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BIOENERGETIC FUNCTIONS OF MITOCHONDRIA IN LIVER, PANCREATIC ACINAR CELLS, AND SPERM CELLS OF RATS FED SHORT-TERM HIGH-FAT OR HIGH-FAT HIGH-SUGAR DIETS

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An unhealthy diet often is a cause of obesity, chronic inflammation, and metabolic disruption in multiple organs. However, the direct influence of elevated lipid or sugar consumption on liver, pancreatic, and sperm mitochondria is not well understood. The aim of the study was to investigate the functional activity of mitochondria of liver, pancreatic acinar cells, and sperm cells in rats on a short-term (7 weeks) diet with high fat or high fat and high sugar content. Male Wistar rats were on a basic, high-fat or high-fat high-sugar diet for 7 weeks. At the end of the experiment, visceral fat mass, blood glucose and lipids were measured. Mitochondrial functional activity was evaluated with oxygen consumption assay. In isolated pancreatic acinar cells, NAD(P)H autofluorescence and mitochondrial membrane potential were also studied. No difference in body mass was observed between the 3 groups at the end of the experiment. Visceral fat mass was slightly but significantly elevated in rats on a high-fat high-sugar diet. Both diets did not affect plasma glucose or triglyceride levels but caused a modest elevation of total plasma cholesterol. Respiration and oxidative phosphorylation of isolated liver mitochondria were not affected by any experimental diet. In pancreatic acinar cells, a high-fat diet caused a significant decrease of basal respiration by ~15%, but no effects were observed on the maximal rate of uncoupled respiration, mitochondrial membrane potential, or NAD(P)H autofluorescence. In these cells, a ketone body 3-hydroxybutyrate caused elevation of uncoupled respiration and NAD(P)H level irrespectively of the diet. Diets did not cause any change in sperm concentration, viability or motility. Surprisingly, in animals on a high-fat high-sugar diet, a significant increase in both basal and maximal respiration of sperm cells was observed. Collectively, these data show that while the elevated fat and sugar content in the diet does not cause significant obesity, no detrimental effects on mitochondria of the liver, pancreas, and sperm cells are observed.

K e y w o r d s: mitochondria, liver, pancreatic acinar cells, sperm, diet.

The complex relationship between dietary patterns, metabolic responses, and the risk of obesity is a subject of substantial scientific interest. Obesity is a pressing global concern and understanding how changes in diet influence metabolism is crucial for elucidating the broader health implications. While genetic factors undeniably play a role in an individual's susceptibility to obesity [1, 2] , the significant rise in overweight populations during the modernization of previously underdeveloped countries underscores the substantial influence of environmental factors. Among these factors, the adoption of a high-energy density, high-fat diet is believed to be a key contributor to this trend [3]. It remains unclear whether or in what manner dietary fat alone or changes in metabolism serve as the predisposing factor.

An approach to investigate the multifaceted systemic consequences of obesity and the associated metabolic alterations in the human body involves utilizing animal models [4]. There are several types

of high-calorie diets considered quite effective for modeling obesity: high-fat (HF) diet, high-sugar diet, and combined high-fat and high-sugar (HFHS) diet. Studies on rats have shown that the application of HF diet induces adipocyte hypertrophy, weight gain, insulin resistance, glucose metabolism disturbances, dyslipidaemia, and liver steatosis [5]. High-sugar diets, due to their taste qualities and high caloric content, also contribute to a positive energy balance and increased body weight. However, there is evidence that high-sugar diets are less effective than HF diets [5]. Combined HFHS diets are considered to mimic the diet of modern humans (rich in fats and carbohydrates), which is why they are often used for experimental modelling of obesity [6]. It has been previously confirmed that the changes observed in mice on HFHS diets are similar to those on HF diets, including increased body weight, abdominal fat, hyperinsulinemia, and hyperglycaemia [7].

Diseases of many organs are associated with unhealthy diet, metabolic syndrome, and obesity. In liver, the current understanding is that hyperlipidemia due to unhealthy diets and obesity is the main cause of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis [8]. This is supported by animal studies. In rats, a long-term high-sugar diet of 20 weeks has caused elevation of body mass, blood glucose levels [9]. This was accompanied by a compensatory intensification of oxygen computation rate of rat liver cells [9]. The adaptation of mitochondria to HF diet was also confirmed in a long-term (30 weeks) study on diabetic mice [10]. Yet other study showed loss of mitochondrial respiratory functions in mice subject to 12-week long HF diet [10]. A short-term high-fat diet of 7 weeks has shown no impact on body mass or oxygen update on rat liver cells [11]. Thus, the role of mitochondrial adaptation to diets and obesity in sustaining normal liver functions requires further elucidation.

