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ЗМІСТ

ALEKSANDROVYCH VERONIKA, WOLNICKI MICHAL, GIL ANNA, GIL KRZYSZTOF Relation between ureteral telocytes and the hydronephrosis development in children 13
HANCHEVA O. V., HREKOVA T. A., KADZHARIAN IE. V. Age-related morphological and functional changes in endocrine pancreas of wistar rats in physiological aging..... 14
LANOVENKO I. I. The experimental study of the reactivity of erythrocyte glutathione in hemic hypoxia of hemolytic genesis..... 16
LYSIUK ROMAN M., SABET MOHAMED ASHRAF Substantiation on choice of ingredients for herbal collection of radioprotective action..... 17
OGNEVA L., LISINA D. Reactivity mechanisms adaptation medical students to study load..... 18
POLUPAN Y. S., SAFARGALINA-KORNILOVA N. A. Pathophysiological bases of oxidative stress development by example of huntington's disease..... 19
SLEIMAN F., MYRONCHENKO S. New approaches to allergen-specific immunotherapy..... 21
ZAPLATNIKOV Y., BASHYNSKA V., MOSEIKO V., MURLANOVA K., KOLIADA A. Association of SNPs in coagulation factor genes with ischaemic stroke among ukrainian population..... 22
АБДУЛЛАЕВА АЗИЗА, РЫБАК В. А., КОРОЛЬ В. В. Изучение особенностей фармакотерапии хронического гастрита..... 24
АКИМОВ О. Е. Роль активации NF-кВ фактора в развитии гиперпродукции оксида азота в семенниках крыс при хронической фторидной интоксикации... 26
АНДРЮШАЄВ О. В., РУБАН О. А., МАСЛІЙ Ю. С., ЯРЕМЕНКО М. С. Перспективність розробки нових препаратів на основі листя лепехи звичайної 28
АРТЮШЕНКО В. В., МАМОТЕНКО А. В. Оцінка функціонального стану серцево-судинної системи учнів 10-х класів за пробою за В.К. Добровольського..... 30
БАЙКОВА К. О., САФАРГАЛІНА-КОРНІЛОВА Н. А. Патогенетична роль оксидативного стресу у розвитку артеріальної гіпертензії..... 31
БЕЛЯЕВА Т. М. Вклад взаимодействия полиморфных локусов гена *FLG* в формирование хронической истинной экземы 33
БОЛБАТ Е. З., ИОТЧЕНКО Д. О. Патофизиологические и клинико-морфологические особенности красного плоского лишая слизистой оболочки полости рта..... 34
БРАТЧУК К. В., САФАРГАЛІНА-КОРНІЛОВА Н. А. Взаємозв'зок між SARS-COV-2 та ренін-ангіотензиновою системою..... 36
ВАСИЛЕНКО І. В., САХАРОВА Т. С., АНДРЄЄВА О. О. До питання безпеки застосування жовчогінних препаратів у межах самолікування..... 38
ВАШКОВА Д. Н., ЧЕРЕНКЕВИЧ Т. В. Механизмы развития и ранняя диагностика глаукомы 39

AGE-RELATED MORPHOLOGICAL AND FUNCTIONAL CHANGES IN ENDOCRINE PANCREAS OF WISTAR RATS IN PHYSIOLOGICAL AGING

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Introduction. Growing evidence suggests that physiological aging is crucial in the progressive deterioration of organs in adulthood and the pancreatic islet morphological and functional decline pathogenetically associated with chronic and metabolic diseases, including impaired glucose tolerance, obesity, arterial hypertension as well as type 2 diabetes mellitus, is beyond argument. Although the endocrine pancreas is the main objective of numerous universal in-depth studies, some aspects still remain to be resolved giving rise to some forms of potentially serious limitations. For example, it is not clear whether the islet changes are due to aging itself, or the result of another additional factor which is merely associated with increasing age. It is quite probable that sex differences as well as laboratory rats' subjection to life-long inactivity tending toward obesity affect islet morphology and function. The impact of inactivity predisposing for obesity and possible influence of breeding on islet morphology and insulin secretion could not have been evaluated in our original study.

