

<https://doi.org/10.15407/ujpe63.9.809>

A.A. GUSLISTY,¹ N.P. MALOMUZH,² A.I. FISENKO²

¹ Odesa Regional Medical Center of Mental Health
(9, Vorobyov Str., Odesa 65006, Ukraine; e-mail: aguslisty@gmail.com)

² I.I. Mechnikov National University of Odesa
(2, Dvoryans'ka Str., Odesa 65026, Ukraine; e-mail: mnp@onu.edu.ua)

OPTIMAL TEMPERATURE FOR HUMAN LIFE ACTIVITY

The optimal temperature for the human life activity has been determined, by assuming that this parameter corresponds to the most intensive oxygen transport in arteries and the most intensive chemical reactions in the cells. The oxygen transport is found to be mainly governed by the blood saturation with oxygen and the blood plasma viscosity, with the both parameters depending on the temperature and the acid-base balance in blood. Additional parameters affecting the erythrocyte volume and, accordingly, the temperature of the most intensive oxygen transport are also taken into account. Erythrocytes are assumed to affect the shear viscosity of blood in the same way, as impurity particles change the suspension viscosity. It is shown that the optimal temperature equals 36.6 °C under normal environmental conditions. The dependence of the optimal temperature for the human life activity on the acid-base index is discussed.

Keywords: blood shear viscosity, erythrocytes, hemoglobin saturation with oxygen, acid-base balance.

1. Introduction

Regularities in the existence of living matter attracted the interest of physicists since the very beginning of their discovery. This is quite natural, because living organisms consist of molecules and ions, and the motion of those components and their interaction with one another are governed solely by physical laws. The temperature value required for the normal human life, which is equal to 36.6 °C, is not an exception from this picture. The corresponding deviations arise as a result of some diseases. If the temperature increases to 42 °C or decreases to 32 °C, there appears the danger for a person to die [1].

As a rule, the threshold temperature magnitude is associated with peculiarities of the protein functioning in living organism [1–3]. But proteins are not isolated objects. Usually, they are located in an aqueous biophysical solution. Therefore, a hypothesis was put forward in works [4–7] that this is the properties of water that determine the existence limits for the living matter. In particular, this is the temperature interval of human life. In agreement with this

hypothesis, in works [8–10], it was shown that 42 °C is a temperature, at which the crystal-like character of the thermal motion of water molecules disappears. In a more general sense, this is a temperature of a dynamic phase transition in water, which results in the change of the molecular thermal motion and in the loss of an ice-like structure by water at higher temperatures [5].

In works [11], it was found that 36.6 °C is also a characteristic temperature for water. It is a temperature, at which the isobaric heat capacity of water is minimum under the normal pressure. This combination forms the most favorable conditions for the heat exchange in mammalian organisms. In turn, the thermal processes in a living organism are closely related to oxidation ones. In other words, the oxidation processes should also be characterized by extreme properties at a temperature of 36.6 °C.

In this work, we found that the rate of oxygen transport by human blood vessels is really characterized by a maximum at 36.6 °C. Furthermore, we established some important factors that can shift this temperature to either side. It is probable that those factors are responsible for an appreciable shift of the temperature for the normal life activity of birds and some mammals [12]. The regulation of the oxygen

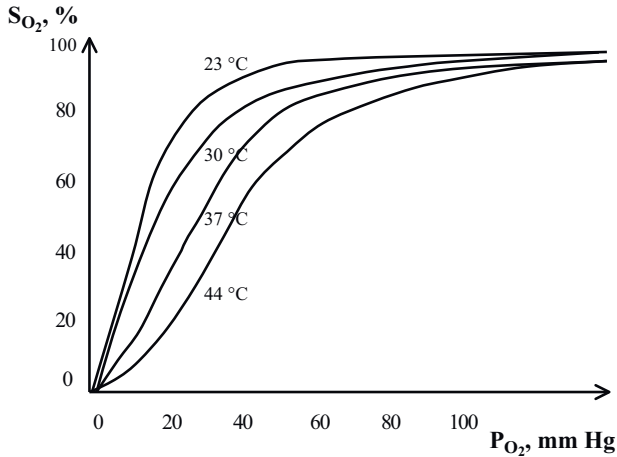


Fig. 1. Dependences of the degree of saturation of hemoglobin with oxygen in blood, S_{O_2} , on the partial oxygen pressure P_{O_2} at various temperatures (according to work [14])

