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Calcitonin Secretion under Insulin Hypoglycemia

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Abstract

The increasing secretion of calcitonin and hypocalcemia under insulin hypoglycemia, induced with insulin injection (1 IU/100g), was established. Physiological mechanisms of the stimulating effect of insulin hypoglycemia on calcitonin secretion were studied in double-side adrenalectomized and pancreatectomized rats and under the blocking synaptic transmission in sympathetic ganglions or via peripheral cholino- and adreno-receptor structures. Insulin hypoglycemia didn't expose the increasing secretion of calcitonin in rats under adrenalectomy and pancreatectomy. Ganglion-blocker pentamin (2.5 mg/100g body weight), blocker of M-cholino-receptors atropine (0.2 ml), α-adreno-blocker tropaphen (0.1 mg/100g), and β-adreno-blocker obzidan (0.1 mg/100g) evoked the inhibiting effect on calcitonin secretion in spite of simultaneously increasing of hypoglycemia. Corticosteroids and, obviously, glucagon and also the tone of autonomic nervous system via peripheral M-cholinoreactive and α- and β-adrenoreactive structures take part in the activation of calcitonin secretion under insulin hypoglycemia.

Keywords

Calcitonin, Insulin Hypoglycemia, Autonomic Nervous System, Glucocorticoids, Glucagon

1. Introduction

The calcitonin (CT) effect on carbohydrate metabolism is well-known. In part, it possesses hyperglycemic action [1]-[8]. However, for CT secretion, it is indifferent and is the state of carbohydrate metabolism. In our previous investigations, hypocalcaemia and increasing of CT secretion under insulin hypoglycemia (IH) [9] [10] were established. The mechanisms of the hypoglycemia stimulating effects on CT secretion have yet been stu-

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died in detail. On the basis of this phenomenon, various mechanisms—nervous (the alteration of tone automatic (vegetative) nervous system) and hymoral-hormonal (the increasing of the production of hormones of glucose-elevating action and others) can exist. Glucocorticoids and biologically active products of pancreas play the definite role in the changing of CT secretion, too. For elucidation of significance of the latest ones, some experiments were carried out on the double-sided adrenalectomized and pancreatectomized animals. So far as hypoglycemia evokes the significant alterations of tone of vegetative nervous system (especially the increasing of the activity of sympatho-adrenal part), one could suppose that the indicative shifts cause the increasing of CT secretion under IH. In this connection, the investigations were fulfilled whose aim was to elucidate how calcitonin-activity (CT-activity) of blood serum and the total calcium content will change under IH in the conditions of blocking of synaptic transmission in sympathetic ganglions or peripheral cholino- and adreno-receptor structures.

2. Method of Investigation

Experiments were performed on 130 mature male Wistar rats weighing 270 - 300 g, which were divided into 11 groups. Rats of the 1st group served as control. Rats of the 2nd group received an intramuscular injection of insulin, 1 IU/100g body weight. Rats of the 3rd group received the analogues dose of hormone and after 30 and 60 minutes-intraperitoneally 2 ml glucose for the avoidance of the significant decreasing of the blood glucose level. 2 hours after insulin administration blood samples from the femoral vein were taken in rats of the 2nd and 3rd groups under the light ether anesthesia. To rats of the 4th and 5th groups, double-sided adrenalectomy [11] was conducted. On the 4th day after operation fulfilled experiments were on these animals. Rats of the 4th group served as control, and rats of the 5th group underwent the effect of exogenous insulin, administrated analogically animals of the 2nd group. To rats of the 6th and 7th groups pancreatectomy by well-known method was conducted [11]. 1 hour after operation animals were taken for experiment. The 6th group of animals was control, and rats of the 7th group received insulin analogically animals of the 2nd group. Animals of the 8th group were administrated with ganglion-blocker pentamin (2.5 mg/100g body weight), 9th group-blocker of M-cholino-receptor atropine (0.2 ml), the 10th group-α-adreno-blocker tropaphen (0.1 mg/100g body weight) and the 11th group-β-adreno-blocker obzidan (0.1 mg/100g body weight) intramuscular. 30 minutes after injection of the indicative preparations animals received insulin analogically rats of the 2nd group.