Excessive accumulation of fat in pancreas was recently discussed as a risk factor for many pancreatic diseases, including acute pancreatitis, chronic pancreatitis, pancreatic cancer, and type 2 diabetes mellitus [12]. It is also well known that patients with lipoprotein lipase deficiency display elevated blood lipid levels and high incidence of acute pancreatitis. *In vitro* studies showed harmful effects of free fatty acids on both mitochondrial ATP synthesis and viability of pancreatic acinar cells [13, 14]. It is thus important to study the effects of dietary fat on exocrine pancreas. In rat models, long-term HF diet (12-20 weeks) caused hyperlipidemia, weight gain and, importantly, lipid droplets accumulation within the pancreatic tissue with signs of pancreatic injury [15, 16]. However, to date, no studies of HF or HFHS diets effects on mitochondria of pancreatic acinar cells were performed.

Obesity and modern dietary changes seem to negatively contribute to sperm function and energy metabolism in men [17]. Ferramosca et al. [18] showed that 4-week HF diet resulted in significant weight gain in rats leading to the decrease of mitochondrial respiration and sperm motility. However, it is not clear if dietary fat alone (without causing obesity) can cause significant damage to energy metabolism or sperm quality – both in humans and animal models. Interestingly, one-week high-sugar diet in healthy men caused an increase of a subset of small non-coding RNA (tRNA-derived small RNA) both nuclear and mitochondrial, accompanied by an improvement in sperm motility [19]. Additional analysis of RNA data from clinically obese patients [20] showed opposite changes in sugar-sensing RNAs [19]. However, mitochondrial respiration and oxidative phosphorylation were not evaluated in these studies.

Short-term high-calorie diets are interesting models for assessing the adaptive capabilities of the organism, especially when clear signs of obesity are either absent or just beginning to appear. Our particular interest is in changes in metabolism and mitochondrial activity when transitioning to a high-fat or high-fat high-sugar diet. In particular, it is important that the presented models allow us to evaluate the global effect of diet on the body and to study several organs at the same time. Thus, the aim of the study was to investigate the direct effects of short-term HF and HFHS diets on respiration of rat liver mitochondria, pancreatic acinar cells, and sperm cells.

Materials and Methods

Reagents used in experiments were purchased from Sigma-Aldrich (St. Louis, Mo): sucrose, sodium chloride, glucose, N-2-hydroxyethylpiperazine-N′-2-ethanesulfonic acid (HEPES), soybean trypsin inhibitor, bovine serum albumin (BSA), sodium pyruvate, glutamine, malate, ADP, succinate, glutamate, 3-hydroxybutyrate, FCCP, collagenase type IV, bromopyruvic acid, tetramethylrhodamine methyl ester perchlorate (TMRM); Merck Chemicals

(Burlington, Mass) – Calcium chloride dihydrate. All other reagents were of the purest available grade.

All manipulations with animals are accomplished in accordance with the European Convention for the Protection of Vertebrate Animals used for Experimental and other Scientific Purposes (Council of Europe No 123, Strasbourg 1986). The work was carried out without violations of international standards for the humane treatment of laboratory animals (according to the protocol of the bioethics committee of the Faculty of Biology of Lviv National University No. 40-09 dated September 8, 2023). Experiments were carried out on 30 male Wistar rats. At the start of the experiment, animals weighed 250–300 g. Animals were kept under the standard conditions of a vivarium at a constant temperature with 12:12 h light-dark cycle. Animals were split into three groups based on the diet: standard diet, high-fat diet and high-fat high-sugar diet. Detailed food composition is presented in Table 1. Animals were sacrificed on day 50 of an experiment by decapitation after 12 h of starvation.

A suspension of isolated pancreatic acini was obtained with collagenase (type IV, 0.2 mg/ml) as previously reported [21] in basic incubation medium, containing (mM): NaCl – 140.0, KCl – 4.7, $CaCl_2$ – 1.3, $MgCl_2$ – 1.0, HEPES – 10.0, glutamine – 2.0, sodium pyruvate – 2.0, glucose – 10.0; BSA – 2.5 mg/ml; soybean trypsin inhibitor – 0.1 mg/ml and essential MEM amino acid supplement; pH set at 7.4 with NaOH. Cell counting was performed with a hemocytometer.

Rat liver mitochondria were isolated with a standard differential centrifugation technique according to the protocol of Frezza et al. [22] in a sucrose-based medium. After decapitation, the extracted liver is minced, then the obtained suspension is filtered, passed through a press, and homogenized with glass-Teflon potter at 1600 r.p.m. in a cooled homogenizer. Following homogenization, the solution is transferred to another chilled test tube and centrifuged at 600 g at 4°C. After the first centrifugation, the solution must be centrifuged two more times at 7000 g at 4°C. After that, the supernatant is removed, and the obtained mitochondria from the precipitate are resuspended and transferred to a cooled container. Isolation medium contains (mM): sucrose – 250.0, EGTA – 1.0, HEPES – 10.0. The incubation medium contains (mM): sucrose – 250.0, EGTA – 0.1, HEPES – 10.0, K_2HPO_4 – 2.0, and $MgCl_2$ – 0.5. pH of both media was set at 7.2 with KOH.

