=0.83$, $p<0.01$). Muscle lactate concentration averaged 6.9 (range 2.1-12.6) mmol·kg^{-1} w.w. The muscle to blood lactate gradient as well as the change in blood lactate concentration (prior to and 1 min post exercise) were correlated to muscle lactate concentration ($r=0.89$, $p<0.001$ and $r=0.71$, $p<0.05$). It is concluded that great individual variations in the muscle/blood lactate gradient do exist during submaximal steady-state exercise, performed at a certain blood lactate level.

INTRODUCTION

The rate of muscle lactate accumulation during so called "steady-state" exercise is primarily a function of oxygen supply, relative number of muscle fibers recruited and that muscle's potential for formation, release, uptake and oxidation of lactate (cf. Jorfeldt 1970, Karlsson 1971, Karlsson 1979). Primarily these factors determine the rate of lactate accumulation in blood. Other factors to be considered are blood flow, blood volume and the potential for other tissues to metabolize lactate (Cori & Cori 1929, Ekblom et al. 1976, Folkow & Halicka 1967, Klausen et al. 1974). An increased rate of blood lactate accumulation is suggested to reflect a shift from predominantly aerobic to more anaerobic metabolism with a concomitantly enhanced glycogenolytic activity (cf. Skinner & McLellan 1980). During steady-state exercise conditions this increased lactate formation has been reported to occur at exercise intensities in the order of 60-70% of maximal oxygen uptake (Karlsson 1971, Knuttgen & Saltin 1972).

It is generally agreed that the main muscle fiber type to be recruited below this level is the slow twitch (ST or type I) fiber whereas progressively more fast twitch (FT or type II) fibers are brought into play as the energy demand increases (cf. Burke & Edgerton 1975). The latter fiber type has a greater potential for lactate formation than the ST fiber, as indicated by a higher activity of lactate dehydrogenase (LDH) and a more muscle specific LDH isozyme pattern (Sjödin 1976). In concert, a lactate concentration gradient between fiber types was recently demonstrated following maximal non steady-state exercise (Tesch et al. 1978, Tesch 1980). Hence, lactate was shown

to accumulate at a higher rate in individuals with muscles rich in FT fibers than in those with predominantly ST fibers in their exercising muscles. The significance of muscle fiber type distribution and the related metabolic properties for blood lactate accumulation during submaximal exercise has also been documented. Thus, in both cycling (Ivy et al. 1980, Tesch et al. 1981) and running (Sjödin & Jacobs 1981, Jacobs 1981) experiments the exercise intensity, at which the initial increase in lactate accumulation in blood occurs was positively related to the percentage of ST fibers in the exercising muscle. Moreover, the capacity for pyruvate oxidation (Ivy et al. 1980), the balance between oxidative and glycolytic enzyme activities (Sjödin et al. 1981, Jacobs 1981) as well as the capillary density (Tesch et al. 1981, Sjödin & Jacobs 1981, Sjödin et al. 1981) of the musculature were found to influence this relationship.

In light of these findings it was of interest to study muscle lactate concentration at i given blood lactate level, assumed to represent onset of blood lactate accumulation, during "steady-state" conditions.

Material and methods

Subjects were 10 healthy males accustomed to physical exercise. Their age, height and weight were (mean±SD) 29 (±6) yrs, 178 (±4) cm and 79 (±9) kg, respectively. After being informed of the purpose of the study and the possible discomfort associated with the experiments, written consent was given. Maximal oxygen uptake ($\dot{V}_{O_{2max}}$) was measured during cycling (60 rpm) on a Monark ergometer and defined according to the "leveling off"

