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UDC 612.128:616.379-008:612.393.1
DOI – <https://doi.org/10.14300/mnnc.2018.13092>
ISSN – 2073-8137

CHARACTERIZATION OF THE METABOLIC DISORDERS IN RATS WITH ALLOXAN-INDUCED DIABETES AND CHRONIC ALCOHOLIC INTOXICATION

Bykov I. M.¹, Popov K. A.¹, Berberidy H. P.¹, Tsymbalyuk I. Yu.¹,
Ermakova G. A.¹, Pavlyuchenko I. I.¹, Storozhuk P. G.¹, Ovsyannikov V. G.²

¹ Kuban State Medical University, Krasnodar, Russian Federation

² Rostov State Medical University, Rostov-on-Don, Russian Federation

ОСОБЕННОСТИ МЕТАБОЛИЧЕСКИХ НАРУШЕНИЙ У КРЫС С АЛЛОКСАНОВЫМ ДИАБЕТОМ, РАЗВИВШИМСЯ НА ФОНЕ ХРОНИЧЕСКОЙ АЛКОГОЛЬНОЙ ИНТОКСИКАЦИИ

И. М. Быков¹, К. А. Попов¹, Х. П. Бербериди¹, И. Ю. Цымбалюк¹,
Г. А. Ермакова¹, И. И. Павлюченко¹, П. Г. Сторожук¹, В. Г. Овсянников²

¹ Кубанский государственный медицинский университет, Краснодар,
Российская Федерация

² Ростовский государственный медицинский университет,
Ростов-на-Дону, Российская Федерация

We investigated the metabolic disorders in rats with alloxan-induced diabetes and chronic alcoholic intoxication. Rats were divided into four groups: the first group was untreated controls, the second group was administered alloxan to induce diabetes, the third consumed ethanol for 2 months, and the fourth group consumed ethanol for 2 months and were injected with alloxan half way through this period. Plasma glucose concentrations in rats with both alcoholic intoxication and alloxan-induced diabetes were 22 % higher than in those with alloxan-induced diabetes alone. Plasma ALT activity was 1.7 and 2.2 times higher in rats in groups 3 and 4, respectively, than in control rats. Rats in group 4 showed a 30 % higher GR activity and 29 % and 20 % reductions in CAT and SOD activities in erythrocytes, respectively, versus controls. In addition, the concentration of GR in erythrocytes was a mean 31 % lower in rats in groups 2–4 than in controls. These data reveal more severe disruption of carbohydrate metabolism and a more severe imbalance in the antioxidant system in rats with diabetes mellitus if they have already been subjected to chronic alcohol intoxication.

Keywords: alloxan diabetes, alcohol, oxidative stress, antioxidant system

Исследование особенностей углеводного обмена, цитолиза гепатоцитов и антиоксидантной системы при экспериментальном диабете на фоне хронической алкоголизации проведено на белых крысах, разделенных на 4 группы. Первую группу составили интактные животные. Группу 2 составили животные с моделированием аллоксанового диабета. Животные 3-й группы подвергались алкоголизации в течение 2 месяцев. Группу 4 составили крысы, которым по той же схеме проводили алкоголизацию, но спустя месяц начала эксперимента вводили аллоксан. В ходе исследований было установлено, что концентрация глюкозы плазмы крови при сочетанном течении алкогольной интоксикации и аллоксанового диабета превышала значения аналогичного показателя животных только с диабетом на 22 %. Исследование активности АЛТ показало значительное ее увеличение в плазме крови животных 3–4-й групп по сравнению с контролем в 1,7–2,2 раза. Эритроциты крыс 4-й группы характеризовались увеличенными значениями активности глутатионредуктазы на 30 %, сниженной на 29 и 20 % соответственно каталазной и супероксиддисмутазной активностью. Концентрация восстановленного глутатиона в эритроцитах была снижена на 31 % у животных всех групп. Представленные данные отражают более тяжелое течение нарушений углеводного обмена и дисбаланса антиоксидантной системы при развитии экспериментального диабета на фоне хронической алкоголизации.