For evaluation of hypoglycemia degree the blood glucose level was measured by the colorimetric method of Frank-Kirberger [12]. To assess the state of CT secretion we determined CT-activity using the method of Laljee [13] with the help of salmon CT as standard (method of biological test). The total calcium content was assayed by complexono-metric method [14]. The indicator of corticosteroid function served the concentration of 11-oxycorticosteroids (11-OCS) in the blood serum by the method of Usvatov and Pankov [15]. For biological test were used 190 mice. The data processed statistically using Student-Fisher tests.

3. Results and Discussion

The values of glucose, total calcium and 11-OCS of the blood serum in control (intact) rats were, respectively, 5.6 ± 0.3 m·mol/l, 2.2 ± 0.08 m·mol/l and 158 ± 6 m·kg/l, *i.e*. in norm for this type of laboratory animals. Serum CT-activity in basal conditions did not determine (**Table 1**).

Insulin injection led to the decreasing of the blood glucose level to 2.4 ± 0.2 m·mol/l ($P_1 < 0.001$) and increasing of serum CT-activity to 23.1 ± 3.5 IU/ml. High level of CT-activity accompanied with the decreasing of the total calcium content in the blood serum to 1.4 ± 0.09 m·mol/l ($P_1 < 0.001$). The content of 11-OCS in the blood serum increased under IH to 312 ± 27 m·kg/l ($P_1 < 0.001$). There is a close negative correlation established between calcium level and 11-OCS content in the blood serum ($r = -0.89$, $P < 0.05$), and also between glucose level and 11-OCS content ($r = -0.87$, $P < 0.05$) that testifies about interaction of regulating effects of calcium and carbohydrate metabolism.

Pathogenic significant of hypoglycemia in the increasing of serum CT-activity is clear from the experiments in which the development of hypoglycemia prevented by intraperitoneal glucose administration. Under this state of experiment the glucose content in blood decreased only to 5.2 ± 0.2 m·mol/l, $P_1 > 0.1$ (**Table 1**).

For this serum CT-activity didn't reveal, but the total calcium content formed 2.1 ± 0.01 m·mol/l that was a little (although and reliably) below than in control rats. Thus, one can make the conclusion that CT secretion after insulin administration is caused by hypoglycemic state.

For studying of the role of the hormones of adrenal glands in the activation of CT secretion under IH we re-

Table 1. Effect of insulin administration on glucose, calcium, CT-activity and 11-OCS in intact, adrenalectomized and pancreatectomized rats.

Experimental groups	Glucose, m·mol/l (M ± m)	11-OCS, m·kg/l (M ± m)	Calcium, m·mol/l (M ± m)	CT-activity, IU/ml (M ± m)
		Intact rats		
Control	5.6 ± 0.3	158 ± 6	2.2 ± 0.08	0
n	10	5	10	10
Insulin administration	2.4 ± 0.2	312 ± 27	1.4 ± 0.09	23.1 ± 3.5
n	20	5	20	20
P_1	<0.001	<0.001	<0.001	
Insulin + glucose administration	5.2 ± 0.3	-	2.1 ± 0.01	0
n	10		10	10
P_1	>0.1			
		Adrenalectomized rats		
Control	5.2 ± 0.3	29 ± 5	1.8 ± 0.05	0
n	5	5	5	5
P_2	<0.05	<0.001	<0.001	
Insulin administration	1.6 ± 0.2	32 ± 6	1.45 ± 0.05	2.6 ± 0.8
n	7	5	7	7
P_1	<0.001	>0.5	<0.001	
P_2	<0.001	<0.001	>0.1	<0.001
		Pancreatectomized rats		
Control	4.9 ± 0.5	-	2.0 ± 0.09	0.5
n	7		7	7
P_2	<0.01		<0.001	
Insulin administration	2.3 ± 0.3	-	1.9 ± 0.5	0.5
n	7		7	7
P_1	<0.001		>0.2	
P_2	>0.5		<0.05	

Note: P_1—The reliability of results differences under control group; P_2—The reliability of results differences under analogues group of intact rats; *n*—the amount of animals.

sorted to the making of experimental adrenal deficiency too. On the 4th day after operation the blood glucose level of adrenalectomized animals compiled 5.2 ± 0.3 m·mol/l, that was reliably lower than in rats with intact adrenal glands (5.6 ± 0.3 m·mol/l, $P_2 < 0.05$).