criterion. Onset of blood lactate accumulation (OBLA) was determined using the experimental protocol described by Tesch et al. (1981) based on procedures introduced elsewhere (Mader et al. 1976, Jacobs 1981). Briefly, continuous cycling exercise was performed at a pedaling frequency of 60 rpm and with 30 W increments every fourth minute until near voluntary exhaustion. Oxygen consumption, respiratory parameters and heart rate were monitored during the final 30 seconds at each exercise intensity. Simultaneously, blood samples were collected from an antecubital vein through an indwelling catheter for subsequent spectrophotometric analysis of lactate concentration (Sigma Technical Bulletin 826, 1968). OBLA was defined as the exercise intensity corresponding to a lactate concentration of 4 $mmol \cdot l^{-1}$ blood. Within a week the test-subjects were re-examined using the same protocol, but exercise was stopped after 4 min at the intensity, calculated to correspond to OBLA. In addition to the blood sample taken just prior to termination of exercise another blood sample was obtained 1 min later for lactate determination. A muscle biopsy (Bergström 1962) was obtained from m.vastus lateralis at cessation of exercise with the subject still sitting on the cycle ergometer. The tissue sample was immediately frozen in liquid nitrogen for subsequent analysis of lactate concentration in freeze dried dissected out muscle fiber fragments (Karlsson 1971, Tesch 1980). Muscle biopsies were also taken at rest for determination of muscle fiber type distribution (% FT fibers, % FT area) mean fiber area (Tesch 1980) as well as capillary density ($cap \cdot mm^{-2}$) and frequency ($cap \cdot fib^{-1}$) according to Andersen & Henriksson 1977.

RESULTS

Maximal oxygen uptake averaged (±SD) 3.84 (±0.51) $l \cdot min^{-1}$. Mean (±SD) values for percentage of and relative area occupied by FT fibers, mean fiber area, capillary density ($cap \cdot mm^{-2}$) and capillary frequency ($cap \cdot fib^{-1}$) were 50 (±14)%, 55 (±15)%, 57 (±13) $\cdot 100 \mu m^2$, 297 (±84) $cap \cdot mm^{-2}$ and 1.62 (±0.34) $cap \cdot fib^{-1}$.

The exercise intensity, calculated to correspond to OBLA was 159 (±34)W or equivalent to 65 (±9)% of maximal oxygen uptake. Measured oxygen consumption and pulmonary ventilation during exercise at the calculated OBLA were 2.31 (±0.43) $l \cdot min^{-1}$ and 56.6 (±14.8) $l \cdot min^{-1}$, respectively. The predicted value for oxygen consumption at OBLA, based on the initial cycling task, was slightly (2.44 $l \cdot min^{-1}$) but significantly ($p<0.05$) higher whereas the predicted ventilation was the same 53.9 (±11.8) $l \cdot min^{-1}$. Muscle lactate concentration averaged 6.9 (range 2.1-12.6) $mmol \cdot kg^{-1}$ w.w. (Table I). Mean values for blood lactate concentration immediately prior to and 1 min after cessation of exercise were 3.0 (range 2.1-3.6) and 3.5 (range 2.6-4.3) $mmol \cdot l^{-1}$. Muscle lactate was not significantly correlated to blood lactate concentrations following exercise nor to any of the histochemical variables studied. However, positive relationships were established between muscle lactate concentration and the muscle to blood lactate gradient ($r=0.89$, $p<0.001$, Fig. 1) and the 1 min increase in blood lactate concentration ($r=0.71$, $p<0.05$, Fig. 2, Table II), respectively. Thus, only a small further increase in blood lactate concentration occurred following exercise in subjects with low muscle lactates whereas a more exaggerated increase was demonstrated in those who exhibited high muscle lactate levels. The exer-

intensity corresponding to ($OBLA_W$), as well as the change in blood lactate concentration were related to capillary frequency ($r=0.83$, $p<0.01$, Fig. 3 and $r=-0.76$, $p<0.01$, Fig. 4, Table II).

