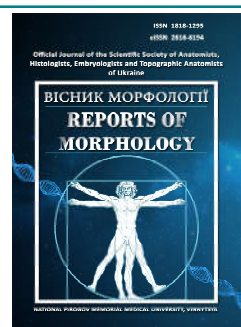




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Comparative characteristics of the manifestations of damage and reparative processes in the mucous membrane of the duodenum of rats under the conditions of skin burns and skin burns associated with diabetes

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Currently, severe thermal injury is becoming one of the most important problems of practical medicine. Diabetes is also recognized as another global medical and social challenge of our century. The emergency situation for the treatment and prevention of the consequences of these pathologies is a consequence of the lack of a reliable theoretical basis for solving specific clinical problems regarding the course of burns, diabetes and their complications. The aim of the study is to establish the patterns of structural changes in the mucous membrane of the duodenum after burn injury of the skin of rats under conditions of experimental diabetes mellitus. The study was performed on 63 laboratory white adult male rats weighing 180-210 g, which were divided into 3 groups: intact animals, rats with skin burns and rats with skin burns on the background of diabetes. The model of experimental diabetes mellitus was reproduced by administering Streptozotocin to rats intraperitoneally once at a dose of 50 mg/kg, pre-dissolved in 0.1 M citrate buffer solution (pH=4.5). The control of the development of hyperglycemia in the experimental groups was the level of glucose in the blood 24.24 ± 0.79 mmol/l. In the control group this index was 8.03 ± 0.4 mmol/l. Rats with skin burns revealed destructive manifestations, which are accompanied by an active inflammatory reaction and corresponding necrotic changes, while rats with skin burns on the background of diabetes mellitus pathological processes are not just "summed up", but in some way adaptively modified with the involvement of stress mechanisms of the endoplasmic reticulum and associated autophagy.

Key words: duodenum, thermal trauma, diabetes.

Introduction

Medium molecular weight proteins, which are products of proteolytic cleavage of blood plasma and tissue peptides, various toxins, proteolytic enzymes, mediators of immune reactions, prostaglandins, and cytokines, play an important role in the development of burn endogenous intoxication. The result of the rapid development of endogenous intoxication is the appearance of other disorders typical of burns - multiorgan failure syndrome, hypermetabolism syndrome, systemic inflammatory and apoptotic response. Thermal burn injury causes structural and functional changes in the small intestine, the nature and depth of which in some way correlate with the stage of the burn. Intestinal dysfunction (based on reactive, compensatory-adaptive and destructive changes in the cells of the digestive

system) plays an important role in the development of multiorgan failure typical to burns.

Complications of diabetes mellitus and their symptoms are caused by abnormal gastrointestinal motility, which is a consequence of diabetic autonomic neuropathy. Currently, the most common is the streptozotocin model of experimental diabetes mellitus [2, 3, 10, 16, 17, 20]. Streptozotocin is an antibiotic (2-deoxy-2-methylnitrosamino-carbonyl-amino-O-glucopyranose) that is isolated during the fermentation of a culture of *Streptomyces aerotogenes*. Streptozotocin in the chemical sense is N-acetylglucosamine, which has in the position of acetate a residue of nitrosourea. According to the peculiarities of hormonal and metabolic changes, streptozotocin-induced

diabetes is similar to type 1 diabetes in humans, although under the conditions of this experimental model absolute insulin deficiency is a consequence of direct toxic damage to beta cells and occurs without autoimmune mechanisms, which is characteristic of their destruction. type 1 diabetes mellitus in humans.

It is generally accepted that the destructive processes of various genesis in the mucous membrane of the gastrointestinal tract not only cause changes in its barrier function, but also have critical effects on the course of carbohydrate, protein and lipid metabolism in the body [11, 14, 15, 19, 21]. Therefore, the study of polycasual reaction [14] of the mucous membrane of the gastrointestinal tract, as part of the general maladaptation syndrome in diabetes mellitus (especially in combination with diabetes and other pathological conditions) remains relevant [19].

The aim of the study is to establish the patterns of structural changes in the mucous membrane of the duodenum after burn injury of the skin under conditions of experimental diabetes mellitus.

