Relationships among body composition, hemorheology and exercise performance in rugbymen

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Abstract. We investigated relationships among body composition, blood rheology, and exercise performance in 14 rugbymen (19–31 yr, weight 65.8–109.2 kg, height 1.7–1.96 m, body mass index 21.7–33.1 kg/m²) who underwent a standardized sub-maximal exercise session on cycloergometer corresponding to 225 kJ over 30 min. The rheologic response to exercise was measured with the MT90 viscometer and the Myrenne aggregometer. Dehydration, evaluated by precision weighing, resulted in a loss of 360 to 973 g water; i.e., 1.69 to 4.32 g/kJ. This loss of water is not correlated to plasma volume contraction as assessed by the equation of Greenleaf. Hemorheologic changes are observed, but they are correlated neither to water loss, nor to plasma volume contraction. A 36% increase in blood viscosity (p < 0.01) is mainly explained by a red blood cell rigidification (p < 0.02), although hematocrit and plasma viscosity also increase (p < 0.01). Isometric adductor strength (specific ergometer) is correlated to erythrocyte flexibility (r = 0.680, p < 0.01). Red cell aggregability (Myrenne aggregometer) is correlated to fat mass measured by bioelectrical impedance (r = 0.634, p < 0.02). Aerobic working capacity index W170 is negatively correlated to the increase in plasma viscosity during exercise (r = −0.546, p < 0.05), suggesting that this event is less important in stronger individuals. This study shows that fat mass, even within a physiological range, is a determinant of erythrocyte aggregability, suggesting that training-induced alterations in body composition play a role in the specific hemorheologic profile of athletes. In addition, both erythrocyte flexibility and the magnitude of fluid shifts during exercise appear to be related to fitness in these sportmen.

Keywords: Blood viscosity, hematocrit, exercise, rugby, body composition, bioelectrical impedance, hemorheology, erythrocyte deformability, erythrocyte aggregation, fitness

1. Introduction

The relevance of hemorheological investigations to sports medicine is increasingly suggested by recent literature. First, 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]. By contrast, the acute effect of exercise is rather a transient impairment in blood fluidity [9–13]. Hyperviscosity resulting from an increase in plasma viscosity and hematocrit has been repeatedly found [9–13], but changes in RBC rigidity are only found in several protocols [4–6,11]. Erythrocyte

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aggregability, which impairs erythrocyte distribution in muscle microcirculation [14] has been reported to be related to the magnitude of blood lactate response [15,16] as well as the lactate thresholds during a maximal exercise-test [17].

The training-induced decrease in blood viscosity that is chronically observed in trained sportsmen has been mainly described as the result of an “autohodilution” increasing plasma volume [8]. However, there is another aspect which has retained until now little attention: training-induced changes in body composition, hormonal status and fuel metabolism are likely to induce further beneficial changes in blood rheology and to explain some of the rheologic characteristics of athletes [18]. The interest of investigating this point is that it could give some light on the mechanisms of exercise-induced improvement in cardiovascular risk, blood rheology being a potential link between these two aspects. Therefore, in this study, we aimed at determining whether body composition in athletes is related to hemorheologic parameters, the latter being themselves determinants of exercise performance.

2. Subjects and methods

Subjects used in this study were 14 rugby men submitted to a daily physical training program. They were volunteers and gave their informed consent in accordance to the local ethical regulation. Their mean age was 19–31 yr; their mean weight was 65.8–109.2 kg; their mean height was 1.7–1.96 m. Their mean waist to hip ratio was 0.92 ± 0.02. Measurements of isometric handgrip strength and isometric strength of the tight adductors were performed with specific home-made ergometers (INSERM U103-Biomécanique, Montpellier). These ergometers comprise a strength analyzer shaped in order to fit with anatomic characteristics of the subjects, and an electronic analysis system with liquid crystal screen display. Accuracy of the device is regularly reevaluated [19,20].

