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THE NADH-UBIQUINONE REDUCTASE AND SUCCINATE DEHYDROGENASE ACTIVITY IN THE RAT KIDNEY MITOCHONDRIA UNDER THE CONDITIONS OF DIFFERENT PROTEIN AND SUCROSE CONTENT IN THE DIET

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The NADH-ubiquinone reductase (EC 7.1.1.2) and succinate dehydrogenase (EC 1.3.5.1) activity, the levels of total ubiquinone and its redox forms, and the degree of oxidative modification of mitochondrial proteins in the rat kidney were investigated. It was found that when consuming a low-protein diet there is a decrease in NADH-ubiquinone reductase and succinate dehydrogenase activity by 1.4-1.7 times, a 20% decrease in total ubiquinone and a quantitative redistribution of its oxidized and reduced form with a predominance of oxidized form. Under the studied conditions, there is no accumulation of carbonyl derivatives, but the level of free SH-groups is significantly reduced compared with control. At the same time, in animals consuming a high-sucrose diet there is an increase in NADH-ubiquinone reductase and succinate dehydrogenase activity by 1.5-2 times and maintenance of the total ubiquinone at the control level against the background of redistribution of its redox forms, namely a decrease in reduced ubiquinone and an increase in oxidized ubiquinone on average by 1.5 times. In addition, there is an intensification of the reactions of free radical damage of mitochondrial proteins in kidney cells, as evidenced by an increase in the level of carbonyl derivatives and a significant decrease in the level of free protein SH-groups by approximately 1.4-1.5 times. The most pronounced changes in the studied indicators are found in animals that consumed a low-protein/high-sucrose diet. In particular, an excessive consumption of sucrose on the background of protein deficiency is accompanied by a reduction of NADH-ubiquinone reductase and succinate dehydrogenase activity by 1.7-2 times, a decrease in total ubiquinone level by approximately 1.4 times, and a two-fold decrease in reduced ubiquinone against the background of intensification of the free radical oxidation of mitochondrial proteins, which can be considered as a prerequisite for the impairment of the renal function under the conditions of carbohydrate-protein imbalance.

K e y w o r d s: nutrients, kidney, NADH-Ubiquinone reductase, succinate dehydrogenase, ubiquinone, carbonyl derivatives, protein SH-group.

It is known that excessive consumption of sucrose induces metabolic disorders that precede the development of diabetes mellitus [1]. Our previous studies have shown that in the liver mitochondria of animals kept on a high-sucrose diet there is an activation of free radical processes, including intensification of hydroxyl radical generation, accumulation of primary (diene conjugates, ketodienes) and secondary (TBA-active) lipid peroxidation products, along with a two-fold increase in ratio of cholesterol/phospholipids, as well as the accumulation of products of oxidative protein modification [2]. The interaction between protein and carbohydrate intake is also important. There is growing evidence from studies on a wide range of species that, rather than macronutrients acting singly, it is their interactive effects (their balance) that are more important for health. Many of these macronutrient studies used

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a nutritional geometry approach, where the effects of macronutrients, calories, and food interrogated across a wide topology of diets differing in macronutrient content [3]. It is shown that metabolic disorders under the conditions of excessive consumption of sucrose occur in the cells of the liver, pancreas, kidneys, accompanied by the development of dyslipidemia and hyperglycemia [1, 4]. Persistent chronic hyperglycemia is known to contribute to the development and progression of diabetic nephropathy [5]. At the same time, the study of the mechanisms of development of the metabolic disorders in the kidneys under the conditions of nutritional imbalance has become especially relevant [6, 7].

Homeostatic function of the kidneys is provided by a number of processes that require a significant expenditure of ATP energy – maintenance the balance of electrolytes and acid-base status, excretion of toxic substances, reabsorption of nutrients [8-10]. Mitochondria play a key role in ensuring the functional activity of the kidneys, while their dysfunction primarily leads to disturbances of the reabsorption and filtration capacity of the kidneys, which underlies the pathogenesis of many diseases [11]. The functional activity of the kidneys is largely determined by the state of the energy supply system, while the activity of enzymes of I and II complexes of the electron transport chain and the content of ubiquinone and its redox forms are important indicators of its state. Therefore, to understand the mechanisms of the kidney dysfunction under the conditions of nutritional imbalance, in particular, the simultaneous deficiency of protein and excess of sucrose in the diet, it is important to study the respiratory chain enzyme activity. In the current research the NADH-ubiquinone reductase (EC 7.1.1.2) and succinate dehydrogenase (EC 1.3.5.1) activity, the relationship between the quantitative content of the redox forms of ubiquinone and the degree of oxidative modification of mitochondrial proteins in the kidneys under the conditions of different protein and sucrose content in the diet will be investigated for the first time.