transport is tightly related to directions of the development of drugs. Therefore, the formulation of a new viewpoint on this problem is very important.

2. Definition of Optimal Temperature

Let us define the temperature that is optimal for the human life as follows. We assume that the optimal temperature T_0 corresponds to the maximum of the oxygen flux $J_{O_2}(T, \text{pH}, q)$ through human vessels:

$$J_{O_2}(T = T_0, \text{pH}, q) = \max, \quad (1)$$

where T is the temperature, pH the measure for the concentration of hydrogen ions in arterial blood, and q the set of all other essential parameters. We have to note at once that the pH index of acid-base balance is the sum of two terms corresponding to a reducible, $\text{pH}(T)$, and irreducible, pH' , components:

$$\text{pH} = \text{pH}(T) + \text{pH}'.$$

In what follows, the main attention is focused on the dependence of $J_{O_2}(T, \text{pH}, q)$ on the irreducible component pH' . The same is true for the parameter set q .

Table 1. $\alpha(T, \text{pH}_0)$ -values at various temperatures

$T, ^\circ\text{C}$	23	30	37	44
$\alpha(T)$	2.66	2.33	1.66	1

In a more general sense, the condition

$$J_{O_2}(T = T_0, \text{pH} = \text{pH}_0, q = q_0) = \max \quad (2)$$

determines all basic parameters of the human state. But, in this work, our main task is to determine the optimal temperature at the equilibrium values of all other parameters. Actually, the maximum flux of oxygen is a necessary condition for the most intensive development of all other processes in human organism. The oxygen transport by a vessel per unit time, J_{O_2} , is proportional to the product of the blood flow in this vessel, Q_B , and the degree of hemoglobin oxygen saturation in blood, S_{O_2} :

$$\begin{aligned} J_{O_2}(T, \text{pH}, q) &= \\ &= Q_B(\Delta P)n(T, \text{pH}, q)S_{O_2}(T, P_{\text{osm}}, \text{pH}, q). \end{aligned} \quad (3)$$

Here, $n(T, \text{pH}, q)$ is the concentration of hemoglobin molecules in blood, P_{osm} the osmotic pressure of oxygen in erythrocytes, and ΔP stands for the pressure drop between two vessel cross-sections separated by a distance l . The relation between the pressure drop, vessel radius r , length l of a vessel section, and blood flow through the vessel cross-section reads [13]

$$Q_B(\Delta P) = \frac{\pi r^4}{8l\eta} \Delta P, \quad (4)$$

where η is the dynamic shear viscosity of blood.

The dependences of the degree of hemoglobin oxygen saturation in blood, S_{O_2} , on the oxygen osmotic pressure and the temperature were determined in works [14–16] (see Fig. 1). They can be approximately described by the formula

$$S_{O_2}(T, P_{\text{osm}}) = \text{th}(\alpha(T, \text{pH}_0), P_{\text{osm}}), \quad (5)$$

where the values of the coefficient $\alpha(T, \text{pH}_0)$ are quoted in Table 1. Hence, the oxygen transport through the vessel depends on the temperature according to the formula

$$J_{O_2}(T, \text{pH}_0, q_0) = \chi(r, l, \Delta P)F(T, P_{\text{osm}}, \text{pH}_0, q_0), \quad (6)$$

where

$$\chi = \frac{\pi r^4}{8l} n(T, \text{pH}, q) \Delta P,$$

$$F(T, P_{\text{osm}}, \text{pH}_0, q_0) = \frac{S_{O_2}(T, P_{\text{osm}}, \text{pH}_0, q_0)}{\eta(T, \text{pH}_0, q_0)}.$$

