Physiological examination of exercise-induced hemoconcentration and postexercise hemodilution

Abstract of PhD Thesis

Zsolt Komka M.D.

Doctoral School of Sport Sciences
Hungarian University of Sports Science

Supervisor: Miklós Tóth professor, MD. PhD. DSc

Official reviewers: Katalin Müllner associate professor, MD. PhD
Mária Szekeres associate professor, MD. PhD

Budapest
2025

1 INTRODUCTION

The circulatory system, the body's fluid balance, and the finely coordinated regulation of these systems are essential for athletes' optimal performance and health preservation. The intravascular fluid can change dynamically during exercise and physical activity, but its regulation is not sufficiently clarified, although these changes basically determine hemodynamics and physical performance. It is important to understand the role of blood density on circulation, as well as its effects and changes related to athletic performance. The main goal of our research was to better understand the reorganization of fluid spaces during the short-term maximum load of normally hydrated (euhydrated) athletes and their dehydrated state as a result of long-term physical exercise.

1.1 HEMOCONCENTRATION

It is known that the intramuscular concentration of metabolites increases during physical exertion, their accumulation can change the osmotic gradient. This, in addition to increased arterial pressure and sympathetic nervous system activity, results in filtration of plasma into the interstitial space, known as the plasma shift mechanism. Cellular blood components and red blood cells remain inside the blood vessels, so their concentration increases, the blood becomes denser, so the plasma shift that occurs during exercise leads to hemoconcentration.

Regarding the nature of the physical load, there is no difference based on the literature data, the phenomenon of hemoconcentration appears both in the case of static resistance load and dynamic cyclic loads, and there are even data on hematocrit changes caused by psychological stress.

In recent years, in addition to the plasma shift mechanism, some observations have shown that the human spleen - like in animal species - is capable of contraction regulated by catecholamines, especially in response to hypoxia. The human spleen contains smooth muscle cells, the contraction of which is controlled by epinephrine and norepinephrine. In a stressful situation, the human spleen, by forming the body's red blood cell reserve, can also contribute to systemic hemoconcentration with its contraction.

1.1.1 Positive effects of hemoconcentration

Several beneficial consequences of hemoconcentration during short-term exercise are known. The increased number of red blood cells due to hemoconcentration - in addition to circulatory redistribution regulated by the autonomic nervous system - can locally reduce peripheral vascular resistance by releasing the vasodilator nitric oxide (NO) and ATP, which can stimulate the formation of NO in endothelial cells. Both processes result in dilation of precapillary resistance arterioles, thereby increasing local (muscle) blood flow. As a result of hemoconcentration, during acute intense exercise, the concentration of hemoglobin and hematocrit increases, so with the circulation of denser blood at the same cardiac output, the body improves the O_2 supply to the tissues, thereby ensuring more economical functioning of the muscles and nervous system during physical exercise.

1.1.2 Negative effects of hemoconcentration

High hemoglobin and hematocrit values measured at rest are known to lead to hemodynamic instability and circulatory failure, and may be responsible for the development of thrombosis and thromboembolism due to slowed capillary circulation.
The hemoconcentration phenomenon develops in stressful situations and during intense exercise. In this case, the increased cardiac output is associated with a higher flow rate, and also causes a significant shear stress of the endothelium. The increased blood viscosity due to the higher hematocrit - which can be harmful to health in the case of a high hematocrit at rest - can be compensated during exercise by the increased flow rate and the shear stress-dependent processes of the endothelium (NO release), which improve the local capillary circulation. The warming up of the skeletal muscles during exercise, body temperature is raised to 38-40 °C, can further reduce plasma viscosity. Regarding these facts, hemoconcentration can increase performance and efficiency in the short term.

However, intravascular hemoconcentration can increase further during long-term training or exercise in extremely hot and/or humid environments - with inadequate fluid replacement. As a result of the above, the adaptive, positive effects of hemoconcentration may gradually disappear, performance may decrease and in some cases even health damage or a dangerous condition may develop.

1.2 HEMODILUTION

Fewer data are available on the cessation of hemoconcentration, the subsequent back-dilution, the process of hemodilution. Some research groups have shown that concentrated hemoglobin and hematocrit return to resting levels 30 minutes after a single acute anaerobic exercise, and there are data on the normalization of hemoglobin and hematocrit values measured 3 and 6 hours after high-intensity interval training (HIIT). However, the question arose, if hemoconcentration occurs in a few minutes, why was hemodilution only documented hours later? One of our main goals during our initial research was to clarify this phenomenon.