Materials and methods

The study was performed on 63 laboratory white adult male rats weighing 180-210 g. The control group - 21 intact rats without signs of somatic pathology, the first experimental group included 21 rats with experimentally simulated skin burn injury, the second experimental group included 21 experimental rats simulated skin burn injury and diabetes. The experimental diabetes mellitus model was replicated by administering intraperitoneal streptozotocin to rats once at a dose of 50 mg/kg, pre-dissolved in 0.1 M citrate buffer solution (pH=4.5). The control of the development of hyperglycemia in the experimental groups was the level of glucose in the blood - 24.24 ± 0.79 mmol/l. In the control group this level was 8.03 ± 0.4 mmol/l.

The department of the duodenum was taken for morphological examinations, fragments of which were examined by light and electron microscopy.

Results

Damage to the epitheliocytes of the duodenal mucosa is based on deep destructive changes (mostly necrotic), which after 21 days (in the stage of septicotoxemia) are usually irreversible and develop against the background of significant intoxication of the body.

The revealed structural changes of epitheliocytes are evidence of violation of the structural integrity of the intestinal epithelial barrier (Fig. 1). In animals of the first experimental group, the damaged epithelium of the duodenal mucosa critically weakens the adequacy of its interface function between the mucosa and the intestinal lumen environment and is structurally unable to provide an effective barrier against toxins, pathogens and antigenic molecules.

The course of structural changes in the mucous membrane of rats of the first experimental group in terms of development over time is staged and it can be divided

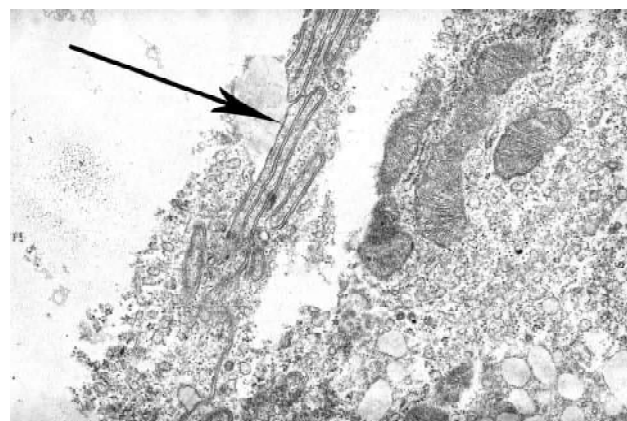


Fig. 1. Destruction of adjacent areas of adjacent epitheliocytes with a brush border in the duodenal mucosa of the rat of the first experimental group 7 days after the burn. The arrow marked the preserved area of intraepithelial contact, preserved in a fragment of intestinal cellular detritus. Electronic microphotography. x30000.

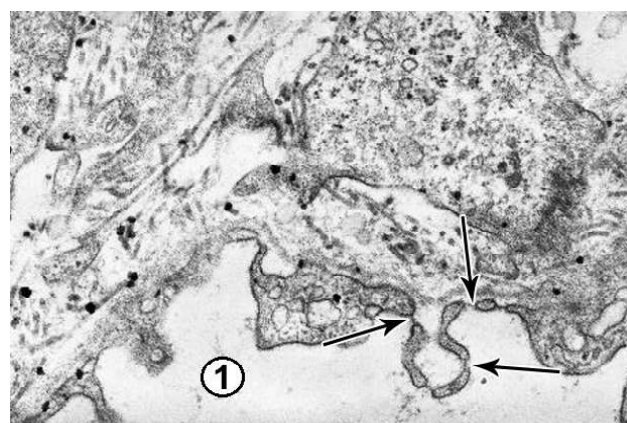


Fig. 2. Detachment of fenestrated blood capillary endothelium from the preserved basement membrane of the blood capillary in the loose connective tissue of the duodenal mucosa of the rat of the first experimental group 7 days after the burn. Arrows marked fenestrae in the endothelial cell. 1 - lumen of the blood capillary. Electronic microphotography. x30000.

into three stages and two phases. Seven days after the burn, structural manifestations of the overload stage are registered (which consists of a combination of total and subtotal destruction of some cells and with structural manifestations of the functional stress phase of other structurally preserved cells) (Fig. 2).

Fourteen days after the burn, a fixed structural picture of mucosal changes unfolds, which corresponds to the stage of relative stabilization of hyperfunction. Twenty-one days after the burn, the structural manifestations of gradual depletion and the development of decompensation are registered (Fig. 3).

