Body Composition - Height, weight, Body Mass Index (BMI), percentage of total and visceral fat using bioimpedance meter weighing scale (OMRON BF 511) were estimated. To assess the insulin resistance degree a small model of homeostasis (Homeostasis model assessment – HOMA) was used, calculated by means of the HOMA Calculator Version 2.2 Diabetes Trials Unit at the University of Oxford (UK). All individuals underwent a single serum 25 (OH) D. An electrical and chemiluminescent method was used to determine the level of 25 (OH) D in the blood serum. The study was performed using the Elecsys 2010 device (Roche Diagnostics, Germany) using cobas test systems.

Behavioral Outcomes, minutes per week of moderate and vigorous physical activity were used in analyses, dichotomized as less than/greater than 90 minutes/week, based on the median reported time. Patients were asked to report number of hours/days spent sitting or reclining on a typical weekday to examine sedentary behavior. Basic descriptive statistics described the sample. Pearson correlations examined the relationships between the vitamin D content and the fitness, insulin resistance and physical activity. Significance levels were set at $p<0.05$. The average BMI of the subjects was 34.4 ± 5.4 kg / m2, visceral fat 10.4 ± 5.2%, the total fat content in the body was 29 ± 5.3%. Vitamin D insufficiency was found in 91.6% of patients, and vitamin D deficiency in 4.5%. Physical activity more than 90 minutes/week was reported by 8% of patients enrolled into the study. The level of vitamin 25 (OH) D in serum correlated negatively with BMI ($r= - 0.414$, $p<0.05$), the content of visceral fat ($r=-0.626$, $p<0.05$), total fat ($r=-0.398$, $p<0.05$) and HOMA-IR ($r=-0.487$, $p<0.05$).

The following conclusions can be drafted: Sedentary lifestyle, higher content of visceral and total fat contributed to vitamin D deficiency. 25 (OH) D deficiency and insulin resistance are interrelated. It is necessary to prescribe cholecalciferol in order to prevent and correct its deficiency in obese individuals and improve insulin sensitivity.

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OBESITY AND HYPERANDROGENISM IN WOMEN – MECHANISMS OF MUTUAL DEVELOPMENT AND PECULARITIES OF METABOLIC DISORDERS

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Obesity is a non-infectious epidemic of our century. All over the world, people of different ages tend to suffer from it. If current trends continue, by 2025 2.7 billion people will be overweight, more than 1 billion people will be obese, and approximately 177 million people will be severely affected by obesity. As a matter of fact, every 4th adult Ukrainian is obese. Obesity is associated with many comorbid metabolic diseases and complications. Among those are diabetes mellitus (DM) type 2, cardiovascular diseases, non-plastic processes, osteoporosis, polycystic ovary syndrome and hyperandrogenism.

In case of obesity, the formation of active androgens in peripheral tissues increases. Obesity causes a decrease in the concentration of sex-binding globulins, which leads to an increase in the fraction of free androgens in the blood. Obese women are almost three times more likely to develop polycystic ovary syndrome, whereas 50-80% of women with polycystic ovary syndrome are overweight or obese. In adipocytes of visceral adipose tissue, there is a decrease in the number of insulin receptors. As a result, relative hyperinsulinemia and compensatory insulin resistance develop which leads to impaired glucose tolerance and the development of hyperglycemia, DM type 2. Hyperinsulinemia affects both the ovaries and adrenal glands, increasing the production of androgens. Due to the disorders of the formation and secretion of gonadotropins, the same changes are observed with gonadotropic hormones. The secretion and level of luteinizing hormone increases, consequently, follicle stimulating hormone decreases. In response to these changes, under the influence of luteinizing hormone, the production of androgens in the theca cells of the follicle increases. The maturation of follicles is deteriorated and their atresia occurs. In atresia follicles, the formation of estradiol decreases and androgen production increases. In adipocytes of visceral adipose tissue, the content of androgen receptors and aromatase increases, resulting in the

autonomous formation of androgens and the development of relative hyperandrogenism. In addition, obesity is accompanied with an increase in the level of 5-alpha-reductase. Thus, apart from the ovaries and adrenal glands, adipose tissue is the source of androgen production in obese persons. Women with obesity and polycystic ovary syndrome have more severe insulin resistance and hyperandrogenism, a more unfavorable lipid profile and a reduced quality of life. Thus, the reduction of the body weight causes impairment of clinical hyperandrogenism manifistations, which is an important component of treatment for such a comorbid pathology.

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TUBULOINTESTITIAL SYNDROME DEVELOPMENT IN THE DYNAMICS OF ALLOXAN-INDUCED EXPERIMENTAL DIABETES MELLITUS

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The aim of the presented study was to explore the peculiarities of tubulointerstitial syndrome development in the dynamics of alloxan-induced experimental diabetes mellitus.

The experiments were carried out on 60 white non-linear mature male rats, 30 with experimental diabetes mellitus (EDM) induced by intraperitoneal administration of alloxan at a dose of 160 mg/kg of body weight, 30 intact rats served as the control group. After 10, 25 and 45 days of the experiment the animals with EDM of corresponding duration and 10 animals of the control group were withdrawn from the experiment. The kidneys, removed after decapitation of rats, were dissected to 3 parts – renal cortex, medulla and papilla, sodium and potassium content was determined in water-extract of the corresponding part of the renal parenchyma, and papillary-cortical, papillary-medullar and medullary-cortical concentration ion gradients were calculated.

Calculation of the concentration gradients of sodium ions revealed a two-fold decrease in the papillary-medullar gradient and a two-fold increase in the medullary-cortical gradient with practically unchanged papillary-cortical gradient when comparing the results of animals with 11-day EDM with control indices. At the same time, there was a significant increase in papillary-cortical and medullary-cortical potassium gradients (1,6 times and 2,6 times, respectively) with a 1,5-fold decrease in papillary-cortical potassium gradient in animals of this experimental series.

Significant suppression of papillary-medullar and papillary-cortical concentration sodium gradients, as well as a slight limitation of its medullary-cortical gradient were established in case of 26-day long EDM. The concentration potassium gradients were significantly reduced.

The papillary-cortical and medullary-cortical concentration sodium gradients were found to be significantly increased, while the papillary-medullar sodium gradient was reliably decreased in 46-day long EDM. Similar changes concerned the concentration potassium renal gradients.

Thus, the results of the study of tubulointerstitial disorders in the dynamics of alloxan-induced EDM suggest that changes in the concentration gradients of sodium and potassium are already observed at the early stages of pathology and indicate the initiation of tubular dysfunction accompanied by intensification of natriuresis and kaliuresis. It is disorders of tubular sodium and potassium transport, redistribution of their content between the vascular, tubular and interstitial compartments of the kidneys that lead to changes in local hemodynamics in the kidneys, hydrophilicity and osmolarity of the interstitium, limitation of regulatory influence of the renal countercurrent multiplication system, disturbance of urine concentrating mechanisms and water-osmotic balance regulating system. Further development of glomerular-tubular and tubular-tubular imbalance, suppression of aldosterone- and vasopressin-dependent mechanisms of interstitial osmolarity regulation will contribute to secondary damage of tubules and interstitium of kidneys as well as the progression progression of renopathy.