Effects of hydration and dehydration on blood rheology in sickle cell trait carriers during exercise

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Running title: Blood viscosity at exercise and water deprivation

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Abstract

This study compared the hemorheological responses of a group of sickle cell trait (SCT) carriers with those of a control group in response to 40 minutes of submaximal exercise (exercise intensity: 55% aerobic peak power) performed in two conditions: one with water offered ad libitum (HYD condition) and one without water (DEHYD condition). Blood and plasma viscosities, as well as red blood cell rigidity, were determined at rest, at the end of exercise, and at two hours recovery with a cone plate viscometer at high shear rate and 37°C. SCT and CONT groups lost 1 ± 0.7 kg and 1.6 ± 0.6 kg of body weight, respectively, in the DEHYD condition, indicating a significant effect of water deprivation compared with HYD condition, in which body weight remained unchanged. Plasma viscosity increased with exercise and returned to baseline during recovery independently of the group/condition. As previously demonstrated, resting blood viscosity was greater in SCT carriers than in the control group. Blood viscosity increased by the end of exercise and returned to baseline at two hours recovery in the control group in both conditions. The blood viscosity of SCT carriers did not change in response to exercise in the DEHYD condition and remained elevated at two hours recovery. This extended hyperviscosity, in association with other biological changes induced by exercise, could be considered as a risk factor for exercise-related events in SCT carriers, similar to vaso-occlusive crises, notably during the recovery. In contrast, HYD condition normalized the hyperviscosity and red blood cell rigidity of the SCT carriers, with blood viscosity values reaching the same lower values as those found in the control group during the recovery. Adequate hydration of SCT carriers should be strongly promoted to reduce the clinical risk associated with potential hyperviscosity complications.

Key words: sickle cell trait, blood viscosity, red blood cell rigidity, endurance effort, water ingestion
**Introduction**

Sickle cell trait (SCT; i.e., the heterozygous form of sickle cell anemia) is common in people of African origin and is generally considered as a benign condition. However, epidemiological data from the US Army demonstrated that exercise-related sudden death was 30 times more common in black recruits with SCT than in black recruits without SCT (29, 35). Since then, the benign nature of SCT during exercise has been questioned (7, 17, 36) and recent case studies of exercise-related sudden death (2, 37) or medical complications (31) have revived the debate.

Several mechanisms have been proposed to explain these adverse events. Notably, many authors suggested that hemorheological abnormalities (19, 40, 48) and inflammation-vascular adhesion mechanisms (41, 42, 47) might be involved in microcirculatory alterations leading to sickle cell anemia-like vaso-occlusive crisis. The hyperthermia and dehydration induced by physical exercise are thought to play a role in the occurrence of exercise-related sudden death in this population (29, 35). Accordingly, it has been recommended that SCT carriers should re-hydrate adequately during exercise (2, 20, 31, 34, 35) to reduce the risk for adverse events potentially related to the red blood cell sickling process observed by Bergeron et al (8).

However, the effects of hydration/dehydration on whole blood rheology have never been investigated. In the study of Bergeron et al (8), the percentage of venous sickled red blood cells in the two exercising SCT carriers deprived of water did not reach the values usually observed in patients with sickle cell anemia experiencing vaso-occlusive crisis (5, 43). This observation suggests that other parameters, such as blood rheology (20), could be modulated by hydration/dehydration and potentially play a contributing role in microcirculatory and vascular alterations.

The present study examined the hemorheological responses of SCT carriers and a control group during submaximal exercise in two conditions: with water offered *ad libitum* and
without water. We hypothesized that water deprivation during exercise would impair blood rheology to a greater extent in SCT carriers than in control subjects. We further assumed that study results confirming this hypothesis would provide a strong basis for more actively promoting adequate hydration in SCT carriers during exercise as a means to prevent microcirculatory and vascular disorders.

Materials and methods

Subjects

Eleven SCT carriers (SCT group) and twelve subjects with normal hemoglobin (CONT group) participated in the study. All subjects were athletes (> 10 hours of team sports/week) and students at the National Institute for Popular Education and Sport (INSEPS) of the Cheikh Anta Diop University in Dakar, Senegal. Subject characteristics are presented in Table 1. The major exclusion criteria were anemia and/or α-thalassemia, hypertension and malaria.

