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MAXIMAL OXYGEN UPTAKE AND LACTATE THRESHOLDS DURING EXERCISE ARE RELATED TO BLOOD VISCOSITY AND ERYTHROCYTE AGGREGATION IN PROFESSIONAL FOOTBALL PLAYERS

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ABSTRACT

Correlations among values at rest of blood viscosity (MT 90, high shear rate), RBC Aggregation (Myrenne) and four markers of fitness were investigated in 21 professional football players during a triangular maximal exercise test. Maximal O_2 uptake ($\dot{V}O_2$ max, directly measured) was correlated with resting plasma viscosity ($r=-0.666$ $p<0.01$) and blood viscosity at corrected hematocrit 45% ($r=-0.426$ $p<0.05$). The physical working capacity $\dot{W}170$ was correlated with $\dot{V}O_{2max}$ ($r=0.645$ $p<0.01$) and with both resting plasma viscosity ($r=-0.524$ $p<0.02$) and hematocrit ($r=-0.524$ $p<0.05$). Two determinants of the 4 mmol lactate threshold were found: red cell aggregation 'M' ($r=-0.529$ $p<0.02$) and 'M1' ($r=0.477$ $p<0.05$). Thus, markers of aerobic working capacity are negatively correlated with plasma viscosity and hematocrit, while the 4 mmol.l-1 lactate threshold which measures the ability to avoid blood lactate increase is negatively correlated to RBC aggregation.

Key words: Blood viscosity, hematocrit, exercise, football, $\dot{V}O_2$max
hemorheology, erythrocyte deformability, erythrocyte aggregation, blood lactate.

INTRODUCTION

A correlation between blood fluidity and several indices of fitness has been reported by several investigators (1-7). It is mostly explained by body water and plasma volume changes after training (8). However, O_2 supply to exercising muscles is a major determinant of muscular performance (9) and O_2 supply to tissues is influenced by both vascular and rheologic factors (10-12). Thus, on a theoretical point of view, increased blood fluidity may improve O_2 delivery to muscle during exercise in trained individuals. However, this question remains not completely clarified. There are several biological indicators of fitness, which are relevant to different kinds of exercise. The most popular is maximal oxygen uptake ($\dot{V}O_2$ max), which has not been (as far as we know) studied in connection to blood rheology despite the theoretical link between O_2 supply and rheology indicated above. Another important parameter is the ability to avoid hyperlactacidemia, indicated by the so-called 'anaerobic thresholds' or 'lactate thresholds' (13-14). In a previous report, we observed that blood viscosity and erythrocyte aggregation were positively correlated to lactate accumulation in blood during exercise (15). Thus, the aim of this study was to investigate the relationships among hemorheologic parameters and four indices of fitness: $\dot{V}O_2$ max, physical working capacity ($\dot{W}170$ see below), 2mmol lactate threshold and 4mmol lactate threshold.

SUBJECTS AND METHODS.

Subjects used in this study were 21 professional football players submitted daily to a physical training program. They were tested together during three days at the end of the sportive season. Their main age was 23.5 ± 0.68 yr; their main weight was 75.7 ± 3.5 kg; their mean height was 180.2 ±1.06 cm. The year of the investigation they won the French National Football Cup. They underwent a triangular maximal exercise test on Cycle ergometer 4000, with progressively increasing work loads until $\dot{V}O_2$ max. $\dot{V}O_2$, heart rate, and blood lactate was monitored during the test. Samples were drawn before exercise (T0), during the test below (T1) and above (T2) the 4 $mmol.l^{-1}$ lactate threshold, and during recovery (T3). Pedal speed was kept constant at 60 rpm by the subjects. Some data derived from the same experiment has previously been presented (15) but did not involve the same parameters. $\dot{V}O_2$ max was directely measured as the maximal value of $\dot{V}O_2$. Physical working capacity $\dot{W}170$ was also calculated, this being the work in watts that the subjects were able to perform at a heart rate of 170 $b.min^{-1}$ (16-17). A percentage of maximal heart rate for each step of exercise was calculated according to the tables of the American Heart Association. Lactate thresholds at 2 $mmol.l^{-1}$ and 4 $mmol.l^{-1}$ were determined as the percentage of $\dot{V}O_2$max at which blood lactate reached this value (14).