11-OCS concentration in serum compiled only 29 ± 5 m·kg/l (for 158 ± 6 m·kg/l in intact rats), *i.e.* a marked corticosteroid deficiency arose. But the total calcium concentration was reliably decreased, serum CT-activity didn't reveal.

Insulin injection to adrenalectomized rats led to more large decreasing of the blood glucose level than in rats with intact adrenal glands (1.6 ± 0.2 and 2.4 ± 0.2 m·mol/l, responsibility, $P_2 < 0.001$), that, obviously, explains by the decreasing of cathecholamines and glucocorticoids level in their blood. 11-OCS level in adrenalectomized animals didn't practically change despite so significant hypoglycemia that additionally indicated for the marked hypocorticism. Serum CT-activity in these conditions compiled only 2.6 ± 0.8 IU/ml, *i.e.* the stimulation of CT secretion was small in spite of harp decreasing of the blood glucose concentration. The fact is that the total calcium content in blood of adrenalectomized rats after insulin administration decreased to 1.45 ± 0.05

m·mol/l pays attention. Apparently, under 11-OCS deficiency even small increasing of CT secretion (increasing of CT secretion to 2.6 ± 0.8 IU/ml) is enough for the significant hypocalcaemia effect. Thus, IH induced in adrenalectomized rats the decreasing of calcium level and non-significant increasing of the serum CT-activity. Taking into consideration the fact that glucocorticoids stimulate CT secretion [16], one can make a conclusion that these hormones take participant in the regulation of CT secretion under IH.

In pancreatectomized rats 1 hour after operation glucose level was 4.9 ± 0.1 m·mol/l, *i.e*. it wasn't even higher but even lower than in intact rats. It indicated on that fact that during the period, lasting after operation, the disturbances of carbohydrate metabolism, which are typical for diabetes mellitus, hadn't yet developed. Initial content of the total calcium compiled 2.0 ± 0.09 m·mol/l that was reliably below than in intact rats. CT-activity was 0.5 IU/ml.

Glucose level after insulin administration in pancreatectomized rats decreased to 2.3 ± 0.3 m·mol/l; the total calcium content in serum decreased too, but only to 1.9 ± 0.1 m·mol/l. CT-activity didn't increase 0.5 IU/ml. So, in pancreatectomized rats IH didn't induce the increasing of CT secretion.

As it is known, pancreas produces a great number of biologically active substances, secreting in blood: insulin, glucagon, somatostatin, pancreatic gastrin, lipokain, kalecrein, and pancreatic enzymes, too. Glucagon has the direct relation to CT secretion; under its influence the output of CT by C-cells [17] is activated. It is also known that under hypoglycemic states one of the first reactions of organism is the intensification of glucagon production. The latest one, together with adrenalin, promotes the dissociation of liver glycogen and reduction of normal blood glucose level. From these concepts one can suppose that the absence of the increasing of the serum CT-activity in pancreatectomized rats is explained in significant degree by glucagon absence. However, it isn't necessary categorically to deny the possibility of the effect of others biologically active substances produced by pancreas.

Against the background of pentamin the decreasing of glucose level occurred to 1.8 ± 0.3 m·mol/l, the total calcium content—to 1.6 ± 0.05 m·mol/l. CT-activity increased only to 2.3 ± 0.5 IU/ml (**Table 2**).

In rats with atropine administration was observed much more decreasing of the blood glucose level under control—1.4 ± 0.1 m·mol/l, however, the total calcium content stayed in normal value-2.0 ± 0.09 m·mol/l, and CT-activity didn't determine.

The blood glucose level in rats with tropaphen administration, compiled 2.0 ± 0.2 m·mol/l after insulin injection, the total calcium content—1.6 ± 0.02 m·mol/l, the value of CT-activity—4.56 ± 0.6 IU/ml.

In rats, which received obzidan injection, hypoglycemia achieved the lowest values—1.3 ± 0.3 m·mol/l. The

Table 2. The effect of pentamin, atropine, tropaphen and obzidan on calcitonin secretion and total calcium level in blood serum under insulin hypoglycemia.