Protocol

The subjects were informed of the procedures and purposes of the study, which was approved by the National Ethics Committee of Senegal, and gave written informed consent to participate. The protocol was in accordance with the guidelines set by the Declaration of Helsinki.

On the first experimental day, each subject performed a progressive and maximal exercise test on a mechanically braked ergometer (Monark 824E, Stockholm, Sweden). The test began with a 5-min warm-up at 30 W. Explicit standardized instructions were given before each test. Pedaling speed remained constant (70 rpm) throughout the test, and a 30-W load was
increased stepwise every minute until exhaustion (peak power output; Ppeak). Heart rate (HR) was measured continuously (S810, Polar Electro Oy, Kempele, Finland) and maximal HR was noted.

Two weeks later, the same subjects participated in two randomized sessions (3-5 days apart), consisting of pedaling for 40 min at 55% Ppeak (i.e., prolonged submaximal exercise). Subjects did not perform any other exercise for two days before each session. In one session, subjects could not drink water (DEHYD) whereas in the other they could drink water *ad libitum* (HYD) during exercise performance. The volume of water ingested during the HYD condition was measured accurately. Rectal temperature (YSI thermistor thermometer, Yellow Springs Instrument, Inc., Yellow Springs, OH, USA) and HR were measured continuously. Whatever the experimental conditions, subjects consumed no fluids for 3 hours prior to exercise testing and no hydration was allowed during the first 2 hours of recovery. Subjects were weighed before and immediately after exercise. Venous blood samples were drawn from the antecubital vein of the nondominant arm at rest, at the end of exercise, and at two hours recovery for hemorheological measurements. The laboratory temperature and hygrometry varied between 25-28°C and 50-65%, respectively, with no differences between exercise conditions or groups.

*SCT diagnosis and blood analysis*

To test for the hemoglobin type, venous blood was drawn at rest into tubes containing EDTA and screened by isoelectric focusing. The results were confirmed by citrate agar electrophoresis. Various hemoglobin fractions were quantified by high performance liquid chromatography (HPLC). A test of solubility confirmed the presence of Hb S. Hematological data determined with a hematology analyzer (Cell Dyn 3700, Abbott Laboratories, Abbott Park, IL, USA) were used for the indirect diagnosis of anemia and alpha-thalassemia, which
result in hematological modifications (28). To confirm the absence of alpha-thalassemia, we used the technique from Chong et al (12) with a single-tube multiplex-PCR assay. The mean concentration of HbS for the SCT group was 38.0 ± 0.9%.

Hemorheological measurements

Blood for hemorheological measurements was sampled in EDTA tubes. Hemorheological parameters were measured within 3 hours of sampling. Measurements of blood viscosity ($\eta_b$) and plasma viscosity ($\eta_p$) were performed with a cone plate viscometer (Brookfield DVII+, with CPE40 spindle) at 37°C. Blood viscosity was determined at a shear rate of 225 s$^{-1}$. To avoid as much as possible the “surfactant layer effect” (6) on the measurements of $\eta_p$, the analysis was done at very high shear rate (i.e., 750 s$^{-1}$). Under the present experimental conditions, the flow instability of the sample in the gap between the cone and the plate should not occur in this type of viscometer (30). Hematocrit (Hct) was measured after blood microcentrifugation (JOUAN-HEMA-C, Saint Herblain, France), and hemorheological measurements were performed according to the recent guidelines for hemorheological laboratory techniques (6).

In addition, the index of red blood cell rigidity from Dintenfass (25) was calculated according to the following equation:

$$Tk = (\eta_r^{0.4} - 1) / (\eta_r^{0.4} \ast \text{Hct})$$

with $\eta_r$ corresponding to the relative blood viscosity; i.e., the ratio $\eta_b/\eta_p$.

Statistics

All results are expressed as means ± standard deviation (SD). Subject characteristics, MAP, HR$_{max}$ and the volume of water ingested were compared between the two groups using an unpaired Student’s $t$ test. The time courses of hemorheological parameters, rectal temperature,
weights and HR during the prolonged exercise (HYD and DEHYD conditions) were compared between the two groups using a two-way analysis of variance (ANOVA) with repeated measures. Pair-wise comparisons (Fisher LSD post-hoc tests) were used when necessary to locate where significant differences had occurred. The significance level was defined as $P < 0.05$. Analyses were conducted using Statistica (v. 5.5, Statsoft, Tulsa, OK, USA).