Blood samples for hemorheological measurements (7 ml) were drawn with potassium EDTA as the anticoagulant in a vacuum tube (Vacutainer). Viscometric measurements were done at very high shear rate (1000 s^{-1}) with a falling ball viscometer (MT 90 Medicatest, F-86280 Saint Benoit) (18-19). Accuracy of the measurements was regularly controlled with the Carrimed Rheometer 'CS' (purchased from Rhéo, 91120 Palaiseau, France)(20). The coefficient of variation of this method ranged between 0.6 and 0.8% (21). With this device we measured apparent viscosity of whole blood at native hematocrit, plasma viscosity, and blood viscosity at corrected hematocrit (0.45) according to the equation of Quemada (22). Dintenfass' 'Tk' index

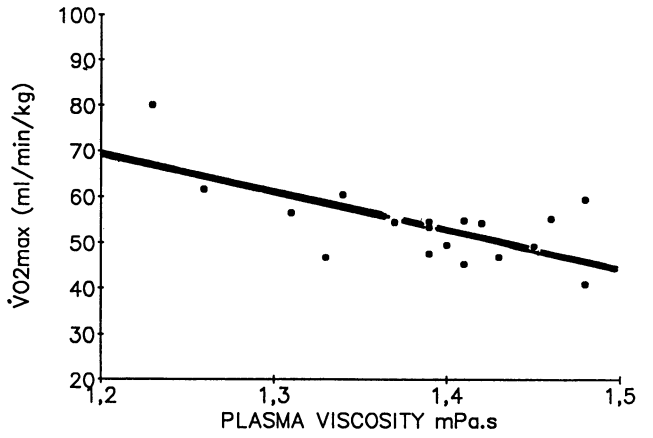

FIG. 1

Correlation between plasma viscosity at rest and $\dot{V}O_2max$ in 21 footballers. $r=0.666$ $p<0.01$.

of erythrocyte rigidity was calculated (23-24). RBC aggregation was assessed with the Myrenne aggregometer (25) which gives two indices of RBC aggregation: 'M' (aggregation during stasis after shearing at 600 s^{-1}) and 'M1' (facilitated aggregation at low shear rate after shearing at 600 s^{-1}). The hematocrit/viscosity (h/η) ratio, an index of oxygen supply to tissues, was calculated according to Chien (26) and Stoltz (27), with hematocrit (as percentage) divided by viscosity at high shear rate determined as described above. Lactate was assayed with a kit from DuPont specially adapted to the DuPont de Nemours automatic clinical analizer. This assay was based on NADH production by rabbit lactate dehydrogenase. Coefficients of variation range beween 0.7 and 5.6 %.

Statistics.

Correlations were tested by least square fitting for linear, exponential, logarithmic and power relationships. Partial correlation analyses were also performed when two hemorheological determinants of a fitness parameter were correlated to each other. This procedure allows to 'neutralize' one of the factors and calculate what would be the correlation if this factor were constant. (28). Results are presented as mean ± the SE of the mean. A value of $p<0.05$ was considered as significant. A correspondence factor analysis was performed on the 21 sets of values in order to give a comprehensive picture of the relationships among all the parameters (29).

RESULTS

responses to exercise

$\dot{V}O_2max$ was found to range between 40.8 and 80 ml.min^{-1}.kg^{-1}. $\dot{W}170$ ranged between 2.37 and 4.17 watt/kg. Lactate peak ranged between 3.2 and 11 mmol.l^{-1}. The 2 mmol.l^{-1} lactate threshold ranged between 33 and 83% of $\dot{V}O_2max$, and the 4 mmol.l^{-1} lactate threshold ranged between 45 and 100% of $\dot{V}O_2max$. Exercise increased significantly (table I) blood viscosity ($p<0.01$), plasma viscosity($p<0.01$), and Tk($p<0.01$).

TABLE I.

	TO	T1	T2	T3
blood viscosity	2.78(0.06)	2.89(0.06)*	3(0.09)***	3.04(0.07)***
corrected viscosity $\eta 45$	2.35(0.03)	2.36(0.03)	2.37(0.03)	2.37(0.04)
plasma viscosity	1.38(0.01)	1.42(0.01)*	1.42(0.01)***	1.43(0.02)***
"Tk" (RBC rigidity)	0.56(0.01)	0.56(0.02)	0.58(0.02)***	0.58(0.01)***
h/η ratio	0.15(0.004)	0.15(0.002)	0.15(0.002)	0.14(0.002)