Materials and Methods

Experimental design and procedures. In the study, 9-10 week old white nonlinear rats weighing 130-140 g were used. The animals were separated into so*litary plastic cages with sand bedding and* ad libitum access to water. The animals were monitored daily, and weighed three times/week. The experiment was conducted in accordance with the rules set by the 'European convention for the protection of vertebrate animals used for experimental and other scientific purposes' (Strasbourg, 1986).

This research was performed on 36 white nonlinear rats. The animals were divided into the following experimental groups (n = 9): I – animals receiving full-value semi-synthetic ration (control group); II – animals receiving low-protein ration; III – animals receiving high-sucrose diet; IV – animals receiving low-protein high-sucrose diet.

Diets were manufactured at Institute of Biology, Chemistry and Natural Resources, stored at 4°C plastic containers with tight-fitting lidsfor a maximum of fourteen days. Dietary composition is detailed in Table 1. Animals received feed of 30 g/ 100 g BW. In order to estimate total macronutrients from the diet, a weighed quantity of food were added daily at the same time of day (09:00 to 10:00 hours). Rationing of the daily diet was conducted taking into account the principle of dual nutrition. The animals of the control group consumed 4.2 g of protein/100 g BW, 3.0 g of fat / 100 g BW, and 3 g of sucrose/100 g BW. The animals of the group II consumed isoenergetic ration containing 1.4 g of protein/100 g BW, 3 g of sucrose/100 g BW and 3.0 g of fat/100 g BW. The animals of the group III consumed high-sucrose diet containing 4.2 g of protein/100 g BW, 12 g of sucrose/100 g BW and 3.0 g of fat/100 g BW, balanced by all other essential nutrients [2]. The animals of the group IV consumed isoenergetic ration containing 1.4 g of protein/100 g BW, 12 g of sucrose/100 g BW), and balanced by all other essential nutrients. The animals were maintained on the corresponding diet during four weeks. The body weight (BW) was measured weekly and the food intake was assessed daily.

Cervical dislocation was performed under the light ether anesthesia on day 29 of the experiment.

Mitochondrial fraction of the kidneys homogenate was separated by differential centrifugation (Heraeus Biofuge, Germany) in the following buffer medium: 250 mM sucrose, 1 mM EDTA, 10 mM Tris-HCl; pH 7.4 at 0-3°C [12].

Enzyme assays. NADH:Q reductase activity was determined by spectrophotometrically, reactions were monitored at 340 nm at 25°C, using an molar absorption coefficient of $6.22 \cdot 10^3$ $M^{-1} \cdot cm^{-1}$ to calculate enzymatic activity [13].

The succinate dehydrogenase activity was measured based on restoration of potassium ferricyanide ($K_3[Fe(CN)_6]$) to potassium ferrocyanide

Table 1. Ingredient composition of the diets (g/kg diet)

Ingredient, g/kg	Diet			
	C	LP	HS	LP/HS
	3601.0 kcal/kg	3601.0 kcal/kg	3797.7 kcal/kg	3797.7 kcal/kg
Cornstarch	620.7	714.1	320.7	414.1
Casein	140	46.6	140	46.6
Sugar	100	100	400	400
Fiber (cellulose microfiber)	50	50	50	50
Mineral mix[a]	35	35	35	35
Vitamin mix[a]	10	10	10	10
L-Cystine	1.8	1.8	1.8	1.8
Choline bitartrate	2.5	2.5	2.5	2.5
Soy oil	40	40	40	40

[a]Mineral and Vitamin mix – based on the AIN-93G vitamin and mineral mixes

Table 2. Indicators of average body weight of animals under the conditions of different protein and sucrose content in the diet