Results

Subject characteristics and maximal exercise responses

As shown in Table 1, no significant difference between the two groups was observed for height, weight or age. In addition, the $P_{\text{peak}}$ and HRmax determined during the progressive and maximal exercise tests were not different between the SCT group and CONT group (Table 1). 

Exercise responses during the prolonged submaximal exercise

Body weight in the two groups remained constant during the HYD condition but decreased under resting value during the exercise conducted in DEHYD condition (Table 2). The percentage of weight loss during the DEHYD condition was not significantly different between the two groups and was $2.0 \pm 0.2\%$ and $1.5 \pm 0.3\%$ in the CONT and SCT groups, respectively. The volume of water ingested during the 40-min exercise period conducted in HYD condition did not differ between the two groups and was $604 \pm 125$ ml and $700 \pm 73$ ml in the CONT and SCT groups, respectively. HR and rectal temperature increased above baseline in the two groups and in the two conditions (Table 2). No significant difference was observed between the two groups.
Hct and ηp parameters are presented in Table 3. Hct did not differ between the two groups at any time and did not change with exercise. Plasma viscosity increased above baseline in response to exercise in the SCT group (HYD and DEHYD conditions) and the CONT group (HYD condition only) and then returned to baseline during the recovery. The change observed for ηp in the CONT group during the DEHYD condition was not statistically significant.

Resting ηb was higher in the SCT group than in the CONT group in the two conditions (Figure 1). Exercise increased ηb in the CONT group in the two conditions and ηb then returned to baseline during the recovery. The ηb did not change with exercise during the DEHYD and HYD conditions in the SCT group. During the recovery, ηb decreased under resting level in the HYD condition only, reaching a value close to the values found in the CONT group (HYD and DEHYD conditions) at that time. Blood viscosity in the SCT group did not change during the recovery of the DEHYD condition and, therefore, remained elevated. The CONT and SCT groups exhibited significantly lower values during the recovery of the HYD condition as compared with the DEHYD condition of the SCT group. The difference between the SCT group and CONT group during the recovery of the DEHYD condition did not reach statistical significance (P < 0.1).

On the whole, baseline red blood cell rigidity was greater in the SCT group than in the CONT group in the two conditions (Figure 2). Exercise did not significantly affect red blood rigidity in the CONT group. Red blood cell rigidity in the SCT group decreased under resting value in the HYD condition at the end of exercise and during the recovery. In contrast, it exhibited no change at the end of exercise compared to baseline in the DEHYD condition and then increased above baseline during the recovery. The CONT group exhibited significantly lower values during the recovery in both conditions as compared with the DEHYD condition of the SCT group. The difference between the DEHYD and HYD conditions of the SCT group during the recovery did not reach statistical difference (P < 0.1).
Discussion

Main study outcome

The main finding of the present study was the marked effect of *ad libitum* hydration on the $\eta_b$ and red blood cell rigidity of exercising SCT carriers, with $\eta_b$ and cell rigidity decreasing to the same values of the CONT group within two hours of recovery.

Exercise responses

Our study confirmed previous findings showing that SCT carriers are able to perform progressive and maximal exercise or prolonged submaximal exercise at the same mechanical level as subjects with no hemoglobinopathy [see (20) for review]. In addition, there were no unusual symptoms of general or local discomfort reported by either SCT carriers or CONT subjects. The magnitude of physiological stress was the same in the two groups whatever the condition tested, as indicated by the outcome measures (i.e., HR and rectal temperature). Although the DEHYD condition induced a weight loss in both groups, HR and rectal temperature did not differ between HYD and DEHYD conditions at the end of exercise. This unexpected finding cannot be explained by environmental differences between the HYD and DEHYD conditions since humidity and external temperature were the same in the two conditions. In addition, the exercise intensity was exactly the same for each subject in the two conditions. Although the pre-exercise body weight difference between the HYD and DEHYD conditions was less than 1% in both groups, the pre-exercise hydration level of the subjects was not evaluated (i.e., plasma osmolality or urine specific gravity) as recommended (38, 39) and this is clearly a shortcoming of the study. The body weight loss during the DEHYD condition remained within an acceptable range [i.e., $\approx 1.5$-2%; (1)] and was apparently of
limited effect on HR and rectal temperature. Recent studies comparing the effects of neutral and hot temperature exposures on cycling responses reported that HR and rectal temperature did not differ between the two environmental conditions (32, 33). However, subjects exhibited higher skin temperature changes (not measured in our study) in hot condition, suggesting hyperthermic strain as compared with neutral condition (32, 33).