*Modifications (mean ± SEM) of rheologic parameters during maximal exercise in study subjects. * $p<0.05$; *** $p<0.01$.*

relationships between blood rheology at rest and fitness parameters

The correlations are shown on table II. Two significant determinants of $\dot{V}O_2max$ were found: resting plasma viscosity (see fig.1) and blood viscosity at corrected hematocrit 45% ($r=-0.426$ $p<0.05$). Those two rheologic parameters are correlated ($r=0.596$ $p<0.02$). A partial correlation analysis indicates that, when viscosity at corrected hematocrit 0.45 is kept constant, the correlation between $\dot{V}O_2max$ and resting plasma viscosity remains significant ($r=-0.551$ $p<0.01$) while when plasma viscosity is kept constant, the correlation between $\dot{V}O_2max$ and blood viscosity at corrected hematocrit 0.45 disappears ($r=-0.10$ ns). Thus, the only hemorheological determinant of $\dot{V}O_2max$ which remains after this partial correlation analysis is plasma viscosity. Blood viscosity at native hematocrit was not correlated with $\dot{V}O_2max$. $\dot{W}170$ was correlated with $\dot{V}O_2max$ ($r=0.645$ $p<0.01$). Two determinants of $\dot{W}170$ were found: resting plasma viscosity ($r=-0.524$ $p<0.02$, see fig.2) and hematocrit ($r=-0.524$ $p<0.05$, see fig.3). Those two rheologic parameters are correlated ($r=0.471$ $p<0.05$) and the partial correlation analysis is unable to select one of them: when one is kept constant, the other is no longer correlated with $\dot{W}170$ ($r=0.369$ ns). No rheologic parameter was correlated with the 2 mmol lactate threshold. Two determinants of the 4 mmol lactate threshold were found: red cell aggregation 'M' ($r=-0.529$ $p<0.02$, see fig. 4) and 'M1' ($r=0.477$ $p<0.05$), which are highly correlated to each other ($r=0.830$), so that it is impossible to select one of the aggregation parameters and to eliminate the other. Partial correlation analysis suppresses the correlation between M1 and the threshold ($r=0.080$) as well as the correlation between M and the threshold ($r=0.271$ ns) when the other measurement of aggregation is supposed to be constant.

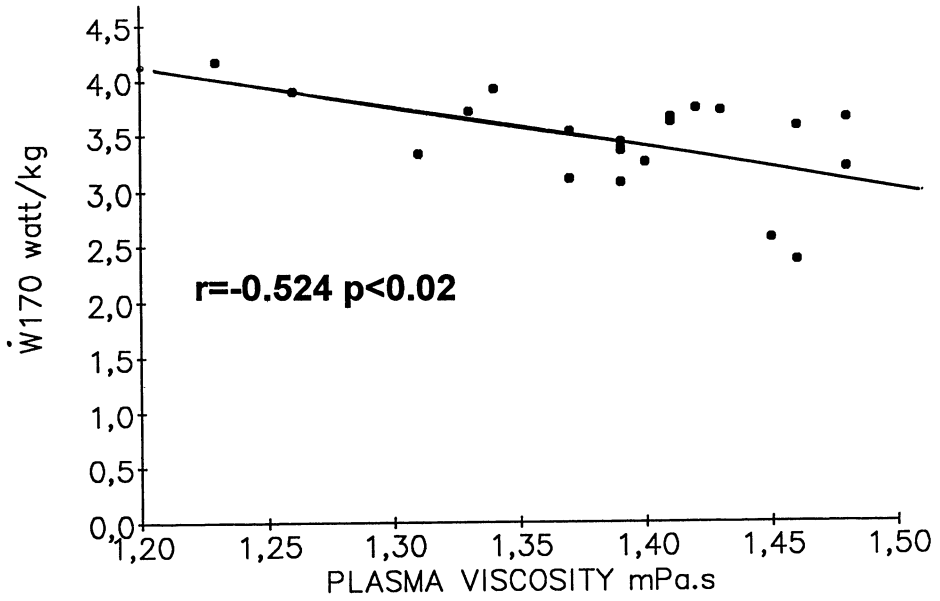

FIG. 2

Correlation between plasma viscosity and Ẇ170 in 21 footballers. r=0.524 p<0.02.

Correspondence factor analysis (fig. 5).

This multivariate analysis shows an opposition on axis 1 (which represents 68.38% of the total variance) beween lactate thresholds and RBC aggregation. Axis N°2 (15.42% of variance) opposes aerobic parameters ($\dot{V}O_2max$ and Ẇ170 which are quite close) to high shear rate viscometric data, including Tk and hematocrit. According to their individual characteristics, subjects are scattered on this first factorial plan which summarizes almost 79% of all the data of the study.