The structural expression of the overload stage is not only the destruction of the plasmalemma followed by total or subtotal cell destruction, but also the changes of the organelles of the preserved cells detected by us (characteristic of the functional stress phase).

This is an expansion of the lumen and an increase in

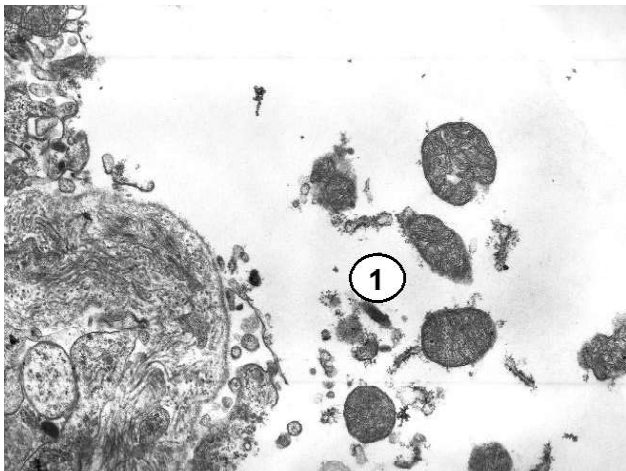


Fig. 3. Cellular detritus (1) in the intestinal space of the duodenum of rats of the first experimental group 21 days after the burn. Electronic microphotography. x20000.

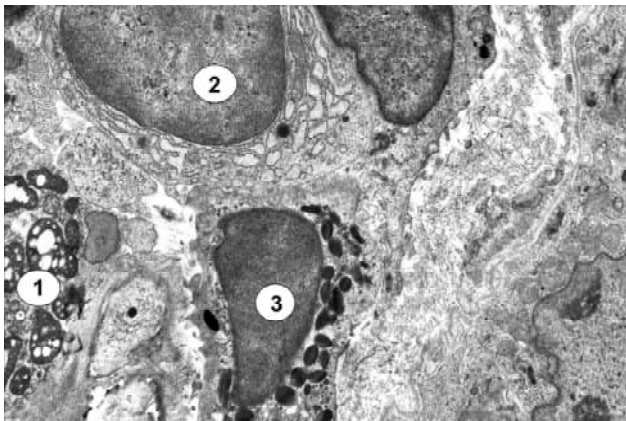


Fig. 4. Loose connective tissue of the duodenal mucosa of the rat of the third experimental group 21 days after the start of the experiment. 1 - accumulation of microbial bodies; 2 - plasma cell nucleus; 3 - leukocyte nucleus. Electronic microphotography. x10000.

the number of tubules of the granular endoplasmic reticulum (as well as an increase in the number of ribosomes attached to the tubular wall); increase in the number of ribosomes and polysomes; Golgi complex hypertrophy; increase in the number and variability of mitochondria (presence of large, old and small "young" mitochondria). But such intensive functioning, in our opinion, accelerates the rate of cell depletion, increases the accumulation in the cytoplasm of substances and damaged (defective) organelles, which activates intracellular mechanisms that lead to cell death (excessive autophagy and necrosis).

The phase of functional stress (and all its above manifestations) under the condition of favorable for the maintenance of cell life coincides with the phase of consolidation of compensatory-adaptive processes (which precedes the stage of relative stabilization of hyperfunction). But prolonged compensatory hyperfunction leads to the destruction of cellular organelles and cytoplasmic matrix,

promotes the transition to the stage of gradual depletion and the development of decompensation.

Thus, not only the primary direct destruction of intestinal epithelial cells, but also organelle hyperplasia, probably leads to a disorder of the intracellular self-regulation system, disruption of intercellular interactions (including due to the destruction of dense intraepithelial contacts). It is probable that in the stage of stabilization of structural bases of function compensation the development of compensatory adaptations of intestinal epithelium ends and the point of dichotomy appears: the process goes towards full restoration of functions or towards decompensation of functions (in case of loss of structural support, even partial restoration of functions).

In contrast to the above changes, in the duodenal mucosa of rats of the second experimental group, the dynamics of morphological changes during different periods after burns (7 days - stage of shock and early toxemia; 14 days - stage of late toxemia; 21 days - stage of septicotoxicity) differs from this in animals of the first experimental group. Comparison of the obtained data with the previously revealed ones gives grounds to believe that the time intervals and the nature of the adaptive changes of the duodenal mucosa are significantly prolonged and worsened.