DISCUSSION

The present study describes a wide variation in the muscle/ /blood lactate gradient among individuals working at a submaximal exercise intensity corresponding to a lactate concentration of approximately 4 $mmol \cdot l^{-1}$ blood. The mean lactate concentration (6.9 $mmol \cdot kg^{-1}$ w.w), is in good agreement with other reports of muscle lactate levels at similar relative exercise intensities (Karlsson 1971, Linnarsson et al. 1974). Since 8 out of 10 subjects demonstrated muscle lactate values above 4 $mmol \cdot kg^{-1}$ w.w, it was obvious that muscle and blood lactate concentrations did not parallell each other.

Although no relationship was established between absolute muscle and blood lactate levels, individuals exhibiting high muscle lactate concentrations demonstrated higher muscle to blood lactate gradients, which confirms findings reported for contracting in situ dog muscle (Graham et al. 1976). These subjects also possessed greater increases in blood lactate concentration following exercise than subjects with low muscle lactate levels. This relationship can be interpreted as indicating that translocation hindrances of lactate from muscle exists even at low muscle lactate concentrations as suggested by Jorfeldt et al. (1978). They demonstrated that the rate of lactate release from muscle during cycling exercise increases linearly with exercise intensity, blood flow and arterio-venous O_2 difference up to muscle lactate concentrations of 4-5 $mmol \cdot kg^{-1}$ w.w. A further increase in exercise intensity did not result in a greater rate of release. Thus lactate began to accumulate in muscle at a higher rate than in blood in spite of a sufficient oxygen supply as indicated by a further linear increase in blood flow. The marked elevation in muscle lactate

concentration occurred at a relative exercise intensity corresponding to approximately 70% of maximal oxygen uptake, which is in concert with the present and previous findings (Karlsson 1971, Knuttgen and Saltin 1972). Furthermore, when steady-state exercise at approximately 70% of $\dot{V}_{O_{2max}}$ is maintained following muscle lactate accumulation below 4 $mmol \cdot kg^{-1}$ w.w a reduction in lactate may occur both in muscle and venous blood, while prolonged exercise slightly above this level evokes a reverse pattern (Jorfeldt et al. 1978, Karlsson 1980). A high muscle to blood lactate gradient will favor the efflux of lactate (Harris et al. 1981) and is reflected in that individuals possessing high muscle lactate levels exhibited the greatest rise in blood lactate concentration at cessation of exercise.

In contrast to what has been demonstrated for maximal short term exercise (Tesch et al. 1978, Tesch 1980), but in concert with findings reported in connection with submaximal exercise (Jacobs 1981) muscle lactate accumulation was not related to the proportion of FT fibers in the exercising muscle. Very likely, due to the experimental design used here, muscle fibers with the greatest potential for lactate formation (FT fibers) are far from maximally recruited irrespective of individual variations in fiber type composition. During progressively increased exercise intensity more and more fibers are brought into play. At low intensities ST fibers are exclusively recruited (Gollnick et al. 1973, 1974), small amounts of lactate are produced (Karlsson 1971, Gollnick et al. 1973), which can be oxidized whithin the muscle (Jorfeldt 1970, Essén et al. 1975), or released to the blood stream. As exercise intensity is further increased an augmented portion of FT fibers is involved

(Gollnick et al. 1974) concomitant to an accelerated rate of lactate formation and accumulation (Karlsson 1971) and reduced lactate release (Jorfeldt 1970, Jorfeldt et al. 1978).

Neither fiber type composition nor capillary supply could explain the variation in muscle/blood lactate gradients. As mentioned above blood samples obtained at termination of exercise revealed that in individuals exhibiting high muscle lactate levels an exaggerated rise in lactate took place indicating an increased release after exercise. It is tempting to suggest that the capillary density or frequency is of importance for elimination of lactate from the muscle. Since a negative relationship was found between these two variables and the change in blood lactate concentration at termination of exercise it can be speculated that the magnitude of the vascular bed is decisive for rate of lactate disappearance during exercise. Thus a well developed capillary network provides the muscle with the potential for an efficient elimination of lactate and muscle concentration can be maintained fairly constant at low levels. A greater lactate release from leg muscles with a predominance of ST fibers as compared to muscles with a high percentage of FT fibers has been implicated following repeated maximal contractions (Tesch 1980), and submaximal cycling exercise (Bonen et al. 1978, Graham et al. 1978). This is consistent with the observed differences in blood flow (Frisk-Holmberg et al. 1981) and capillary supply (Andersen 1975, Brodal et al. 1977) with regard to fiber type composition. Uptake of lactate may also be influenced by variations in fiber type composition and capillary supply. Since a larger blood flow, as demonstrated in individuals possessing high percentage of ST fibers, implies a larger