Subjects underwent a standardized submaximal exercise session on cycloergometer corresponding to 225 kJ over 30 min. Pedal speed was kept constant at 60 rpm by the subjects. Physical working capacity \( W_{170} \) was calculated, this being the work in watts that the subjects were able to perform at a heart rate of 170 \( b/min^{-1} \) [21]. Dehydration was evaluated by precision weighing (Sartorius model P 150-S-F2, France) and the water loss was expressed as a percentage of plasma volume (assumed to represent 4.3% of body weight [22]). The total heat production during exercise was calculated from the work output measured on cycloergometer and was corrected by cutaneous surface \( (S^2 \text{ (skin)}) \) calculated from height \( (H \text{ (cm)}) \) and weight \( (W \text{ (kg)}) \) with the formula of DuBois and DuBois [23]:

\[
S^2 = H^{0.725} \times W^{0.425} \times 71.84 \times 10^{-4}.
\]

Body composition was assessed with a four terminal impedance plethysmograph BIA 101/s from Akern RJL Systems (Detroit, MI, USA). The four electrode method minimizes contact impedance and skin-electrode interactions. Measurements were made in fasting subjects after 15 min resting in a supine position. A current of 800 \( \mu \text{A} \) and 50 kHz is introduced into the subject and the measurement of the voltage drop allows the determination of total body reactance and impedance. These values are used with a software provided by the manufacturer for calculating body water, fat mass, fat-free mass, and body cell mass [24].

2.1. Laboratory measurements

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) [25,26]. Accuracy of the measurements was regularly controlled with the Carriemed Rheometer ‘CS’ (purchased from Rhéo, 91120 Palaiseau, France) [27]. The coefficient of variation of this method ranged between 0.6 and 0.8% [28]. 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 Quezada [29]. Dininfass’ ‘T’k’ index of erythrocyte rigidity was calculated [30]. RBC aggregation was assessed with the Myrenne aggregometer [31] 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 [32] and Stoltz [33], with hematocrit (as percentage) divided by viscosity at high shear rate determined as described above.

The AFFIBIO-SEFAM aggregometer was used for a more precise assessment of RBC aggregation. This device is based upon the experiments of Mills [34,35] on cell disaggregation behavior in shear flow. This device measures the changes in backscattered light which are observed when sheared RBC suspensions are abruptly brought to a full stop. The decrease in the optical signal reflects the formation of RBC aggregates [36–38]. Some parameters are derived from the curve of light intensity as a function of time. The aggregation time is the reciprocal of the initial slope (calculated between 0.5 and 2 s after the shear has stopped). The aggregation index at 10 s is a measurement of the extent of erythrocyte aggregation and is the relative surface area above the curve calculated over the first 10 s. This device measures also disaggregation thresholds, by submitting blood to a succession of shear rates from 600 to 7 s⁻¹. The total disaggregation threshold is the shear rate below which the backscattered light intensity starts to decrease, indicating that the shear stress applied to aggregates is no longer sufficient for allowing complete dispersion of RBC aggregates. The partial disaggregation shear rate is defined as the shear rate corresponding to the intersection point of the two asymptotes drawn from the extremes (maximum and minimum shear rate).

Changes in plasma volume

A formula for calculating plasma volume changes (% ΔPV) during exercise from hematocrit changes has been published by investigators of the NASA-Ames Research Center [41] who demonstrated its validity in moderate as well as maximal exercise. We applied this formula to our data. The equation is:

\[
% \Delta PV = \frac{100}{(100 - H_o)} \times 100\left[\frac{(H_o - H)}{H_o}\right],
\]

where Ho is resting hematocrit and H hematocrit during exercise.

2.2. Statistics

Results are presented as mean ± SE of the mean. A value of \( p < 0.05 \) was considered as significant. Comparisons were made with Mann, Whitney and Wilcoxon nonparametric tests [42]. Correlations were tested by least square fitting for linear, exponential, logarithmic and power relationships.