Ключевые слова: аллоксановый диабет, алкоголь, окислительный стресс, антиоксидантная система

For citation: Bykov I. M., Popov K. A., Berberidy H. P., Tsymbalyuk I. Yu., Ermakova G. A., Pavlyuchenko I. I., Storozhuk P. G., Ovsyannikov V. G. CHARACTERIZATION OF THE METABOLIC DISORDERS IN RATS WITH ALLOXAN-INDUCED DIABETES AND CHRONIC ALCOHOLIC INTOXICATION. *Medical News of North Caucasus*. 2018;13(3):511-515. DOI – <https://doi.org/10.14300/mnnc.2018.13092>

Для цитирования: Быков И. М., Попов К. А., Бербериди Х. П., Цымбалюк И. Ю., Ермакова Г. А., Павлюченко И. И., Сторожук П. Г., Овсянников В. Г. ОСОБЕННОСТИ МЕТАБОЛИЧЕСКИХ НАРУШЕНИЙ У КРЫС С АЛЛОКСАНОВЫМ ДИАБЕТОМ, РАЗВИВШИМСЯ НА ФОНЕ ХРОНИЧЕСКОЙ АЛКОГОЛЬНОЙ ИНТОКСИКАЦИИ. *Медицинский вестник Северного Кавказа*. 2018;13(3):511-515. DOI – <https://doi.org/10.14300/mnnc.2018.13092>

ALT – Alanine Transaminase
AST – Aspartate Transaminase
CAT – Catalase
DM – Diabetes Mellitus

GPO – Glutathione Peroxidase
GR – Glutathione Reductase
KubSMU – Kuban State Medical University
SOD – Superoxide Dismutase

One of the most urgent medical and social concerns in modern health care is alcoholism, which can be accompanied by the development of a variety of changes consistent with systemic intoxication [1]. Among the multiple visceral pathologies induced by alcoholism that influence quality of life and lifespan, the most severe occurs in the liver. The liver plays an important role in ethanol metabolism, which can lead to the preferential accumulation of ethanol and the toxic products of its oxidation by alcohol dehydrogenase, including acetaldehyde and acetic acid [2]. Thus, the liver can be considered to be the principal site of the toxic effects of ethanol and it manifests the principal defects in the pathogenesis of alcoholism. However, the liver is also the main organ responsible for glucose and energy homeostasis. One of the key defects caused by the chronic alcoholic intoxication that develops during the first 2–4 months is hypoglycemia, which probably occurs as a result of the suppression of gluconeogenesis and glycogenesis in the liver [3].

According to a previous study [4], chronic alcoholic intoxication is accompanied by diabetes mellitus (DM) in 8–10 % of patients. Considering that hypoglycemia develops following temporary alcoholic intoxication, whereas hyperglycemia is the main problem in DM, the effects of each on carbohydrate metabolism are clearly different in these two conditions. Therefore, the molecular mechanisms involved in the effects of alcoholization and DM together are of great interest. A number of previous studies have shown that chronic alcoholization in patients suffering from type II DM not only inhibits insulin secretion but also aggravates insulin resistance in tissues, potentiating hyperglycemia and upregulating non-fermented glycation of proteins [5–6]. One of the main pathophysiological links between DM and alcoholism is oxidative stress, which may aggravate the metabolic disorders involved in these diseases.

It is of note that in most studies in laboratory animals a model of DM is created and alcohol is then only administered for a matter of days or a month after this. Such models have a number of limitations. Firstly, animals with alloxan or streptozotocin-induced diabetes have a short lifespan, making it difficult to superimpose chronic alcoholic intoxication. Secondly, to more accurately model the real-life situation it would be more appropriate to induce DM against a background of pre-existing chronic alcoholic intoxication, because alcohol addiction typically develops at a young age.

We aimed to characterize the metabolic disorder that develops in rats with experimentally induced DM superimposed upon pre-existing chronic alcoholic intoxication.

Material and Methods. The study was performed using 80 male rats initially weighing 210–240 g. The test animals were housed in the vivarium of the KubSMU. The study was carried out in accordance with the «Rules of the European convention on the protection of vertebrate animals» (Strasbourg, 1986) and was approved by the ethics committee of the KubSMU (report № 54 of October 11th 2017).