open capillary surface, more favorable conditions for both uptake and release of lactate could probably be achieved by these individuals. According to Jorfeldt (1970) muscles least prone to produce lactate are those with the greatest potential to take up lactate, i.e. ST muscle fibers. Moreover, the quantity of carbons originating from lactate incorporated into CO_2 is greater for red (ST) than white (FT) muscles (Bär & Blanchaer 1965), indicating higher potentials for ST fibers to catabolize lactate.

The number of capillaries surrounding each muscle fiber is highly correlated to the mitochondrial content of the fiber (Brodal et al. 1977). Hence it can be concluded that capillary density or frequency reflects both the oxidative metabolic potential of the muscle and other properties associated with the transport of oxygen, metabolites and substrates.

The large variation in terms of training status among the subjects studied (i.e. they were all accustomed to heavy physical exercise but type of training varied considerably), likely influenced the lactate accumulation pattern observed. Support for this was recently demonstrated when a team of homogeneously trained soccer players was examined. A more narrow range was present for $\dot{V}_{O_{2max}}$, exercise intensity corresponding to OBLA as well as histochemical variables studied (Kaiser & Tesch, 1981) than in the present study. Muscle lactate concentration at OBLA ranged 1.4-6.9 $mmol \cdot kg^{-1}$ w.w. Thus, besides the possible factors already discussed, which may determine variations in the muscle/blood lactate gradient, the activity of some of the key enzymes reflecting metabolic regulation and known to be influenced by physical training should be considered

(Holloszy 1973, Henriksson & Reitman 1976, Sjödin 1976, Spryna-rova et al. 1980).

In conclusion, the present study clearly shows that blood lactate concentration does not covary with muscle lactate accumulation under exercise conditions generally termed "steady state". Thus despite the possible use of blood lactate accumulation as a predictor for physical performance (Mader et al. 1976, Farrell et al. 1979, Sjödin & Jacobs 1981) great individual muscle/blood lactate gradients may be present. It is very likely that multiple factors governed by both environmental and genetic influences contribute to such variations.

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TABLE I

Subj.	muscle lactate concentration, $mmol \cdot kg^{-1}$ w.w.	exercise intensity watt	exercise intensity % $\dot{V}_{O_2max}$	% FT area	$cap \cdot mm^{-2}$	$cap \cdot fib^{-1}$
I.	2.1	213	72	59	489	2.24
II.	2.3	141	57	45	335	1.75
III.	5.7	150	58	64	218	1.60
IV.	6.3	117	65	61	172	1.12
V.	6.6	193	63	47	333	1.77
VI.	7.1	150	74	78	265	1.55
VII.	7.7	144	58	53	307	1.57
VIII.	8.4	216	84	21	276	1.95
IX.	10.2	147	55	64	296	1.16
X.	12.6	126	59	57	276	1.52
mean	6.9	159	65	55	297	1.62
SD	±3.2	±34	±9	±15	±84	±0.34

TABLE II

	Δ blood lactate	OBLA, w	OBLA % $\dot{V}_{O_{2max}}$	% FT area	cap·mm^{-2}	cap·fib^{-1}
muscle lactate	.71*	-.39	-.11	.01	-.45	-.52
Δ blood lactate		-.70*	-.46	.30	-.45	-.76**
OBLA, w			.68*	-.52	.63*	.83**
OBLA, % $\dot{V}_{O_{2max}}$				-.36	.13	.51
% FT area					-.44	.30
cap·mm^{-2}						.75**

TABLE I. Individual values for muscle lactate concentration, exercise intensity corresponding to onset of blood lactate accumulation (OBLA) and histochemical parameters.