Significant lesions of the mucous membrane should lead to disruption of the digestive system, parietal digestion and absorption, as well as immunological protection (due to the fact that the mucous membranes are the first zone of contact with environmental antigens and a leading link in immune protection) which, no doubt, affects the condition of the body of the burned and, to a large extent, determines the development of burns, as well as the course of diabetes.

Evidence of dysfunction of the immune component of the intestinal barrier is our structural signs of stress function (adaptation) of various immunocompetent cells (lymphocytes, plasma cells, macrophages, which provide the functions of antigen presentation and secretion of local inflammatory response mediators), and subsequently to their partial destruction (failure of adaptation). This leads to the further development of an inflammatory reaction involving leukocytes, which also undergo a stage of activation and subsequent depletion (a sign of which is the destruction of leukocytes). These structural transformations are accompanied by the appearance in the cytoplasm of epitheliocytes and in the loose connective tissue of the duodenal mucosa of microbial bodies, which is a sign of microbial infiltration and general shifts of the intestinal microbiota (which, under normal conditions, acts as a biological intestinal filter) (Fig. 4).

The permeability of the duodenal mucosa barrier is also impaired in the association of experimental skin burn injury with streptozocin-induced diabetes mellitus. We have found structural transformations of goblet cells that produce intestinal mucus, which forms a layer on the surface of the intestinal mucosa.

A gradual change in the types of secretion of goblet

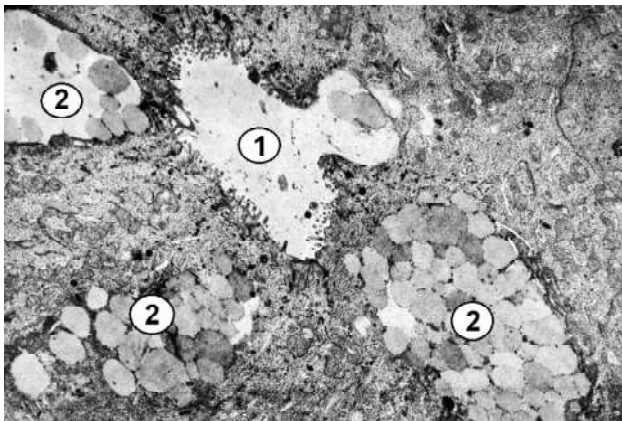


Fig. 5. Destruction of secretory granules with content of different degrees of electron density in the apical parts of goblet cells in the duodenal mucosa of rats of the third experimental group 21 days after the start of the experiment. 1 - lumen, 2 - secretory granules with content of different degrees of electron density. Electronic microphotography. x20000.

cells has been registered: from merocrine type (by exocytosis of mature secretory granules) to apocrine type (by rejection of the apical part of goblet cells together with secretory granules and individual organelles due to clasmatosis), and further - to the holocrine type (by complete destruction of the apical part of goblet cells and even complete destruction of these cells) (Fig. 5).

This leads to structural signs of changes in the intestinal mucosa (which at the electron microscopic level consists of small globular content of low electron density, secretory granules with preserved membrane and cell detritus, and on histological sections stained with hematoxylin and eosin in the lumen of the intestinal lumen). eosinophilic content). The structural changes of epitheliocytes detected under the conditions of the experiment are evidence of violation of the structural integrity of the intestinal epithelial barrier.

Thus, the permeability of the intestinal mucosal barrier is altered and these changes can be interpreted as contributing to microbial invasion. The results of studies have shown that the basis of damage to the epitheliocytes of the duodenal mucosa are profound destructive changes, which after 21 days (in the stage of septicotoxemia) are usually irreversible and develop against the background of significant intoxication. In animals of the second experimental group, the damaged epithelium of the duodenal mucosa critically weakens the adequacy of its function of the interface between the mucosa and the intestinal lumen environment and is structurally unable to provide an effective barrier against toxins, pathogens and antigenic molecules.

In particular, it should be noted that the structural changes of the mucous membrane in rats after burn injury of the skin associated with diabetes develop over time so rapidly that the stage of relative stabilization can be considered significantly unsettled.