The rats were divided into four groups of 20. Group 2 (DM group) had DM induced by a single intraperitoneal injection of alloxan monohydrate (15 mg/100 g body mass) after a day of fasting. Group 3 (A group) underwent alcoholization for 2 months. During the first week these rats were given

10 % ethanol in place of water, during the second week 20 %, then 30 % for the remaining period. Group 4 (A+DM group) underwent alcoholization as described above but 1 month after ethanol consumption commenced they were also injected with alloxan as described above. Group 1 (control) were housed under the same conditions but were not given ethanol and were injected with 1-ml vehicle intraperitoneally in place of alloxan. One month after the alloxan injection rats were anesthetized by intramuscular injection of 10 mg/kg tiletamine and zolazepam (Zoletil 100, Virbac, France) and their blood was sampled for further laboratory investigation.

Plasma glucose concentration, alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activity were determined using kits (Vital Development Corporation, Russia). To evaluate the status of the antioxidant system the activities of superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPO), and glutathione reductase (GR) were determined. CAT activity was determined by measuring the recycling speed of hydrogen peroxide, measured using the absorption of light at 260 nm [7]. SOD activity was determined using the deceleration stage of quercetin autooxidation [8]. GPO activity was determined by the rate of oxidation of glutathione during its reaction with tert-butyl hydroperoxide [7]. GR activity was determined by measuring the consumption of NADPH during the restoration of the oxidized form of glutathione [7]. The content of reduced glutathione (GSH) in erythrocytes was determined by quantifying its reaction with dithiobisnitrobenzoic acid after deproteinization using sulfosalicylic acid [7].

The oxidative modification of proteins was evaluated by measuring the amount of bityrosine remaining in the blood and the autofluorescence of tryptophan in proteins. The evaluation of the autofluorescence of tryptophan was carried out after the dilution of the blood plasma to contain 0.5 mg/ml protein using an excitation wavelength of 280 nm and an emission wavelength of 330 nm. The determination of bityrosine content was performed using a fluorometric method involving an excitation wavelength of 325 nm and an emission wavelength of 418 nm [9]. The content of glycation products in the blood plasma was measured fluorometrically using an excitation wavelength of 370 nm and an emission wavelength of 404 nm [10]. Measurement of fluorescence intensity was performed using a CM2203 spectrofluorometer (SOLAR, Belarus).

Data processing. Stat plus LE was used to analyze the data. They are presented as median (Me), 25th, and 75th percentiles (p0,25/p0,75) and were analyzed using the Mann – Whitney U-test. $p < 0.05$ was considered to represent statistical significance.

Results and Discussion. The rates of mortality in the first 2–4 days after alloxan injection in rats in Groups 2 and 4 were 28 % and 32 %, respectively, which is typical for this experimental model [11]. The plasma glucose concentration in rats with alloxan-induced diabetes increased by 66.4 % in the first month after the administration of alloxan (Table 1). In animals that underwent alcoholization for 2 months plasma glucose was similar to that of control rats. It is well established that hypoglycemia occurs following chronic alcoholization, but in a previous study [5] in which the plasma glucose concentrations had halved, the rats had been given ethanol for more than 16 weeks. However, in another study [3] gavage of ethanol for 14 days caused slight hyperglycemia (29 % increase), although by the 29th day, glycemia had

returned to normal. Finally, in a third study [12] 2-month alcoholization caused a non-significant lowering of plasma glucose concentration. Thus, our data are consistent with some of the findings of earlier studies. The plasma glucose concentration of rats with alloxan-induced diabetes was two-fold higher than that of the control group and was 21.5 % higher than that of rats in Group 2.

Table 1
Plasma glucose and aminotransferase activity in rats with alloxan-induced diabetes, chronic alcoholization, or both

Group, NN	Indices		
	Glucose, mmol/l	ALT, units/l	AST, units/l
1 (Control) group	6.40 (5.90/6.91)	23.51 (19.41/24.52)	44.64 (42.89/47.93)
2 (DM) group	10.65* (10.26/11.56)	23.40 (22.00/24.33)	47.55 (44.17/50.46)
3 (A) group	6.76 (6.08/7.02)	40.19* (35.83/48.9)	45.28 (40.33/47.61)
4 (A+DM) group	12.93*^# (12.19/14.68)	50.70*^ (42.60/65.62)	49.30 (47.84/55.54)

Note: * – statistically significant difference ($p < 0.05$) versus Group 1; ^ – statistically significant difference ($p < 0.05$) between Groups 2 and 4; # – statistically significant difference ($p < 0.05$) between Groups 3 and 4.