TABLE II. Correlation matrix for muscle lactate concentration, increase (Δ) in blood lactate concentration during the initial phase of recovery from exercise, exercise intensity and histochemical variables. Correlation coefficients and levels of significance ($p<0.05$=*, $p<0.01$=**) are denoted.

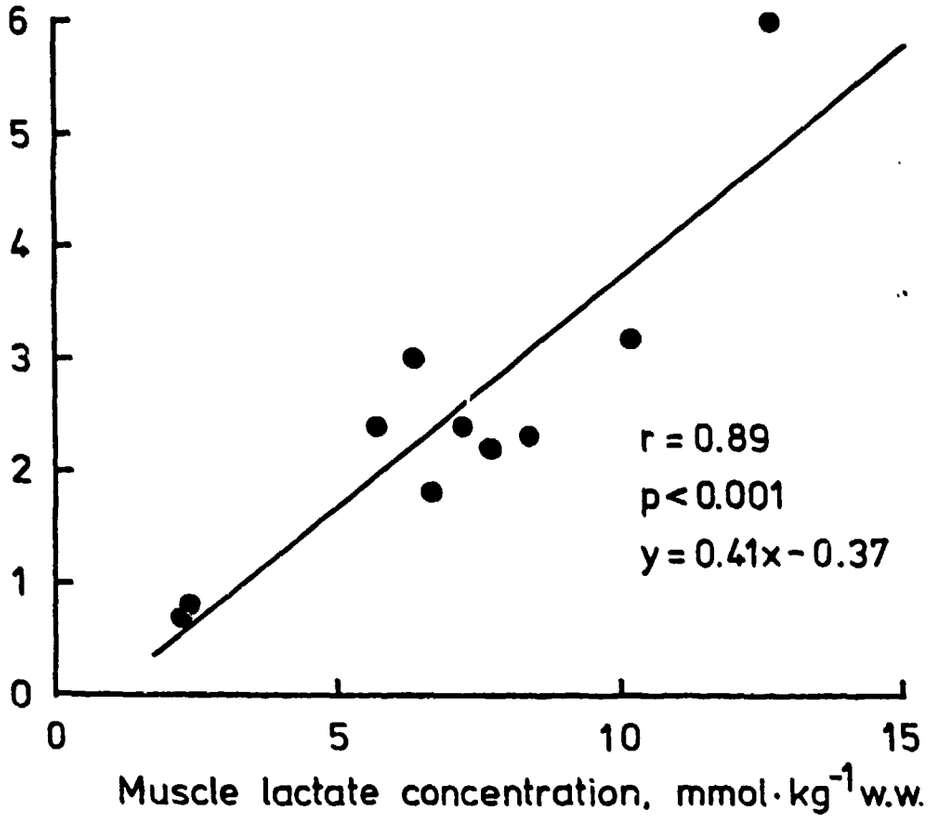
FIGURE LEGENDS

Fig. 1. The relationship of muscle/blood lactate concentration gradient to muscle lactate concentration.

Fig. 2. The relationship of increase in blood lactate concentration during 1 min after cessation of exercise to muscle lactate concentration.

Fig. 3. The relationship of the exercise intensity (W) corresponding to OBLA, to capillary frequency expressed as $cap \cdot fib^{-1}$.

Fig. 4. The relationship of increase in blood lactate concentration to capillary frequency.


6
5
4
3
2
1
0
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10
15
r = 0.89
p < 0.001
y = 0.41x - 0.37
Muscle lactate concentration, mmol·kg⁻¹ w.w.