Conditions of experiment	Glucose, m·mol/l (M ± m)	P_1	P_2	Calcium, m·mol/l (M ± m)	P_1	P_2	CT-activity, IU/ml (M ± m)	P_2
Intact rats	5.6 ± 0.3			2.2 ± 0.08			0	
n	10			10			10	
Insulin	2.4 ± 0.2	<0.001		1.4 ± 0.09			23.1 ± 3.5	
n	20			20			20	
Pentamin + insulin	1.8 ± 0.3	<0.001	<0.01	1.6 ± 0.05	<0.001	<0.001	2.3 ± 0.5	<0.001
n	5			5			5	
Atropin + insulin	1.4 ± 0.1	<0.001	<0.001	2.0 ± 0.09	<0.001	<0.001	0	
n	8			8			8	
Tropaphen + insulin	2.0 ± 0.2	<0.001	<0.01	1.6 ± 0.02	<0.001	<0.001	4.6 ± 0.6	<0.001
n	10			10			10	
Obzidan + insulin	1.3 ± 0.3	<0.001	<0.001	1.9 ± 0.01	<0.001	<0.001	0.63 ± 0.2	<0.001
n	5			5			5	

Note: P_1—The reliability of results differences under the intact rats; P_2—The reliability of results differences under insulin; *n*—The amount of animals.

total calcium level decreased to 1.9 ± 0.01 m·mol/l, CT-activity increased only 0.63 ± 0.2 IU/ml, that was in normal level.

The ganglion-blockers inhibit the transmission of nervous excitation from pre-ganglions on post-ganglions fibres of vegetative nerves; vegetative ganglion (sympathetic or parasympathetic) becomes non-sensitive to the stimulating action of irritants; M-cholino-blocker inhibits the conduction of excitation, inhibiting the receptors of post-ganglions parasympathetic, but α- and β-adreno-blockers block the receptors of post-ganglions sympathetic fibres as a result the functions of all organs, supplying with vegetative innervation, are changed.

One can suppose that in our investigations the injection of ganglion-blocker pentamin, M-cholino-blocker atropine, α-adreno-blocker tropaphen and β-adreno-blocker obzidan against the background of IH induces the inhibition of the functional activity of thyroid C-cells, produced CT, in this connection the decreasing of blood serum CT-activity were observed. The results of studying about the alteration of thyroid C-cells activity in rats with experimental hyperthyroidism under the administration of β-adreno-blocker propranolol [18] can serve as the confirmation to it.

The decreasing of the total calcium content in the blood serum under IH against the background of pentamin, tropaphen and obzidan, despite the inhibition of CT secretion is connected, apparently, with the known hypocalcaemic effect of insulin. As for atropine administration, it didn't evoke a significant hypocalcaemia. It is known that M-cholino-blocker atropine influences on the peripheral M-cholino-receptors, makes it non-sensitive to acetylcholine, produced in the region of the endings of post-ganglions parasympathetic nerves. In the present it is established that parasympathetic nervous system stimulates insulin secretion, and sympathetic-inhibits [19]. So, the injection of atropine, blocking the conduction of excitation on parasympathetic fibres, induced the inhibition of insulin secretion, as a result the significant decreasing of the total calcium content in the blood serum in rats under IH didn't occur.

Unlike ganglion-blocker pentamin, tropaphen and obzidan-α- and β-adreno-blockers interrupt the conduction of efferent nervous excitation, acting on post-ganglions signals and non-effecting on the transmission of excitation in ganglions. α-adreno-blocker blocks mainly the stimulating effects, connecting with the excitation of α-adreno-receptors, it is known that it doesn't block all adrenaline effects. Its hyperglycemic effect changes a little. In this connection against the background of tropaphen administration under IH glucose level decreases significantly less than against the background of pentamin, atropine and obzidan administration. B-adreno-blockers block the effects, connected with the action of sympathetic nervous impulses and sympathetic substances on β-adreno-receptors. Obzidan possesses only blocking effect on β-adreno-receptors, inhibiting simultaneously β_1- and β_2-receptors. As mentioned above, sympathetic nervous system inhibits insulin secretion [19]. As far as obzidan administration blocked sympathetic nervous impulses, coming to pancreas β-cells, so the inhibition of insulin secretion didn't occur, and in our investigations more significant hypoglycemia under insulin injection observed.