Blood rheology at rest
Blood viscosity was higher at rest in SCT carriers than in CONT group. Since $\eta_p$ and Hct were not different between the two groups, the difference in $\eta_b$ was related to the reduced resting red blood cell deformability (i.e., higher Tk index reflecting higher red blood cell rigidity), as previously demonstrated (9, 18, 22). Although most of the hemorheological alterations usually found in SCT carriers may be considered as subclinical (45), blood viscosity values can be as high as in patients with sickle cell hemoglobin C disease (45), who are known to develop frequent thromboembolic complications. The mean value of blood viscosity in sickle cell hemoglobin C disease is about 6 mPa.s$^{-1}$ (45) and five of the 11 SCT carriers in the present study exhibited greater values at rest, indicating the presence of hyperviscosity and highlighting the wide heterogeneity in the hemorheological profile of SCT carriers. In support of this, the results from Austin et al (4) strongly suggest that sickle cell trait is a risk factor for venous thromboembolism. SCT carriers with elevated baseline blood viscosity values could be considered as potentially at risk for microcirculatory disorders and hydration levels should be carefully controlled in these individuals. Strategies to normalize blood viscosity in SCT carriers would be beneficial for their health.

Blood rheology in response to exercise in CONT group
The increase of $\eta_b$ in the CONT group during the cycling exercise conducted in both conditions is a classical finding (10, 14, 23, 26) and is related to the changes in $\eta_p$. Although not significant, the changes in red blood cell rigidity during the DEHYD condition also contributed to the increase in $\eta_b$. The increase in $\eta_b$ during the DEHYD condition resulted in values in the CONT group close to the values found in the SCT group, in both conditions. The $\eta_b$ of the CONT group then returned to baseline during the recovery, partly in relation with the return to baseline of $\eta_p$ and the non-significant reduction in red blood cell rigidity. The lack of difference between the HYD and DEHYD conditions in the CONT group was very surprising but indicated that dehydration did not exert any adverse effects on blood rheology during a 40-min submaximal exercise. The plasma volume change in both conditions was minimal and can be evaluated (50) at 2-3% after exercise (and less during recovery) as compared with resting value. The plasma shifts between intravascular and extravascular spaces may play a role in this equilibrium (27).

Blood rheology in response to exercise in SCT carriers

In contrast, it seems that water hydration played a very important role in the exercising SCT carriers. When the SCT carriers performed the exercise protocol in DEHYD condition, $\eta_b$ remained at the same elevated value as observed at baseline and this effect was persistent at two hours recovery. Given the blood viscosity range values published by Tripette et al (45) for healthy individuals and subjects with either SCT or sickle cell disease, the difference between the DEHYD and HYD conditions observed in the SCT group during recovery is of clinical relevance. The persistent hyperviscosity during the recovery period, in association with the other physiological/biological changes previously observed in the hours following an exercise bout in SCT carriers, might have increased the risks for clinical complications in this population at that time. Tripette et al (47), for example, reported that exercise increases the
plasma L-selectin level of SCT carriers above baseline during the recovery, and Hedreville et al (31) observed a further deterioration in the autonomic nervous system activity of SCT carriers during the recovery. Altogether, hyperviscosity, a decrease in red blood rigidity, an increase in adhesion molecules, and autonomic imbalance could indicate increased risk for adverse events during the recovery of an exercise. Nevertheless, as at rest, hyperviscosity is not always observed in SCT carriers in response to exercise (40). Although not addressed in this study, this heterogeneity could result from hemorheological compensation, such as lowered hematocrit to compensate for the reduction in RBC deformability (40). Improving physical fitness might potentially be a way to improve the hemorheological profile of SCT carriers, as demonstrated in non-carriers (10, 11). Moreover, the SCT sportsmen from the study of Monchanin et al (40) were fitter than the SCT sportsmen from the present study, as indicated by the difference in Ppeak reached during the maximal incremental exercise test. The recent findings of Aufradet et al (3) support the hypothesis that training and physical fitness can modulate vascular adhesion phenomena, but this hypothesis needs to be rigorously tested. In addition, it should be noted that hemorheological responses to exercise are dependent on the type of exercise (16), with some being more stressful for SCT carriers (40, 48) than others (14, 22).