DISCUSSION

Although several papers are in agreement for a relationship between blood fluidity and fitness, we were not aware of studies evaluating a link between blood rheology and either $\dot{V}O_2max$ (directly measured during maximal exercise) or lactate thresholds. This paper shows that these two indices of fitness are correlated with hemorheological parameters.

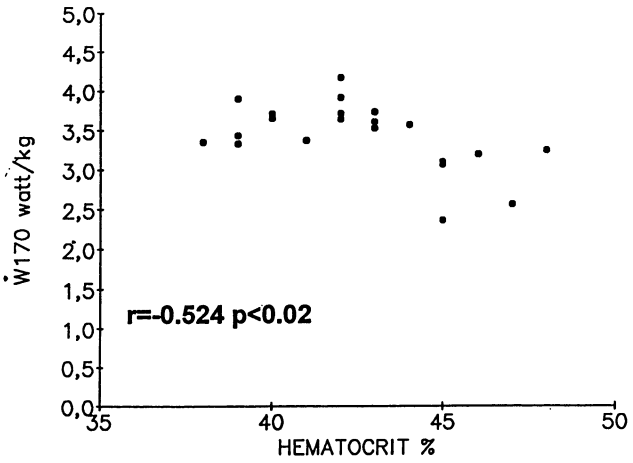

FIG. 3

Correlation between Ẇ170 and baseline hematocrit (r=0.524 p<0.02) in the 21 subjects of the study..

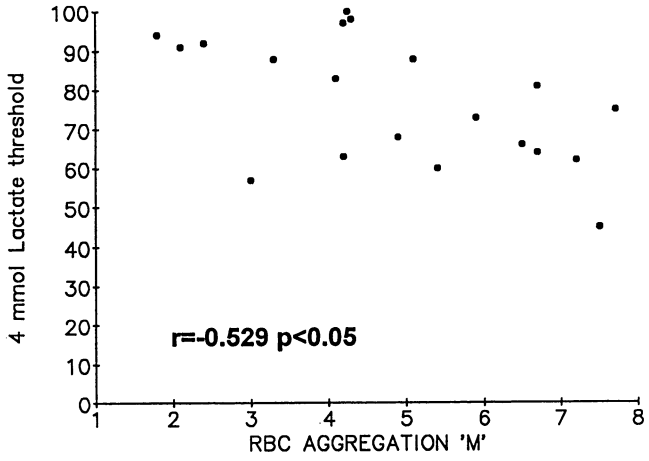

FIG. 4

Correlation between RBC aggregation 'M' (aggregation under stasis) at rest and the 4 mmol lactate threshold during exercise (r=0.529 p<0.02) in the 21 subjects of the study.

TABLE II.

	$\dot{V}O_{2max}$	$\dot{W}170$	[Lac] 2mmol	[Lac]4 mmol
blood viscosity	-0.138	-0.356	0.060	0.330
corrected viscosity $\eta 45$	-0.426*	-0.060	-0.359	0.080
plasma viscosity	-0.666***	-0.524**	-0.270	0.166
"Tk" (RBC rigidity)	-0.307	0.133	0.273	0.290
hematocrit	-0.279	-0.524**	0.070	0.327
h/η ratio	0.0004	0.103	-0.100	0.144
RBC aggregation 'M'	0.346	0.340	-0.386	-0.529**
RBC aggregation 'M1'	0.285	0.296	-0.361	-0.477*

*Linear correlations (r coefficients) among rheologic parameters before the exercise test and fitness parameters during exercise in the 21 subjects. * $p<0.05$; ** $p<0.02$; *** $p<0.01$.*

The correlations found here are sometimes wide, and do not imply a causal relationship. However it should be pointed out that this study group is homogeneous in terms of size and weight, while ergometric and hemorheologic data exhibit a wide variability. For this reason, we think that they may have some biological meaning which can be discussed in the light of current literature and from a theoretical point of view.