Thus, administration of ganglion-blocker, M-cholino-blocker, α- and β-adreno-blockers led to sharp decreasing of CT secretion under IH that indicates on the participant of sympathetic and parasympathtetic parts of vegetative nervous system via synaptic transmission of nervous excitation in ganglions and via tissue's adreno- and cholino-receptor structures.

The increasing of CT secretion under IH established in our investigations has biological significance as CT and the decreasing of out-cell calcium concentration inhibit endogene insulin secretion [20] [21]—the main sugar-decreasing hormone and by this way prevent organism from excess decreasing of the blood glucose level. Moreover, under CT the secretion of sugar-increasing hormones-cathecholamines and cortisol [22] increases. On the other hand, CT has hyperglycemic effect [23] [24], contra-acting to hypoglycemia too.

4. Conclusions

Summing the results of our investigations, one can make the conclusion:

1) IH induces the activation of CT secretion, which is expressed in the increasing of serum CT-activity and decreasing of the total calcium content;

2) Combined administration of insulin and glucose, which doesn't induce hypoglycemia, also doesn't reflect significantly on serum CT-activity and calcium content. It indicates the role of hypoglycemia in the stimulation of CT secretion;

3) Against the background of glucocorticoids deficiency, evoked by double-sided adrenalectomy, IH leads to

the decreasing of the total calcium level and non-significant increasing of CT-activity. It lets us consider that glucocorticoids take part in the activation of CT secretion under IH;

4) The absence of the increasing of serum CT-activity in pancreatectomized rats under IH gives the basis to suppose that in significant degree it is associated with the absence of glucagon;

5) Pentamin, tropaphen, obzidan and especially atropine invoke the inhibiting effect on CT secretion, despite simultaneous intensifying of hypoglycemia, induced by insulin injection;

6) Sympathetic and parasympathetic parts, peripheral M-cholino-receptor, α- and β-adreno-receptor structures of vegetative nervous system take part in the regulation of CT secretion under IH.

References

[1] Butakova (Moisa), S.S. and Nozdrachev, A.D. (2010) Effect of One-Time Injection of Calcitonin Preparations on Glucose and Calcium Level in Rats of Different Age Groups. *Advances of Gerontology*, **23**, 93-97.

[2] Butakova (Moisa), S.S. and Nozdrachev, A.D. (2010) Effect of Calcitonin on the Type of Alimentary Hyperglycemia in Rats of Different Age and Sex. *Advances of Gerontology*, **23**, 213-220.

[3] Butakova (Moisa), S.S. and Nozdrachev, A.D. (2010) Calcitonin-Glucoregulating Hormone. *Vestik of Russian Military Medical Academy*, 188-196.

[4] Moisa, S.S. and Nozdrachev, A.D. (2011) Mechanisms of Hyperglycemic Effect of Calcitonin. *Bulletin of Experimental Biology and Medicine*, **150**, 320-323. http://dx.doi.org/10.1007/s10517-011-1132-3

[5] Moisa, S.S. and Nozdrachev, A.D. (2011) Mechanisms of Regulation of Calcium and Carbohydrate Metabolism (Monograph). LAP LAMBERT Academic Publishing GmbH & Co. KG, Saarbrucken, 319 p.

[6] Moisa, S.S. (2013) Calcitonin Contra-Insulin Action on Glucose Metabolism. *Bulletin of Experimental Biology and Medicine*, **156**, 183-185. http://dx.doi.org/10.1007/s10517-013-2314-y

[7] Moisa, S.S. and Nozdrachev, A.D. (2013) One-Time Injection of Calcitonin Induces Glucose Intolerance in Children with the 1st Degree Obesity. *Health*, **5**, 9-13. http://dx.doi.org/10.4236/health.2013.56A1002

[8] Moisa, S.S. and Nozdrachev, A.D. (2014) Calcitonin and Parathyrin Are Glucoregulating Hormones. *Journal of Molecular and Genetic Medicine*, **2**, S1-S24. http://dx.doi.org/10.4172/1747-0862

[9] Butakova (Moisa), S.S. (2005) Calcitonin Secretion under the Different State of Carbohydrate Metabolism in Ontogenesis in Rats. *Materials of the VII All-Russian Conference* "*Neuroendocrinology*-2005", St.-Petersburg, 25-27 April 2005, 36-37.