The testes appendages were quickly removed immediately after the decapitation of the animals, weighed, and then carefully the tail portion of the epididymis (cauda epididymis) was separated. The tissue was transferred to a Petri dish containing 3–4 ml of preheated to 37°C modified Tyrode's solution, containing mM: NaCl – 131.89, KCl – 2.68, $MgCl_2 \cdot 6H_2O$ – 0.49, KH_2PO_4 – 1.2, $CaCl_2$ – 1.8, HEPES – 20 mM, glucose – 5.56, sodium pyruvate – 2 mM and 4 mg/ml bovine serum albumin; pH set at 7.4 with NaOH.

Four transverse and two longitudinal incisions were made on each caudal part of the epididymis, after which it was left in Tyrode's solution for 5-10 min to allow the sperm cells to diffuse into the solution. The cell suspension was diluted with warm Tyrode's solution in a ratio of 1:20 (0.02 ml of the suspension was mixed with 0.38 ml of Tyrode's solution). Sperm cell counting was performed in a haemocytometer, assessing each sample in two aliquots.

Sperm motility was determined as the percentage of motile sperm cells relative to the total number of counted sperm cells. To assess the motility of the reproductive cells, a drop of sperm suspension was

T a b l e. Food composition of the diets used in experimental groups

Components (per 100 g)	Diets		
	Balanced (control)	HF diet 45% kcal of fat	HFHS diet
Balanced premade fodder, g	100.0	85.0	60.0
External animal fat, g	–	15.0	18.0
External sugar (saccharose), g	–	–	22.0
Energy value (kcal/g)	3.03	3.92	5.08
Proteins, %	21.5	20.0	10.0
Fats, %	6.5	45.0	39.0
Carbohydrates, %	72.0	35.0	51.0

placed on a preheated to 37°C glass slide. The assessment was carried out using light microscopy by counting a minimum of 200 cells. Sperm cells in which flagellar movement was observed were considered motile.

The rate of oxygen consumption of biological suspensions was measured with Clark oxygen electrode at 37°C using SI929 6-channel Oxygen Meter (Strathkelvin). Respiration of pancreatic acini was studied in respiration medium containing (mM): NaCl – 140.0, KCl – 4.7, $CaCl_2$ – 1.3, $MgCl_2$ – 1.0, HEPES – 10.0, glucose – 10.0; BSA – 2.5 mg/ml and soybean trypsin inhibitor – 0.1 mg/ml; pH set at 7.4 with NaOH. In some experiments, pyruvate or 3-hydroxybutyrate (2 mM) was added. Respiration of sperm cells was studied in Tyrode's medium with or without lactate (10 mM). In cell respiration studies, protonophore FCCP was added stepwise into the respiration chamber in 0.5 μM aliquots (2.0 μM total) to reach the maximal uncoupled respiration rate as described earlier [23].

Respiration of isolated mitochondria was studied in the medium containing, mM: saccharose – 250.0, EGTA – 0.1, HEPES – 10.0, K_2HPO_4 – 2.0, and $MgCl_2$ – 0.5. Oxidative substrates succinate (5 mM), glutamate (5 mM) + malate (5 mM), or 3-hydroxybutyrate (5 mM) were added to the medium in respiratory cells at the beginning of the experiment before mitochondria. State 4 respiration was recorded after phosphorylation of initial small ADP aliquot (0.1 μM). A second ADP aliquot (0.85 μM) was added to study State 3_{ADP} respiration followed by FCCP (0.1-0.3 μM) to reach maximal State 3_U respiration.

Fluorescent imaging was performed using IX73 microscope and DP-74 camera (Olympus, Tokyo, Japan). Images were processed using ImageJ 1.53e software (NIH, Bethesda, Md). Mitochondrial membrane potential in pancreatic acini was assessed with tetramethylrhodamine methyl ester perchlorate (TMRM) fluorescence (excitation, 530–550 nM; emission >575 nm). TMRM (20 μM) was loaded for 25 minutes at 37°C. Acini were washed once, and fluorescence was studied immediately. In parallel, reduced nicotinamide adenine dinucleotide (phosphate) (NAD(P)H) autofluorescence (excitation, 340–390 nM, emission >420 nm) was studied. Fluorescence intensity was analysed with ImageJ software (NIH) using the blue channel for NAD(P)H autofluorescence and the red channel for TMRM fluorescence measurement. In each experiment, at least 50 different acini from at least 5 different photographs were analysed.

Sperm viability was assessed with propidium iodide (1 μM) staining using IX73 microscope and DP-74 camera (excitation, 530–550 nM; emission >575 nm). In each experiment, at least 200 cells were counted. Preparations with viability of less than 30% were not studied further.

Blood was collected after decapitation, plasma was immediately separated by centrifugation and stored at -20°C. Glucose concentration was determined with “Glucose-MONO” kit (“Filisit-diagnostics, Ukraine) using DeNovix DS-11+ spectrophotometer (Denovix Inc., USA) at 550 nm absorbance. Plasma triglycerides, total cholesterol, high-density lipoprotein (HDL) and low-density lipoprotein (LDL) concentrations were determined with enzymatic colorimetric methods (kits of “Filisit-diagnostics”, Ukraine) using Stat-fax-303 (Awareness Technology Inc., USA).

