International Journal of Medical Sciences And Clinical Research

VOLUME Vol.05 Issue02 2025
PAGE NO. 102-104
DOI 10.37547/ijmscr/Volume05Issue02-19

The effect of glyderinine on glucose transport into muscle tissue depending on the level of insulin in the blood

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Received: 24 December 2024; **Accepted:** 26 January 2025; **Published:** 28 February 2025

Abstract: The paper studied glucose in glycerin (18-dehydroglycerretic acid) by comparing the level of transport to diaphragm muscle tissue with the insulin effect under in vitro conditions. In this case, a 53% decrease in glucose content in the incubation medium in muscle tissue was to a certain extent the reason for its accumulation as glycogen (24%). Since the observed changes were similar to the qualitative changes determined by the action of insulin, the amount of insulin and S-peptide was measured using radioimmun analysis in the rat mine, where glycerin was sent in this experiment. The results of these studies showed that in animal experiments conducted in the glyderin laboratory, it was shown that insulin and C-peptide act in both directions. No significant glycemic reactions to adrenaline were observed in animals taking antidepressants. Therefore, the observed metabolic changes may be related to the stimulation of insulin secretion.

Keywords: Diabetes, hexokinase, glycogen, phosphorylase, insulin, C-peptide, radioimmune, incubation.

Introduction: Normalizing metabolic processes in diabetes mellitus with the prevention of its various complications is the object of close attention of physicians, pharmacologists, biotechnologists and biochemists. Pharmacotherapy in diabetes mellitus provides for aspects of increased insulin secretion depending on the type of diabetes, replacement of insulin in case of its deficiency and normalization of existing metabolic disorders. Synthetic derivatives of sulfonylureas and biguanides and their subsequent analogues remain the main oral treatment for patients with non-insulin-dependent diabetes. Unfortunately, due to the side effects of addiction and, in some cases, direct toxicity, they have limited use. Therefore, attempts to develop new antidiabetic drugs that are convenient for patients and have relatively minimal side effects are still relevant today.

In all tissues, under physiological conditions, glucose transport determines its intracellular metabolism, estimated by the oxidation of glucose to carbon dioxide. Obviously, transport is the primary limiting reaction in the utilization of glucose by cells, since in the absence of insulin, the flow of transferred glucose is always less than the rate of glucose phosphorylation (1). The intensity of the main metabolic pathways of glucose depends on the structural and functional characteristics of individual tissues.

For example, in human erythrocytes and in animal livers, the highest rate of glucose transport is observed, exceeding, by about an order of magnitude, the corresponding values in other tissues. In muscle and some other tissues, an increase in the rate of glycolysis is accompanied by stimulation of glucose extraction and activation of hexokinase (2).

METHODS

Taking into account the above, and based on the observed changes in glucose, glycogen, phosphorylase and hexokinase activity under the action of glyderinine, in this work we studied the effect of the drug on glucose transport into muscle tissue, while comparing the results with the effect of insulin in similar experimental conditions.

For this purpose, in in vitro experiments, the diaphragm was incubated in a glucose medium and glucose consumption was determined by its loss from the incubation medium and the glycogen content in the diaphragm (3).

Determination of insulin in blood serum was performed using radioimmunological kits. The insulin content in the blood serum of rats according to this method is 30.6 = 7.2 mkED / ml in 1 ml of plasma.

RESULTS AND DISCUSSION

We found (see Table 1) that the drug under study contributes to the intake of glucose by the diaphragm by 53% and the accumulation of glycogen in it by 24%. As can be seen from the data in the table, the effects of glyderinine on glucose transport and its incorporation into glycogen are similar to those of insulin, i.e. glyderinine, like insulin, stimulates glucose transport through cytoplasmic membranes.

Table 1.

The effect of glyderinine on glucose intake and glycogen content in the diaphragm (the experiments are the average of 6 definitions)

	Indicators	Glucose in microns / g	Glycogen in mg%
1	before incubation (control)	2,81 ± 0,55	54,5 ± 5,02
2	after incubation	4,28 ± 0,59*	67,8 ± 6,13
3	insulin in iu/ml	4.40 ± 0.54*	68,5 ±4,6*

The differences are significant - p<0.05 from the control.