Indicator	I group, (control)	II group, (low-protein ration)	III group, (high-sucrose ration)	IV group, (high-sucrose–low-protein ration)
Body weight:				
- begining of the experiment, g	135 ± 4	134 ± 4	133 ± 3	134 ± 3
- end of the experiment, g	192 ± 5	155 ± 6[a]	208 ± 10a	160 ± 7[a]

[a]$P \leq 0.05$ vs control

($K_4[Fe(CN)_6]$) by the action of succinate dehydrogenase [14]. The mitochondria (0.1 mg of protein) were suspended in 50 mM (K) phosphate buffer (pH 7.4) containing 3 mM potassium ferricyanide (III) acting as an exogenous electron acceptor. A decrease in the absorption (420 nm) upon the addition of 50 mM succinate was followed to measure the rate of potassium ferrocyanide (II) formation at 30°C for 2 min. The reaction rate was calculated as nmol ferrocyanide formed per minute per mg protein.

Protein determination. The protein content was determined according to the method of Lowry et al.

Assay of ubiquinone. Mitochondrial fraction of the kidneys containing 10-30 mg of protein was placed in a conical centrifuge tube and denatured by the rapid addition of 4 ml of cold methanol. Light petroleum (5 ml) was added immediately and the mixture was shaken rapidly for 1 min. The tube was then given a short spin in a clinical centrifuge to separate the layers. The upper light petroleum layer was transferred to another centrifuge tube and was extracted again with 3 ml light petroleum. The combined light petroleum extracts were treated with 2 ml of 95% methanol and the mixture was shaken for 30 sec. After separation of the layers the light petroleum layer was transferred and evaporated in a vacuum desiccator. The residual lipid was redissolved in 3 ml spectroscopically pure ethanol. The presence of ubiquinone was indicated by absorption with a maximum of 275 nm. The ubiquinone was reduced by the addition of a small crystal of sodium borohydride followed by rapid stirring. Measurements were carried out by spectrophotometry (CARY 60, USA) at a wavelength of $\lambda = 275$ nm. From the decrease in extinction at 275 nm the concentration of ubiquinone in the lipid extract was calculated by using the molecular extinction coefficient for the difference in absorption of the oxidized and reduced forms of ubiquinone $(\varepsilon_{ox} - \varepsilon_{red})_{275} = 12250\ M^{-1} \cdot cm^{-1}$. Ubiquinone concentrations are expressed as nmol/mg of protein [15].

Mitochondrial proteins were extracted by method [16] that is based on principle of insolubility of products of mitochondrial translation in 0.05 M sodium-phosphate buffer with pH 11.5. Mitochondria suspension was stored at -20°C overnight. After thawing the suspension was centrifuged at 30 000 g for 20 min. The sediment was resuspended in 10 ml of 0.05 M sodium-phosphate buffer (pH 11.5) and centrifuged at 30 000 g for 20 min. The pellet was suspended in 10 ml of 50 mM sodium phosphate buffer (pH 11.5) and resedimented at 30 000 g for 20 min. The final pellet was the pH 11.5 insoluble fraction.

Protein carbonyl derivatives content assay. Protein carbonylation was assessed via amount of 2.4-dinitrophenylhydrazone derivatives, produced in reactions of oxidized amino acid residues with 2.4-dinitrophenylhydrazine, and expressed as nmol/ mg of protein [17].

Protein SH-group content assay. Protein SH-group content was determined with method based on reaction of Ellman's reagent (5,5′-dithiobis-(2-nitrobenzoic acid) (DTNB)) with SH-groups [18]. The products are disulfide and thionitrophenyl anion (TNPA). The amount of TNPA is directly proportional to the amount of SH-groups of proteins that reacted with DTNB. The content of free SH-groups was calculated basing on molar extinction coefficient of 11400 $M^{-1} \cdot cm^{-1}$ and expressed as nmol/ mg of protein.

Data analysis and statistics. The data were compared and analyzed by using unpaired T-test. Characteristics of the studied groups were expressed as Mean ± SD for normal distribution. For all statistical calculations, significance was considered to be a value of $P < 0.05$.

