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Brief Communication

BLOCKED UTILIZATION OF OXYGEN IN PIGS DURING EXERCISE *

When the oxygen consumption is insufficient for the energy expenditure during exercise blood lactic acid will increase. This is due to anaerobic breakdown of glycogen to lactic acid. The cause may be an inadequate supply of oxygen or an insufficient utilization of oxygen by the muscles.

The conventional pig is known to develop high blood lactic acid even at mild exercise. The elevation is significantly lower in pigs which have been trained by treadmill running (*Lindberg et al.* in press).

The present paper reports on blood oxygen, carbon dioxide, pH and lactic acid after treadmill exercise in trained and untrained pigs.

Animals and methods have been described (*Lindberg et al.*). Blood samples were obtained by puncturing the fibular artery (*Lannek*, to be publ.) or, for determination of lactic acid, the vena cava cranialis.

The results of 3 experiments are shown in Table 1. The figures represent the conditions about 5 min. after terminated exercise.

It is seen that lactic acid is significantly higher and pH consequently lower in untrained pigs compared with trained animals. Lowering of $P\,CO_2$ corresponds to increased utilization of the $\frac{HCO_3^-}{CO_2}$ buffering system with blowing off of CO_2 by lung ventilation.

It appears further that the relative anaerobosis in untrained pigs is associated with elevated $P\,O_2$ in arterial blood as compared with trained pigs. This supports the hypothesis that the anaerobosis is caused by blocked peripheral utilization of oxygen. Thus, the effect of training would mainly be an improvement

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Table 1. Arterial blood $P O_2$, $P CO_2$ and pH and venous blood lactate in trained (T) and not trained (NT) pigs after treadmill exercise, 2.5 m per sec. for 4 min. $P O_2$, $P CO_2$ and pH were calculated for 40°C. (Number of pigs within brackets).

Experiment	107		
	T	NT	P
$P O_2$ mm Hg	107.90 ±12.48 (9)	122.93 ± 6.27 (16)	< 0.005
$P CO_2$ mm Hg	26.86 ± 3.46 (7)	21.57 ± 2.46 (12)	< 0.005
pH	7.17 ± 0.04 (9)	7.02 ± 0.07 (15)	< 0.005
Lactate meq./l	11.04 ± 3.06 (10)	19.69 ± 2.59 (9)	< 0.001

Table 1 (continued).

Experiment	108		
	T	NT	P
$P O_2$ mm Hg	110.79 ± 3.93 (8)	123.38 ± 8.08 (15)	< 0.001
$P CO_2$ mm Hg	32.63 ± 3.29 (6)	27.30 ± 2.99 (9)	< 0.01
pH	7.18 ± 0.10 (9)	7.08 ± 0.10 (15)	< 0.025
Lactate meq./l	19.26 ± 4.68 (10)	24.86 ± 4.50 (15)	< 0.01

Table 1 (continued).

Experiment	109		
	T	NT	P
$P O_2$ mm Hg	113.42 ± 6.10 (9)	127.00 ± 5.87 (9)	< 0.001
$P CO_2$ mm Hg	30.36 ± 2.69 (9)	26.32 ± 1.79 (9)	< 0.005
pH	7.33 ± 0.09 (9)	7.17 ± 0.05 (9)	< 0.001
Lactate meq./l	9.62 ± 2.93 (9)	20.25 ± 1.85 (9)	< 0.001

of the capacity of the oxidative energy processes in the tissues (*Holloszy* 1967). The elimination of an inadequacy of the peripheral circulation cannot be excluded.

The conditions are in some respects similar to Halothane-induced anaerobosis in pigs (*Berman et al.* 1970). In their experiments lactacidosis also developed in spite of high arterial oxygen tension.

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