Measurement of plasma hepatic transaminase activities demonstrated significantly higher ALT activity in Groups 3 and 4 (1.7 and 2.2 times control levels, respectively; Table 1), but no differences between these groups. Plasma AST activity in Groups 2–4 was no different from control values. However, such changes in markers of hepatocyte cytolysis are characteristic of a chronic hepatic disorder.

Study of the activity of antioxidant enzymes showed lower levels of SOD and CAT activity in erythrocytes in Groups 2–4 (Table 2). SOD activity in rats in Groups 2, 3, and 4 was 10.5 %, 13 %, and 29.5 % lower than in control rats, respectively. CAT activity in erythrocytes from animals in Groups 2 and 3 was 31 % and 43 % lower than in control rats, indicating a decrease in the resistance of the rats to cellular peroxidation due to the formation of active forms of oxygen, meaning that there is a greater risk of oxidative damage to cellular structures [13]. However, CAT activity in erythrocytes from rats in Group 4 showed a smaller (20 %) difference from control rats. Nevertheless, relative insufficiency of SOD activity in the erythrocytes rats with alloxan-induced diabetes and 2-month alcoholization implies impaired inactivation of the superoxide anion and thus the presence of oxidative stress, which represents an adverse prognostic factor [14].

Table 2
Activity of enzymes involved in antioxidant protection in erythrocytes from rats with alloxan-induced diabetes, chronic alcoholization, or both

Group, NN	Indices	
	SOD, con. un.	CAT, mmol/l×min
1 (Control) group	65.22 (62.89/67.05)	16.24 (15.84/20.85)
2 (DM) group	56.77 (56.02/58.00) *	9.42 (7.96/10.74) *
3 (A) group	58.40 (56.7/62.7) *	11.24 (10.54/15.02) *
4 (A+DM) group	46.07 (45.14/50.10) *^#	13.08 (11.39/15.25) *^

Note: * – statistically significant difference ($p < 0.05$) versus Group 1; ^ – statistically significant difference ($p < 0.05$) between Groups 2 and 4; # – statistically significant difference ($p < 0.05$) between Groups 3 and 4.

The enzymes of glutathione metabolism neutralize secondary and tertiary radicals and regenerate other antioxidants, representing a second line of defense against free radicals and reactive molecules, and a more adaptive mechanism. Increases in activity of both GPO and GR during the initial stages of development of the pathological process and prolonged high levels of activity have been demonstrated in other similar studies [15–17]. Measurement of the activity of these enzymes of glutathione metabolism in animals with alloxan-induced diabetes revealed respectively 68 % and 37 % higher activity levels of GPO and GR (Table 3). In rats that had undergone chronic alcoholization the GPO activity was 50 % higher and the GR activity was 88 % higher than controls. The erythrocytes of rats in Group 4 were characterized by 30 % higher GR activity, but there was no difference from controls in their GPO activity. This may imply a decompensation of the thiol link in the antioxidant system of animals in Group 4. The concentration of reduced glutathione in the erythrocytes was lower in Groups 2–4 than controls by a mean 31 %, but there were no significant differences between the three experimental groups.

Table 3
Indices of the metabolic state of glutathione in erythrocytes of rats with alloxan diabetes, chronic alcoholization, or both

Group, NN	Indices		
	GPO, $\mu\text{mol/l} \times \text{min}$	GR, $\mu\text{mol/l} \times \text{min}$	GSH, $\mu\text{mol/ml}$
1 (Control) group	330.2 (300.55/358.69)	742.23 (650.30/800.25)	2.44 (2.29/2.62)
2 (DM) group	553.30* (502.10/585.96)	1,021.03* (911.80/1,131.57)	1.68* (1.61/1.74)
3 (A) group	496.59* (423.14/507.09)	1,392.1* (1,297.9/1,411.5)	1.66* (1.51/2.06)
4 (A+DM) group	347.99^# (328.69/414.68)	961.9*# (889.14/1,124.81)	1.69* (1.63/2.14)

Note: * – statistically significant difference ($p < 0.05$) versus Group 1; ^ – statistically significant difference ($p < 0.05$) between Groups 2 and 4; # – statistically significant difference ($p < 0.05$) between Groups 3 and 4.