Hemorheological determinants of $\dot{V}O_2max$ and $\dot{W}170$ are quite the same. This is not surprising since these two parameters are highly correlated to each other and are both indices of aerobic exercising capacity. Plasma viscosity, in these study conditions, is the best statistical determinant of aerobic performance. This is consistent with previous findings (2, 5, 7). However, hematocrit is also negatively correlated with aerobic performance. This requires some comments. $\dot{V}O_2max$ is a measurement of body's ability to increase O_2 transfer from air to muscles and depends on several steps. The limiting step is not the same in all sportsmen (9). When arterial circulation is considered, $\dot{V}O_2max$ is equal to the maximal value of $\dot{Q}.CaO_2$, $\dot{Q}$ being cardiac output and CaO_2 the O_2 content of blood (30-34). This formula $\dot{V}O_2max = \dot{Q}.CaO_2$ can be written as a function of hematocrit and viscosity if one applies Hagen-Poiseuille law (35). It becomes $\dot{V}O_2max$ = constant x (h/η) x $(\Delta P/Z)$, where ΔP is the pressure drop and Z is hindrance. Thus $\dot{V}O_2max$ should be proportional to (h/η). However it is not the case in this study (table II) and $\dot{V}O_2max$ is totally decorrelated to (h/η). Hematocrit behaves mainly as a factor of viscosity (as evidenciated by its negative correlation with $\dot{W}170$ and its location on fig. 5): it is negatively related to fitness. One could suggest that this comes from the fact that fitness is accompanied by blood dilution which lowers hematocrit, but results in increased cardiac output. However, systemic hematocrit influences blood flow in tissues (36). Murray and Escobar (37) have shown that the decrease in hematocrit is primarily responsible for the rise in cardiac output after acute experimental hemodilution. Furthermore, regional vascular beds have markedly different blood flow responses to alterations in hematocrit (36, 38-41). It has been shown that hematocrit directly reduces blood flow in some tissues (38-41). Our data, consistent with previous papers (2, 4, 5, 8) show that training and improved performance are associated with low hematocrit.

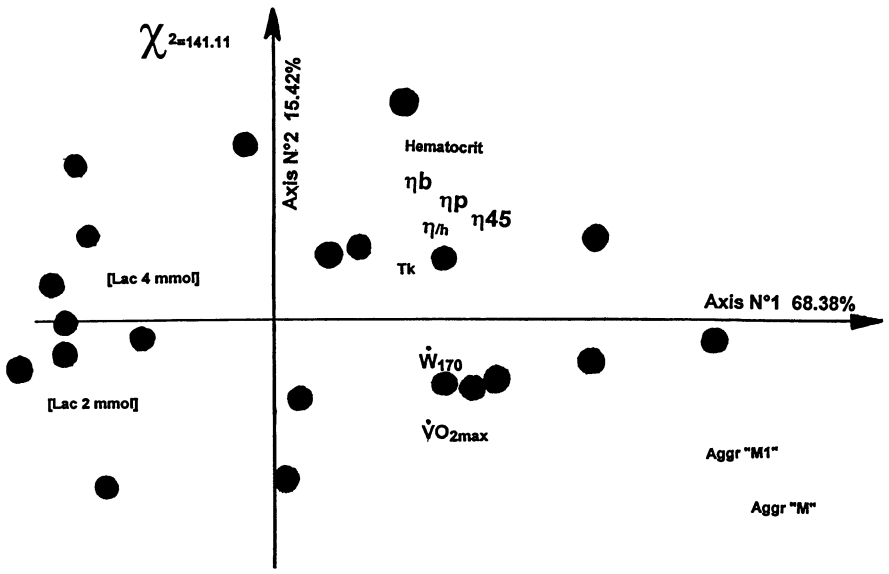

FIG. 5

Correspondence factor analysis showing the relationships between factors of blood viscosity at high shear rate, RBC aggregation and the fitness parameters $\dot{V}O_{2max}$, $\dot{W}_{170}$, 2 mmol lactate threshold and 4 mmol lactate threshold in the 21 footballers of the study. (●) individual subjects.

However, low hematocrit may be also found associated with a reduction of performance in 'sports anemia' (42). Thus, variations of hematocrit among sportsmen are not specific and may reflect either an increase in plasma volume (related to improved fitness), or anemia resulting from increased iron loss and repeated intestinal bleeding (42), which markedly impair performance. For this reason, it is necessary in such studies to investigate homogeneous series of sportsmen, e.g. the same team explored during the same period, in order to minimize this possible confusing factor. However, it remains difficult to give a simple interpretation of the relationships between hematocrit and performance.

Another point raised by this study is a possible influence of RBC aggregation on muscular lactate metabolism, as reflected by the lactate thresholds. Increased RBC aggregation may impair microcirculation in muscles, as experimentally shown by Vicaut (43). We previously reported positive correlations in sportsmen between lactate accumulation and RBC aggregation (15). Thus, although RBC aggregation is beneficial to some extent for microvascular perfusion (10), its increase, even within a physiological range, might impair aerobic metabolism in muscle, resulting in higher blood lactate. More studies will be needed for clarifying this question.

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