Fig 1

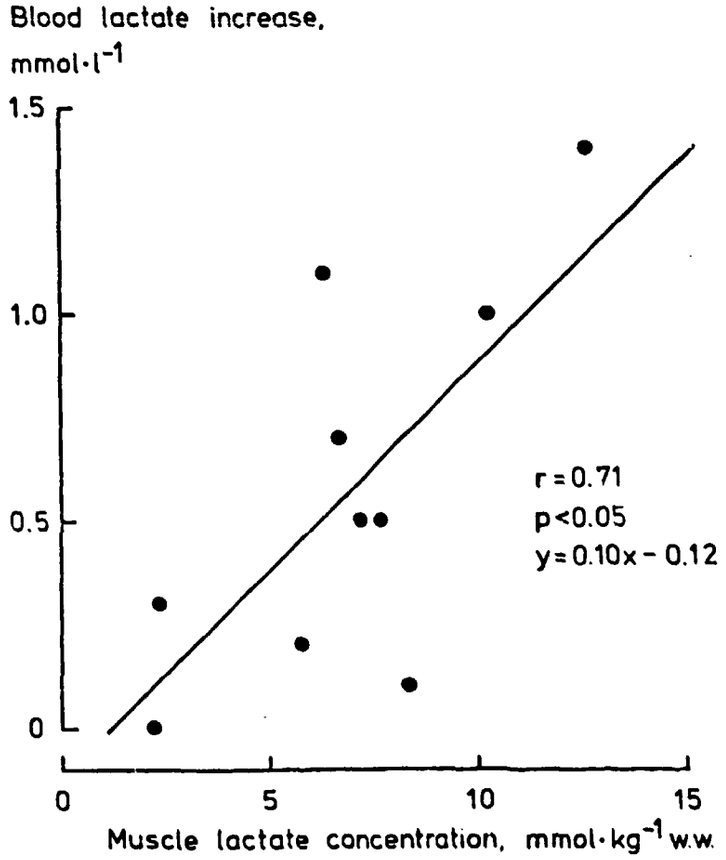
Blood lactate increase,
mmol·l⁻¹
1.5
1.0
0.5
0
0
5
10
15
r = 0.71
p < 0.05
y = 0.10x - 0.12
Muscle lactate concentration, mmol·kg⁻¹ w.w.

Fig 2

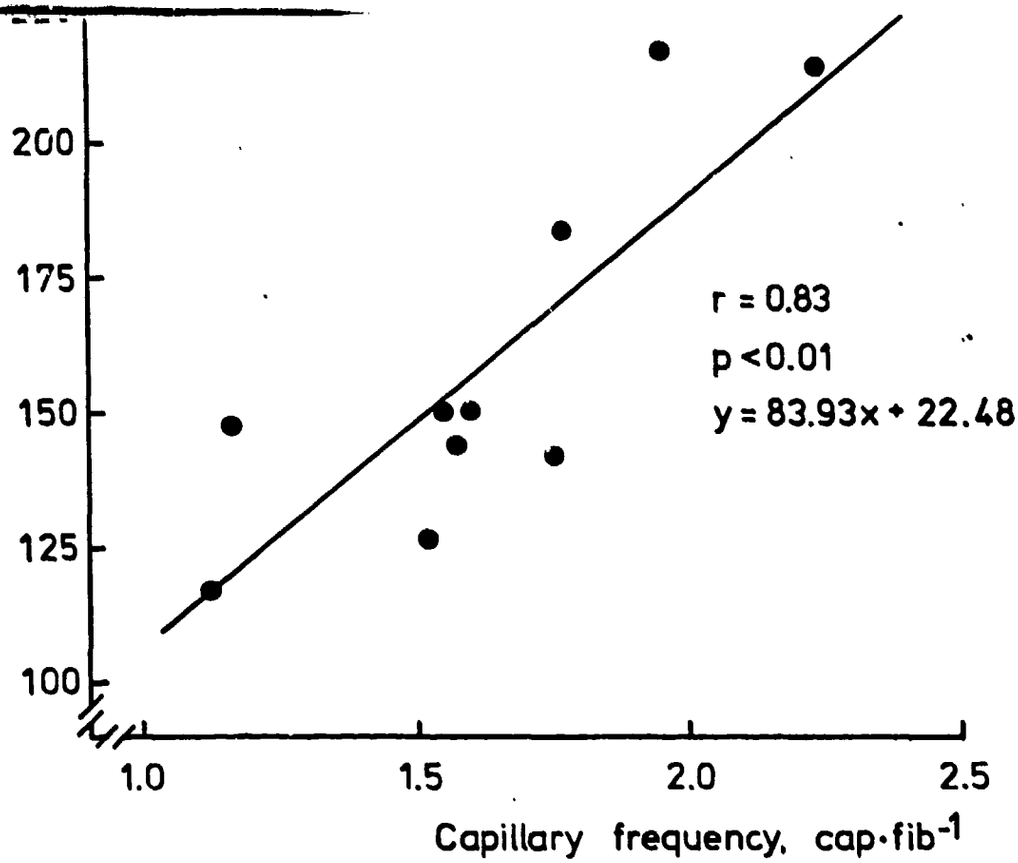
200
175
150
125
100
r = 0.83
p < 0.01
y = 83.93x + 22.48
1.0
1.5
2.0
2.5
Capillary frequency, cap·fib⁻¹

