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Distribution of sodium, potassium, magnesium and calcium in blood plasma

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The distribution of alkaline (Na, K) and alkaline earth (Mg, Ca) metals in blood plasma from patients infected with *hepatitis C* has been analysed.

Problems with blood composition or circulation can lead to downstream tissue disfunction.[1,2] The physiological importance of metals in human organisms, especially in blood, is well known. At low concentrations, metals play an important role in metabolism and biological processes as enzyme activators, stabilizers, functional components of proteins, *etc*. The toxic doses of metals can lead to serious health problems.[3,4]

Moreover, the distribution of metals between blood plasma and blood cells could serve as very important clinical information.[5,6] The role of alkaline and alkaline earth metals in clinical medicine, nutrition and physiology is of interest.[7–11] For example, the concentration of sodium has a beneficial effect on patients with hypo-volemic shock.[12] Potassium has a potent protective effect against cardiovascular diseases.[13] For instance, the deficiency of magnesium in blood (hypomagnesemia) causes among others cardiac arrhythmia and increased irritability of the nervous system with tetany. An excess of calcium within a cell may damage it or even cause it to undergo apoptosis.[14–16] Information on the concentrations of alkaline earth metals in the human body is also useful for diagnostics, including cancer.[6,17]

The aim of this work was to study the distribution of Na, K, Mg and Ca in blood plasma from patients infected with *hepatitis C* and to compare the results with analysis data from non-infected samples.

The amount of metals in blood samples was determined by flame atomic absorption spectrometry (FAAS) using a Hitachi 170-50 spectrometer. The following FAAS conditions were used: (i) absorption line, 589.0 nm (Na), 766.5 nm (K), 285.2 nm (Mg), 422.7 nm (Ca); (ii) electric current, 10 mA (Na, Mg) or 15 mA (K, Ca); (iii) flame, propane–butane (Na, K) or acetylene (Mg, Ca); (iv) gas pressure, 1.47×10^4 Pa (Na), 9.81×10^5 Pa (K), 2.45×10^4 Pa (Mg) or 2.94×10^4 Pa (Ca). The pressure of air was 1.47×10^5 Pa. Double-distilled water and analytical-grade reagents were used.

The blood samples were taken from 36 volunteer patients infected with *hepatitis C*. The determination of alkaline and alkaline earth metals in blood was performed without preconcentration. The samples were burnt in an ordinary furnace at 600 °C. The obtained residuals were dissolved in 10 ml of nitric acid (1:1), transferred into a 25 ml volumetric flask and diluted with double-distilled water. For comparison, 36 blood samples from patients non-infected by *hepatitis C* were also analysed. For the analysis of metals distribution in blood plasma, the immediate separation of plasma from cells was performed by centrifugation (8000 rpm). The dependence of recovery of blood plasma on the duration of centrifugation is shown in Figure 1. The full separation of plasma from cells was achieved after 8 min.

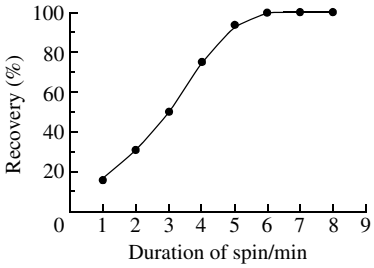

Figure 1 Dependence of the recovery of blood plasma on the duration of centrifugation.

The are summarised in Table 1. The RSD values obtained in these determinations (7.5–11.7%) indicate a high degree of homogeneity, which could be expected for blood samples.

Evidently, the concentration of potassium in blood plasma is much higher in comparison with other elements. The levels of sodium and potassium did not vary much in blood plasma from different patients. Thus, the amount of alkaline metals in blood plasma seems independent of the infection. An opposite situation was observed for the distribution of alkaline earth metals in blood plasma from different patients. Interestingly, the concentration of magnesium in the infected blood plasma (70.21 μg g^{-1}) is almost two times higher than that in the non-infected blood samples (35.91 μg g^{-1}). On the other hand, the concentration of calcium in the infected blood plasma (106.44 μg g^{-1}) is more than five times lower than that in the non-infected blood samples (580.85 μg g^{-1}). Such different behaviours of Mg and Ca are very interesting and unexpected, since the complexation ability

Table 1 Determination of Na, K, Mg and Ca in blood cells from *hepatitis C* infected and non-infected patients.

Metal	Blood cells			
	Infected by *hepatitis C* (36 samples)		Non-infected (36 samples)	
	Amount of metal[a]/μg g^{-1}	RSD (%)	Amount of metal[a]/μg g^{-1}	RSD (%)
Na	987.44	9.9	1006.31	10.2
K	1834.91	11.0	1998.96	11.7
Mg	70.21	8.4	35.91	8.8
Ca	106.44	7.5	580.85	7.9

[a]Average of five independent determinations.

