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detoxication of xenobiotics, which damaging effect can be realized during gametogenesis, fertilization, implantation and embryogenesis. Variability of glutathione -S-transferase (GSTM1, GSTT1 μ GSTP1) genes, which control synthesis of phase 2 detoxication enzymes, was analyzed by PCR (DNA "Prep") in women with endocrine and endometriosis-associated infertility history, control group and their kids. Results and discussion: GSTT1 gene deletion polymorphism analysis in women with endometriosis-associated infertility history revealed, that part of homozygotes with zero genotype (13%) is 2.7 times less than in the "copy-couple" group (34,8%). Frequency of genotype GSTP1 Val105/Val105 (reduced catalytic activity) is definitely higher in women with infertility history compared to control group. In fertile women children the most functionally favorable M1+/T1+/P1 Ile105/Ile105 genotype had maximal frequency, compared to children of women with infertility history. Differences in frequencies of allelic variants of glutathione - S-transferase genes and their inter locus combinations suppose to be the additional laboratory criteria in reproductive diseases early differential diagnosis and prognosis. Conclusion: disbalance of detoxication system genes in women with infertility history, especially endometriosis-associated infertility, and in their offsprings was proved. Analysis of glutathione - S-transferase genes (responsible for detoxication) is highly recommended for female infertility prognosis.

■ P-119 > THE INFLUENCE OF THYROGLOBULIN AUTOANTIBODIES ON THYROGLOBULIN CONCENTRATIONS MEASURED BY AN IMMUNORADIOMETRIC ASSAY

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Background: Thyroglobulin autoantibody (TgAb) interference with Tg measurements remains the most serious problem limiting the clinical significance of serum Tg values in patients with differentiated thyroid cancer. The aim of this study was to explore the influence of TgAbs on Tg concentrations measured by an immunoradiometric assay (IRMA). Design: We have used standard Tg concentrations (50, 100 and 200 ng/ml), THYRO, CIS biointernational, France, and 30 patient's sera with TgAbs. Standard Tg concentrations were preincubated with patient's sera or zero standard of TgAbs (volume ratio 1:1) during 30 min and Tg concentrations were measured. Tg concentrations obtained in the presence of patient's sera were compared with Tg concentrations measured in the presence of zero standard of TgAb. The Tg concentrations in the range of 80-120% of expected values were considered unvaried. Results: The standard Tg concentrations measured in the presence of patient's sera with TgAb were unvaried (76.7%), decreased (16.7%) or increased (7.6%), respectively. These results were obtained with all three standard Tg concentrations that were analysed. It was interesting that maximal influence of TgAbs on Tg concentrations were not obtained with patients sera containing utmost TgAb concentrations, indicated that not concentration, but specificity of TgAbs in patient's sera has predominant influence on Tg measurements. Conclusion: Our results show that TgAbs in some, but not all patient's sera have influence on Tg concentrations measured by the IRMA assay. The degree of influence of TgAbs on Tg measurements depends predominantly on autoantibody specificity.

■ P-120 > RESEARCH OF CORRELATIVITY BETWEEN THE PLASMA HOMOCYSTEINE AND ENDOGENOUS CARBON MONOXIDE OF PENILE CORPUS CAVERNOSUM IN TYPE 2 DIABETES

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Background: Erectile dysfunction (ED) is a common disease in urinary surgery, diabetes mellitus is one of the high risk factors in ED patients. The aim of this experiment is to explore whether homocysteine (Hcy) is an independent effect factor for endogenous carbon monoxide (CO) of rat penile corpus cavernosum of type 2 diabetic erectile dysfunction of adult male Wistar rats. Design: Ten rats were randomly selected from fifty Wistar male rats of three months supplied by normal feeding, as control group (A group). Other forty rats were fed by high-fat and high glucose. After four weeks they were injected in caudal vein with streptozotocin (30mg/kg). Filtered thirty diabetes mellitus divided into three groups: diabetic rats (B group), diabetic rats with insulin treatment (C group),

diabetic rats therapy in folic acid and vitamin B12 groups (D group). A group supplied by normal feeding, B, C, D group were fed by high-fat and high glucose. Results : the level of plasma total Hcy of B group was obviously increased compared with the controls, the erection rate of the penile cavernous tissue and the activity of CO in penile corpus cavernosum decreased significantly. The level of plasma total Hcy of C and D group was obviously decreased, while the activity of CO and the erection rate were higher than B group. Conclusions: The high level of plasma total Hcy of type 2 diabetes maybe a molecular mechanism that induce the decline of the activity of CO in penile corpus cavernous, and then cause diabetic erectile dysfunction(DMED).