Fig 3

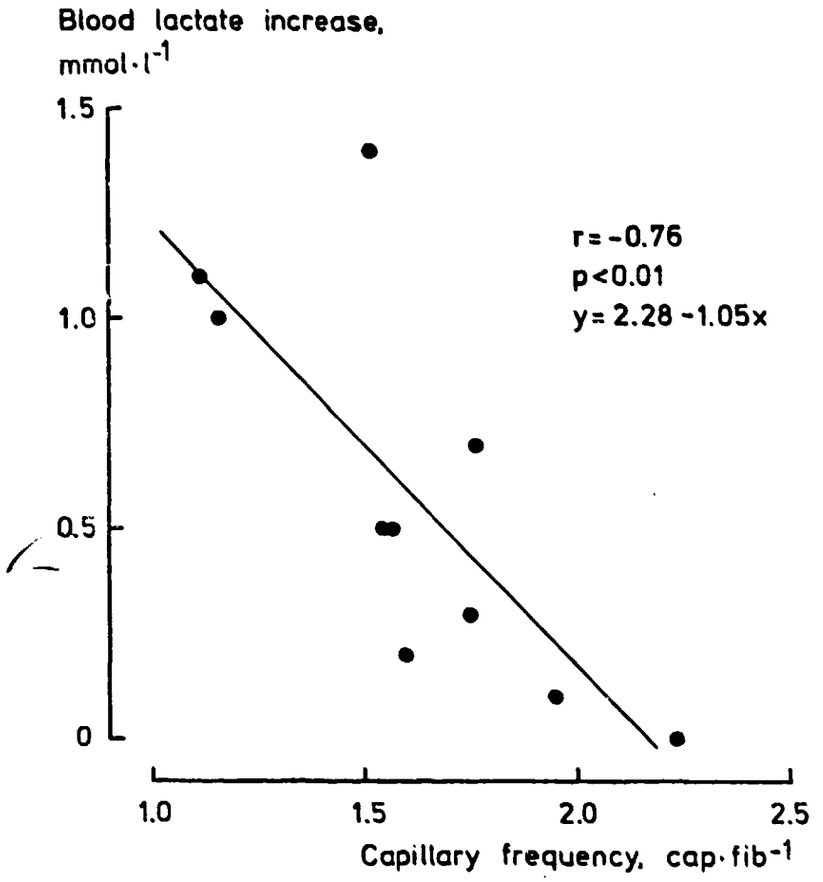
Blood lactate increase,
mmol·l⁻¹
1.5
1.0
0.5
0
r = -0.76
p < 0.01
y = 2.28 - 1.05x
1.0
1.5
2.0
2.5
Capillary frequency, cap·fib⁻¹

Fig 4