Results and Discussion

According to the results of the study, a decrease in NADH-ubiquinone reductase and succinate dehydrogenase activity by 1.4-1.7 times is found in the mitochondria of the kidney of rats under the conditions of low-protein diet (Fig. 1, Fig. 2). Apparently, the alimentary protein deficiency results in an impairment of the synthesis and regulation of the activity of the studied enzymes at the posttranslational level. On the other hand, the decrease in the activity of enzymatic complexes of the respiratory chain may be caused by a decrease in the energy demands of the kidney due to reduction of the filtration load under conditions of protein deficiency in the diet. Dietary protein had the greatest influence on kidney function, where chronic low protein intake decreased glomerular filtration rates and kidney mass, whereas it increased kidney immune infiltration and structural injury [19].

At the same time, in animals maintained on the high-sucrose diet, a doubling of NADH-ubiquinone reductase activity (Fig. 1) and an increase in succinate dehydrogenase activity by 1.5 times (Fig. 2) in the mitochondrial fraction of rat kidney is observed. Presumably, hyperglycemia due to the excessive sucrose consumption leads to excessive formation of oxidation substrates for the complexes I and II of the electron transport chain and increases the need for ATP required for glucose reabsorption in the proximal tubules of the kidneys, accompanied by activation of the Krebs cycle and subsequent intensification of oxidative phosphorylation [20].

The most pronounced changes in the studied indicators are found in animals that consumed a low-protein/high-sucrose diet. For this group of animals, the nature of changes in the studied parameters is similar to those found in animals with alimentary protein deficiency; the differences concern only the severity of the determined changes. In particular, an excessive consumption of sucrose on the background of protein deficiency is accompanied by a decrease in NADH-ubiquinone reductase activity by 1.6-1.7 times (Fig. 1) and a two-fold decrease in succinate dehydrogenase activity (Fig. 2) compared to control.

Apparently, the lack of protein in the diet is a determining factor of metabolic disturbances in the kidneys under the conditions of nutritional carbohydrate-protein imbalance. The consequence of these changes may be not only impairment of the electron transport and oxidative phosphorylation processes in the mitochondria of the kidney cells, but also disruption of the energy supply system of the kidney as a whole, which will lead to the progression of their injury. As stated by Sun et all [21], impaired oxidative phosphorylation in the kidney in diabetic nephropathy leads to mitochondrial dysfunction, in particular impaired ATP production due to loss of mitochondrial membrane potential and changes in cationic gradients, and increased ROS production. Thus, there is a "vicious circle" of disorders, which ultimately predict the development of renal dysfunction.

Given that the coenzyme of the studied enzymes is ubiquinone (coenzyme Q), the next step of the research was the analysis of the total ubiquinone

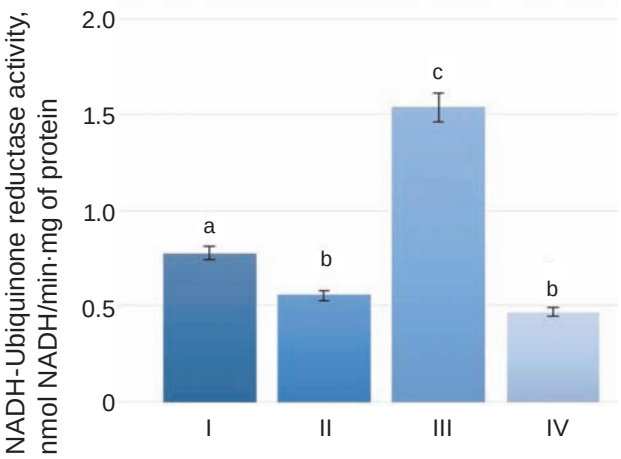

Fig. 1. The NADH-ubiquinone reductase activity in kidney cell mitochondria from rats that received diets with different contents of sucrose and protein (M ± m, n = 9): I – control rats that receive the adequate semisynthetic diet; II – rats that receive the low-protein ration; III – rats that receive the high-sucrose diet; IV – rats that receive the high-sucrose–low-protein diet. Different letters indicate significant differences inside the parameters and the same letters indicate no difference at P ≤ 0.05

