хрупкости эритроцитов. В результате мы определили скорость истечения коньюгата динитрофенола — глутатиона из контрольных эритроцитов крысы и эритроцитов, подвергнутых окислительному стрессу. В случае контрольных эритроцитов скорость истечения коньюгатов равна 12,8 нмоль/мин. В случае эритроцитов, подвергнутых воздействию окислительного агента, скорость истечения коньюгатов глутатиона равна 10,7 нмоль/мин.

**Выводы.** Таким образом, мы выяснили, что воздействие гипохлорной кислоты приводит к выраженному повреждению ABC-транспортеров и ингибированию экспорта конъюгата глутатиона из клетки. Ингибирование экспорта ксенобиотиков, возможно, связано с окислением внутриэритроцитарного глутатиона.

## Литература:

- 1. Dean, M. The human ATP-binding cassette (ABC) transporter superfamily / M. Dean, A. Rzhetsky, R. Allikmets. Genome Res. 2001. Vol. 11. P. 1165–1166 c.
- 2. Venkatakrishnan, K. Effects of the antifungal agents on oxidative drug metabolism / K. Venkatakrishnan, L. L. von Moltke, D. J. Greenblatt. Clinical relevance; Clin. Pharmacokin, 2000. 111 c.
- 3. Grapefruit-drug interactions: can interactions with drugs be avoided? / S.U. Mertens-Talcott [et. al.]. J. Clin. Pharmacol, 2006. 46 c.
- 4. Bergamottin contribution to the grapefruit juice-felodipine interaction and disposition in humans / Goosen TC [et. al.]. Clin. Pharmacol. Ther, 2004. 78–79 c.

## STUDY AND COMPARATIVE OF THE LEVEL OF SOME SEXUAL HORMONE IN BLOOD SERUM MEN WITH TYPE 2 DIABETES

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**Introduction.** Sexual hormones (as estradiol, progesterone, androstenedione, or testosterone) that are produced especially by the ovaries, testes, or adrenal cortex and that exerts estrogenic, progestational, or androgenic activity on the growth or function of the reproductive organs or on the development of secondary sex characteristics [1].

In addition, we can define them as a chemical substance produced by a sex gland or other organ that has an effect on the sexual features of an organism. Like many other kinds of hormones, sex hormones may also be artificially synthesized [2].

The sex hormones of the male follow a much simpler pattern than do those of the female, although the same principle of interaction exists between the pituitary gland and the gonads. The latter organs, the testes, secrete steroids called androgens, which are responsible for the maintenance of male characteristics and behavior [3]. FSH from the pituitary gland stimulates the growth of the seminiferous tubules that constitute much of the structure of the testes and promotes within them the cell divisions that result in the production of mature sperm. LH from the pituitary gland promotes the development within the testes of endocrine tissue, which is composed of groups of cells (interstitial tissue) between the seminiferous tubules. The interstitial tissue of certain bony fishes, however, is represented by cells, called lobule boundary cells, situated within the tubule tissue [4].

Under the influence of LH, the interstitial tissue secretes the steroid hormone testosterone, which is the most important vertebrate androgen. The fact that it is an intermediate compound in the metabolic pathway of estrogen synthesis accounts for the origin of some forms of abnormal sexual organization in humans; for example, the testes may secrete predominantly estrogen instead of androgen, resulting in markedly female appearance and behavior in a male. Although testosterone may be secreted by the adrenal cortex, occasionally producing sexual disturbances, the amount of secretion is not normally significant. Testosterone, which is bound to a protein as it circulates in human blood, can be converted to the compound (androstenedione) from which it is formed, especially in the liver and in muscle; both compounds are metabolized, mainly in the liver, to substances that are excreted in urine [5]. Very small quantities of testosterone can also be excreted in urine, and the quantities of testosterone and compounds derived from it frequently are measured to provide an index of testicular condition [6].

In addition to promoting male characteristics, male behavior, and the maintenance of the spermatic tubules, testosterone, in the presence of normal amounts of growth hormone, also promotes growth of the bony skeleton. The reason for rapid growth at puberty is that the secretion of androgen markedly increases. The hormone brings about the closure of the epiphyses (ends) of the long bones, which completes the process of growth (estrogens have a similar action in the female). Thus, as often occurs among animals, growth ceases before full reproductive activity is attained, and competition between two processes, both of which make heavy demands upon the resources of the body, is avoided [6].

In vertebrates, the muscular and secretory activities of the alimentary canal and its associated glands are regulated by nervous and hormonal mechanisms. The hormones constitute a self-contained complex in which the digestive hormones regulate the system that produces them, functioning largely independent of the rest of the endocrine system [7].

The functions of digestive hormones are best understood in mammals, in whom at least six are well characterized. Three well-known digestive hormones are gastrin, secretin, and cholecystokinin (CCK). Other digestive hormones include ghrelin, motilin, and gastric inhibitory peptide [8].