In other words, the temperature optimal for the human life activity, T_0 , is determined from the condition

$$F(T = T_0, P_{\text{osm}}, \text{pH}_0, q_0) = \max. \quad (7)$$

As one can see, the value of the optimal temperature for the life activity substantially depends on the blood shear viscosity $\eta(T, \text{pH}_0)$.

3. Blood Shear Viscosity

This issue is considered in detail in our separate work. We assume that the blood viscosity is determined by the same mechanism as the viscosity of suspensions, with erythrocytes playing the role of suspension particles, and the blood plasma the role of solvent. All other components – such as leukocytes, platelets, and blood plasma proteins – occupy a much smaller fractional volume and make a significantly smaller contribution to the shear viscosity. Note that although the linear dimensions of leukocytes are only 1.5–2 times smaller than those of red blood cells, their concentration in the blood is two orders of magnitude lower.

According to this qualitative picture, the blood shear viscosity can be written in the form (see work [17])

$$\eta(T, p) = \eta_p(T, \text{pH})\Gamma(\Psi), \quad (8)$$

where $\eta_p(T, \text{pH})$ is the blood plasma viscosity,

$$\Gamma(\Psi) = \frac{\Psi(1 - \Psi)}{\Psi(1 - \Psi) + 1 - \sqrt{1 + \Psi^2(1 - \Psi)}},$$

and the quantity

$$\Psi = \frac{R_{\text{er}}^3}{R^3} \quad (9)$$

is the ratio between the average erythrocyte volume and the volume of a net cell with radius R . Note that formula (8) was obtained, by using the cell method, which is completely consistent with Einstein's [18, 19] and Batchelor's [20] formulas for dilute and moderately dilute suspensions, respectively. The cell radius R , in turn, is related to the mean distance R_G between erythrocytes by the formula

$$R = (\alpha_0 + \alpha_1\Phi + \alpha_2\Phi^2 + \dots) R_G, \quad (10)$$

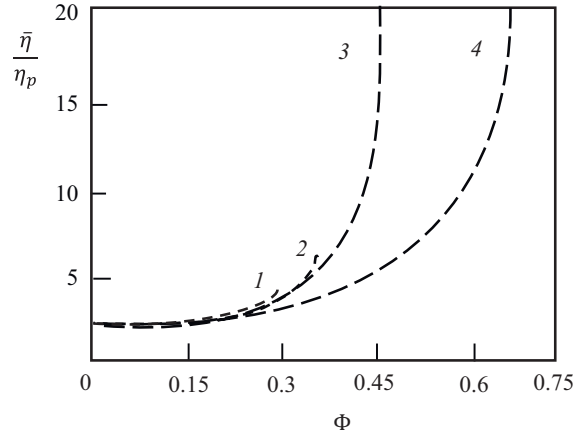


Fig. 2. Dependences of the relative blood viscosity with respect to the plasma viscosity on the specific volume Φ . Curve 1 corresponds to Einstein's formula for dilute suspensions [18], curve 2 to Batchelor's formula [20], curve 3 was plotted according to formulas (8)–(11), and curve 4 corresponds to a model formula for a dense suspension [28]

where $\Phi = \frac{\pi}{3} \frac{R_{\text{er}}^3}{R_G^3}$ is the volume fraction of blood plasma occupied by erythrocytes. According to work [17], the coefficients α_i in expansion (10) equal

$$\begin{aligned} \alpha_0 &= \left(\frac{6}{2.5\pi}\right)^{1/3} = 0.93, \\ \alpha_1 &= \frac{\pi\alpha_0^4}{18} \left(\left(\frac{6}{\pi\alpha_0^3}\right) - 5.2\right) = 0.127, \\ \alpha_2 &= 0.03. \end{aligned} \quad (11)$$