Results are presented as means ± S.E.M. Statistical analysis was performed using Origin Pro 2018 (Northampton, Mass) software. Respiration rates were calculated automatically using custom software written in Python language. No deviations from the normal distribution in data were found according to the Shapiro-Wilk test. The significance of the difference between the groups was determined with one-way or two-way ANOVA (where appropriate) followed by a Turkey-corrected post-hoc *t*-test in case of significant difference according to ANOVA.

Results and Discussion

Over the course of a 7-week diet and daily body weight measurements during ad libitum feeding, no differences were observed between the experimental groups and the control group (Fig. 1, *A*). Despite controversial results in other similar high-fat diet models [11, 15, 16], the absence of changes in our experiment can be explained by the relatively short diet duration for the used model rat breed. Similarly, liver mass was not affected by the studied diets (Fig. 1, *C*).

A slightly different diet outcome was found with quantities of visceral fat, which were also measured. The experimental group with the high-fat high-sugar diet had an increased amount of visceral fat by 25% (2.85% in the HFHS diet compared to 2.14% in the control; Fig 1, *B*; $P < 0.05$, $n = 10$), while the high-fat and control groups did not differ.

Fig. 2 illustrates changes in total cholesterol, low-density and high-density lipoproteins, triglyce-

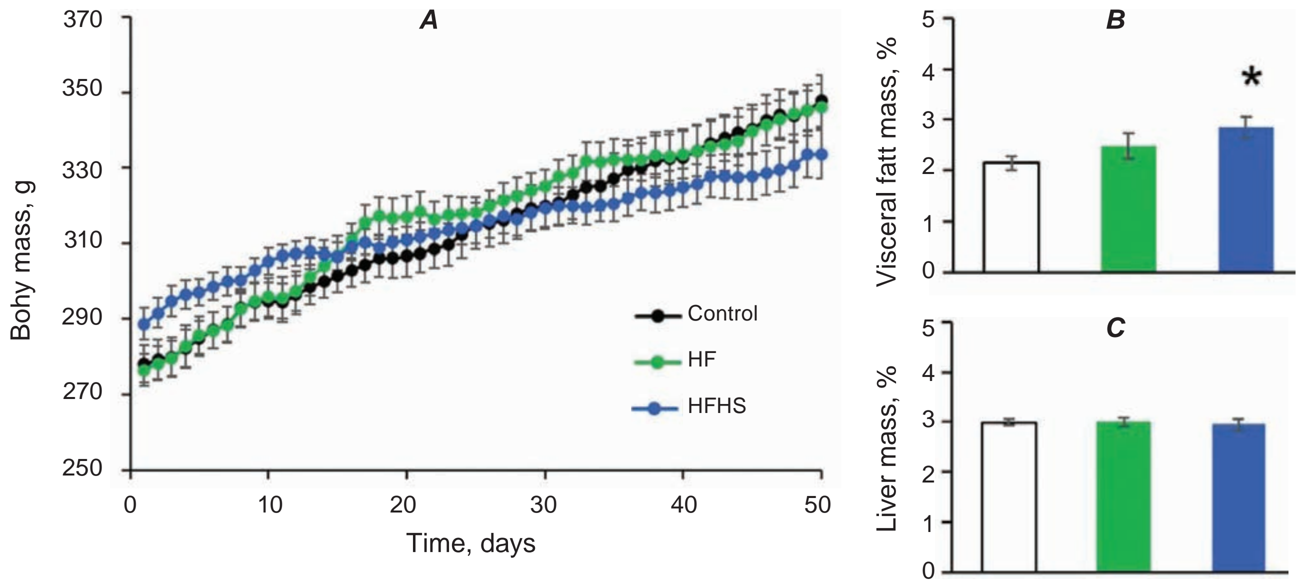

*Fig. 1. Body mass (**A**), visceral fat (**B**) and liver mass (**C**) of rats on a basic (white), HF (green) or HFHS diet (blue): *significant difference compared to control according to one-way ANOVA and post-hoc Holm-Bonferroni test with P < 0.05, n = 10*

