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№ 2(78)/2015

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МЕДИЦИНА. ГЕОГРАФИЯ

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УНИВЕРСИТЕТІНІҢ
ХАБАРШЫСЫ

ВЕСТНИК
КАРАГАНДИНСКОГО
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1st Conference on the Pathogenesis and Epidemiology of Diabetes
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**Diabetes Research Group, Research Park of Biotechnology
and Ecomonitoring of the Karaganda State University**

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On the protective effect of 2,3-dimercaptopropanol for destructive action of zincbinding chemicals on pancreatic B-cells

2,3-Dimercaptopropanol is a substance able to re-activate sulfhydryl groups of enzymes and has the property to form temporary complexes with metals, including zinc. It is also known that certain amino acids, particularly cysteine and glutathione also contain in its composition mole coli SH-groups. Administration of these amino acids in to animals result prevention developing of experimental diabetes caused zinkbinding diabetogenic chemicals. It is confirmed that this effect is determined by their ability to form non-toxic temporary complexes with zinc in B-cells of pancreatic islets that protect cells of the destruction caused by diabetogenic chelating agents. The authors have shown that 2,3-dimercaptopropanol at doses of 60 and 120 mg/kg is able to prevent the development of diabetes in almost all experimental animals. Authors found that this ability 2,3-dimercaptopropanol is explained by its property through SH-groups included in its composition, to form non-toxic complexes with zinc in pancreatic cells that protect cells of death.

Key words: B-cells, SH-groups, experimental diabetes, zinc, 2,3-dimercaptopropanol.

2,3-Dimercaptopropanol (DMP) is known as re-activator of SH-group of enzymes and possess ability to form stable complexes with metals. However it is known that some aminoacids contains SH-groups in molecule as Cystein and Glutathuone reduced form protect developing of diabetes caused by chelat active chemicals. This effect determined by high affinity of SH-group for zinc and cadmium [1]. 2,3-Dimercaptopropanol is able in added to destroy other complexes, previously formed with zinc by chelators, that accompanied by re-placing atom of chelator from complex [2] and formation of complex DMT-metal via SH-group.

Aim of work: to investigate state of histostructure of pancreatic islets and possible interaction of Zn^{+2} -ions in B-cells with DMP and Dithizon, a diabetogenic chelator.

Material and methods

Animals: 10 rabbits 2,200–2,650 g were used. Group 1: control intact animals: A-injection of Dithizon, 48,9 mg/kg; B-intact rabbit; Group 2: injection of DMP («SIGMA») in doses 60 and 120 mg/kg to 8 animals; 30 min later water solution of Dithizon 46,6–49,7 mg/kg was injected to 6 animals; animals killed 10 min past injection of DZ. Group 3: injection of DMP, 60 and 120 mg/kg to 2 animals; animals killed 10 min past injection. Group 4: injection of DMP, 60 and 120 mg/kg to 2 animals; 30 min later injection of Dithizon 48 mg/kg; animals killed 5 days past injection of DZ.

Frozen sections of pancreas were investigated using dark microscopy for revealing of DZ- Zn^{+2} -complex in B-cells. The high specific fluorescent method revealing of free Zn^{+2} -ions in B-cells by fluorochrom 8-para(toluenesulphonylamino)quinolin [8PTSQ] was used [3, 4]. 3 animals were killed 5 days past injections of DMP and DZ. Pancreas was fixed in alcohol 70° + H₂S. Staining of sections by aldehyde-fuchshine [5–7] and 8PTSQ.

Results

Group 1. Control intact animals section o pancreas. 20 frozen sections of pancreas tissue were investigated using dark microscopy. Cytoplasm of all investigated islets contain a large amount of red chelat complex Zn^{+2} -Dithizon [1] which concentrated on the all surface of cytoplasm, maximally around blood islet's capillaries (fig. 1.1). Reaction for Zn^{+2} -ions: intensive fluorescence of B-cells (fig. 1.2). Group 2. Administration of 2,3-dimercaptopropanol and of Dithizon result prevention of formation in majority of B-cells of Zn^{+2} -Dithizon complex which is partially formed in cells located around blood capillaries (fig. 1.3). Pro

protective effect determined by not diabetogenic binding of Zn^{+2} -ions by DMT. It is known that DMT possess high affinity for Zn^{+2} -ions.

