FATTY ACID ANALYSIS OF BREAST MILK OF ATOPIC AND NON ATOPIC WOMEN

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Impaired function of the enzyme δ-6-desaturase has been proposed as a pathogenetic factor for atopic eczema. Such a defect would cause an increase in linoleic acid and a decrease of its metabolic products (e.g. γ-linolenic acid and arachidonic acids) and should be detectable in human milk. To examine this hypothesis we analysed the fatty acid patterns of breast milk samples at 31 healthy and 33 atopic mothers. The atopic individuals were suffering from allergic rhinitis, allergic bronchial asthma or atopic eczema (n = 10). Serum-IgE was above 100 kU/l in all atopic individuals. The quotient of linoleic acid and the sum of Dihomo-γ-linoleic acid plus arachidonic acids is considered the best measure of δ-6-desaturase activity. Both atopic and non-atopic mother's diet did not differ. Breast milk samples were gained by manual expression under standardized condition and immediately frozen in liquid nitrogen. Cold fat extraction and mehtylation preceeded fatty acid analysis by means of gas-chromatography. Results: Quotients of atopic mothers' milk (11.22 ± 3.24) were not different from those of non-atopic mothers' (10.89 ± 3.24). This was valid for both the total group of all atopic mothers as well as the subgroup of exzematous mothers (11.57 ± 3.38). Conclusion: Our results do not confirm an impaired activity of δ-6-desaturase in atopic individuals. Thus, the biochemical basis and rationale for supplementing lactating atopic mothers with γ-linolenic acid preparations seems questionable.

GASTRO-OESOPHAGEAL REFLUX DISEASE IN CYSTIC FIBROSIS: AN ALTERNATIVE TO ATOPY AS A CAUSE OF BRONCHOSPASM.

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Gastro-oesophageal reflux disease(GORD) has been documented in cystic fibrosis(CF) but reported values vary due to patient selection and different diagnostic techniques. GORD can result in persistent lung problems. To gauge the prevalence of GORD and the concomitant respiratory status in CF, 24hr. oesophageal pH and spirometric studies were carried out in an unselected group, n=20 of children, median age 9, range 4-15.

RESULTS: GORD was present and asymptomatic in 17/20 children, 84% Those with severe bronchospasm (SB) and poor growth profiles (PGF) had significantly greater oesophageal acid scores than those with mild bronchospasm (MB) or normal growth profiles (NGP). *P<0.01, °P<0.01. The Schwachman clinical score successfully predicted patients with severe disease, P<0.01.

Acid score:-	SB, n=5	MB, n=15	PGP, n=4	NGP, n=16
Median	*58	25	°69	25
Range	51-99	9-104	9-104	13-100

CONCLUSIONS: GORD is grossly underestimated in CF patients but the Schwachman score is a new useful indicator of disease status. Treatment of GORD may improve bronchospasm and poor growth.

SPECIFIC IgE TO A WHEY HYDROLYSATE FORMULA (HF) IN CHILDREN WITH IgE-MEDIATED ALLERGY TO COWS MILK (CMA)

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There are several protein HFs which have been defined as "hypoallergenic" and are devised for children with allergy to cows milk (CM). HFs are processed using two main techniques: heat denaturation and enzymatic hydrolysis to reduce the molecular weight (mw) of peptides and their allergenic power. However, cases of anaphylaxis in children with IgE-mediated CMA and fed whey HFs have been reported. We have selected 20 children (15 M, 5 F) aged 5 mo-3 yr (median age 1 yr + 6 mo) with IgE-mediated CMA (positive STs and RAST), 15 of whom had immediate-type (urticaria, angioedema, shock, asthma, vomiting, diarrhea), and 5 delayed-type reactions (atopic dermatitis). Diagnosis was done on the basis of anamnesis, elimination-provocation (open) tests under medical surveillance, positive STs, and specific IgE to CM proteins. IgE to HFs were measured using an immunoenzymatic technique (Phadezym RAST, Pharmacia). IgE to CM were detectable in 19 babies, to a-lactalbumin in 20, to β-lactoglobulin in 19, to casein in 16, to a partially HF (Nidina HA) in 7, and to an extensively HF (Prophylac) in 4. This data demonstrates that a cross-reactivity between CM proteins and the epitopes present in HFs is a distinct possibility. In conclusion, the peptides of HFs still have allergenic potency and can be recognized by the cell-bound IgE of a child allergic to CM. Therefore the use of whey HFs should be avoided in children with IgE-mediated CMA.