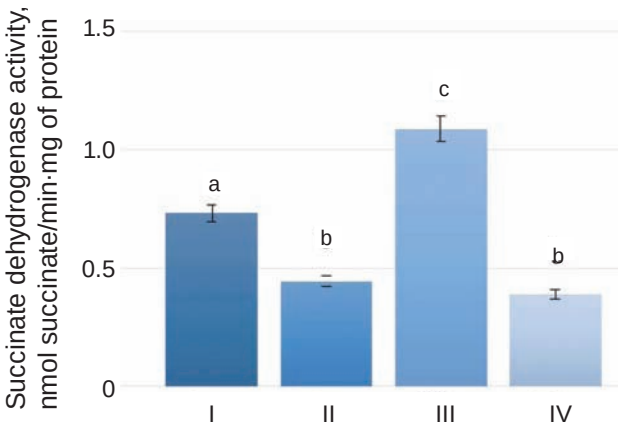

Fig. 2. The succinate dehydrogenase activity in kidney cell mitochondria from rats that received diets with different contents of sucrose and protein (M ± m, n = 9): I – control rats that receive the adequate semisynthetic diet; II – rats that receive the low-protein ration; III – rats that receive the high-sucrose diet; IV – rats that receive the high-sucrose–low-protein diet. Different letters indicate significant differences inside the parameters and the same letters indicate no difference at P ≤ 0.05

and its redox forms levels. In addition to being an important component of the mitochondrial electron transport chain, coenzyme Q acts as an electron transporter from complexes I and II to the cytochrome system [9, 21], and functions as a potent endogenous fat-soluble antioxidant [5].

According to obtained results, there is a decrease in total ubiquinone level in the mitochondria of the kidney of rats maintained on the low-protein diet by 20% compared with the control (Fig. 3). Apparently, the lack of dietary protein results in the disruption of synthesis of the benzoquinone ring of coenzyme Q molecule, which precursor is tyrosine [22]. Under the current experimental conditions, there is an increase in the level of the oxidized form of ubiquinone against the background of a simultaneous decrease in the level of its reduced form (Fig. 4) in group II compared to control.

It should be noted, that under the conditions of a high-sucrose diet in the mitochondria of the kidney the level of total ubiquinone is preserved (Fig. 3) against the background of redistribution of its redox forms, in particular a decrease in reduced ubiquinone and an increase in oxidized form by 1.5 times on average (Fig. 4) compared to control. Under the

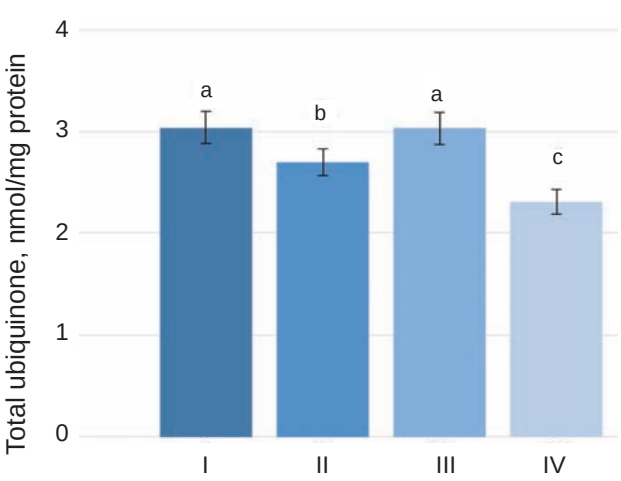

Fig. 3. The content of total ubiquinone in kidney cell mitochondria from rats that received diets with different contents of sucrose and protein (M ± m, n = 9): I – control rats that receive the adequate semisynthetic diet; II – rats that receive the low-protein ration; III – rats that receive the high-sucrose diet; IV – rats that receive the high-sucrose–low-protein diet. Different letters indicate significant differences inside the parameters and the same letters indicate no difference at P ≤ 0.05

conditions of the oxidative stress observed in hyperglycemia [5], a decrease in the level of reduced ubiquinone is associated with its participation in the antioxidant defense reactions. It has been shown that under the conditions of hyperglycemia, ubiquinone may be involved in maintaining renal functional activity by reactivating mitophagy via the Nrf2/ARE pathway. High concentrations of glucose in glomerular endothelial cells cause insufficient mitophagy, mitochondrial dysfunction and apoptosis, which are eliminated by ubiquinone [21].