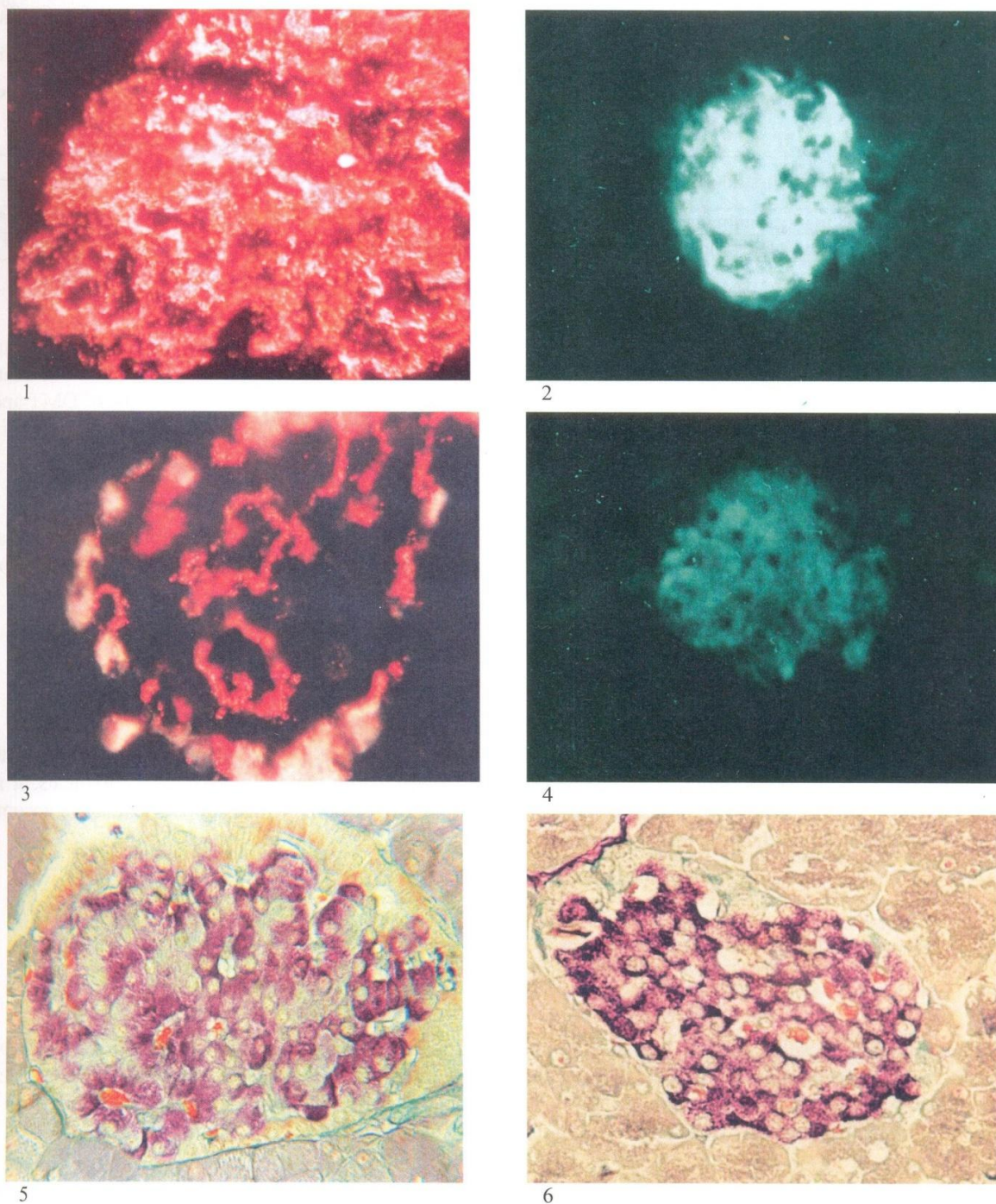


Figure 1. Pancreas tissue

Group 3. Investigation of free Zn^{+2} -ions content in B-cells past injection of DMT showed a negative reaction for Zn^{+2} in islets (fig. 1.4). A few B-cells contains minimal amount of Zn^{+2} in cytoplasm. This result determined by forming by DMT of not visible Zn^{+2} -DMT complex.

Group 4. Investigation of effect of DMT on diabetogenic property of Dithizon showed that administration of it accompanied by absence of any histological changes in pancreatic islets (fig. 1.5; 1.6).

Table

Results of investigation of blood glucose level

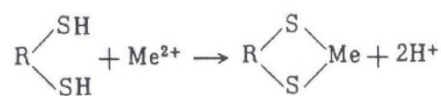
№	Conditions	Blood Glucose (mmol/l), day				
		Before	1	3	6	7
1	Dithizon, 46,8–48 mg/kg 2 animals	5,1±0,3	3,0±0,4	9,6±1,2 [■]	12,6±2,2 [●]	16,2±2,6 [*]
2	DMT, 102–110 mg/kg + Dithizon, 48,8 mg/kg, 4 animals	4,9±0,4	5,2±0,4	5,6±0,5 [■]	5,8±0,7 [●]	5,4±0,6 [*]

Note. * — $p < 0,001$; ● — $p < 0,005$; ■ — $p < 0,01$.

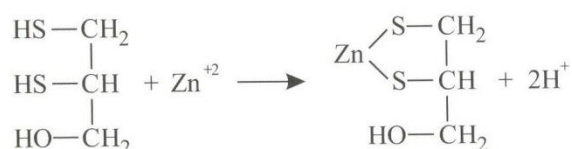
Results of investigation of blood glucose level (table 1) demonstrated that injection of Dithizon accompanied by marked decreasing of blood glucose level that is determined by release of a large amount of insulin as result of destruction within short time of majority B-cells. In other animals, past injections of DMT and followed past 1, 2 and 3 h injections of diabetogenic doses of Dithizon not accompanied by hyperglycemia in animals. We observed only not reliable increasing of blood glucose level until 5,6–5,8 mmol/l (Table).