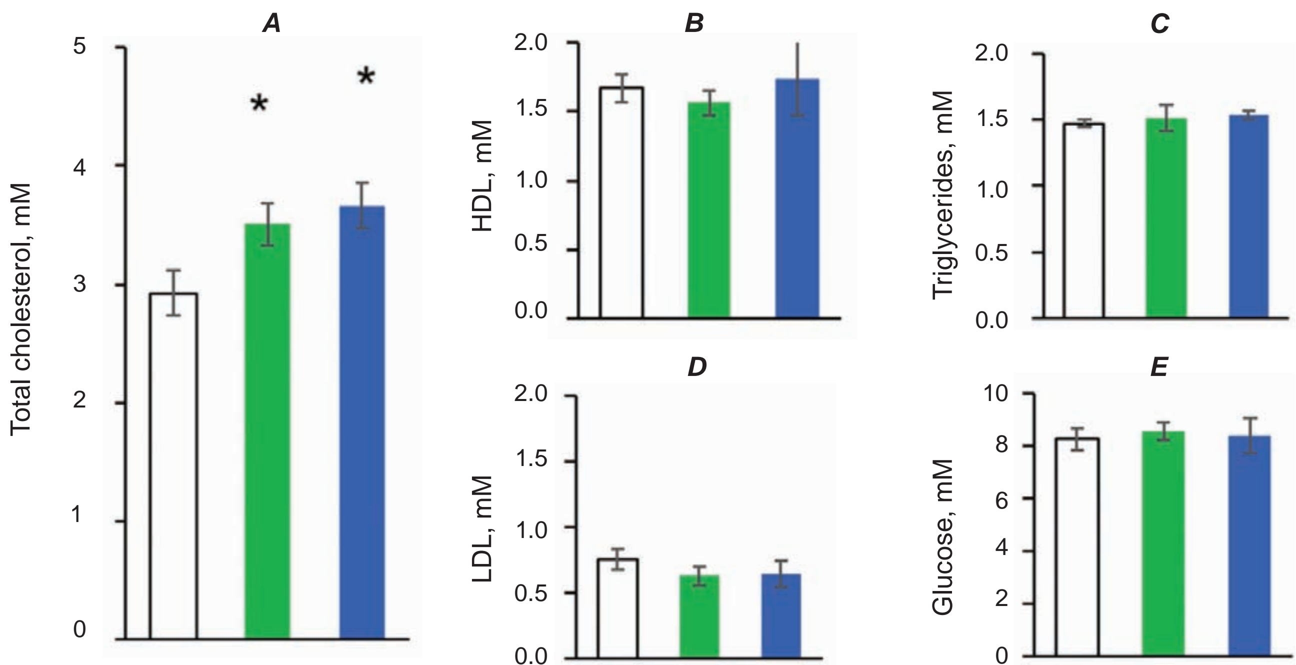

*Fig. 2. Concentration of total plasma cholesterol (**A**), high-density (**B**) and low-density (**D**) lipoproteins, triglycerides (**C**) and glucose (**E**) in rats on a basic (white), HF (green) or HFHS diet (blue): *significant difference compared to control according to one-way ANOVA and post-hoc Holm-Bonferroni test with P < 0.05, n = 10*

rides, and glucose in the blood plasma on day 50 of the experiment. In both HF and HFHS diets, the total cholesterol level statistically significantly increased compared to the control (2.93 mM) by 20% (up to 3.51 mM) and 25% (up to 3.66 mM) in HF and HFHS diets, respectively (Fig. 2, *A*; $P < 0.05$, $n = 10$). This modest increase, however, was still in the range of normal cholesterol level, and was not due to high-density and low-density lipoproteins change (Fig. 2, *B*, *D*). Plasma glucose and triglycerides maintained

a consistent level across all experimental groups (Fig. 2, *C*, *E*).

Overall, these results confirm that 7-week HF or HFHS diet does not induce obesity or metabolic syndrome in rats, as shown previously in long-term HF or high-sugar diet studies [11, 9].

We have also studied the effects of HF and HFHS diets on the respiration of liver mitochondria. The State 4, State 3, and State3_U (uncoupled with FCCP) respiration of isolated liver mitochondria were similar in all experimental groups irrespective of the oxidative substrate used (Fig. 3, *A*–*C*). The respiratory control indices (RCI) of isolated liver mitochondria were in the range of 3.9–5.6, indicating good quality of mitochondrial preparations from all animal groups (Fig. 3, *D*). Thus, within the experimental time frames, the studied diets do not affect the respiratory function of liver mitochondria, in agreement with the previous studies [11]. Our results support the conclusion that short-term HF or HFHS diet exerts no direct effect on liver mitochondria in rats with no signs of obesity.

In cell studies, unlike isolated mitochondria, it is not possible to modulate State 3 respiration with external ADP addition, because it does not penetrate plasma membrane and intracellular ATP concentration is high. To investigate the maximal respiratory capacity, we used uncoupler FCC, which elicits an adaptive respiratory response previously described in isolated pancreatic acini [23]. Basal respiration represents the metabolic flow of mitochondria in unstimulated cells. Basal respiration rate of isolated pancreatic acini decreased by about 15 % in animals on a high-fat diet compared to the control, irrespectively of oxidative substrate in solution, as shown in Fig. 4, *A* ($P < 0.05$, $n = 10$). Meanwhile, a high-fat, high-sugar diet had no such effect, and basal rates were close to control levels. No differences between experimental groups were found in measured maximal uncoupled respiration (Fig. 4, *B*). We have observed the positive effect of 3-hydroxybutyrate on maximal respiration of pancreatic acini regardless of the diet, indicating the utilization of ketone bodies by mitochondria of pancreatic acinar cells.

Mitochondrial membrane potential, measured with TMRM fluorescence, was the same for all experimental groups, and no statistical difference was recorded between the experiments with different substrates used (Fig. 4, *C*). FCCP induced sharp depolarization of mitochondrial membrane potential confirming the good quality of the experimental setup.