Measurement of the fluorescence indices in the plasma revealed a larger quantity of products of non-fermented glycation of proteins in rats with alloxan diabetes alone and in those that had also been subjected to chronic alcoholic intoxication. However, in Group 2 this was 69 % higher, while in Group 4 it was only 33 % higher than in controls. The bityrosine residue content was higher than in controls in all the experimental groups by 10–20 %. Bityrosine is generated by the monoelectronic oxidation of the tyrosine residues of protein molecules, with the fusing of two such residues. This is a predictable product of protein oxidation that is suitable for use as a marker of oxidative damage because it has a long half-life and is accumulated in the body [18]. The fluorescence of tryptophan residues in protein molecules in the blood plasma was 26–30.5 % lower in Groups 2–4 than in controls. The fluorescence of tryptophan residues in proteins depends on their surroundings; therefore, this provides confirmation of oxidative changes in molecules or the effects of endotoxins (Table 4).

Table 4

Plasma concentrations of products of glycation and oxidative modification of proteins in rats with alloxan diabetes, chronic alcoholization, or both

Group, NN	Indices		
	PGP, conv. units	Bityrosine, conv. units	Fluorescence of tryptophanlys, conv. units
1 (Control) group	0.80 (0.76/0.90)	1.61 (1.50/1.68)	27.2 (27.0/28.6)
2 (DM) group	1.35 (1.23/1.44)*	1.93 (1.83/2.05)*	18.9 (18.45/19.35)*
3 (A) group	0.70 (0.68/0.82)	1.70 (1.64/1.77)	19.7 (19.6/20.3)*
4 (A+DM) group	1.06 (1.00/1.15)*^#	1.88 (1.82/1.98)*	20.0 (19.4/20.4)*

Note: * – statistically significant difference ($p < 0.05$) versus Group 1; ^ – statistically significant difference ($p < 0.05$) between Groups 2 and 4; # – statistically significant difference ($p < 0.05$) between Groups 3 and 4.

Conclusions. Our data demonstrate the complex disorders of carbohydrate metabolism when experimental diabetes is induced against a background of chronic alcoholization, involving a number of pathological processes. Interestingly, the quantity of products of non-fermented glycation of plasma proteins in rats that had alloxan-induced diabetes and had undergone chronic alcoholization was lower than in the animals with diabetes alone. This could be the result of the temporary appearance of hyperglycemia in animals consuming ethanol, which could be especially apparent in the early stages of the pathological process. The severity of liver damage in laboratory animals with both diabetes and chronic alcoholic intoxication was no different to that in rats with either diabetes or alcohol intoxication alone, but there was a tendency towards higher ALT activity in the former. There were also effects on the antioxidant system in rats with diabetes and chronic alcoholic intoxication, demonstrated by lower SOD, GPO, and GR activity versus the comparison groups, implying a more pronounced decompensation of the non-specific antioxidant resistant system in these animals.

Disclosures:

The authors declare no conflict of interest.

Acknowledgements. This work was supported by the State Task of the Ministry of Health Care of the Russian Federation by state assignment (28.01.2015, part 1, chapter 1). We thank Mark Cleasby, PhD, from Edanz Group (www.edanzediting.com/ac) for editing a draft of this manuscript.