Discussion

Molecule of 2,3-Dimercaptopropanol ($C_3H_8OS_2$ m.m. 124,22) contains two SH-groups. It is known that some metals (Me) as mercury, arsenic, cadmium, lead, zinc interacted with chemicals contains SH-groups and formed stable cyclic mercaptide:



As bivalent metal Zn^{+2} -ions interacts with 2 SH-groups of molecule of 2,3-Dimercaptopropanol with forming of cyclic mercaptide which are more stable in compared with some chelat active chemicals. It is known that 2,3-Dimercaptopropanol is able to destroy complexes previously formed with chelators accompanied by replace atom of chelator from complex [2].



Thus, obtained results showed that 2,3-Dimercaptopropanol protect B-cells of destruction caused by Dithizon and of developing of diabetes. Investigation of interaction in B-cells between Zn^{+2} -ions and 2,3-Dimercaptopropanol evidently showed that DMT protect B-cells of formation of toxic Dithizon- Zn^{+2} complex by intersection of Zn^{+2} -ions and forming new complex DMT- Zn^{+2} .

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Диабетогенді мырыш байланыстырушы қосылыстармен туындайтын панкреатит В-жасушаларының бұзылуын болдырмауда 2,3-димеркаптопропанолдың қабілеті туралы

2,3-Димеркаптопропанол өзінің молекулалық құрамында екі сульфгидрильді топ (SH-топ) болуымен, сульфгидрильді фермент тобы реактиватор ретінде белгілі және оның SH-тобы арқылы байланысатын ауыр металдармен уақытша кешен түзуге бейімді. Сонымен қатар құрамында SH-тобы бар L-гистидин және цистеин аминқышқылдары В-жасушаларының Zn^{+2} ионымен уақытша байланыстыруға бейімді, осылайша диабетогенді мырыш байланыстырушы қосылыстармен өзара әрекетіне және жойылуын болдырмауға әрекет етеді. Авторлар көрсеткендей, жануарларға 2,3-димеркаптопропанол 60 және 120 мг/кг мөлшерде енгізу В-жасушалардағы мырышты толық тосқауылдатады. 2,3-Димеркаптопропанол превентивті әрекеті оның құрамында сульфгидрильді топ молекулаларының бар болуымен сипатталады деген қорытынды жасалды.

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О способности 2,3-димеркаптопропанола предотвращать разрушение панкреатических В-клеток, вызываемое диабетогенными цинксвязывающими соединениями

2,3-Димеркаптопропанол является веществом, способным реактивировать сульфгидрильные группы ферментов и обладает свойством формировать временные комплексы с металлами, включая цинк. Известно также, что некоторые аминокислоты, в частности, цистеин и глутатион также содержат в составе своей молекулы SH-группы. Эти аминокислоты при парентеральном введении предотвращают развитие экспериментального сахарного диабета, вызываемого цинксвязывающими диабетогенными веществами. Доказано, что этот эффект обусловлен их способностью формировать нетоксичные временные комплексы с цинком В-клеток панкреатических островков, защищая их таким образом от разрушающего воздействия диабетогенных хелатообразователей. Авторами показано, что 2,3-димеркаптопропанол в дозах 60 и 120 мг/кг способен предотвращать развитие диабета почти у всех опытных животных. Авторами установлено, что эта способность 2,3-димеркаптопропанола обусловлена его свойством через SH-группы, входящие в его состав, формировать нетоксичные комплексы с цинком панкреатических В-клеток, препятствуя этим повреждающему действию диабетогенных цинксвязывающих веществ.

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Preface

Diabetes mellitus is a severe disease, and its prevalence is dramatically increasing world-wide. The complications associated with the disease include cardiovascular disease, blindness, amputations, end-stage renal disease, kidney dialysis, and kidney transplantations and present major public-health problems. Furthermore, diabetes costs are exceeding meanwhile billions of dollars annually and put tremendous burden on national health care systems. Recent years have seen a significant progress in basic knowledge on diabetes due to enormous research efforts, making possible the development of new technology and therapeutics for diabetes management and care.

It was just diabetes research, when Dr. G. A. Meyramov from the Karaganda State University joined me in 1977 in Karlsburg and started out to study how tryptophan metabolites could induce diabetes in animals. Since then we look back on a very fruitful cooperation between the Karaganda State University and the Institute of Diabetes Karlsburg.

It was a landmark decision of the Faculty of Biology of the University and the Publishing House of this journal to issue a volume focusing on problems in diabetes. Of note, this is the first time in the history of Kazakhstan and Central Asia that there is a special issue of this distinguished journal, exclusively devoted to diabetes research. The Editors of this journal, by inviting contributions from international as well as national diabetes experts, made an important step in enhancing and disseminating knowledge about diabetes but, beyond that, lay emphasis on care and management of diabetes in Kazakhstan.

We trust that readers will welcome the present issue and benefit from the contributions herein provided by experts in the field.



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