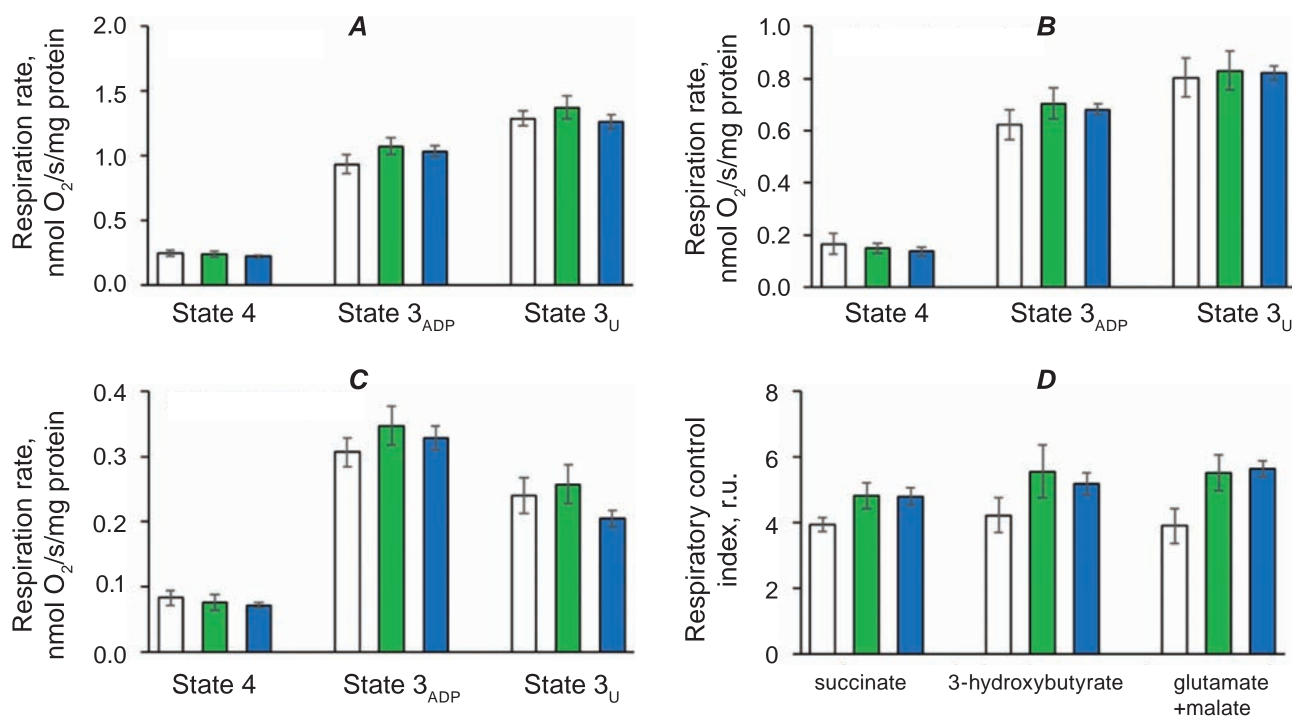

Fig. 3. Respiration of isolated liver mitochondria from rats on basic (white), HF (green) or HFHS (blue) diet; oxidative substrates were: ***A*** *– succinate (5 mM),* ***B*** *– glutamate + malate (5 mM),* ***C*** *– 3-hydroxybutyrate (5 mM);* ***D*** *– respiratory control indices for experiments of all substrates, n = 10*

