

TABLE I.

Metal Ion	Concentration	lag phase (min)	growth rate ($\Delta A/\text{min}$)
–	–	16	0.050
Sm^{3+}	$\left\{ \begin{array}{l} 20 \mu\text{M} \\ 50 \mu\text{M} \\ 100 \mu\text{M} \end{array} \right.$	9	0.085
		4	0.130
		0	0.150
La^{3+}	100 μM	8	0.155
Er^{3+}	100 μM	8	0.085
Lu^{3+}	100 μM	1	0.020
Ca^{2+}	2.5 mM	7	0.095

TABLE II.

Concentration of Sm^{3+} (μM)	Average fibril thickness (nm)
0	320 \pm 62
25	210 \pm 49
50	130 \pm 31
100	78 \pm 17

Arrhenius plots of the rates of polymerisation at different temperatures gave an activation energy (E_a) of 45.8 \pm 5.6 kcal/mol for the nucleation phase and 57.2 \pm 5.8 kcal/mol for the growth phase. Ca^{2+} (5 mM) and Sm^{3+} (25 μM) reduced the E_a of the growth phase to 34.8 \pm 2.3 kcal/mol and 18.0 \pm 1.1 kcal/mol respectively. However, the E_a for the nucleation phase was little changed by either 5 mM Ca^{2+} (E_a = 38.2 \pm 4.3 kcal/mol) or 25 μM Sm^{3+} (E_a = 38.0 \pm 2.3 kcal/mol).

The ability of physiological concentrations of Ca^{2+} to influence the rate of polymerisation of collagen suggests a modulating role for calcium *in vivo*. The ability of low concentrations of Ln^{3+} to mimic these effects suggests that they may prove useful probes of the interaction between Ca^{2+} and collagen.

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The Effect of Silicon Compounds on Blood Cell Membranes

M. G. VORONKOV, V. B. KAZIMIROVSKAYA, L. I. KHOLDEEVA and T. V. SHEPKOVA

Institute of Organic Chemistry, Siberian Division of the USSR Academy of Sciences, 664033 Irkutsk, U.S.S.R.

Silicon compounds play an essential role in metabolic processes, participate in oxidative reactions and are able to change the surface potential of blood cells, which is determined by the structural integration of cell membranes [1].

If found, the compounds of this element, increasing the structural stability of cell membranes would allow one to control the sensitivity of cells to external environmental effects and provide a remedy for curing cells and the organism as a whole.

We have established that in the development of haemolytic anaemia of different types the introduction of silatranes, $\text{XSi}(\text{OCH}_2\text{CH}_2)_3\text{N}$ with $\text{X} = \text{CH}_3$, ClCH_2 , $\text{C}_2\text{H}_5\text{O}$, $(\text{CH}_3)_2\text{CHO}$ and HO which are efficient silicon donors [2, 3] increases the stability of blood red cell membranes manifested in a high osmotic and chemical resistance.

The haemolytic processes are always accompanied by the peroxidation of lipids. *In vivo* the silatranes studied inhibit free-radical and peroxidation reactions of erythrocytary membranes.

These data show that silatranes are worth studying as a promising means of prophylaxis of thrombosis. The use of $\text{I-CH}_2\text{ClSi}(\text{OCH}_2\text{CH}_2)_3\text{N}$ decreases or completely inhibits thrombocyte aggregation induced by ADP, adrenaline, thrombin and collagen. Such a combination of antioxidative and antiaggregate action is observed in neither of the known inhibitors of this process which are able to suppress the aggregation caused by only one specific inductor.

$\text{I-CH}_2\text{ClSi}(\text{OCH}_2\text{CH}_2)_3\text{N}$ increases almost two-fold the blood heparin level forming blood protein complexes which stimulate the anticoagulation system and non-enzymatic fibrinolysis in the organism.

All these effects are of great importance in natural prophylaxis of intravascular blood coagulation.

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