The obtained results can be used to develop methods

to prevent the development of negative changes in the intestinal epithelial barrier of the duodenum and enterocytes in particular and to effectively treat their possible consequences.

Discussion

The prevalence of diabetes, its specific complications, and the presence of other comorbidities that often accompany diabetes make it one of the major social and public health problems. The steady increase in information about the etiology and pathogenesis of diabetes mellitus, as well as its chronic complications, leads to the need for timely supplementation and clarification of existing scientific concepts, accompanied by constant reassessment of already established pathophysiological processes and structural mechanisms that provide them.

In the modern scientific literature there is an idea that "diabetes is predominantly an intestinal disease". It is well known [9] that the small intestine plays an important role in both digestion and the endocrine response in regulating blood glucose levels. Hormones (GLP-1 and GIP) released from the small intestinal mucosa in response to ingestion of nutrients modulate the secretory response of insulin and glucagon to this food.

The data obtained by us may indicate the greatest contribution to the development of diabetic enteropathy of structural changes in the mucous membrane of the duodenum. In addition, it should be noted that the structural transformation of the duodenal mucosa under the conditions of the applied model of experimental burns is deepened in its association with diabetes mellitus.

Also important is the dependence of morphological changes of the duodenal mucosa on the course characteristic of [4, 7, 12, 13, 18, 22] for burn disease complexes of syndromes (endotoxic syndrome, hypermetabolism syndrome, systemic inflammatory response and their general manifestation in the form of internal dysfunction). It should be noted that the excretion of toxins from the body of burnt [13] is carried out in several ways (skin, kidneys, lungs, liver and gastrointestinal tract), but these ways in the development of burn disease are blocked by "actual intraorgan afterburn pathology". In addition to the fact that the mucous membrane of the duodenum is affected by endotoxins of various origins (from foci of ischemia and necrosis in the burn area and from initially intact internal organs), It is worth noting the local pathogenic effects of leukocytes, which we found in large numbers in the mucous membrane of the duodenum of rats of the first and especially the third experimental group. It should be noted that their main foci of toxic products are formed due to the interaction of leukocytes and their main forms of oxygen with damaged cells. This phenomenon is called "oxygen-metabolic, or respiratory explosion" [8]. The chain connection of this phenomenon is that against the background of activation of neutrophils occurs almost fifteen times, which in turn induces the relaxation of smooth muscle. Under the combination of burn disease with diabetes mellitus, these

processes are superimposed on the typical for diabetic enteropathy changes in intestinal motility in the form of alternating diarrhea with constipation [1, 5, 6, 23-25], which should generate a vicious circle of temporary intraluminal intestinal endotoxins accumulation and subsequent intestinal release from them. In general, this variant of the dynamics of changes in pathological mechanisms, which increases the uncertainty and adds instability to the nature of intestinal motility, should affect the course of diabetic enteropathy in terms of its association with burn injury. These data obtained by us and made on the basis of their assumptions are theoretically plausible, but need to be confirmed in clinical trials.

Conclusions

In the mucous membrane of the duodenum after burn injury of the skin associated with diabetes there is a deterioration of the adaptive response and prolongation of destructive processes, accompanied by disruption of intercellular interactions in cytoarchitecturally altered and deformed villi and crypts. Structural changes in the duodenal mucosa of rats in experimental burn skin injury under experimental streptozotocin-induced diabetes mellitus 7, 14 and 21 days after the start of the experiment indicate structural features (peculiarity of morphological features) of enteropathy in burnt with diabetics.

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ПОРІВНЯЛЬНА ХАРАКТЕРИСТИКА ПРОЯВІВ ПОШКОДЖЕННЯ ТА РЕПАРАТИВНИХ ПРОЦЕСІВ У СЛИЗОВІЙ ОБОЛОНЦІ ДВНАДЦЯТИПАЛОЇ КИШКИ ЩУРІВ ЗА УМОВ ОПІКОВОЇ ТРАВМИ ШКІРИ ТА ОПІКОВОЇ ТРАВМИ ШКІРИ, АСОЦІЙОВАНОЮ З ЦУКРОВИМ ДІАБЕТОМ

Тимошенко І.О., Сокурєнко Л.М., Янчишин А.Я., Пастухова В.А.