As was shown in work [17], the cell approach used by us allows the behavior of the suspension shear viscosity to be successfully described up to $\Phi \approx 0.45$, which far exceeds the upper application limits for the expressions obtained in the framework of the hydrodynamic perturbation theory [21, 22]. The function $\Gamma(\Psi)$ immediately depends only on the specific volume Φ ($\Psi = \Psi(\Phi)$; and the values of R_G are also determined by the quantity Φ : $R_G = R_G(\Phi)$). But the quantity Φ itself depends on the erythrocyte size, which varies with changes in both the temperature [23, 24] and the acid-base index [25, 26], as well as on the presence of certain ingredients, in particular, lactate [27]. The dependence of the shear viscosity on the specific volume is shown in Fig. 2.

To a certain extent, we may neglect the dependence of the erythrocyte volume on the temperature and

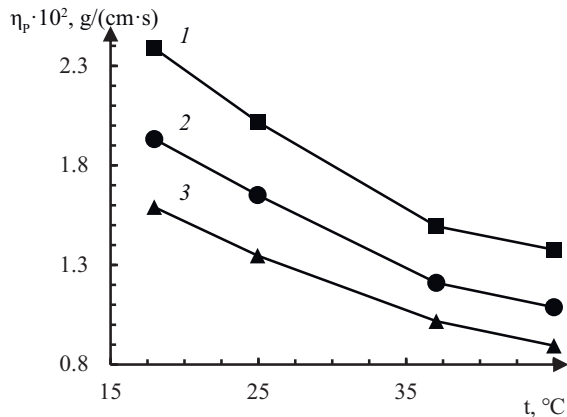


Fig. 3. Temperature dependences of the blood plasma (1), blood serum (2), and blood serum at myeloma (3) [30]. The serum is obtained by centrifuging the blood plasma

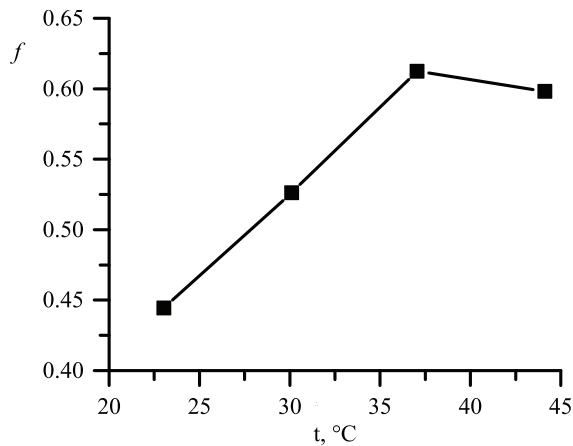


Fig. 4. Temperature dependence of the function f [Eq. (13)] for fixed values of blood parameters

Table 2. $f(T, P_{osm}, pH_0, q_0)$ -, S_{O_2} -, and η_p -values at various temperatures

$T, ^\circ C$	23	30	37	44
S_{O_2}	0.98	0.95	0.92	0.88
η_p	2.2	1.8	1.5	1.45
$f(T, P_{osm}, pH_0, q_0)$	0.45	0.53	0.61	0.60

the acid-base index and assume that all other parameters of blood, q , acquire the corresponding equilibrium values. In this approximation, the optimal temperature of human life is determined by the relation

$$f(T = T_0, P_{osm}, pH_0, q_0) = \max, \quad (12)$$

where, unlike the function $F(T, P_{osm}, pH_0, q_0)$, the function $f(T, P_{osm}, pH_0, q_0)$ depends only on the shear viscosity of blood plasma:

$$f(T, P_{osm}, pH_0, q_0) = \frac{S_{O_2}(T, P_{osm}, pH_0, q_0)}{\eta_p(T, pH_0, q_0)}. \quad (13)$$

4. Optimal Temperature under Various Conditions

Let us determine the optimal temperature for the human life activity, provided that the acid-base index has normal values $pH_0 = 7.35 \div 7.44$, and a change in the erythrocyte volume can be neglected. In this case, the optimal temperature T_0 is determined by formula (12), in which the degree of blood oxygen saturation is determined either from Fig. 1 or by the approximate formula (5). Let us use the temperature dependence for the shear viscosity of blood plasma that was determined in work [29] (see Fig. 3).

