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SPINK5 G1258A (Glu420Lys) polymorphism and asthma

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Background: Serin protease inhibitor “Kazal type 5” (SPINK5, LEKTI) gene is located on chromosome 5q31-32 region. Interestingly, this region contains the so called cytokine cluster including IL-4, IL-5, IL-9 and IL-13. Up to now, 32 single nucleotide polymorphisms have been identified on SPINK5 gene. In several studies it has been shown that G1258A (Glu420Lys) substitution is associated with atopy, atopic dermatitis, high levels of serum IgE and asthma.

Aim: This study aimed to determine the relationship of (Glu420Lys) substitution in SPINK5 gene with asthma and asthma phenotypes in Turkish children.

Methods: The frequency of G1258A (Glu420Lys) polymorphism was investigated by PCR-RFLP analysis using Hinf I restriction endonuclease in 291 asthmatic

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The effects of nos1 gene and environmental factors on asthma and rhinitis in Taiwan

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Introduction: Asthma and rhinitis is a complex disorder which is known to be affected by interactions between genetic and environmental factors. NOS (nitric oxide synthase) influences the production of innate NO (nitric oxide) that could participate the inflammatory response and represent a key mechanism in asthma.

Methods: The aim of the study was to investigate the polymorphisms of AAT repeats in intron 20, CA repeats in exon 29 and GT repeats in intron 2 of the NOS1 gene in asthmatic and rhinitis children in Taiwan, and their interactions with environmental factors. One hundred and fifty-five asthmatic children, 115 rhinitis children and 186 control children aged 5 to 12 years were recruited in southern Taiwan. In the present study, we addressed genetic effects of NOS 1 gene and interaction effects with environ-

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Clinical respiratory allergy patterns in pairs of siblings

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Background: The allergic disease has multifactorial aetiology. The interaction of genetic and environmental agents induces particularities in the clinical expression of the respiratory allergy.

Objective: To analyse the clinical patterns of respiratory allergy in pairs of siblings.

Methods: It was included 18 pairs of siblings observed in our allergy department. We analysed the clinical allergy patterns concerning respiratory presentation (bronchial asthma (BA) and/or allergic rhinitis (AR) and/or conjunctivitis (C) and/or atopic dermatitis (AD)), its age of beginning and BA and AR severity. We denominated “homogeneous” when the clinical presentation was similar in both siblings of each pair and “heterogeneous” when the clinical presentation was different. The clinical patterns were compared in each pair of siblings (1st and 2nd brother).

Results:

Table 1 for abstract 1665.

| | n | Current age (yrs) | 1st consult (yrs) | 1st symptom (yrs) | Beginning of symptoms: BA (yrs) | Beginning of symptoms: AR (yrs) | Beginning of symptoms: C (yrs) | Beginning of symptoms: AD (yrs) |
|---------------------------------------|----|-------------------|-------------------|-------------------|---------------------------------|---------------------------------|--------------------------------|---------------------------------|
| Total | 18 | 17.1 ± 5.9 | 13.1 ± 4.3 | 6.4 ± 5.3 | 6.6 ± 5.3 | 8.2 ± 4.6 | 12.5 ± 3.8 | 3.4 ± 6.5 |
| 1st | – | 19.0 ± 6.1 | 13.6 ± 4.4 | 7.3 ± 5.8 | 8.3 ± 5.5 | 9.0 ± 4.2 | 11.6 ± 5.0 | 4.7 ± 5.1 |
| 2nd | – | 15.3 ± 5.2 | 12.5 ± 4.3 | 5.4 ± 4.6 | 4.2 ± 3.7 | 7.4 ± 4.9 | 12.7 ± 3.2 | 0.7 ± 0.3 |
| Homogeneous clinical pattern: total | 6 | 17.2 ± 7.8 | 12.2 ± 4.6 | 4.7 ± 4.4 | 4.7 ± 4.4 | 6.7 ± 3.9 | – | – |
| Homogeneous clinical pattern: 1st | – | 20.5 ± 7.3 | 13.1 ± 2.9 | 6.1 ± 5.6 | 6.1 ± 5.6 | 8.5 ± 3.3 | – | – |
| Homogeneous clinical pattern: 2nd | – | 14.0 ± 7.5 | 11.3 ± 6.0 | 3.4 ± 2.4 | 3.4 ± 2.4 | 5.0 ± 3.8 | – | – |
| Heterogeneous clinical pattern: total | 12 | 17.1 ± 4.8 | 13.5 ± 4.2 | 7.2 ± 5.5 | 7.7 ± 5.4 | 9.0 ± 4.8 | 12.1 ± 4.1 | 3.9 ± 4.8 |
| Heterogeneous clinical pattern: 1st | – | 18.2 ± 5.6 | 13.9 ± 5.1 | 8.0 ± 6.0 | 9.4 ± 5.3 | 9.3 ± 4.8 | 11.6 ± 5.0 | 4.7 ± 5.1 |
| Heterogeneous clinical pattern: 2nd | – | 16.0 ± 3.8 | 13.1 ± 3.4 | 6.4 ± 5.2 | 4.9 ± 4.6 | 8.8 ± 5.0 | 12.7 ± 3.2 | 0.7 ± 0.3 |

children and 215 healthy controls. Allele frequencies of G1258A polymorphism were compared between asthmatic and healthy cohorts. Association between the polymorphism and asthma phenotypes including eosinophil counts, total IgE and FEV1 were analysed.

