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НАЦІОНАЛЬНИЙ ФАРМАЦЕВТИЧНИЙ УНІВЕРСИТЕТ
ХАРКІВСЬКИЙ НАЦІОНАЛЬНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ

І Науково-практична конференція студентів та молодих вчених з міжнародною участю

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15 ТРАВНЯ 2019
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NATIONAL UNIVERSITY OF PHARMACY
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«FROM EXPERIMENTAL AND CLINICAL PATHOPHYSIOLOGY TO THE ACHIEVEMENTS OF MODERN MEDICINE AND PHARMACY»

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ЗМІСТ

ABDIHOSSEINABAD MOHAMMAD, PISARIK D. M. High blood pressure response to exercise as a predictor of future hypertension development in young bodybuilder 13
AMAOGE CHIOMA BLESSING, E. N. LUCHKO, O. V. FILIPTSOVA Asthma 15
COLLINS NWACHUKWU, FILIPTSOVA O. V., STOROZHENKO G. V. Stroke 18
JEDI B. DADI The importance of regulatory ubiquitination in neurodegenerative disease 20
FEDOTOV V. V., OGNEVA L. G., SHUTOVA I. V. Influencing risk factors for complications in children with insuline-dependant diabetes 21
GREKOVA T. A., KADZHARYAN E. V. Comparative morphofunctional analysis of chronic hyperglycemia influence on pancreatic islets in spontaneous hypertensive and wistar rats 22
JURAEV SH., MOROZ V. A. Analysis of antihypertensive drugs spectrum used in the district hospital of Uzbekistan 24
KHUDENKO N. V., SARNATSKA V. V, YUSHKO L. O., PAZUK L. M., MASLENNIY V. M., BUBNOVSKA L. M., NIKOLAEV V. G. Study of metabolic shifts caused by development of breast cancer in rats with walker 256 carcinosarcoma 26
KRYVSUN K. V. Morpho-densitometric changes in the parameters of the hypothalamic neurons as a result of stress with prolonged restriction of living space 28
KUCHERIAVYI YU. M., BILAI I. M., MANDZII T. P. Antipyretic properties of 1,2,4-triazole derivatives 30
MOHISEN R., ZHADAN S. A. Musculoskeletal disorders in multiple sclerosis 31
MORAD KATA, E. N. LUCHKO, O. V. FILIPTSOVA HIV virus 32
NAI ABRAHAM Autophagy: cellular degradation 33
RUSANOV O. D., SHUTOVA I. V. Pathophysiological mechanisms of migraine development 34
SULEIMAN OMER KHALID, GERASYMOVA O. O. Assortment of antibacterial medicines for the treatment community-acquired pneumonia in adults at the pharmaceutical market of ukraine 36
АКІМОВ О.Є. Вплив суспензії на основі активованого вугілля на процеси деструкції сполучної тканини в слизовій оболонці шлунка за умов хронічної нітратно-фторидної інтоксикації 37
АШФЕННАР САРА, ГЕРАСИМОВА О. А. Ассортимент антисекреторных лекарственных средств для фармакотерапии язвенной болезни двенадцатиперстной кишки на фармацевтическом рынке Украины 39

COMPARATIVE MORPHOFUNCTIONAL ANALYSIS OF CHRONIC HYPERGLYCEMIA INFLUENCE ON PANCREATIC ISLETS IN SPONTANEOUS HYPERTENSIVE AND WISTAR RATS

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Introduction. The rapidly increasing incidence and prevalence of dyslipidemia, obesity and type 2 diabetes mellitus (DM) are becoming a major global health issue in industrialized countries, however, the need for identification of relevant molecular targets and devising effective preventive and therapeutic algorithms is met by only modest advancement of our knowledge. Systemic arterial hypertension (AH) and DM represent two important risk factors for cardiovascular disease, the leading cause of morbidity and mortality in the world. The coexistence of AH and diabetes is commonly observed in adults and has been associated with a higher risk of cardiovascular events. These two conditions often coexist and have in common the metabolic features of hyperinsulinemia and glucose intolerance. In fact, both are considered as disorders of insulin resistance.

Materials and methods.

A total of 36 adult male spontaneously hypertensive rats (SHR) (n = 18) and Wistar rats (n = 18) bred in the PE "Biomodelservis" (Kyiv) were used in the study. At the initiation of experimentation, Wistar rats and SHR were at an average body weight of 250-270 g and 270-300 g, respectively. All the animals were housed in a controlled environment (temperature 22°C on a 12:12-h light-dark cycle with standard laboratory food and tap water *ad libitum*. After acclimatization, in all overnight-fasted animals blood was withdrawn from tail vein for glucose (glucometer Gluco Card-II, Japan) measurements by the glucose oxidase method following the indications of the manufacturer. SHR were severely hypertensive (system of non-invasive arterial pressure measurement BP-2000, Visitech Systems, USA). At the end of the experiment, rats were euthanized and the pancreas was then rapidly removed and fixed in Bouin's solution for 20 h at room temperature, dehydrated in graded ethanol, paraffin embedded and 5 μm sections were obtained (a rotary microtome Microm-325, MicromCorp, Germany). Insulin and glucagon were stained in pancreas by immunohistochemical and immune fluorescence techniques using the standard immunohistochemistry protocols and their contents were quantified by ELISA using commercial kits (Peninsula Laboratories, San Carlos, CA, USA). The sections were examined with ultraviolet microscopy (AxioScope microscope, Carl Zeiss, Germany) in AxioVision 40 V 4.8.2.0 software program (License No. 3005339) with an excitation wavelength 390 nm, using a filter 38HE with high emission (Carl Zeiss, Germany).

Results. In SHR, plasma glucose level was 2 times higher ($p<0,05$) than those in the age-matched control Wistar rats. The hyperglycemia in SHR was associated with marked hyperplasia of pancreatic beta-cells as their mean area was increased by 11% ($p<0,05$) compared to Wistar rats, and the rats remained markedly hyperglycemic until the end of experiment at the age of 24 months. The beta-cell numbers in pancreatic

islets of SHR were 29% ($p<0,05$) higher than those in the aged-matched Wistar rats. In addition, glucagon secretion in these rats was relatively suppressed as the number of alpha-cells was reduced by 80% ($p<0,05$) and their mean area was less by 5% ($p<0,05$) then those in Wistar rats.

Conclusions. SHRs develop moderate hyperglycemia which rapidly progresses to a marked hyperglycemia with age. This is the result of an age-dependent pancreatic islet hyperplasia which can be observed as early as 7 month of age. Compensatory pancreatic beta-cells hyperplasia develops to maintain glucose homeostasis in state of chronic hyperglycemia. Compensation is maintained by increase in beta-cell number and function. However, inadequate compensation of hyperglycemia due to progressive beta-cell dysfunction leads to obesity associated type 2 diabetes, a well noted feature in human subjects and animal models alike.