The numerical values of the function $f(T, P_{osm}, pH_0, q_0)$ for the osmotic oxygen pressure $P_{osm} = 100$ mm Hg, which is typical of pulmonary arteries, are quoted in Table 2. Attention is drawn by the fact that the function $f(T, P_{osm}, pH_0, q)$ has a real maximum at a temperature of 37 °C (Fig. 4). Unfortunately, because of the lack of experimental data concerning the temperature dependence of the shear viscosity of blood plasma, we cannot determine the optimal temperature T_0 more accurately.

Let us consider, at the qualitative level, the influence of two important factors, the lactate and alcohol levels, on the position of the $f(T, P_{osm}, pH_0, q)$ maximum, i.e. on the optimal temperature value. It is well known that, when children eat food with a high level of carbohydrates and fats, the lactate level in their organisms increases, which is accompanied by the elevation of their body temperature. With the growth of the lactate level in blood, the level of hemoglobin oxygen saturation in the blood decreases, and the blood viscosity increases due to an increase of the average erythrocyte volume. As a result, the maximum of the dependence $f(T, P_{osm}, pH, q)$ shifts toward higher temperatures, which correlates with the temperature elevation in children. In the case of adults, the counteraction of a compensatory mechanism [43, 44] has to be taken into account as well.

Besides all that, there are a few important details. In particular, as the lactate level increases from 0.9 to 1.9 mmol/l, the hemoglobin ability to bind

oxygen decreased by 5%. If the lactate level grew to 11.04 mmol/l, this hemoglobin property decreased by 15%. At the same time, the pH index became shifted toward the acid side to $\text{pH} = 7.4 \div 7.0$ [36], which stimulated, in turn, the growth of the average erythrocyte volume. If the lactate content in blood increased from 1.9 to 12.3 mmol/l, the erythrocyte volume increased from 91.4×10^{-15} to 93.9×10^{-15} cm³ [37]. As was shown in work [27], the increase of the lactate content in blood from 1 to 10 mmol/l led to the increase of the blood viscosity from 5.2 to 6 cP at a shear rate of 0.1 s^{-1} .

If alcohol penetrates into blood, erythrocytes change their volume and shape. They become shrinkaged (it is common to say that the erythrocytes transform into echinocytes). In particular, the erythrocyte volume amounts to 91.5×10^{-15} cm³ at the occasional use of alcohol. At the regular use of alcohol, this volume reaches 96.2×10^{-15} cm³ for men and 98.0×10^{-15} cm³ for women [35]. The penetration of alcohol into blood shifts the pH value toward the acid side [38, 39]. This is a result of the alcohol oxidation to water and carbon dioxide in liver. Carbon dioxide penetrates into erythrocytes in blood, where, under the carbamide action, it transforms into the carbonic acid. The latter dissociates into the H^+ and HCO_3^- ions. If the alcohol dose equals 0.11–3.91 g/kg (the alcohol mass per kilogram of body mass), the blood viscosity increases by 7.4% [34].

The hemoglobin oxygen saturation in blood also changes. In particular, according to work [40], an alcohol dose of 0.5 g/kg resulted in the reduction of the hemoglobin saturation by 0.9%, by 2.9% for a dose of 0.78 g/kg [41], by 6% for a dose of 1.1 g/kg, and, when the dose was 1.5 g/kg, the reduction of the hemoglobin oxygen saturation reached 10% [42].