It should be noted that the most pronounced changes in the level of total ubiquinone and changes in the ratio of its redox forms in the mitochondrial fraction of the kidney were registered in animals maintained on the high-sucrose diet against the background of nutritional deprivation of protein. In particular, there was a decrease in the level of total ubiquinone by 1.4 times (Fig. 3) with a simultaneous decrease in the level of reduced ubiquinone by 2.0 times compared with control (Fig. 4). Given that a decrease in ubiquinone content by 25% is accompanied by an impairment of energy metabolism, and a 75% reduction leads to cell death [23], the consequences of these changes may be critical not only for the energy biotransformation system, but also for the functioning of kidney cells in general. Apparently, the established reduction of the level of total ubiquinone and its reduced form can be considered as one of the reasons for the decrease in the activity of I and II complexes of the respiratory chain. It is known that ubiquinone deficiency leads to dysfunction of the respiratory chain [24].

Given the role of coenzyme Q as a major antioxidant in mitochondria, the determined redistribution of the ubiquinone redox forms in favor of the oxidized form in the mitochondrial fraction of rat kidney under the conditions of nutrient imbalance is likely to be accompanied by an activation of the free radical damage to the mitochondrial proteins. In the mitochondrial proteins of the kidney of rats kept on a low-protein diet an increased accumulation of carbonyl derivatives is not found (Fig. 5), but the level of free SH-groups is significantly reduced compared with control (Fig. 6). It can be explained by the fact that SH-groups play a protective role as their oxidation prevents the irreversible accumulation of carbonyl derivatives [25]. The found changes can be considered as an adaptive mechanism aimed at protecting mitochondrial proteins from oxidative damage. On the other hand, in animals maintained on a high-sucrose diet, there is an increased accumulation of the carbonyl derivatives in the mitochondrial proteins of the kidney (Fig. 5) accompanied by a de-

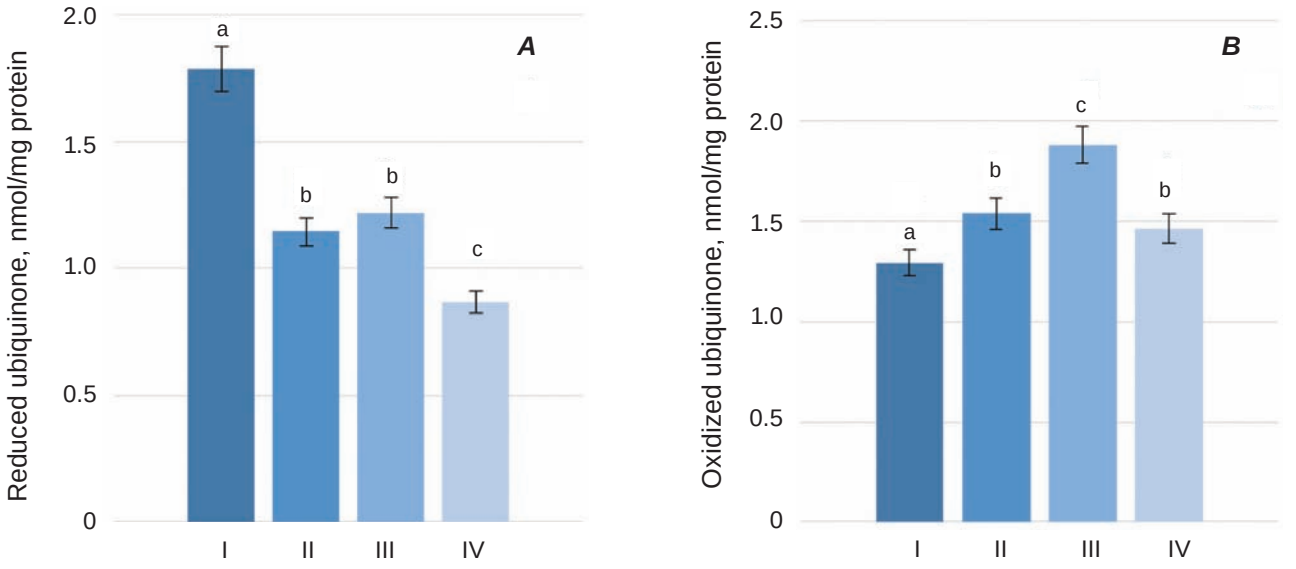

*Fig. 4. The content of reduced (**A**) and oxidized (**B**) ubiquinone in kidney cell mitochondria from rats that received diets with different contents of sucrose and protein (M ± m, n = 9): I – control rats that receive the adequate semisynthetic diet; II – rats that receive the low-protein ration; III – rats that receive the high-sucrose diet; IV – rats that receive the high-sucrose–low-protein diet. Different letters indicate significant differences inside the parameters and the same letters indicate no difference at P ≤ 0.05*