■ P-125 > PATHOPHYSIOLOGIC ROLE OF NOVEL KIDNEY CORTICAL TUBULAR INCLUSION BODIES

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Renal tubular epithelium inclusion bodies are typically seen with viral infections or lead poisoning, but very rarely associated with drug administration. This poster describes and discusses the pathophysiologic role of a novel finding of renal cortical tubular epithelial cells intranuclear and intracytoplasmic inclusion bodies associated with oral administration of a norepinephrine/serotonin reuptake inhibitor (NSRI) in Sprague Dawley (SD) rats. Rats were given an NSRI daily for four weeks and histopathologic, electron microscopic, and immunohistochemical examination were performed. Round eosinophilic intranuclear inclusion bodies were observed histologically in tubular epithelial cells of the renal cortex in rats given the NSRI compound. There was no evidence of cellular degeneration or necrosis in the inclusion-containing renal tubular epithelial cells. By electron microscopy, inclusion bodies consisted of finely granular, amorphous, and uniformly stained non membrane-bound material. By immunohistochemistry, inclusion bodies contained D-amino oxidase protein. In addition, similar inclusion bodies were noted in the cytoplasmic tubular epithelial compartment by electron and immunohistochemical examination. DAAO is an enzyme that catalyzes oxidative deamination of D-amino acids. This is the first description of these novel renal inclusion bodies after an NSRI drug administration in SD rats. Such drug- induced renal inclusions are rat specific, does not represent an expression of nephrotoxicity, and not considered relevant to human risk assessment.

■ P-126 > NON-SELECTIVE BETA-ADRENERGIC RECEPTOR BLOCKADE PREVENTS HEART DYSFUNCTION VIA PRESERVING CAMKII-DEPENDENT HYPERPHOSPHORYLATION OF RYR2 IN DIABETES

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Activation of beta-adrenergic receptors plays important role in modulation of cardiac function in responses to pathological conditions. Stimulation of beta-receptors results in activation of cAMP-dependent PKA cascade. In addition, there is growing evidence supporting the critical role of CAMKII-phosphorylation induced by beta-adrenergic receptor stimulation. We have shown that diabetes-induced phosphorylation of CAMKII, similar to PKA, could induce marked phosphorylation of major components of cardiac excitation-contraction coupling, such as cardiac ryanodine receptors (RyR2) and depletion of the stabilizing FK506 binding protein, FKBP12.6. We have also reported an important therapeutic effectiveness of administration of non-selective beta-adrenergic receptor antagonist timolol, which preserved heart dysfunction via attenuating cardiac remodeling in a rat model of type1 diabetic cardiomyopathy. Since potentiation of the RyR2 by phosphorylation plays a dominant role in the actions of positive inotropic agents such as beta-receptor stimulation, in the present study we examined both RyR2 phosphorylation and protein level, and FKBP12.6 protein level in RyR2 macromolecular complex as well as a role with ERK-phosphorylation on this complex. Beta-blocker, timolol reduced left ventricular volume and restored beta-agonist response in the heart from diabetic rats with cardiomyopathy. Furthermore, our present data showed that the improved cardiac function was associated with restoration to normal RyR2 and FKBP12.6 levels, in part, due to restoration of not only CAMKII but also PKA and ERK phosphorylation levels. Consequently, our results demonstrate the cardioprotective and survival benefits of long-term therapy with beta-blocker in a model of diabetic cardiomyopathy. (Supported by TUBITAK SBAG-COST-109S267)