Hence, the use of alcohol leads to the growth of the blood viscosity and the reduction of the hemoglobin oxygen saturation. As a result, the maximum of the function $f(T, P_{\text{osm}}, \text{pH}_0, q_0)$ becomes shifted toward higher temperatures. If the compensatory mechanisms are not capable of lowering the optimal temperature, the probability of acute ischemic states will grow, which often takes place.

5. Discussion of the Results Obtained

Hence, in this work, we found an important relation between a temperature of 36.6 °C and a temperature

that is optimal for the human life activity, i.e. the temperature corresponding to the maximum oxygen transport to human tissues. As a result, conditions for the maximum intensity of chemical reactions in human cells are created.

Another factor that provides a sufficient condition for the chemical reactions to achieve the maximum intensity is the outflow of carbon dioxide from the tissues. This is a factor that regulates the consistency of the optimal temperature definition made above with the human states at rest or at the execution of hard work. In both cases, a healthy person retains the same value of the normal temperature. This fact means that the difference between the indicated human states is mainly reduced to a change of the balance between the oxygen flux in human arteries and the counterflux of carbon dioxide in human veins. This circumstance is tightly connected with another important issue concerning the shift mechanism of the oxygen-carbon dioxide balance in human lungs. This is a challenging problem, which requires an urgent solution.

For a further progress in the study of the optimal temperature for the human life activity, detailed researches of the degree of arterial blood saturation with oxygen and the venous blood saturation with carbon dioxide, as well as their dependence on the acid-base pH index and all other important blood parameters q , are required.

Furthermore, in the next work, we plan to present the results of our research concerning the character of deviations from the optimal human condition and their dependence on changes in the temperature and acid-base balance, as well as on the blood parameters that are the most varied due to various diseases. In other words, we plan to construct a multidimensional surface of human states, where, besides the global maximum that is observed at $T_0 = 36.6$ °C and $p_0 = 744$ mm Hg, there are also local maxima corresponding to the relative stability of a human state.

To summarize, we would like to sincerely thank Prof. Leonid Bulavin and the participants of the seminar chaired by him at the Chair of Molecular Physics of the Kyiv National University, as well as the participants of the international conference PLMMP-2018 – first of all, these are Prof. Mykola Lebovka and Prof. Longin Lysetskyi – for a detailed and topical discussion of the results of this work.