2 AIMS AND HYPOTHESIS

The main goal of our research was to better understand the rearrangement of fluid spaces and the phenomenon of exercise-induced hemoconcentration-hemodilution. In our initial research, we studied normally hydrated athletes during maximum dynamic load in comparison with an untrained control group.

1. In addition to the development of the methodology, the aim of our initial study was to clarify whether the phenomenon of hemoconcentration can be considered a training adaptation, or is it a phenomenon that can be detected in everyone?
2. Examining several parameters with a high sampling frequency, we asked at what intensity hemoconcentration occurs in a normally hydrated body.
3. Our aim was to collect data on the level of hemoconcentration in normally hydrated athletes and untrained persons, and to get closer to the regulation of hemoconcentration by comparing it with other blood components.
4. Our main objective was to more accurately document the phenomenon of hemodilution following exercise-induced hemoconcentration with frequent, dense sampling frequency in time, to observe its short-term dynamics.
5. The purpose of our study was also to compare hemodilution with the other investigated variables, which allows us to get closer to the regulation of back-dilution.
6. Our question also was, do the cardiac output (high flow rate) and core temperature that compensate for the high hematocrit viscosity during exercise also compensate for the process during the recovery period?

Thinking further about our results, we examined the phenomenon of hemoconcentration-hemodilution later, comparing the normal hydrated and dehydrated state of the athlete's body, under standard laboratory conditions.

1. Our goal was to better understand changes in fluid spaces and blood components in a hydrated and dehydrated states during dynamic exercise.
2. We asked, to what extent does dehydration affect the level of hemoconcentration?
3. Our aim was to examine the degree of hemodilution after maximal exercise in hydrated and dehydrated conditions, thus providing additional information for its regulation.

3 METHODS

In our initial research, we examined 12 healthy, male Hungarian kayak-canoeists (aged 18-24) at the national team level, and six age- and gender-matched, healthy, untrained control volunteers who had not previously participated in regular sports. In our follow-up study investigating dehydration, we examined 12 adult elite Hungarian kayak-canoe athletes on two occasions, 1 week apart in time, in a self-controlled manner.

In both of our studies, the subjects had to come to the examination after 2 hours fasting, we excluded the use of any medication affecting fluid balance, and the consumption of alcohol and caffeine. A dietician prescribed a diet with a standard composition in the preceding 24 hours.

In our initial study, the subjects performed incremental maximal exercise on a recumbent bicycle ergometer with a ramp protocol by spiroergometry. VO_2, VCO_2, and the respiratory quotient (RQ) were documented as continuous variables from breath to breath along with the heart rate throughout the exercise and until the end of the recovery period. In addition, data were collected at rest and during exercise in the aerobic intensity range of RQ=0.9, at RQ=1.0, which we defined as the aerobic-anaerobic transition in intensity, and at the maximum of the exercise (Max). We recorded the first 5 minutes of the recovery period with minute-by-minute sampling, as well as the 7th, 10th, and 30th minutes of restitution.

At the sampling times, heart rate, upper arm blood pressure, and core temperature were recorded, and blood samples were taken, from which total protein, albumin, glucose, hemoglobin, hematocrit, ions (Na^+, K^+), acid-base parameters (pH, lactic acid, bicarbonate) and osmolality were measured (chemistry lab and ABL 800 Flex Radiometer).

In our following study, we investigated the influencing role of dehydration in hemoconcentration and hemodilution. We examined the athletes twice, both times starting from a normal hydrated condition.

Short protocol: on the first test day (short protocol, hydrated state, HS), the subjects performed an incremental test with spiroergometry. Sampling occurred at rest, in the aerobic range (RQ=0.9), at the anaerobic threshold (RQ=1.0); at maximum load; and in minutes 5 and 30 of the recovery period. The investigated parameters were the same as in our previous study protocol.

Long protocol: on the second test day (long protocol, examination of the dehydrated state, DHS), one week later, a 120-minute aerobic preload was performed followed by spirometry on a bicycle ergometer to achieve the dehydrated condition. The exercise test was interrupted every 20 minutes for sampling. After 120 minutes, the intensity was gradually increased until maximum exhaustion, the same as the first load performed in a hydrated state. Data collection and sampling were performed at rest, before exercise, every 20 minutes during the 120-minute exercise, immediately after maximal exercise, and at 5th and 30th minutes of the recovery period.