3. Results

Body composition as assessed by bioelectrical impedance measurements showed a fat free mass of 70 ± 3.1 kg (i.e., 81.7 ± 1.04% of body weight) and a fat mass of 26.65 ± 11.7 kg, i.e., 18.3 ± 1.04%. Body water averaged 49.7 ± 1.8 kg, i.e., 58.3 ± 1.2% of body weight.
Fig. 1. Correlation between water loss (expressed as a percentage of plasma volume) and heat production corrected by cutaneous surface ($r = 0.539$, $p < 0.05$).

3.1. Responses to exercise

Hemorheologic changes are shown in Table 1. There is a 36% increase in blood viscosity ($p < 0.01$) and an increase in the red cell rigidity index ‘Tk’ ($p < 0.02$). Red cell aggregation measured with the Myrenne aggregometer does not change while an increase in aggregation is evidenced with the SEFAM aggregometer (lower aggregation time $p < 0.01$). Hematocrit and plasma viscosity also increase ($p < 0.01$). A stepwise regression analysis selects the increase inTk as the main determinant of this change in blood viscosity, since it explains 34% of the variance of this parameter in this situation. By contrast, changes in hematocrit and plasma viscosity are not significantly correlated to the increase in blood viscosity at high shear rate, suggesting that they do not explain most of this phenomenon in this experimental situation.

3.2. Dehydration

Dehydration, evaluated by precision weighing, resulted in a loss of 360 to 973 g water, i.e., 1.69 to 4.32 g/kJ. This loss of water is not correlated to plasma volume contraction as assessed by the equation of Greenleaf ($r = 0.001$ ns). Water loss (expressed as a percentage of theoretical plasma volume) is correlated to heat production corrected by cutaneous surface (Fig. 1). Rheologic changes are correlated neither to water loss, nor to plasma volume contraction. For instance, $r$ values for the increase in blood viscosity vs water loss is as low as 0.02 and $r$ values for the increase in plasma viscosity vs water loss is 0.06. Thus, rheologic changes seem to be poorly related to dehydratation in these experimental conditions.

3.3. Measurements of fitness

As shown on Fig. 2, the aerobic working capacity index $W_{170}$ is negatively correlated to the increase in plasma viscosity during exercise ($r = -0.546$, $p < 0.05$), suggesting that this event is less important in stronger individuals. A totally different fitness parameter is presented in Fig. 3 which shows that isometric adductor strength (specific ergometer) is negatively correlated to erythrocyte rigidity. This correlation fits better with an exponential curve ($r = -0.680$, $p < 0.01$) than with a linear one ($r = -0.649$,.
Table 1

<table>
<thead>
<tr>
<th></th>
<th>Before exercise</th>
<th>After exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood viscosity</td>
<td>2.9 ± 0.07</td>
<td>3.66 ± 0.17***</td>
</tr>
<tr>
<td>Corrected viscosity $\eta_{IS}$</td>
<td>3.08 ± 0.09</td>
<td>3.44 ± 0.09***</td>
</tr>
<tr>
<td>Plasma viscosity</td>
<td>1.34 ± 0.01</td>
<td>1.45 ± 0.01***</td>
</tr>
<tr>
<td>“Tk” (RBC rigidity)</td>
<td>0.62 ± 0.01</td>
<td>0.65 ± 0.02***</td>
</tr>
<tr>
<td>$h/\eta$ ratio</td>
<td>0.15 ± 0.004</td>
<td>0.13 ± 0.004****</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>42.64 ± 0.94</td>
<td>46.5 ± 1.09***</td>
</tr>
<tr>
<td>RBC aggregation ‘M’</td>
<td>4.3 ± 0.45</td>
<td>4.55 ± 0.37</td>
</tr>
<tr>
<td>RBC aggregation ‘M1’</td>
<td>8.08 ± 0.6</td>
<td>8.12 ± 0.65</td>
</tr>
<tr>
<td>Aggregation time (TA)</td>
<td>2.77 ± 0.28</td>
<td>1.85 ± 0.14***</td>
</tr>
<tr>
<td>Aggregation time (TF)</td>
<td>39 ± 2.9</td>
<td>28.6 ± 2.7***</td>
</tr>
</tbody>
</table>

$p < 0.05$; $***p < 0.01$; $****p < 0.0001$.