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About authors:

Bykov Ilya Mikhaylovich, MD, PhD, Professor, Head of the Department of Fundamental and Clinical Biochemistry;
tel.: +79182125530; e-mail: ilya.bh@mail.ru

Popov Konstantin Andreevich, PhD, Assistant; tel.: +79288824941; e-mail: naftalin444@mail.ru

Berberidi Hristina Panaetovna, Post-graduate student; tel.: +79189045555; e-mail: hristy@mail.ru

Tsybalyuk Igor Yuryevich, Laboratory assistant in the Department of Surgery № 2; tel.: +79284300769; e-mail: igor_ts@inbox.ru

Ermakova Galina Alekseevna, PhD, Assistant in the Department of Psychiatry; tel.: +79182125530; e-mail: ilya.bh@mail.ru

Pavlyuchenko Ivan Ivanovich, MD, PhD, Professor, Head of the Department of Biology; tel.: +79183668281; e-mail: pavluchenkoi@ksma.ru

Storozhuk Petr Grigorievich, MD, PhD, Professor of the Department of Fundamental and Clinical Biochemistry;
tel.: +78612680230; e-mail: ilyaMB@ksma.ru

Ovsyannikov Viktor Grigorievich, MD, PhD, Professor, Head of Department of Pathophysiology;
tel.: +78632014412; e-mail: ovsyannikov_vg@mail.ru

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UDC 615.28

DOI – <https://doi.org/10.14300/mnnc.2018.13093>

ISSN – 2073-8137

EFFECT OF NIOSOMAL ANTIMICROBIAL PEPTIDE hBD-1 ON THE HEALING RATE OF INFECTED WOUNDS IN RATS

Bolatchiev A. D.¹, Baturin V. A.^{1,2}, Bazikov I. A.¹, Maltsev A. N.¹

¹ Stavropol State Medical University, Russian Federation

² Center of Clinical Pharmacology and Pharmacotherapy, Stavropol, Russian Federation

ВЛИЯНИЕ НИОСОМАЛЬНОГО АНТИМИКРОБНОГО ПЕПТИДА hBD-1 НА СКОРОСТЬ ЗАЖИВЛЕНИЯ ИНФИЦИРОВАННЫХ РАН У КРЫС

А. Д. Болатчиев¹, В. А. Батурин^{1,2}, И. А. Базиков¹, А. Н. Мальцев¹

¹ Ставропольский государственный медицинский университет,
Российская Федерация

² Центр клинической фармакологии и фармакотерапии, Ставрополь,
Российская Федерация

In this study we investigated the effect of an antimicrobial peptide hBD-1 capsulated in organosilicon nanocontainers (niosomes) on the healing rate of experimental wounds infected with the clinical strain of *Staphylococcus aureus*. It was shown that the niosomal gel containing recombinant hBD-1 (1 µg/ml) increases the linear healing rate of infected wounds in rats compared to the control group.

Keywords: antimicrobial peptide, defensin, niosome, antibiotic resistance, hBD-1

Определялось влияние антимикробного пептида из класса бета-дефензинов (hBD-1), инкапсулированного в кремнийорганические наноконтейнеры (ниосомы), на скорость заживления экспериментальных ран, инфицированных клиническим штаммом *Staphylococcus aureus*. Было показано, что ниосомальный гель, содержащий рекомбинантный hBD-1 (1 мкг/мл), увеличивал линейную скорость заживления инфицированных ран у крыс по сравнению с контрольной группой.

Ключевые слова: антимикробные пептиды, дефензины, ниосомы, антибиотикорезистентность, hBD-1

For citation: Bolatchiev A. D., Baturin V. A., Bazikov I. A., Maltsev A. N. EFFECT OF NIOSOMAL ANTIMICROBIAL PEPTIDE hBD-1 ON THE HEALING RATE OF INFECTED WOUNDS IN RATS. *Medical News of North Caucasus*. 2018;13(3):515-517. DOI – <https://doi.org/10.14300/mnnc.2018.13093>

Для цитирования: Болатчиев А. Д., Батурин В. А., Базиков И. А., Мальцев А. Н. ВЛИЯНИЕ НИОСОМАЛЬНОГО АНТИМИКРОБНОГО ПЕПТИДА hBD-1 НА СКОРОСТЬ ЗАЖИВЛЕНИЯ ИНФИЦИРОВАННЫХ РАН У КРЫС. *Медицинский вестник Северного Кавказа*. 2018;13(3):515-517. DOI – <https://doi.org/10.14300/mnnc.2018.13093>