The measured variables and parameters were the same as in our previous study.

3.1 STATISTICAL ANALYSIS

The mathematical/statistical analysis of our data was carried out in collaboration with the Budapest University of Technology and Economics. Our calculations were done in the R programming language with the help of the built-in statistical functions, and for the diagrams we used the Plotly and ggplot2 functions.

Our probability variables were not normally distributed in any of the cases, and the standard deviations did not match, so we used the Mann-Whitney U test, and set the significance level at $p<0.05$.

During our second study investigating the effects of dehydration, we performed our calculations in the Python programming language (version 3.7.3) using SciPy (1.5.0) for statistical tests, Numpy (1.19.1) for numerical calculations, Pandas (1.1.3) for data transformation, and Plotly (5.3.1) for visualization.

Our data did not show a normal distribution, nor did the standard deviations match, so we used the Mann-Whitney U test to examine our hypothesis. The threshold for significance was $p<0.05$. When reporting the results, the median values were given using the first and third quartiles.

4 RESULTS

4.1 HEMOCONCENTRATION AND HEMODILUTION IN EUHYDRATED SUBJECTS

4.1.1 Hemoglobin and hematocrit

In our initial study, the average resting hemoglobin (Hgb) and hematocrit (Hct) values of the athletes did not show a significant difference compared to the untrained control group.

During exercise, a significant increase in Hgb and Hct was measured at the anaerobic threshold (at RQ=1.0) and at the maximum of the exercise in the athlete group. Similar rising kinetics were also found in the control group, however, the change in Hgb and Hct did not show a significant difference compared to the resting initial value at any of the measured times. There was no significant difference between the blood density of the two groups at any of the measured time points.

The highest degree of hemoconcentration was measured in the 1st minute of restitution, the changes in hemoglobin levels in our athlete group correspond to an average increase of 9.59±4.18% compared to the resting value, and an even more marked increase of 11.85±2.71% confirmed in the untrained control group. In athletes, a discrete but non-significant decrease in hemoglobin-hematocrit values was observed from the 2nd minute of restitution, when compared with the resting parameters, the significance disappeared at the 10th minute of the recovery period, at the 30th minute of restitution, the parameters returned to the resting baseline values. In the control group, hemoconcentration kinetics similar to those of the athletes were observed, but without statistical significance.

4.1.2 Parameters characterizing circulation

4.1.2.1 VO_2

The VO_2 value (VO_2 = Cardiac output x $AVDO_2$), which originates indirectly from the analysis of gases, but represents the circulatory conditions and the cardiac output very well, increased significantly in both groups compared to the resting values, as expected, their maximum values were measured at the maximum load, the athletes achieved higher values. From the 2-3rd minutes of the recovery period, a rapid VO_2 decrease occurred in both groups. In athletes, the significance disappeared after 5 minutes compared to the resting VO_2 values. In the untrained control group, a slower decrease of

circulation and VO_2 was observed, the significant difference disappeared only in the 30th minute of the recovery.

4.1.2.2 Heart rate

The resting heart rate of the athletes was lower than the untrained controls. During exercise, both groups showed an adequate increase in heart rate, the highest values were measured at the peak load. After exercise, a decrease in heart rate was observed in both groups. Although the heart rate of the athletes decreased faster, we could not show a statistically significant difference between the two groups. We still measured a significantly higher heart rate 30 minutes after exercise in the controls compared to their resting values.

4.1.2.3 Systolic blood pressure

At rest, there was no blood pressure difference between the two groups, but during exercise, the athletes showed higher systolic blood pressure values; both groups reached their highest values at maximum exercise.

The systolic blood pressure values decreased after exercise, and the values returned to normal in the athletes 4 minutes after the maximum load. In the control group, the drop in blood pressure occurred earlier, from the 2nd minute of restitution, and in some cases, the systolic blood pressure even dropped below the physiological range.

4.1.3 Core temperature

In both groups, the core temperature showed a significant increase at the maximum of the load, which continued to rise until the first 3 minutes of the recovery period. After a plateau phase, only in the 30th minute of the recovery returned it to the resting baseline value. In the untrained control group, the body temperature during exercise was lower than the athletes and showed a longer plateau phase in the restitution, the resting temperature was measured in the 30th minute of the recovery period.

