

AMBER ACID – NATURAL PREPARATION FOR DIABETES TREATMENT

In the case of diabetes dependent on insulin, hyperglycemia goes together with other metabolic disorders: hyperlipidemia, hyperlactatemia, and ketosis. Decrease of carbohydrate on purpose to avert hyperglycemia is the main principle of diabetes treatment. The initial criterion of this compensation is absence of sugar in the urine and reduced glucose concentration in the blood during twenty four hours. Such correction of diabetes is carried out by choosing diet, injecting insulin, taking non-hormonic preparations, which reduces amount of sugar or its combinations in the blood.

The usage of glucose in peripheral tissues changes dependent on the concentration of free fatty acid and ketotic corpuscles in blood plasma. Any increase of fatty acid or ketotic corpuscles is a reason of suppression of glucose usage in the tissues. It is visible in the case of both normal and enlarged concentration of insulin. By contrast, injection of glucose reduces oxidation speed of fatty acid and enlarges usage of glucose in peripheries. That is to say, that reduction of fatty acid and ketonic corpuscles level in blood plasma may stimulate usage of glucose in the tissues and reduce amount of glucose in plasma.

In the course of our experiments we noticed that progress of diabetes growth for rats (after injections of alloxan) depends on process of lipid circulation a lot according to glucose concentration in the blood. It was proved that animals, which had injections of alloxan, could be divided into two groups in unison called “diabetic” and “non-diabetic”. Sure enough, injections of alloxan caused increase of glucose in the blood till the trespass of norm for some rats, for others concentration of glucose remained almost unchanged.

Table 1.

Parallel changes of glucose concentration and ketotic corpuscles in the blood of rats causing diabetes with alloxan

Dimensional rate	Groups		
	Control (n-18)	After injection of alloxan	
		diabetic (n-10)	non-diabetic (n-10)
Glucose	9,8±0,4	18,0±1,5	8,7±0,7
3-hydroxybutyrate	127±6	433±66	127±14
Acetoacetate	89±6	222±32	93±10
Amount of ketonic corpuscles	213±11	651±91	212±7

Note. 40 mg/kg of alloxan was injected in proportion to 1 ml saline. Saline was injected for the control group. Three days later, concentration of indicated substances in the blood was measured. Glucose, 3-D -hydroxybutyrate and acetoacetate was measured by fluorimetric fermentative methods. For each alloxan group rats were selected in tens. Criterion for the selection was glucose concentration in the blood: under 10 mg and above 15 mg for “diabetic” and “non-diabetic” groups.

It is shown in the table that level of glucose concentration in the blood does not rise (diabetes does not develop) when injection of alloxan does not cause increase of ketotic corpuscles in the blood. That is to say, such animals do not differ from controlled, their weight does not decrease and there is no polyuria. Those animals may be ascribable to “non-diabetic” group. In other group of rats,

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“diabetic”, increase of glucose concentration and ketotic blood corpuscles, which are caused by alloxan, may be seen. A weight of these rats was 20% lower than a weight of control group. The former drank a lot of water and the polyuria was noticed. Thus, the positive correlation between glucose and amount of ketotic corpuscles in the blood was a feature of rats of “diabetic” group. Spanish researches, who are working with Spreg-Doyli rats causing diabetes with alloxan, got similar (although not identical) results. Glucose accession into peripheral tissues, for example into skeletal muscles and erythrocytes, is a process dependent on insulin, whereas ketotic corpuscles spared over the tissues freely and are independent of insulin. Thus, the findings let to guess, that having diabetes, when permeability of cell membrane disconcerts because of lack of insulin, the amount of glucose concentration in the blood may be diminished by the reduction of concentration of ketotic corpuscles.

We have investigated an impact of various natural preparations on concentration of ketotic corpuscles in the blood, and identified that sodium succinate the most effectively reduces concentration of ketotic corpuscles. It, especially, increases when the animal is starving (Table 2). After twenty four hours of starving concentration of 3-D –hidroxibutirate in blood plasma of the rats used to rise to 1,95 mg, acetoacetate – to 0,5mg (norm approximately 0,2 and 0,05mg respectively), how ever the effect of sodium succinate used to reduce concentration of ketotic corpuscles significantly (to 1,0 and 0,38mg respectively) during the thirty minutes previous to animal death.

Table 2.

An impact of sodium succinate on the concentration of ketotic corpuscles in the blood of starving rats

Dimensional metabolites	Concentration, mg	
	Saline	Sodium succinate
3-O-hidroxibutirate	1,95±0,25	1,00±0,10*
Acetoacetate	0,51±0,10	0,38±0,12
Amount of ketotic corpuscles	2,44±0,33	1,39±0,19*

Note. Rats (in tens in each group) were starving twenty-four hours. Afterwards at 9hr 30min saline or sodium succinate (50mg/kg) was injected for them. After 30 min level of concentration of ketotic corpuscles was researching by the fluorimetric fermentatic method. Deviations of average value and average effective values from normal, *p<0,005, in comparison with control group, are listed.

Mechanism, through which sodium succinate may reduce concentration of ketonic corpuscles and glucose, is indeterminate. It is may be guessed, that oxidation of succinate, which may intensify metabolism in vivo, creates surplus amount of ATP, witch is sufficient for the activity of acetoacetate, lipid biosynthesis, herewith for the stimulation of glucose transmission to peripheral tissues.

We are inclined to think, that sodium succinate may be used for reduction of glucose amount in the blood after causing diabetes with alloxan for animals. Tentative experiments proved that impact of sodium succinate (for animals diabetes was caused by alloxan) reduces not only amount of ketotic corpuscles in blood plasma, but also glucose concentration (more effectively).

Table 3.

Impact of sodium succinate on glucose and ketotic corpuscles concentration in rat blood (diabetes for them was caused by alloxan).

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	Metabolites concentration in the blood	
	Saline	Sodium succinate
Glucose	30±4	16,4±4,2
3-O-hidroxitirata	886±85	530±89
Acetoacetate	369±63	298±60
Amount of ketonic corpuscles, MKM	1250±142	824±204

Note. Alloxan was injected for the rats in 40mg/kg. Three days later, sodium succinate (50mg/kg) was injected for one group of rats, saline – for other group. After half-an-hour all animals were decapitated and metabolites, which are described in table 1) found.

Deviations of average value and average effective values from normal were listed.

In contradistinction to insulin, such unnatural agents like sulphacarbamide may lower glucose amount in plasma without its entrance into tissues stimulation and without utilization in the tissues. In that case the application of preparations, this reduces glucose concentration in the blood through reduction of ketotic corpuscles concentration, stimulation of glucose transmission to cells and its utilization in peripheral tissues without inhibition of gluconeogenesis, may be beneficial.

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