When food enters the stomach, the wall of its pyloric end (the area at which the stomach joins the small intestine) releases gastrin, which promotes the flow of acid from the gastric glands in the stomach. These glands also release pepsinogen, which is the inactive form of the protein-digesting enzyme pepsin, but this process is primarily under nervous control. The entry of the acidified stomach contents into the first part of the small intestine (duodenum) releases secretin and cholecystokinin. Secretin evokes the discharge of fluid and bicarbonate ions from the pancreas (hydrelatic action) and promotes the secretion of bile from the liver (chloretic action). Cholecystokinin, so-called because its two main actions were formerly attributed to two separate hormones, evokes the release of enzymes from the pancreas (ecbolic action) and causes contraction of the gallbladder (cystokinetic action), thereby promoting the entry of bile into the duodenum [9].

Little is known regarding hormonal control of alimentary activities in lower vertebrates; however, hydrelatic, ecbolic, and cystokinetic activities are present in preparations of the alimentary tracts of both agnathans and gnathostomes, indicating that substances able to regulate digestive activity appeared very early in the evolution of the vertebrate alimentary tract. Evidence suggests that the appearance of these hormones may have resulted in molecular diversification similar to examples previously discussed. The structure of the glucagonmolecule from the pancreas, for example, is similar to that of secretin in that each molecule includes the same 15 amino acids located in the same positions. It has therefore been suggested that the two hormones may have evolved from a common ancestral molecule [10].

**Methods.** In the present study, total 50 patients, 25 men aged 3-50 years, which were diagnosed as type 2 diabetes mellitus patients and confirmed by the estimation of fasting plasma glucose (about 130 mg/dl) on two opportunity, were selected from the many hospitals that have situations of diabetes mellitus patients.

The results. The serum total testosterone level of diabetic group was significantly lower than that non-diabetic control group (p-value = 0.000). The mean of serum total testosterone of diabetic group was found  $4.45\pm2.09$  ng/ml and serum total testosterone of non-diabetic control group was  $7.21\pm1.98$ ng/ml. Conclusion: As low serum total testosterone levels are found in type 2 diabetes mellitus patients, this may highlight requirement of urgent implementation of screening programs, in order to detect testosterone deficiency in all type 2 diabetes mellitus male patients at an early stage and to supplement testosterone accordingly.

**Conclusion.** This study has shown that there is a significant reduction in serum total testosterone levels in type 2 diabetes mellitus patients. As low serum total testosterone levels are found in type 2 diabetes mellitus patients, this may highlight requirement of urgent implementation of screening programs, in order to detect testosterone deficiency in all type 2 diabetes mellitus male patients at an early stage and to supplement testosterone accordingly.

## **References:**

- 1. Boyanov, M. A. Testosterone Supplementation in Men with Type 2 Diabetes, Visceral Obesity and Partial Androgen Deficiency / M. A. Boyanov, Z. Boneva, V. G. Christov. Aging Male, 2003. Vol. 6; P. 1–7.
- 2. Kalyani, RR. Androgen deficiency, diabetes and the metabolic syndrome in men. Curr Opin Endocrinol Diabetes Obes / RR. Kalyani, AS. Dobs, 2007. Vol. 14; P. 226–234.
- 3. Association of hypogonadism and type II diabetes in men attending an outpatient erectile dysfunction clinic / G. Corona [et al.]. Int J Impot Res, 2006. Rev. 18; P. 190–197.
- 4. Serum testosterone level in type 2 diabetes mellitus / S. Verma [et al.]. Journal, Indian Academy of Clinical Medicine, 2013. Vol. 14, № 2; P. 115–16.
- 5. A Reference ranges and determinants of testosterone, dihydrotestosterone, and estradiol levels measured using liquid chromatography—tandem mass spectrometry in a population-based cohort of older men / B. B. Yeap [et al.]. Journal of Clinical Endocrinology and Metabolism, 2012. Rev. 97; P 4030–4039.
- 6. Sex differences of endogenous sex hormones and risk of type 2 diabetes: a systematic review and meta-analysis / E. L. Ding [et al.]. JAMA, 2006. Vol. 295; P. 1288–1299.
- 7. Endogenous sex hormones and the development of type 2 diabetes in older men and women: the Rancho Bernardo Study / J. Y. Oh [et al.]. Diabetes Care, 2002. Rev. 25; P. 55–60.
- 8. Type 2 diabetes mellitus and testosterone: a meta-analysis study / G. Corona, [et al.]. Maggi M Int J Androl; 2010. P. 25–26.
- 9. Low sex hormone-binding globulin, total testosterone, and symptomatic androgen deficiency are associated with development of the metabolic syndrome in nonobese men / S.T. Page [et al.]. J Clin Endocrinol Metab, 2006. Rev. 91; P. 843–850.
- 10. Acute sex steroid withdrawal reduces insulin sensitivity in healthy men with idiopathic hypogonadotropic hypogonadism / MA. Yialamas [et al.]. Journal of Clinical Endocrinology and Metabolism, 2007. Rev. 92; P 4254–4259.