На даний час важка термічна травма стає однією з найважливіших проблем практичної медицини. Ще одним глобальним медико-соціальним викликом нашого століття визнано також цукровий діабет. Надзвичайна ситуація щодо профілактики, лікування та попередження наслідків цих патологій є наслідком відсутності надійного теоретичного підґрунтя у вирішенні конкретних клінічних проблем щодо перебігу опікової хвороби, цукрового діабету та їх ускладнень. Метою дослідження є встановлення закономірностей структурних змін в слизовій оболонці дванадцятипалої кишки після опікової травми шкіри щурів за умов експериментального цукрового діабету. Дослідження здійснено на 63 лабораторних білих статевозрілих щурах-самцях масою 180-210 г, котрі були розподілені на 3 групи: інтактні тварини, щурі з опіковою травмою шкіри та щурі з опіковою травмою шкіри на тлі цукрового діабету. Модель експериментального цукрового діабету відтворювали шляхом введення щурам стрептозотоцину внутрішньоочередово одноразово в дозі 50 мг/кг, попередньо розчинивши його в 0,1 М цитратному буферному розчині (рН=4,5). Контролем розвитку гіперглікемії в експериментальних групах був рівень глюкози в крові $24,24 \pm 0,79$ ммоль/л. В контрольній групі цей показник становив $8,03 \pm 0,4$ ммоль/л. У щурів з опіковою травмою шкіри виявлені деструктивні прояви, котрі супроводжуються активною запальною реакцією і відповідними некротичними змінами, в той час як у щурів з опіковою травмою шкіри на тлі цукрового діабету патологічні процеси не просто "сумуються", а певним чином адаптивно модифікуються із залученням механізмів стресу ендоплазматичного ретикулула та асоційованої з ним автофагії.

Ключові слова: дванадцятипала кишка, термічна травма, діабет.

СРАВНИТЕЛЬНАЯ ХАРАКТЕРИСТИКА ПРОЯВЛЕНИЙ ПОВРЕЖДЕНИЯ И РЕПАРАТИВНЫХ ПРОЦЕССОВ В СЛИЗИСТОЙ ОБОЛОЧКЕ ДВНАДЦАТИПЕРСТНОЙ КИШКИ КРЫС В УСЛОВИЯХ ОЖОГОВОЙ ТРАВМЫ КОЖИ И ОЖОГОВОЙ ТРАВМЫ КОЖИ, АССОЦИИРОВАННОЙ С САХАРНЫМ ДИАБЕТОМ

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В данное время тяжелая термическая травма становится одной из наиболее важных проблем практической медицины. Еще одним глобальным медико-социальным вызовом нашего столетия признан также сахарный диабет. Чрезвычайная ситуация по профилактике, лечению и предупреждению последствий этих патологий является следствием отсутствия надежного теоретического основания в решении конкретных клинических проблем о ходе ожоговой болезни, сахарного диабета и их осложнений. Целью исследования является установление закономерностей структурных изменений в слизистой оболочке двенадцатиперстной кишки после ожоговой травмы кожи крыс в условиях экспериментального сахарного диабета. Исследование проведено на 63 лабораторных белых половозрелых крысах-самцах массой 180-210 г, которые были разделены на 3 группы: интактные животные, крысы с ожоговой травмой кожи и крысы с ожоговой травмой кожи на фоне сахарного диабета. Модель экспериментального сахарного диабета воспроизводили путем введения крысам стрептозотоцина внутрибрюшинно однократно в дозе 50 мг/кг, предварительно растворив его в 0,1 М цитратном буферном растворе (рН=4,5). Контролем развития гипергликемии в экспериментальных группах был уровень глюкозы в крови $24,24 \pm 0,79$ ммоль/л. В группе контроля $8,03 \pm 0,4$ ммоль/л. У крыс с ожоговой травмой кожи обнаружены деструктивные проявления, которые сопровождаются активной воспалительной реакцией и соответствующими некротическими изменениями, в то время как у крыс с ожоговой травмой кожи на фоне сахарного диабета патологические процессы не просто "суммируются", а определенным образом адаптивно модифицируются с привлечением механизмов стресса эндоплазматического ретикулула и ассоциированной с ним автофагии.

Ключевые слова: двенадцатиперстная кишка, термическая травма, диабет.