about decrease β-adrenergic reactance, as is meaningful to basis of development atopic diseases.

At atopic diseases decrease β-adrenergic reactance comes to light. It is shown by a smaller degree glikogenolis, lipolis, increases arterial pressure and educations cAMPH in leukocytes at addition of adrenaline. There is growing evidence, that asthma and atop are genetically linked, although the genetic control of both diseases may be present at several levels sensitization and specific IgE responses, mediator release, end organ responsiveness or phenotype expression**.** It is assumed, that asthma may be a result of primary defect of β-2AR function, that an impaired β-adrenergic function may be also related to IgE responsiveness and atopy.

New exciting data are related to the recently found polymorphism of the β-2AR. The gene for the β-2AR, which is localized close to the IL-4 gene duster on chromosome Sq, has been previously cloned, and sequenced. It is established that several point mutations exist within the gene coding region, resulting in changes in the amino acids sequence. It is shown, that the changes in the aminoacid sequence of the extracellular aminoterminus of the receptor affected the function of the receptor. For example, the presence of glycine at position 16 (Glyl6) was associated with enhanced regulation of the receptor in the response to agonist as compared to arginine at this position (Arg16), and substitution of Glu for Gln at position 27 resulted in a decreased regulation of the receptor. It is assumed, that this polymorphism may affect bronchial smooth muscle function in vivo. In fact, study of the Gln/Glu 27 polymorphism, and airway hyperreactivity in a group of patients with mild to moderate asthma revealed that Gln27 homozygotes had a four-fold higher bronchial hyperresponsive-ness to methacholine (lower threshold dose), than patients who were homozygous for the Glu27 form of the receptor; heterozygotes had an intermediate hyperreactivity. Population study, demonstrated similar frequency of Gly/Arg16 and Glu/Gln27 polymorphism in asthmatic and normal subjects. It is indicating, that polymorphism is not a major cause of asthma. However, comparison of the frequency of the β-2AR genetic variants in patients with nocturnal and non-nocturnal asthma reveled, that the Gly 16 allele was significantly more frequent in the nocturnal group as compared to the non-nocturnal group.

These data indicate, that β-2AR polymorphism may be closely related to the asthmatic phenotype, and severity of the disease. Since β-2AR are present on immune cells like lymphocytes, macrophages and granulocytes, being involved in modulation of inflammatory mediators and cytokines release, it has been postulated that, genetic polymorphism of β-2AR may also be associated with the expression of the atopic phenotype.

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THE USE OF INTERFERONS IN CHRONIC VIRAL LIVER DISEASES

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The increased disease a chronic hepatitis and low efficiency of treatment causes necessity of profound their studying immunopathogenesis and development of new methods of therapy. The broad effects of intercellular actions of interferons (IFN) can be roughly divided into: a) antiviral, b) anticancer, c) immunomodulatory. IFN are not produced constantly, so that their concentration in normal tissues is minimal or undetectable. However, after stimulating factors, esp. infections, their level in tissues highly increases, as a result of the activation of immunocompetent cells; later IFN bind to their specific receptors.

The strongest antiviral action exerts IFN-α. It influences all the stages of viral replication, but most important is inhibition of the genome translation, what makes defective the synthesis of viral proteins (kinases phosphorylate). The second mechanism of translation inhibition is the

induction of 2'5'-oligosynthetase which degrades viral RNA. Both mechanisms omit healthy cells in their inhibitory actions. Furthermore, antiviral effect is achieved also by the increased expression of histocompatibility antigens and stimulation of the immune response. Interferons are now produced in the recombinant form, what enhanced their broader use. They are offered for treatment of adult patients and children with a chronic hepatitis. However, IFN-α is most widely used for the treatment of chronic viral hepatitis B and C.

We report results of treatment of 184 children, mostly with chronic aggressive type B hepatitis, HBsAg and HBeAg positive. Treatment consisted of 3 weekly, doses of 3 MU recombinant IFN-a, mean: 4.51 MU/m2 for 16 weeks (ca. 3 months). In the evaluation one year post IFN: 12% of children proved to be complete responders [HBsAg(-), eAg(-)], 53% of children partial responders [HBsAg(+), but eAg(-)], and 35% - nonresponders [HBsAg(+), eAg(+)]. The treatment was quite well tolerated; but such side effects as fever, loss of appetite, headaches, muscle and joint aches were noted in over 10% of children. The reference (unrelated) group consisted of 77 children (53 boys and 24 girls). During the same period, 2.6% of them became complete responders, 22.1% - partial responders and 75.3% remained nonresponders.

Moreover, in part of the treated children (n-23) we have determined pretreatment prognostic factors, by measuring HBeAg, HBsAg and HBV-DNA quantitatively, in addition to ALT and clinical chemistry. We have found differences between arithmetic means of these parameters, but because of big range of results, the statistically significant differences were obtained only in relation to age of children and HBeAg concentrations. We have shown, that low initial level of HBeAg, HBsAg, low level of HBV-DNA, as well as relatively high ALT value, together with rather low age of children were connected with the good prognosis for response. With regard to chronic hepatitis C - female gender, lower level of HVC-RNA, and lower age, but elevated baseline ALT were associated with good prognosis for response to INF-α therapy.

However, monotherapy with INF-α would give sustained response (persistent normalization of transaminases and negative HCV-RNA) in only ca. 20% of patients after 2 years of follow-up. In adults, interferon combined with ribavirin may raise the effectiveness about 2 times. Our current project in children concentrates on the inhibition of metabolites of arachidonic acid formed during the INF therapy. These metabolites, including prostaglandins (PGE2) and tromboxanes could be suppressed by inhibiting cycloxygenase with indomethacin.

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MAJOR FACTORS, INFLUENCING QUALITY OF LIFE IN RUSSIAN PATIENTS WITH DIABETIC FOOT SYNDROME

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Quality of life (QoL) as one of the basic criteria of treatment efficiency, received worldwide circulation in last years. Many authors (Dedov I., 1998; Ashford R., 2000) mark, that the purpose of therapy of chronic diseases is not in treatment itself, but in improvement of patient's life as a result of severity decreasing or restriction of illness progressing.

The augmentation of life expectancy in patients with diabetes results in annual increase of late complications, one of which is the diabetic foot syndrome (DFS) – the condition, combining neuropathic and vascular disorders of the lower extremities. It conducts to occurrence of foot ulcers and other severe complications, such as diabetic gangrene. DFS is recorded in 30-80 % patients with diabetes (Reiber G., 2001).

However, there is a paucity of research into the specific effects of foot ulceration on the QoL of patients (Ashford R., 2000). Further, although studies have reported psychosocial concerns experienced by patients with diabetes and the complications of lower extremity ulcers, there is little qualitative research which outlines patients' perspectives of living with foot ulceration.