LOW BIRTH WEIGHT AND ASTHMA IN CHILDHOOD. RESULTS OF A CROSS-SECTIONAL SURVEY IN SCHOOLCHILDREN.

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Abnormal pulmonary function tests and childhood asthma have been suggested as long term sequelae of Low Birth Weight (LBW) (<2,500 g) (Am Rev Respir Dis 1990; 142:555-562). In order to study whether LBW was associated to childhood asthma, we analyzed the data of a cross-sectional survey in 2929 schoolchildren aged 6-11, randomly selected from three areas of the Lazio region, Italy. Overall LBW rate was 4.9%: LBW was positively associated to childhood asthma (Odds Ratio 2.37; 95% c.i. 1.41-3.95). A multivariate analysis was carried out to adjust the effect of LBW for other known determinants of childhood asthma: sex, maternal age at birth, child age, neonatal feeding practice, parental smoking, socio economic status, area of residence and atopy in parents and sibs. The adjusted OR was 2.39 (95% c.i. 1.41-4.04), practically identical to the unadjusted estimate. Our findings suggest that LBW is a major determinant of childhood asthma: suboptimal intrauterine conditions may have long term consequences for respiratory growth and function even if they do not lead to overt neonatal respiratory difficulties.

Cloning and Expression of the Human High Affinity Receptor for IgE

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Mastcells and basophils express on their cell surface a receptor with high affinity for IgE ($Fc_\varepsilon RI$). Binding of allergen to receptor-bound IgE leads to cell activation and the release of mediators (like histamine) responsible for the symptoms seen in allergic reactions. Understanding the structure and function of this receptor could lead to the development of new therapeutic approaches which would directly interfere with IgE-mediated mastcell activation.

$Fc_\varepsilon RI$ consists of a tetramer of one α, one β and two γ subunits. Our group has already published the cloning of the α and γ subunits (J. Kochan et al., Nucleic Acids Res. 16: 3584-94, 1988 / H. Küster et al., J. Biol. Chem. 265: 6448-52, 1990). Here we report the cloning of the gene and of the cDNA for the β subunit and the expression of the complete human receptor in COS-7 cells. The β subunit is a peptide of 244 aminoacids which crosses the plasma membrane 4 times, both termini being inside the cell. It is encoded by 7 exons spanning over 8 kilobases.

Previously transient expression of the rodent receptor showed that all three subunits are required in order to achieve cell surface expression of the receptor. In contrast for human $Fc_\varepsilon RI$ the α and γ subunit alone are sufficient, the β subunit seems not necessary.

GENETIC PREDISPOSITION FOR ATOPY ALLOWS PREDICTION OF CLINICAL COURSE OF RESPIRATORY SYNCYTIAL VIRUS (RSV) INFECTION IN INFANCY.

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To assess the hypothesis that a family history of atopy can predict the clinical course of infants with RSV infection we studied 172 patients with laboratory documented RSV infection during 1985 to 1989. 99 infants were admitted, 73 infants were treated as outpatients. A positive family history of atopic disease was seen in 61% of hospitalised infants, but only in 21% of outpatients ($p<0.001$). This difference remained after controlling for confounding variables. Length of hospital stay (>6 or >9 days) correlated positively with a family history of atopy ($p<0.02$ and $p<0.005$ respectively). Symptoms were more severe in infants with a positive family history of atopy ($p<0.02$). We conclude that a genetic predisposition for atopy is an important host factor which allows prediction of the clinical course during RSV disease in infancy. Alternatively, the severity of RSV disease in infancy may predict the development of atopic diseases (e.g. bronchial asthma) later in childhood.