4.1.4 Osmolality

Osmolality increased in both groups during exercise. In athletes, the increase in osmolality at the anaerobic threshold (RQ=1.0 and above) became significant, reached its maximum at the peak load, and then a decreasing tendency was recorded. It returned to the resting value in the 10th minute of the recovery period. In the control group, we also

measured the maximum at the peak load, then a decreasing trend appeared, it returned to the rest value more slowly, only at the 30th recovery minute (R30').

Blood osmolality is predominantly dependent on sodium, potassium, chloride and bicarbonate ions, blood urea nitrogen and glucose concentration. The athletes showed a significant sodium ion increase at the maximum load, and then it decreased to the initial value from the 2nd minute of restitution. Although similar Na^+ changes were seen in the controls, we could not prove significance.

During exercise, the serum potassium level increased in both groups, which reached the significance limit at the anaerobic threshold. The highest values were shown at the peak load, after the potassium level began to decrease. The values normalized in the 3rd minute of restitution.

Total protein and albumin levels increased in both groups as a result of exercise. The athletes reached the peak value of total protein in the 3rd minute after the exercise, and in the control group at the peak load. The highest albumin values were measured at the maximum load in both groups.

In both groups, the total protein decreased back to the resting level only at the R30' time point, and the albumin levels normalized in the athletes within 10 minutes. The albumin change of the controls did not show statistical significance.

The serum glucose level of the athletes showed a significant increase at the peak load, continued to rise during the first 3 minutes of the recovery period, and then showed a decreasing trend. The significance of the increase disappeared only 30 minutes after the exercise.

In the untrained control group, we did not verify a significant increase in glucose levels during exercise or during the recovery period.

All participants arrived to the examination in a sufficiently rested and regenerated condition, which was confirmed by their resting lactate values. At the anaerobic threshold, the lactate level of both groups remained below 4.0 mmol/l, and then a significant increase in lactate occurred during the peak load, the maximum value was measured in the 2nd minute of restitution in athletes, and in the 4th minute in controls, followed by a decrease. In both groups, even at the R30' time point, we still measured higher values compared to resting parameters.

4.2 INVESTIGATION OF THE ROLE OF DEHYDRATION ON BLOOD DENSITY

Following our initial results, this study tested 12 kayak-canoe athletes twice in a row, with an average of one week difference between the two. 11 athletes completed both test protocols, 1 athlete complained of feeling unwell during the long protocol, so his test was interrupted.

4.2.1 Hemoglobin and hematocrit

At rest, before exercise, there was no significant difference in hemoglobin (Hgb) and hematocrit (Hct) values between the short (hydrated state, HS) and long (dehydration state, DHS) protocol days.

During the HS load, a significant hemoconcentration occurred at the anaerobic threshold (at RER=1.0), which continued to increase at the maximum load. After exercise, the blood density normalized in the 30th minute of the recovery period (R30').

During the long protocol (DHS), the Hgb-Hct values measured every 20 minutes rose discretely (a significant increase from the 80th minute), and after the 120-minute preload, we noticed an even more significant hemoconcentration at maximum performance, the level of which did not differ from the values measured in the HS protocol. In the 5th minute of the recovery period, the hemoconcentration decreased, and after 30 minutes, it was diluted back to the resting value (hydrated, before the exercise).

4.2.2 Body weight

During the short protocol performed in the hydrated state, the subjects' body weight loss was an average of 1.59% by the end of the exercise.

The data of the body weight and body composition tests performed before the long protocol were the same as those measured before the short protocol. During dehydration, the body weight loss was measured every 20 minutes. At the end of the procedure, the test subjects achieved an average total body weight loss of 3.69%, which corresponds to a loss of 2.8 liters of fluid.

4.2.3 Glucose and lactic acid

During the HS protocol, resting blood glucose and lactate increased at the anaerobic threshold and maximal exercise, increased even more in the 5th minute of restitution, and then began to decrease in the 30th minute of rest, however, compared to the resting value, we still measured significantly higher values.

During our dehydration test (DHS) protocol, blood sugar was between 4 and 5 mmol/l and lactic acid was below 2 mmol/l during the 120-minute preload, so neither substrate availability nor acid-base shift affected performance.

4.2.4 Performance

During the short protocol (HS), the subjects achieved an average maximum power of 301.5W at the end of the gradually increasing intensity. During the resting period, the VO_2 and HR values decreased, after 30 minutes the VO_2 returned to the initial value, but the heart rate was still elevated compared to the resting value.

The long protocol was performed at a constant average of 125W for the first 120 minutes (based on an individually adjusted RER of 0.85-0.95). Heart rate and VO_2 values increased continuously during the 120 minutes. After 120 minutes, a gradually increasing load was applied until the maximum voluntary effort, an average of 240W.