Fig. 2. Negative correlation between aerobic working capacity index $W_{170}$ and the increase in plasma viscosity ($r = -0.546$, $p < 0.05$).

$p < 0.02$). As shown in Fig. 4, red cell aggregability (Myrenne aggregometer) appears to be unrelated to performance but is correlated to the body mass index ($r = 0.565$, $p < 0.05$) and even better to fat mass ($r = 0.634$, $p < 0.02$).

4. Discussion

This study shows that fat mass is correlated to erythrocyte aggregability (Fig. 4), and that the magnitude of the exercise-induced rise in plasma viscosity is related to aerobic working capacity (Fig. 2). In addition, red blood cell deformability is correlated with isometric strength.

Most of the literature on blood rheology and exercise focuses on endurance athletes [18]. To our knowledge, this is the first report of hemorrhology in rugby players. Rugby players belong to several athletic types, ranging from the highly strength-trained to the endurance-trained, so that they could be expected to represent a heterogeneous sample, mostly concerning their body composition [43]. Therefore, these sportsmen were potentially an interesting population for investigating relationships between
training-induced alterations in body composition and hemorheological parameters over a wide physiological range of values.

Our data on body composition are obtained with bioelectrical impedance analysis, which is an indirect, widely used and validated, method for evaluating fat mass and body fluids from measurements of body’s electric properties [24]. This technique is reproducible and accurate when conditions of validity are carefully respected (e.g., rest, fast, absence of ionic disturbances, use of specific equations valid for the population investigated). In the case of sportsmen, the technique can be precise and sensitive if these conditions are well respected [43–45].

An important finding of this study, obtained with this method, is that body fat mass is correlated to erythrocyte aggregability. In obese people, increased red cell aggregability proportional to excessive fat deposits is a classical finding [46,47]. However, bioelectrical impedance analysis of these sportsmen does not support the diagnosis of obesity, although they have a high body mass index. As previously reported, the large body mass index of rugbymen is explained in our sample by an increase in fat-free mass [43] while the percentage of fat remains within a normal range. Thus, the correlation in Fig. 4 shows that fat mass is a determinant of erythrocyte aggregability even within a physiological range, i.e., the lower is
fat mass, the lower is erythrocyte aggregability. The mechanism as well as the physiological meaning of this relationship remain to be investigated. A physiological link between the adipose tissue and red cell aggregability could be hypothesized on the basis of recent reports indicating that the fat cell is able to release substances involved in fibrinogen turnover, such as plasminogen activator inhibitor 1 [48]. Therefore, the correlation between red cell aggregation and adipose mass may not be restricted to obesity, but rather reflect a physiological regulation. Since erythrocyte aggregability impairs oxygen supply by muscle microcirculation [14] and is associated with a higher lactate accumulation into blood during exercise [15–18], this relationship may have some influence on fuel processing at the muscular level. However, blood lactate was not measured in this study, and further investigation is needed for clarifying this point.

Another aspect of body composition in sportsmen that is likely to influence blood rheology is the exercise-induced alteration in body water volumes. We expressed the water loss proportional to body weight, assuming that plasma volume represents 4.3% of this weight, a classical statement in physiological textbooks [22]. However, given the different training backgrounds of our subjects, this should be considered as a theoretical evaluation, in order to assess the magnitude of the body water loss as a fraction of the plasma volume and thus to compare it with the rheologic changes. In fact, no significant relationship between body water and blood rheology is evidenced, either at rest or after exercise. While water supply has been demonstrated to reduce exercise-induced hyperviscosity [13], neither water loss measured by precision weighing nor hemoconcentration evaluated by the formula of Greenleaf are correlated in this study to rheological changes.