These facts can be used to interpret the mechanism of the metabolic effect of the drug. It seems to us that, based on in vitro experiments without insulin or in vivo at steady-state concentrations of insulin in the blood, the established increase in glucose transport and glycogen synthesis in skeletal muscles, combined with the effect of insulin on phosphorylase and hexokinase activity, is to a certain extent due to tissue reaction or the insulin-like effect of the drug, or they are mediated through increased insulin secretion in beta cells of the islets of Langerhans of the pancreas.

Clarifying this may help in understanding the stimulation of membrane glucose transport under the influence of glyderinine. Therefore, in a series of studies, we determined the levels of insulin and C-peptide in the blood by radioimmune analysis in rats treated with glyderinine in these dosages normally and against the background of administration of the counterinsular hormone epinephrine 0.7-0.8 mg/kg body weight 20 minutes before slaughter (see Table 2).

Table 2.

The effect of glyderinine on the blood levels of insulin and C-peptide in rats is normal and against the background of adrenaline administration

Indicators of	Glyderinine		Glyderinine + adrenaline	
The amount of the administered drug is	Insulin in micrograms/ ml	C- peptide in pg/ml C-	Insulin in micrograms/ml	C- peptide in pg/ml
Control (without introduction)	11,85 ± 1,92	0,44 ± 0,14	9,38 ± 1,23	0,233 ± 0,134
once	7,06 ± 1,38*	0,90 ± 0,29*	16,88 ± 2,29	0,650 ± 0,109*

three times	19,40 ± 1,14*	1,13 ± 0,37*	17,76 ± 1,81	0,783 ± 0,107*
Sevenfold	20,88 ± 2 ,86*	1,23 ± 0,45*	21,15 ± 1.82	1,133 ± 0,349*

Currently, the simultaneous determination of C-peptide serves as a yardstick.

* Confidence - p<0.05-0.01.

Currently, the simultaneous determination of C-peptide serves as a measure of the level of insulin secretion and liver extraction. This attitude has developed in view of the significant and highly variable extraction of insulin by the liver. Since it is known that C-peptide and insulin are secreted by β-cells at an equimolar concentration and that C-peptide is not extracted by the liver in significant quantities, the determination of C-peptide in peripheral blood has become widely used to assess the endogenous secretory activity of β-cells.

In our experience, the administration of glyderinine contributed to a significant increase in the level of insulin in the blood, the rise of which depended on the amount of the drug administered. A similar trend took place with respect to the quantitative change of the C-peptide, only with a difference exceeding the control values by more than two times (see Table 2). A marked increase in the level of C-peptide compared with insulin is convincing evidence of stimulation of insulin secretion under the influence of glyderinine. The discrepancy between the quantitative level of insulin and C-peptide is obviously related to the breakdown of insulin in the liver by the enzyme insulinase. Therefore, the ratio of C-peptide to insulin, if taken conventionally equal to 1 normally, increased to 2.7 with repeated administration under the influence of the drug (see Table 2). In the light of the results obtained, it can be assumed that insulin is a possible mediator in the implementation of the metabolic effect of glyderinine at the intracellular level. It is well known from the literature that epinephrine is an antagonist of insulin in the regulation of glucose transport and glycogen synthesis. When adrenaline was administered to animals in amounts that created physiological stress, hyperglycemia was accompanied by a decrease in glucose utilization and insulin secretion (3). In our experiments, after the introduction of adrenaline, there was indeed a decrease in the level of insulin in the blood, but it was statistically unreliable. Therefore, the decreases in insulin and C-peptide detected only with the introduction of epinephrine can be considered as a downward trend. While epinephrine administered while receiving glyderinine could not cause any detectable change. On the contrary, both the amounts of insulin and C-peptide in this series of experiments, as well as the previous series with only one glyderinin, increased 2-3 times compared with the control.

Comparing the indicators shown in the tables, it is easy to see that the adrenaline effect on insulin secretion does not manifest itself when administered concomitantly with glyderinine.

CONCLUSION

Consequently, the glycemic changes we found caused by the studied drug are due to the stimulation of endogenous insulin secretion.

Since the animals received glyderinine previously in vivo, the experimental data can be interpreted as a result of the insulin-like effect of the drug.

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