1. N.P.O. Green, G.W. Stout, D.J. Taylor. *Biological Science* (Cambridge Univ. Press, 1997).
2. G.L. Zubay, W.W. Parson, D.E. Vance. *Principles of Biochemistry: Energy, Proteins, Catalysis* (McGraw-Hill College Division, 1995).
3. F.J. Ayala, J.A. Kiger, jr. *Modern Genetics* (Benjamin, 1980).
4. L.A. Bulavin, N.P. Malomuzh. Upper temperature limit for the existence of living matter. *J. Mol. Liq.* **124**, 136 (2006).
5. A.I. Fisenko, N.P. Malomuzh. The role of the H-bond network in the creation of the life-giving properties of water. *Chem. Phys.* **345**, 164 (2008).
6. A.I. Fisenko, N.P. Malomuzh. To what extent is water responsible for the maintenance of the life for warm-blooded organisms? *Int. J. Mol. Sci.* **10**, 2383 (2009).
7. L.A. Bulavin, N.P. Malomuzh. Dynamic phase transition in water as the most important factor provoking the protein denaturation in warm-blooded organisms. *Fiz. Zhivogo* **18**, 16 (2010) (in Russian).
8. N.P. Malomuzh, A.V. Oleinik. The origin of the kinematic shear viscosity of water. *Zh. Struk. Khim.* **49**, 1093 (2008) (in Russian).
9. L.A. Bulavin, A.I. Fisenko, N.P. Malomuzh. Surprising properties of the kinematic shear viscosity of water. *Chem. Phys. Lett.* **453**, 183 (2008).
10. L.A. Bulavin, T.V. Lokotosh, N.P. Malomuzh. Role of the collective self-diffusion in water and other liquids. *J. Mol. Liq.* **137**, 1 (2008).
11. *The Engineering ToolBox. Water – Thermal Properties* [http://www.engineeringtoolbox.com/water-thermal-properties-d_162.html].
12. D. Randall, W. Burggren, K. French, R. Eckert. *Animal Physiology: Mechanisms and Adaptation* (Freeman, 1997).
13. L.D. Landau, E.M. Lifshitz, *Fluid Mechanics* (Pergamon Press, 1993).
14. B. Tremey, B. Vigue. Changes in blood gases with temperature: Implications for clinical Practice. *Ann. Fr. Anesth. Reanim.* **23**, 474 (2004).
15. S. Mrozek, F. Vardon, Th. Geeraerts. Brain temperature: physiology and pathophysiology after brain injury. *Anesth. Res. Pract.* **2012**, 13 (2012).
16. J.-A. Collins, A. Rudensky, J. Gibson *et al.* Relating oxygen partial pressure, saturation and content: The hemoglobin-oxygen dissociation curve. *Breathe* **11**, 194 (2015).
17. N.P. Malomuzh, E.V. Orlov. A new version of the cell method for determining the viscosity of suspensions. *Kolloidn. Zh.* **64**, 802 (2002) (in Russian).
18. A. Einstein. Eine neue bestimmung der molekuldimensionen. *Ann. Phys.* **19**, 289 (1906).
19. A. Einstein. Berichtigung zu meiner arbeit: "Eine neue bestimmung der molekuldimensionen". *Ann. Phys.* **34**, 591 (1911).
20. *Hydrodynamic Interaction of Particles in Suspensions*. Edited by A.Yu. Ishlinskii, G.G. Chorny (Mir, 1980) (in Russian).
21. J. Happel, R. Brenner. *Low Reynolds Number Hydrodynamics: With Special Applications to Particulate Media* (Prentice-Hall, 1965).
22. J. Happel. Viscosity of suspension of uniform spheres. *J. Appl. Phys.* **28**, 1288 (1957).
23. G.V. Richieri, H.C. Mel. Temperature effects on osmotic fragility, and the erythrocyte membrane. *Biochim. Biophys. Acta* **813**, 41 (1985).
24. J.F. Gillooly, R. Zenil-Ferguson. Vertebrate blood cell volume increase with temperature: implications for aerobic activity. *Peer J.* **2**, 346 (2014).
25. P. Swietach, T. Tiffert, J.M.-A. Maruiz *et al.* Hydrogen ion dynamics in human red blood cells. *J. Physiol.* **588**, 4995 (2010).
26. D. Kuzman, T. Znidartit, M. Gros *et al.* Effect of pH on red blood cell deformability. *Eur. J. Physiol.* **440**, 193 (2000).
27. W.H. Reinhart, R. Gaudenz, R. Walter. Acidosis induced by lactate, piruvate, or HCl increases blood viscosity. *J. Crit. Care* **17**, 68 (2002).
28. P.V. Rand, W.H. Austin, E. Lacombe, N. Barker. pH and blood viscosity. *J. Appl. Physiol.* **25**, 550 (1968).
29. T.S. Chow. Viscosity of concentrated dispersions. *Phys. Rev. E* **48**, 1977 (1993).
30. *Blood Plasma and Serum Viscosity* [<http://www.rheosense.com/application/viscosity-of-blood-plasma-and-serum>].
31. R.I. Weed, A.I. Bowdler. Metabolic dependence of the critical hemolytic volume of human erythrocytes: Relationship of osmotic fragility and autohemolysis in hereditary spherocytosis and normal res cells. *J. Clin. Invest.* **45**, 1137 (1966).
32. E. Naeraa, E.S. Peterson, E. Boye, J.W. Severinghaus. pH and molecular CO₂ components of the Bohr effect in human blood. *Scand. J. Clin. Lab. Invest.* **18**, 96 (1966).
33. L. Cordone, A. Cupane, P.L. San Biagio, E. Vitrano. Effect of some monohydric alcohols on the oxygen affinity of hemoglobin: Relevance of solvent dielectric constant and hydrophobicity. *Biopolymers* **18**, 1975 (1979).
34. T. Hamazaki, H. Shishido. Increase in blood viscosity due to alcohol drinking. *Trombosis Res* **30**, 587 (1983).
35. H. Tonnesen, L. Hejberg, S. Frobenius, J.R. Andersen. Erythrocyte mean cell volume – correlation to drinking pattern in heavy alcoholics. *Acta Med. Scand.* **219**, 515 (1986).
36. W. Stringer, J. Porszasz, K. Wasserman, K. Maehara. Lactic acidosis as facilitator of oxyhemoglobin dissociation during exercise. *J. Appl. Physiol.* **76**, 1462 (1994).
37. J.A. Smith, R.D. Telford, M. Kolbuch-Braddon, M.J. Weidemann. Lactate/H⁺ uptake by red blood cells during exercise alters their physical properties. *Eur. J. Appl. Physiol.* **75**, 54 (1997).
38. A. Lamminpaa, J. Vilska. Acid-base balance in alcohol users seen in an emergency room. *Vet. Hum. Toxicol.* **33**, 482 (1991).