4.2.5 Core temperature

In the short (HS) protocol, the core temperature increased significantly at the maximum load, then a further increase was measured in the 5th minute of the resting period, and it returned to normal 30 minutes after the load.

During the first 120 minutes of the long protocol, the resting core temperature rose continuously and then reached its highest value at the maximum intensity. In the 30th minute of restitution, we still measured significantly elevated temperatures.

4.2.6 Osmolality

Serum osmolality increased during both loads. In the short (HS) protocol, the increase in osmolality became significant at and above the anaerobic threshold

(RER=1.0), reached its maximum at the peak load, and then normalized in the 30th minute of restitution.

During the long DHS protocol, osmolality increased gradually, reaching its maximum value at the peak load. Although the osmolality decreased discreetly during the restitution, it did not reach the starting value in 30 minutes and remained elevated throughout.

5 CONCLUSIONS

The circulatory system, the body's fluid balance, and the finely coordinated regulation of these systems are essential for the optimal performance of athletes and the preservation of their health. Despite several decades of research and numerous publications, however, the optimal hydration of the athlete's body is still not sufficiently clarified. During exercise, the body's fluid spaces are rearranged, the blood thickens, and hemoconcentration develops. Hemoconcentration is a short-term phenomenon that can have many positive benefits. However, the precise role of this, its participation in sports adaptation, and the limit at which the blood density may not increase performance, but deteriorate it, and possibly have harmful consequences for health, are not known. The results presented by our working group can bring us closer to a deeper understanding of the phenomenon and its regulatory processes.

In our studies, we examined first-class male kayak-canoe athletes in comparison with an untrained, healthy, gender-age-matched control group. The test subjects performed a cyclic dynamic load of gradually increasing intensity up to the physiological maximum, with continuous cardio-pulmonary monitoring. Our test protocol is unique in that we used a very high frequency of data collection during loading and restitution, so that rapid changes and causes can be more easily revealed.

Based on our results, hemoconcentration is not an adaptive process related to training, hemoconcentration was detected in the samples of the athletes (9.6±4.2%) and the untrained group (11.8±2.73%). The more significant changes observed in the control group can be explained by the less developed regulatory mechanisms of the untrained body for physical exertion (overcompensation). The hemoglobin concentration began to rise discretely at the beginning of the load, then we verified a significant increase at the anaerobic threshold, which showed a further increase at the maximum load. Hemoconcentration reached its maximum in the first minute of the recovery period. The process continued for 7-10 minutes, then hemodilution occurred. The reported data can be significant, since during high-intensity exercise, the viscosity of thick blood is a compensated process due to the increased flow rate (high cardiac output) and increased core temperature. This compensation can be impaired during the recovery.

The novelty of our finding is that the regulation of processes compensating for hemoconcentration and the consequent high viscosity during exercise is different, which

can shift over time. The circulation was fast regulated by the autonomic nervous system and, based on our data (heart rate, VO_2), occurred within 2-3 minutes. The regulation of the hemoconcentration back-dilution process is not sufficiently clarified, it is probably under complex, non-nervous regulation and only occurred after 7-10 minutes. Due to regulatory differences, the process can slip over time, creating a "gray zone", the so-called first described by us "Critical Hemoconcentration Zone". During the restitution, the already slowing circulation must temporarily circulate denser blood that has not yet been diluted, which may carry the possibility of performance deterioration and the development of possible circulatory disorders.