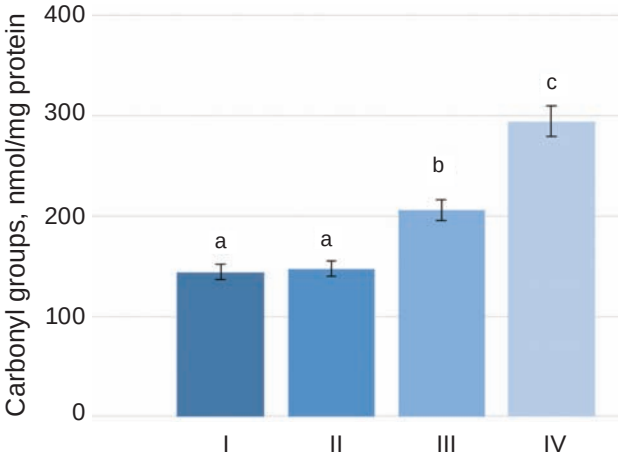

Fig. 5. Protein carbonyl derivatives content in kidney cell mitochondria from rats that received diets with different contents of sucrose and protein (M ± m, n = 9): I – control rats that receive the adequate semisynthetic diet; II – rats that receive the low-protein ration; III – rats that receive the high-sucrose diet; IV – rats that receive the high-sucrose–low-protein diet. Different letters indicate significant differences inside the parameters and the same letters indicate no difference at P ≤ 0.05

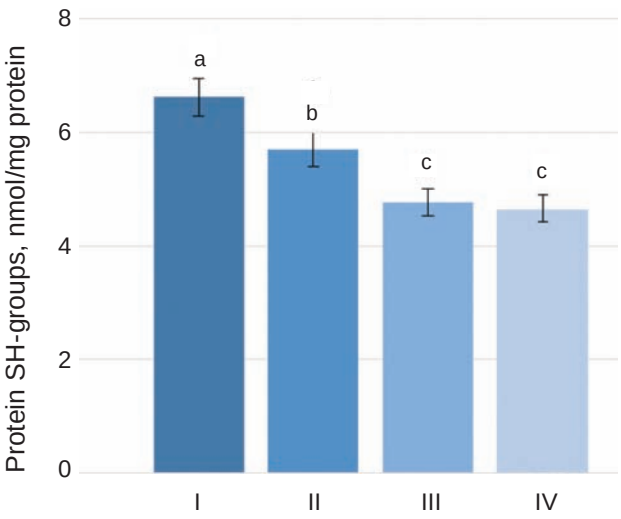

Fig. 6. Free SH-groups content in proteins in kidney cell mitochondria from rats that received diets with different contents of sucrose and protein (M ± m, n = 9): I – control rats that receive the adequate semisynthetic diet; II – rats that receive the low-protein ration; III – rats that receive the high-sucrose diet; IV – rats that receive the high-sucrose–low-protein diet. Different letters indicate significant differences inside the parameters and the same letters indicate no difference at P ≤ 0.05

crease in the level of free protein SH-groups (Fig. 6) by approximately 1.4-1.5 times compared with control, which indicates the intensification of reactions of free radical damage to mitochondrial proteins under the conditions of hyperglycemia. It is shown that persistent hyperglycemia leads to the development of oxidative stress through various mechanisms, including autooxidation of glucose, non-enzymatic glycosylation of proteins, activation and enhancement of the polyol pathway, reduced activity of the antioxidant defense system. The determined intensification of free radical damage to mitochondrial proteins under the conditions of excessive sucrose consumption may also be associated with the decrease in the level of reduced ubiquinone, which normally provides neutralization of ROS and prevents oxidative modification of mitochondrial proteins under physiological conditions. Since it is known that oxidative stress along with hyperglycemia plays an important role in the pathogenesis of functional and structural damage to the kidney glomeruli and tubules, the consequence of observed changes may be a deepening of renal dysfunction [5].