The work was aimed to analyze the local effects of physiological aging on pancreatic islet structure with especial reference to beta-cells morphology and function in Wistar male rats aged between 1 and 24 months.

Materials and methods. A total of 40 male Wistar rats (250-270 g) bred in the PE "Biomodelservis" (Kyiv) were used in the study to compare variations in islet structure and size in physiological aging. To evaluate age-related morphological and functional changes in endocrine pancreas, the animals were randomly assigned to four age-grade groups after 1-week acclimation. All the animal groups 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). Body weight and fasting blood glucose were measured in overnight fasted animals at 1 (the young group), 7 (the adult group), 18 (the middle-age group) and 24 (the aged group) months of age followed by a decapitation sacrifice under deep anaesthesia via intraperitoneal injection of sodium thiopental (40 mg/kg body weight). Just before the pancreas removal, blood was taken from the aorta for measurement of glucose levels by the glucose oxidase method (glucometer GlucoCard-II, Japan, based on the manufacturer's instructions). Following standard histological preparation, immunofluorescence staining for insulin (Peninsula Laboratories Inc., USA) was performed on tissue serial sections (5 μm thick) from different pancreatic areas. Approximately 100 pancreatic islets were evaluated microscopically at least in 5 separate fields for each specimen and the number of positively staining cells was counted. Morphometric characteristics of pancreatic islets and beta-cells were examined under a light microscope (Axioskop, Zeiss, Germany).

Microscopic fields were captured by a high-resolution colour video camera (COHU-4922, COHU Inc., USA) connected to the microscope. Islets and beta-cells in these fields were digitized interactively using a self-written software VIDAS release 2.5 and a Vidas Image Analysis System (VIDAS-386, Kontron Elektronik GmbH/Carl Zeiss, Germany), and digital images were stored. A P value of < 0.05 was deemed statistically significant.

Results. In general, plasma glucose level remained stable with slight variations occurred during the 18-month observational period, but it was significantly 40% increased by the 24 month. The mean volume of islets obtained from 18-month-old rats was substantially greater than the volume of islets of younger rats ($P<0.05$). On the other hand, islets from 24-month-old rats were only small- and middle-sized with little large-sized and no giant islets, mostly obtained from 18-month-old rats. The beta-cells number per islet gradually increased with age ($P < 0.05$) reaching the maximum by the 18th month, but decreased by as much as 40% in aged group animals as compared to middle-age group. The insulin content in beta-cells represented a 30% ($P < 0.05$) increase by the 18th month as compared to 1- and 7-month-old rats, and doubled by the 24th month amounting to the highest value. The insulin content per islet was progressively increased and reached its maximum also in the aged group animals. It is clear that this age-related impairment in insulin secretion per beta-cell was progressive, worsening by the 24th month when hyperglycemia was developed. Insulin secretion per volume islets was such as that the larger islets of 18-month-old rats secreted significantly less insulin than did islets from 1- and 7-month-old rats ($P<0.01$). The decrease in insulin secretory rates noted for islets was even more dramatic when expressed per beta-cell in aged rats.

Conclusions. The data presented here clearly demonstrate that the elevation of fasting blood glucose in aged rats was coincided with a significant decrease in insulin secretion. The ability of beta cells to secrete insulin declined with age and pancreatic islets showed significantly reduced glucose-induced insulin release compared with islets from younger rats. New cells were formed in the middle-aged rats resulting in enlarged islets predominance in order to compensate incipient hyperglycemia mounting with advanced age, but a demand in combination with the age-related decline in beta-cell capacity, eventually led to injury and death of some cells presenting in significantly reduced the mean volume of islets. Certainly, additional studies will be necessary to define the limits of the endocrine pancreas compensatory response and to detail the influence of various external factors on these processes.

Key words: pancreas, beta-cells, insulin, hyperglycemia, aging, immunofluorescence staining