Of greater importance, when ad libitum hydration was offered to the SCT carriers, their $\eta_b$ decreased during recovery and reached the very low values found in the CONT group. Similar to the findings in subjects with no hemoglobinopathy, the Hct of the SCT carriers was unchanged by exercise (plasma volume change was minimal) and $\eta_p$ increased slightly at the end of exercise before returning to baseline during the recovery. Therefore, the decrease in $\eta_b$ in the SCT carriers during the recovery of the HYD condition (-13% from baseline) was related to a decrease in red blood cell rigidity below baseline. This reduction in $\eta_b$ and red blood cell rigidity might limit the risks for microvascular alterations, which was advanced to
explain post-exercise complications in SCT carriers (19, 20). This novel observation
strengthens the findings of Bergeron et al (8) showing beneficial effects of hydration on
sickling in two walking SCT carriers. The reduction in red blood cell rigidity in the exercising
SCT carriers was unexpected, but it had already been described during a short ramp exercise
test (40). As extensively discussed (22, 24, 40), the increases in oxidative stress (44) and
lactate (15) during exercise may have different effects on the red blood cells from trained and
sedentary individuals, with the former being better protected against a reduction in red blood
cell deformability, as observed in the present study. Although the effects of hydration and
dehydration on the exercise physiological responses of SCT carriers appear to be the same, ad
libitum hydration was able to normalize during the recovery the hemorheological alterations
observed at rest. Hydration probably plays a key role in the hydration status of SCT carriers’
red blood cells, which are known to be moderately dehydrated at times (21), leading to a
normalization of their deformability. In contrast, in the DEHYD condition, the red blood cell
rigidity of the SCT carriers increased during recovery above the values found at the end of
exercise and returned to the elevated baseline level; this may explain why $\eta_b$ remained
elevated during recovery in that condition. Although the present study did not specifically
investigate the reason for this late re-increase in red blood rigidity in the DEHYD condition,
recent findings suggest that lipid peroxidation of red blood cells during the recovery of a
submaximal exercise is specific to SCT carriers and that this could affect the red blood cell
membrane (46). We also examined blood smears from two SCT carriers during the DEHYD
and HYD conditions. We observed few sickled red blood cells at rest or at the end of exercise
(< 0.5%), and no change induced by exercise whatever the experimental conditions. The red
blood cell alterations and sickling rate were probably of low magnitude during recovery since
the cell rigidity values did not differ between baseline and recovery periods.
Alternate view and conclusion

The interpretation of hyperviscosity in SCT carriers is difficult since recent findings support the viewpoint that hyperviscosity might be beneficial for vascular adaptation (13, 23). The increase in blood viscosity may increase shear stress and stimulate vasodilation through a nitric oxide-dependent mechanism, thereby promoting adequate oxygen delivery to tissues (49). Unfortunately, hemodynamic parameters like vascular resistance were not measured in the present study and this hypothesis needs to be tested. Vincent et al (51) observed that SCT carriers are characterized by lower capillary tortuosity, reduced counts of microvessels with diameters less than 5 µm, and a higher percentage of broader microvessels, i.e., with diameters greater than 10 µm, compared with individuals without hemoglobinopathy. This suggests that SCT carriers undergo chronic vascular remodeling in order to compensate as much as possible for the hemorheological disorders.