39. S. Zehtabchi, R. Sinert, B.J. Baron, L. Paladino, K. Yadav. Does ethanol explain the acidosis commonly seen in ethanol-intoxicated patients? *Clin.Toxicol.* **43**, 161 (2005).
40. I. Izumi, A. Nasermoaddelia, M. Sekine, S. Kagamimori. Effect of moderate alcohol intake on nocturnal sleep respiratory parameters in healthy middle-aged men. *Environ. Health Prev. Med.* **10**, 16 (2005).
41. P.A. Easton, P. West, R.C. Meatherall *et al.* The effect of excessive ethanol ingestion on sleep in severe chronic obstructive pulmonary disease. *Sleep* **10**, 224 (1987).
42. F.G. Issa, C.E. Sullivan. Alcohol, snoring and sleep apnea. *J. Neurol., Neurosurg. Psychiatry* **45**, 353 (1982).
43. *Biological Chemistry with Exercises and Problems*. Edited by S.E. Severin (GEOTAR-Media, 2011) (in Russian).
44. D.L. Nelson, M.M. Cox. *Lehninger Principles of Biochemistry* (Freeman, 2008).

Received 26.01.18.

Translated from Ukrainian by O.I. Voitenko

А.А. Гуслистий, М.П. Маломуж, А.І. Фісенко

ОПТИМАЛЬНА ТЕМПЕРАТУРА ЖИТТЄВОЇ АКТИВНОСТІ ЛЮДИНИ

Резюме

В роботі приймається, що оптимальна температура життєвої активності людини відповідає максимальному переносу кисню артеріями в одиницю часу. З огляду на це хімічні перетворення в клітинах будуть найбільш інтенсивними. Встановлено, що перенос кисню визначається, перш за все, ступенем насиченості крові киснем та в'язкістю плазми крові. Обидві ці характеристики залежать від температури та показника лужно-кислотного балансу крові. Враховуються також додаткові параметри, які впливають на об'єм еритроцитів, а разом з тим і на температуру максимального переносу кисню. При цьому вважається, що еритроцити впливають на зсуву в'язкість крові у той самий спосіб, як домішкові частинки на в'язкість суспензій. Показано, що за нормальних умов оптимальна температура складає 36.6 °C. Обговорюється залежність оптимальної температури життєвої активності людини від показника кислотно-лужного балансу.