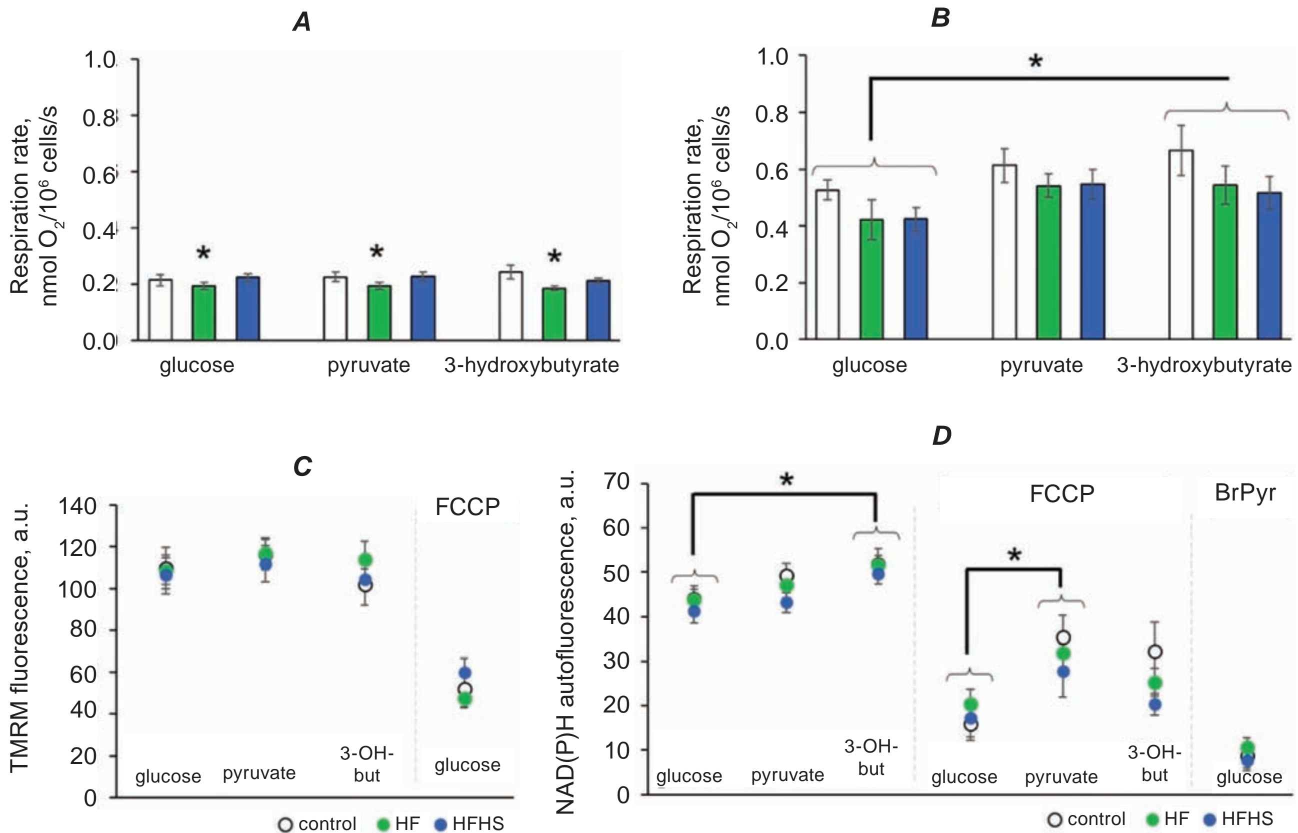

*Fig. 4. Functional parameters of mitochondria of isolated pancreatic acini from rats on a basic (white), HF (green) or HFHS (blue) diet: **A** – basal respiration, **B** – maximal uncoupled respiration, C – mitochondrial membrane potential (TMRM fluorescence), **D** – autofluorescence of HAD(P)H; substrates were glucose (10 mM) alone or in combination with pyruvate (2 mM) or 3-hydroxybutyrate (3-OH-but, 2 mM); *significant difference compared to control according to one-way ANOVA and post-hoc Holm-Bonferroni test with $P < 0.05$, $n = 5–10$*

Total basal cell metabolism activity, measured with autofluorescence of HAD(P)H, was similar within all experimental groups (Fig. 4, *D*). Adding 3-hydroxybutyrate to the medium significantly elevated cytosolic NAD(P)H in all animal groups. Glycolysis inhibitor bromopyruvate (BrPyr, 500 μM) almost completely abolished NAD(P)H fluorescence. Using FCCP to enhance mitochondrial HAD(P)H oxidation caused the decrease of its autofluorescence, which did not depend on the diet but was significantly less pronounced when pyruvate was oxidized, indicating increased tricarboxylic cycle activity (Fig. 4, *D*). This confirms our previous findings that pyruvate enhances mitochondrial functions in isolated rat pancreatic acini [24].

In conclusion, we find mitochondrial functions of pancreas to be largely unaffected by the studied diets. The small decrease of basal respiration in HF diet group was not accompanied by any other changes of mitochondrial parameters. Whether this effect is associated with the lipid transformation of the gland, as described in the literature [15], remains to be elucidated in further studies.

According to research findings, diet-induced obesity has a negative impact on the quantity and motility of sperm in rats [18, 17], but no data is available for short-term HF or HFHS diets with no signs of obesity. Thus, we have studied rat sperm cells freshly isolated from epididymis. Rat sperm cell viability was similar in all three experimental groups (Fig. 5, *A*). Diets also did not cause a reduction of sperm concentration or motility (not shown), in contrast to the previously described negative effects of 4-week HF diet [18]. High-fat diet had no impact on basal and FCCP-induced respiration rates upon glucose or lactate + glucose oxidation. Surprisingly, high-fat high-sugar diet increased both basal and FCCP-induced respiration rates irre-