[10] Butakova (Moisa), S.S. (2007) Serum Calcitonin Activity and Total Calcium Content under the Different State of Carbohydrate Metabolism in Ontogenesis in Rats. Pavlov Institute of Physiology Russian Academy of Sciences. St.-Petersburg, 7-9 October 2007, p. 32.

[11] Nozdrachev, A.D., Poliakov, E.L. and Bagaev, V.A. (2007) Experimental Surgery of the Laboratory Animals. Lan, St.-Petersburg, 125 and 130-132.

[12] Frank, H. and Kirberger, E. (1950) Eine Kolorimetrische Methode zur Bestimmung der "Wahren Glucose" und Galactose in 0.005 rm^3. *Blut. Biochem. Ztschr.*, **320**, 359-367.

[13] Laljee, H.C.K., Smith, K.I. and Dorrington, K.J. (1967) The Assay of Human Thyrocalcitonin in Mice. *Proccedings of the Symposium on Thyrocalcitonin and the C-Cells*, London, 17-20 July 1967, 32-35.

[14] Selochnik, I.I., Briskin, A.I. and Antonova, E.E. (1978) Photoelectrocolorimetric Assay of Calcium Concentration in Serum by the Help of EDTA and Murexide. *Chemical Pharmacological Journal*, **12**, 138-140.

[15] Usvatova, I.Y. and Pankov, Y.A. (1969) Fluorimetrical Methods of the Determination of Steroid Hormones in Biological Fluids. Medicine, Moscow, 38.

[16] Lineberry, M.P. and Waite, L.C. (1978) Calcitonin Responses in Intact and Adrenalectomized Rats. *Life Sciences*, **22**, 511-518. http://dx.doi.org/10.1016/0024-3205(78)90432-0

[17] Shustov, S.B. and Halimov, Y.S. (2001) Functional and Topical Diagnostic in Endocrinology. Elbi, St.-Petersburg, 238 p.

[18] Zbucki, R.L., Dadan, I., Sawicki, B., Bialuk, I., Kosiozek, P., Winnicka, M. and Puchalski, Z. (2004) Propranolol Alters the Activity of Thyroid C-Cells in the Experimental Model of Hyperthyroidism in Rats. *Proceedings of the* 15 *International Congress of the Polish Pharmacological Society*, Poznan, 12-14 September 2004, 223.

[19] Ahren, B. (2000) Automatic Regulation of Islet Hormone Secretion—Implications for Health and Disease. *Diabetologia*, **43**, 393-410. http://dx.doi.org/10.1007/s001250051322

[20] Butakova (Moisa), S.S. (2008) Calcitonin, a Modulater of Pancreas Secret Process. Thesis, Institute of Physiology of the Russian Academy of Sciences, St.-Petersburg, 26-27.

[21] Iaroshevskii, I.A., Darinskii, I.A. and Butakova (Moisa), S.S. (1989) Calcitonin Effect on Insulin and Glucagon Secretion by Panceas. *Problems of Endocrinol*ogy, **35**, 58-61.

[22] Moore, M.C., Lin, D.W., Colburn, C.A., Goldstein, R.E., Neal, D.W. and Cherrington, A.D. (1999) Insulin- and Glucagons-Independent Effects of Calcitonin Gene-Related Peptide in the Conscious Dog. *Metabolism*, **48**, 603-610. http://dx.doi.org/10.1016/S0026-0495(99)90058-6

[23] Moisa, S.S. and Nozdrachev, A.D. (2013) Effect of Calcium-Regulating Hormones on Glucose Tolerance. *Proceedings of Materials of the III International Research and Practice Conference*: *Science*, *Technology and Higher Education*, Westwood, 16 October 2013, 516-522.

[24] Young, A.A., Wang, M.W. and Gedulin, B. (1995) Diabetogenic Effects of Salmon Calcitonin Are Attributable to Amylin-Like Activity. *Metabolism*, **44**, 1581-1589. http://dx.doi.org/10.1016/0026-0495(95)90079-9

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