Figure 1. ***Critical Hemoconcentration Zone***

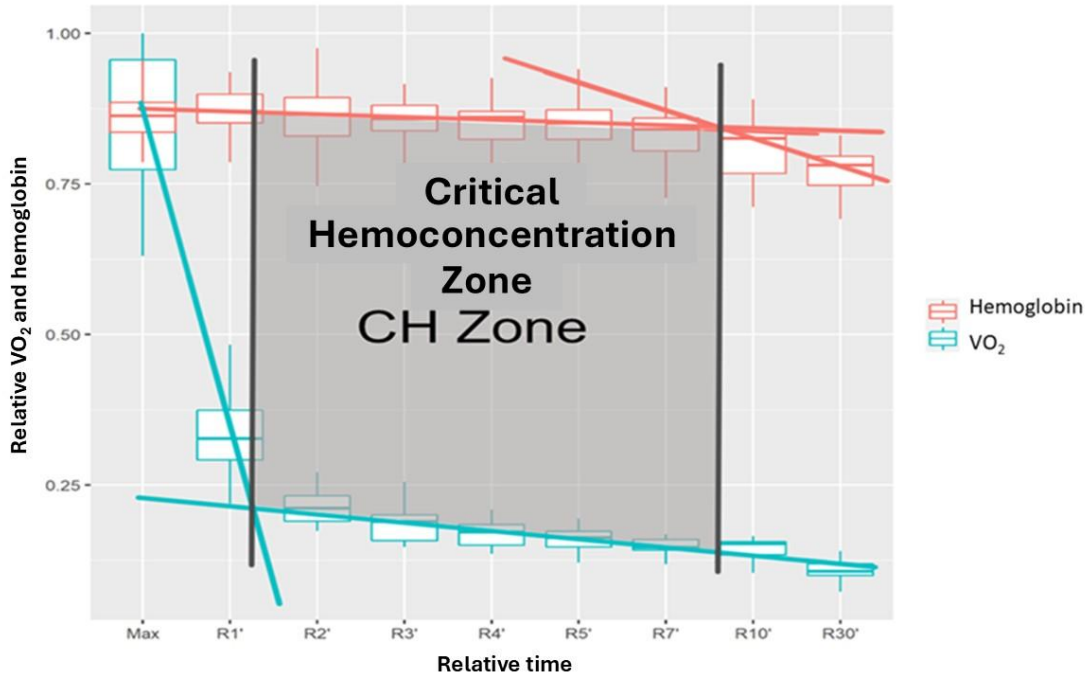

Figure legend:

Max: sampling at the maximum load; R1', R2', ..., R30': samples taken in the 1st, 2nd, ..., 30th minute of restitution, respectively.

The change of Hgb and VO_2 as a function of time during the recovery period for all individuals. Hemoconcentration occurred in all the subjects, regardless of fitness. The kinetics of both circulation (VO_2) and hemodilution during recovery are represented by 2 lines with different inclinations, fitted with small errors. The vertical coordinates of the break points indicate the endpoints of the time interval that define a critical "gray zone", the Critical Hemoconcentration Zone (CH Zone)

We concluded that the hemoconcentration that results from acute intensive anaerobic exercise is a short-term phenomenon, lasting an average of 7-10 minutes. The high blood viscosity caused by the increased hematocrit level was compensated by the increased core temperature, reaching its maximum value in the third minute of restitution and remaining elevated throughout the first 10 minutes of the recovery period. The main drive behind the plasma shift is the effective filtration rate, which, according to our results, was determined by the increase in sodium, glucose, total protein, and albumin concentrations, while the hemoglobin concentration could be influenced by both hemodynamic and adrenergic regulatory mechanisms.

Based on our initial results, we performed further studies to a deeper understanding of the role and importance of osmotic conditions, body water loss, and dehydration in the physiology of hemoconcentration and the kinetics of hemodilution. Both physical exertion and dehydration can alter the distribution and amount of fluid between the fluid spaces. We investigated the effect of acute exercise in normal hydrated and dehydrated conditions on hemoconcentration and post-exercise hemodilution. The significance of our study is that the dehydrated state was achieved by a method that realistically occurs in the everyday life of athletes, physical exertion, not by exogenous methods (e.g. sauna) or by using diuretics.

Our results support the knowledge that hemoconcentration is a phenomenon related to exercise, mostly to its intensity. The significant, 3.69% total body weight loss and dehydration caused during the 120-minute preload did not increase the amount of hemoconcentration as expected. It also had no effect on the dynamics of hemodilution after exercise, although similar hemoconcentration was observed at a lower peak load in the dehydrated state. Hemoconcentration in both hydrated and dehydrated states started to develop in the extensive aerobic intensity range, reaching its maximum at the peak load.

The dynamics of hemodilution were similar in the dehydrated and hydrated state of the athletes. Based on our results, hemoconcentration does not depend on dehydration, it is mainly determined by the intensity of the load. For both the HS and DHS protocols, hemoglobin-hematocrit values returned to resting values by the 30th minute of the recovery period. In the regulation of hemodilution following exercise-induced hemoconcentration, it should be noted that complete hemodilution occurred even in a dehydrated state at the 30th minute of restitution, however, the osmolality value remained

elevated due to lack of fluid. Further studies are needed to clarify these regulatory processes in the future.

6 LIST OF OWN PUBLICATION

6.1 Publications related to the dissertation

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