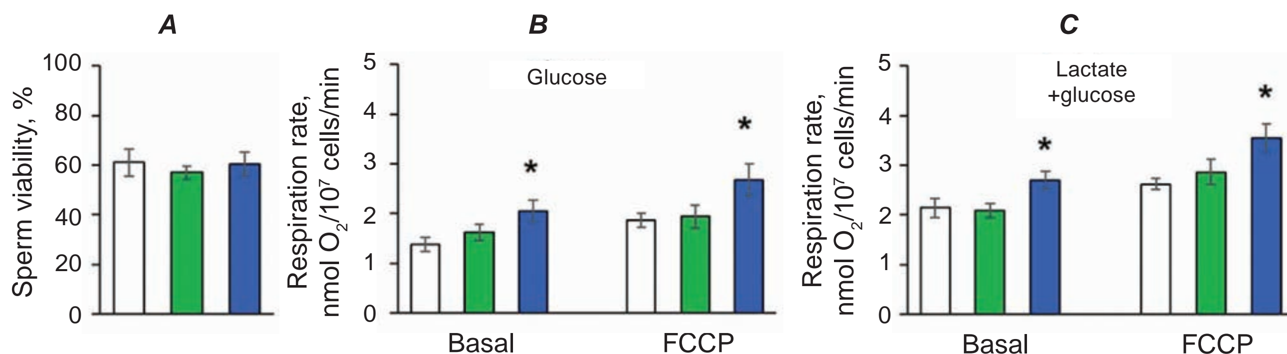

Fig. 5. Rat sperm viability and respiration in rats on a basic (white), HF (green) or HFHS diet (blue), ***A*** *– sperm viability,* ***B*** *– basal and maximal (FCCP) respiration in Tyrode's medium,* ***C*** *– basal and maximal (FCCP) respiration in Tyrode's medium supplemented with lactate (10 mM); *significant difference compared to control according to one-way ANOVA and post-hoc Holm-Bonferroni test with P < 0.05, n = 6–8*

spective of lactate presence (Fig. 5, *B-C*; $P < 0.05$, $n = 6–8$). We assume that this may be related to the previously reported change in mitochondrial sugar-sensing RNAs in a short study on healthy men [23]. These results further support the hypothesis that a high level of sugar in the diet (at least while no obesity or diabetes mellitus is developed) signals positively about the living conditions and improves sperm catabolic activity, quality and motility to facilitate conception.

Conclusions. The obtained results demonstrated that short-term diets with high levels of fat and/or sugar content do not lead to substantial obesity and do not have negative effects on the mitochondria of the liver, pancreas, and sperm cells, while sugar also increases mitochondrial respiration in sperm.

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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БІОЕНЕРГЕТИЧНІ ФУНКЦІЇ МІТОХОНДРІЙ ПЕЧІНКИ, АЦИНАРНИХ КЛІТИН ПІДШЛУНКОВОЇ ЗАЛОЗИ ТА СПЕРМАТОЗОЇДІВ ЩУРІВ ЗА КОРОТКОТРИВАЛОЇ ДІЄТИ З ВИСОКИМ ВМІСТОМ ЖИРУ АБО З ВИСОКИМ ВМІСТОМ ЖИРУ І ЦУКРУ

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Нездорове харчування часто є причиною ожиріння, хронічного запалення та порушення обміну речовин у багатьох органах. Однак прямий вплив підвищеного споживання жирів або цукру на мітохондріальні функції печінки, підшлункової залози та сперми недостатньо досліджено. Метою цієї роботи було дослідити функціональну активність

мітохондрій печінки, ацинарних клітин підшлункової залози та сперматозоїдів у щурів за короткотривалої (7 тижнів) дієти з високим вмістом жиру або жиру та цукру. Щури-самці лінії Вістар були на контрольній, високожировій або високожировій високоцукровій дієтах протягом 7 тижнів. По закінченню експерименту вимірювали масу вісцерального жиру, рівень глюкози та ліпідів у крові. Мітохондріальну функціональну активність визначали за вимірюванням швидкості поглинання кисню. У клітинах підшлункової залози за допомогою флуоресцентного мікроскопа також досліджували автофлуоресценцію NAD(P) H та мембранний потенціал мітохондрій. Наприкінці експерименту різниці у масі тіла між 3 групами не спостерігалося. Кількість вісцерального жиру була незначно, але статистично достовірно збільшена у щурів, які перебували на високожировій високовуглеводневій дієті. Жодна із дієт не впливала на рівень глюкози чи тригліцеридів у плазмі крові, але було зареєстровано (в обох дослідних групах) зростання рівня загального холестерину. Швидкість дихання та окисне фосфорилювання в ізольованих мітохондріях печінки не змінювались за жодної із дослідних дієт. У клітинах підшлункової залози за високожирової дієти спостерігалось збільшення швидкості базального дихання на ~15%, але такий ефект не був отриманий для максимальної швидкості роз'єднаного дихання, мембранного потенціалу мітохондрій чи автофлуоресценції NAD(P)H. У цих клітинах, кетонове тіло 3-гідроксибутират спричинило зростання неспряженого дихання та рівня NAD(P)H незалежно від дієт. Дієти не спричинювали будь-яких змін у концентрації клітин сперми, їх життєздатності або рухливості. Неочікувано, але у разі високожирової високовуглеводневої дієти спостерігали достовірне збільшення як базального, так і максимального дихання сперматозоїдів щурів. Загалом, отримані результати свідчать, що в разі підвищеного вмісту жирів і цукру в раціоні не спостерігали значного ожиріння та негативного впливу на мітохондрії клітин печінки, підшлункової залози та клітин сперми щурів.

К л ю ч о в і с л о в а: мітохондрії, печінка, ацинарні клітини підшлункової залози, сперма, дієта.

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