As noted above, the mean blood viscosity values of the SCT carriers were close to the values found in sickle cell hemoglobin C disease (45), and Austin et al (4) demonstrated that SCT carriers are prone to thromboembolic complications. We therefore suggest that the chronic hyperviscosity (found in approximately one third of SCT carriers), which is associated with other exercise-induced biological changes previously described, should be considered as a risk factor for exercise-related sudden death and exercise-related medical complications in SCT carriers, especially during the recovery. However, the main finding of the present study is that ad libitum hydration is able to normalize blood viscosity in SCT carriers. This normalization, in association with the chronic structural adaptation of microvessels (i.e., larger microvessels), may favor normal blood flow and adequate tissue perfusion in this population. Physicians and sports trainers should vigorously promote adequate hydration in
SCT carriers before, during and after exercise to spare them as much as possible from clinical
problems.

Acknowledgements:

The present study has been funded by the Institut de Recherche et Développement (IRD) with
the CORUS 2 program.
References


Table 1: Subject characteristics and maximal exercise response parameters

<table>
<thead>
<tr>
<th></th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Age (yrs)</th>
<th>HRmax (beats.min⁻¹)</th>
<th>Ppeak</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONT</td>
<td>180 ± 10</td>
<td>70.2 ± 6.6</td>
<td>25.3 ± 1.9</td>
<td>180 ± 14</td>
<td>195 ± 35</td>
</tr>
<tr>
<td>SCT</td>
<td>176 ± 7</td>
<td>65.1 ± 7</td>
<td>26.4 ± 2.0</td>
<td>186 ± 7</td>
<td>180 ± 33</td>
</tr>
</tbody>
</table>

CONT = control group, SCT = sickle cell trait carriers, HRmax = maximal heart rate, Ppeak = maximal power output.

Table 2: Effects of prolonged submaximal exercise and hydration/dehydration on weight, rectal temperature and heart rate in the two groups

<table>
<thead>
<tr>
<th></th>
<th>Weight (kg)</th>
<th>Rectal temperature (°C)</th>
<th>Heart rate (beats.min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>End of Exercise</td>
<td>Rest</td>
</tr>
<tr>
<td>CONT</td>
<td>DEHYD</td>
<td>70.2 ± 6.6</td>
<td>68.6 ± 6.6*</td>
</tr>
<tr>
<td></td>
<td>HYD</td>
<td>69.9 ± 6.2</td>
<td>69.8 ± 6.6</td>
</tr>
<tr>
<td>SCT</td>
<td>DEHYD</td>
<td>65.1 ± 7.0</td>
<td>64.1 ± 7.0*</td>
</tr>
<tr>
<td></td>
<td>HYD</td>
<td>64.6 ± 7.0</td>
<td>64.8 ± 6.0</td>
</tr>
</tbody>
</table>

CONT = control group, SCT = sickle cell trait carriers, DEHYD = dehydration, HYD = hydration. *difference between rest and end of exercise (P < 0.05).

Table 3: Hematocrit and plasma viscosity at rest, at the end of the prolonged submaximal exercise and at two hours recovery in the two groups and in the two conditions (hydration/dehydration)

<table>
<thead>
<tr>
<th></th>
<th>Hematocrit (%)</th>
<th>Plasma viscosity (mPa.s⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>End of Exercise</td>
</tr>
<tr>
<td>CONT</td>
<td>DEHYD</td>
<td>46.8 ± 3.6</td>
</tr>
<tr>
<td></td>
<td>HYD</td>
<td>45.3 ± 3.1</td>
</tr>
<tr>
<td>SCT</td>
<td>DEHYD</td>
<td>46.5 ± 3.0</td>
</tr>
<tr>
<td></td>
<td>HYD</td>
<td>47.0 ± 2.6</td>
</tr>
</tbody>
</table>

CONT = control group, SCT = sickle cell trait carriers, DEHYD = dehydration, HYD = hydration. *difference between rest and end of exercise (P < 0.05); ‡difference between end of exercise and recovery (P < 0.05).
Figure legends:

Figure 1: Blood viscosity at rest, at the end of the prolonged submaximal exercise and at two hours recovery in the two groups and in the two conditions (hydration/dehydration). †statistical difference (P < 0.05).

Figure 2: Red blood cell rigidity (Tk index) at rest, at the end of the prolonged submaximal exercise and at two hours recovery in the two groups and in the two conditions (hydration/dehydration). †statistical difference (P < 0.05).