Table 2 Determination of Mg and Ca in blood plasma from patients differently infected by *hepatitis C*.

Metal	Blood plasma from *hepatitis C* infected patients (25 samples)			
	After diagnosis		After medical treatment	
	Amount of metal[a]/μg g^{-1}	RSD (%)	Amount of metal[a]/μg g^{-1}	RSD (%)
Mg	70.02	9.7	49.56	9.0
Ca	105.33	7.7	325.01	8.5

[a]Average of five independent determinations.

of these two metals with chelating ligands or proteins should be very similar. The concentration of calcium slightly prevails over the concentration of magnesium in the infected and non-infected blood samples.

Table 1 clearly demonstrates that Mg and Ca exist in blood plasma in different physico-chemical forms. Possibly, the metal ions are bound to different chelators or proteins. Therefore, the determined extra amount of Mg^{2+} and hyper-low concentration of Ca^{2+} in the blood plasma from *hepatitis C* infected patients are probably caused by the illness of the patients. Such deviations of concentration of electrolytes could influence the dehydration and osmosis processes and, consequently, promote the progress of disease. In conclusion, the regulation of concentration of magnesium and calcium might be an attractive for anti-viral protection for the *hepatitis C* infected subjects. At any rate, the initial observations show such a tendency that the concentration of Mg and Ca in the blood plasma could be a signal of the seriousness and depth of the disease. Finally, the concentrations of Na and K do not vary significantly in blood plasma from the infected and non-infected patients.

The distribution of magnesium and calcium levels depending on the duration of infection was also investigated. For this, the blood samples from patients differently infected with *hepatitis C* were checked. Table 2 shows the distribution of Mg and Ca in plasma of the patients having slightly different background of infection. The results demonstrate that magnesium concentration slightly decreases and calcium increases in the blood plasma with medical treatment. These results support a new hypothesis that the amount of Mg and Ca in blood plasma of untreated *hepatitis C* patients could be related to the occurrence or progression of infection. The distribution of sodium, potassium, magnesium and calcium concentrations in blood plasma were also estimated in female and male patients infected with *hepatitis C*. Absolutely no tendency, however, in the distribution of these elements between different gender patients was detected.

References

1 V. Lazar, J. Bouska, J. Stavkova and D. Klecker, *Zivocisna Vyroba*, 1990, **35**, 625.
2 L. Merson and N. Olivier, *Blood Rev.*, 2002, **16**, 127.
3 A. Bednarek, K. Pasternak and M. Karska, *Magnes. Res*., 2003, **16**, 271.
4 A. Kindness, C. N. Sekaran and J. Feldmann, *Clin. Chem*., 2003, **49**, 1916.
5 A. L. Dunne, F. Mitchell, K. M. Allen, H. W. G. Baker, S. Garland, G. N. Clarke, A. Mijch and S. M. Crowe, *J. Clin. Virol*., 2003, **26**, 239.
6 N. Hayashi and T. Takehara, *J. Gastroenterol*., 2006, **41**, 17.
7 B. Godlewska-Zylkiewicz, B. Lesniewska, M. Maj-Zurawska and A. Hulanicki, *Magnes. Bull*., 1998, **20**, 65.
8 G. D. Miller, D. D. DiRienzo, M. E. Reusser and D. A. McCarron, *J. Am. Coll. Nutrit.*, 2000, **19**, 147S.
9 S. Wang, E. H. McDonnell, F. A. Sedor and J. G. Toffaletti, *Archiv. Pathol. Lab. Med*., 2002, **126**, 947.
10 C. K. Chow, *Am. J. Clin. Nutrit.*, 2006, **84**, 1552.
11 S. Sankaralingam, K. M. Desai and T. W. Wilson, *Am. J. Hyperten*., 2006, **19**, 1167.
12 E. Hatanaka, F. M. Shimomi, R. Curi and A. Campa, *Shock*, 2007, **27**, 32.
13 H. Matsui, T. Shimosawa, Y. Uetake, H. Wang, S. Ogura, T. Kaneko, J. Liu, K. Ando and T. Fujita, *Hypertension*, 2006, **48**, 225.
14 H. W. de Valk, *Netherl. J. Med*., 1999, **54**, 139.
15 S. Vasdev, L. Longerich and P. Singal, *Nutrit. Res*., 2002, **22**, 111.
16 Z. Katzir, A. Michlin, M. Boaz, A. Biro and S. Smetana, *Isr. Med. Assoc. J*., 2005, **7**, 704.
17 D. Deheinzelin, E. M. Negri, M. R. Tucci, M. Z. Salem, V. M. da Cruz, R. M. Oliveira, I. N. Nishimoto and C. Hoelz, *Braz. J. Med. Biol. Res*., 2000, **33**, 1443.

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