The most pronounced intensification of free radical damage to mitochondrial proteins is found under the conditions of keeping animals on a low-protein/high-sucrose diet, in particular, there is a two-fold increase in the level of carbonyl derivatives compared with control (Fig. 5) and decrease in the level of free protein SH-groups (Fig. 6). According to the literature [26], severe free radical damage to mitochondrial proteins can be caused by both excessive ROS generation and activation of glycosylation of the antioxidant enzymes with their subsequent inactivation, which was found under the conditions of hyperglycemia. One of the consequences of free radical damage to mitochondrial proteins under the conditions of nutrient imbalance may be a found decrease in the activity of enzymes of the respiratory chain.

Thus, the energy deficiency of kidney cells under the conditions of carbohydrate-protein imbalance is due to the intensification of the oxidative processes in mitochondria manifested in the decrease in key enzymatic activities of the mitochondrial respiratory chain, redistribution of redox forms of ubiquinone and intensified accumulation of the products of oxidative protein modification. The found changes can be considered as a precondition for the development of kidney dysfunction under the conditions of carbohydrate-protein imbalance.

Conflict of interest. Authors have completed the Unified Conflicts of Interest form at http://ukr-biochemjournal.org/wp-content/uploads/2018/12/coi_disclosure.pdf and declare no conflict of interest.

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NADH-УБІХІНОНРЕДУКТАЗНА ТА СУКЦИНАТДЕГІДРОГЕНАЗНА АКТИВНОСТІ В МІТОХОНДРІЯХ НИРОК ЩУРІВ ЗА УМОВ РІЗНОЇ ЗАБЕЗПЕЧЕНОСТІ РАЦІОНУ ПРОТЕЇНОМ ТА САХАРОЗОЮ

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Досліджували NADH-убіхінонредуктазну, сукцинатдегідрогеназну активність, вміст загального убіхінону та його редокс-форм, а також ступінь окислювальної модифікації мітохондріальних протеїнів у нирках щурів за різної забезпеченості раціону протеїном та сахарозою. Встановлено, що у разі споживання низькопротеїнового раціону спостерігається зниження NADH-убіхінонредуктазної та сукцинатдегідрогеназної активності в 1,4-1,7 раза, зниження на 20 % вмісту загального убіхінону та кількісний перерозподіл його окисленої та відновленої форми з переважанням окисленої форми. При цьому за досліджуваних умов не спостерігається накопичення карбоніл-дериватів, проте вміст вільних SH-груп вірогідно знижується порівняно з контролем. Водночас у тварин, які споживали високосахарозний раціон, виявлено підвищення NADH-убіхінонредуктазної та сукцинатдегідрогеназної активності в 1,5-2 рази за збереження вмісту загального убіхінону на рівні показників контролю на тлі перерозподілу його редокс-форм, зокрема зниження вмісту відновленого убіхінону та зростання вмісту окисленого убіхінону у середньому у 1,5 раза. При цьому спостерігалась інтенсифікація реакцій вільнорадикального ушкодження мітохондріальних протеїнів у клітинах нирок, про що свідчить підвищення вмісту карбоніл-дериватів, а також вірогідне зниження вмісту вільних протеїнових SH-груп приблизно у 1,4-1,5 раза. Проте максимальні зміни досліджуваних показників характерні для тварин, які споживали низькопротеїновий високосахарозний раціон. Надмірне споживання сахарози на тлі аліментарного дефіциту протеїну супроводжувалось зниженням NADH-убіхінонредуктазної та сукцинатдегідрогеназної активності в 1,7-2 рази, вмісту загального убіхінону приблизно в 1,4 раза та відновленого убіхінону в понад 2 рази на тлі інтенсифікації вільнорадикального ушкодження мітохондріальних протеїнів, що може розглядатись як передумови формування порушення функціональної активності нирок в умовах вуглеводно-протеїнового дисбалансу.

Ключові слова: нутрієнти, нирки, NADH-убіхінонредуктаза, сукцинатдегідрогеназа, убіхінон, карбонільні похідні, протеїнові SH-групи.

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