All the classical components of the exercise-induced rise in blood viscosity are observed in this experiment, as shown on Table 1. However, results concerning erythrocyte aggregability require some comments. In most studies, this parameter is unchanged during exercise when investigators measure it with the Myrenne aggregometer [4,18]. By contrast, we recently observed in footballers that the SEFAM erythroaggregometer, which is a less widely used, but much more sophisticated and reliable device [36–38], detects a significant increase in red cell aggregability during a 25 min submaximal exercise-test [49]. Similar results have been reported by Hardeeman with the LORCA [50]. The reason for the discrepancy among various methods in this situation is unclear, since the Myrenne aggregometer is generally sensitive to all clinical situations characterized by increased red cell aggregability [36–38]. Exercise-induced alterations in disaggregability and aggregability of erythrocytes seem to be related to fibrinogen levels [49] but their mechanism remains to be further clarified.

Finally, we find two relationships between markers of fitness and blood rheology that have not yet been, as far as we know, reported elsewhere. As shown in Fig. 2 there is a negative correlation between aerobic working capacity index $W_{170}$ and the increase in plasma viscosity during exercise. $W_{170}$ is a classical index of an individual’s response to a submaximal work load. Actually, the relationship between the work load and the corresponding steady state of heart rate is more generally expressed as a VO$_2$ max, according to Astrand’s equations and nomograms [51], but one could argue that it is more satisfactory to express such results as watts for a heart rate than as a rate of oxygen consumption, given that oxygen was not measured. $W_{170}$ and indirect VO$_2$ max have the same meaning and are generally well correlated [15]. Obviously, 170 b/min may represent a different percentage of maximal heart rate if subjects have not the same age: however, sportsmen studied in our paper had quite the same age so that their $W_{170}$ could safely be compared.

This finding may suggest that the fitter is the subject, the more moderate is hemoconcentration during exercise. Thus, plasma volume can be better preserved, with presumably beneficial consequences on cardiac output and working capacity [52]. On the other hand, as shown on Fig. 3, the viscometric index
of red cell rigidity ‘Tk’ is negatively correlated with isometric adductor strength. Although various measurements of fitness have been reported to be related to blood fluidity (including VO₂ max [17], W₁₇₀ [5] and exercise duration until exhaustion [3]) we are not aware of studies reporting a relationship between isometric strength and a rheological parameter. Moreover, erythrocyte deformability has not been until now described as a major rheologic marker of fitness, contrasting with a large body of evidence showing that plasma viscosity is a good statistical determinant of aerobic capacity [18]. Thus, whether erythrocyte deformability is a marker of fitness for some sports in which strength is more important than aerobic capacity remains to be studied. Presumably, metabolic alterations associated with training-induced changes in body composition may explain why some sportmen have a lower erythrocyte rigidity [18]. However, trace element deficiencies such as zinc depletion are associated with both a lower strength [19] and a higher red cell rigidity [53] that may explain some of our findings in this study. Since zinc was not measured in this work, this hypothesis remains speculative.

In conclusion, this study shows that red cell aggregation is correlated to fat mass even in sportmen in whom this fat mass is low, suggesting that this aspect of body composition may explain some of the specific rheologic improvements that have been described in athletes. We think that this finding supports our previous assumption of a ‘triphaseic effect’ of exercise on blood rheology [18], i.e., training-related alterations in body composition have specific long-term metabolic effects that further influence the rheologic properties of blood. In addition, aerobic capacity is associated with a lower rise in blood viscosity during exercise, and red cell flexibility is correlated to strength, further supporting the concept of a role of blood fluidity in exercise performance.

References