Results: SPINK5 mutant genotype was significantly more frequent in asthmatic children (0.333) compared to healthy controls (0.242) ($P=0.026$) in a model where the wild type G allele is taken as the dominant allele. Glu420Lys genotype did not show any significant association with asthma phenotypes.

Conclusion: These results suggest that Glu420Lys amino acid changes at SPINK5 gene may be a risk factor for asthma in this population.

mental factors on childhood asthma and rhinitis.

Results: After adjusting for age, sex and potential confounders, only NOS1 gene with 14/14 AAT tandem repeats was significantly associated with asthmatic children (AOR = 3.17, $P=0.022$). The interaction effects between NOS1 14/14 AAT and molds, cockroaches, phadiatop test and high levels of total IgE were from 3.6 to 17.9 folds on asthmatic children. But we did not find the relationship between NOS1 gene and rhinitis.

Conclusions: This study findings provide strong evidence that NOS1 gene with 14/14 AAT tandem repeats has a significantly effect on asthmatic children, and also have interaction effects with environmental factors.

We observed that 6 pairs showed homogeneous clinical pattern characterized by BA and AR. Furthermore, in 5 pairs, both siblings presented the same severity of BA and AR. The first manifestation was equal in 4 pairs. Also 4 pairs have a family background of allergy. All of them were house dust mites (HDM) sensitised. Twelve pairs of siblings presented heterogeneous clinical pattern. In each pair, besides the different clinical presentation we also founded discrepancy in the 1st manifestation and clinical severity. Half of them had a family background of allergy. In 10 pairs, both siblings were HDM sensitised. The results concerning age (current age, 1st consult and beginning of each symptom) in the total group and sub-groups according to

the clinical pattern are presented in the table.

Conclusions: The age of the beginning of the symptoms and the need of an allergy consult was earlier in the 2nd sibling. In the group of siblings with homogeneous clinical pattern characterized by HDM allergic BA and AR, the symptoms began at a pre-school age. In the heterogeneous group, the 1st clinical manifestation was at a school age. Determinant genetic agents must be underlying in the group of siblings with homogeneous clinical pattern.

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Statistical model of interactions between CCR3 and eotaxin genes with eosinophilia

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Introduction: Chemokines, such as eotaxin1, eotaxin2, RANTES, MCP provoke an eosinophilic response in the peripheral blood and airways via CCR3. CCR3 gene SNPs and Eotaxin gene SNPs already reported to be associated with asthma. We would search for the evidence of genetic interaction to blood eosinophilia among SNPs of Eotaxin genes and CCR3 gene in asthma.

Methods: Five hundred and thirty-three asthmatics were enrolled. Asthmatics with eosinophilia ($>0.5 \times 10^9/L$) were compared with those without eosinophilia ($<0.5 \times 10^9/L$). The interactions were tested with two different methods. Chi-square tests were used to compare SNP frequencies. Another interaction model was built with logistic regression.

Results: Eotaxin2 + 304C>A (29L>I) was significantly associated with three of four CCR3 SNPs in asthma with eosinophilia. ($P = 0.037 \sim 0.009$) Eotaxin2 + 304C>A (29L>I) with CCR3 SNPs effect was also significantly associated with blood eosinophilia in a interaction model constructed with logistic regression. ($P = 0.0087$)

Conclusions: This statistical model can probably be one of the evidence that CCR3 and Eotaxin genes interact to the blood eosinophilila in asthma.

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Age, height, weight and body mass index and relationship with exhaled nitric oxide

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Introduction: Values of exhaled nitric oxide (FENO) are variable, even in healthy subjects. This variation can be attributed to some variables, as age, height, weight and body mass index (BMI).

Objectives: To evaluate the relationship between FENO and age, height, weight and BIM in asthmatic patients younger than 18 years old.

Material and methods: During a 2 week period, 86 consecutive asthmatic patients younger than 18 years old, followed in our outpatient clinic, were submitted to a FENO determination (flow rate: 50 mL/sec). We recorded their age, height and weight. The body mass index (BMI) was calculated. We analyzed the relationship between FENO and age, height, weight and BMI.

Results: The mean age was 12.3 years old (SD 3.6), with 68.6% males. The median value for FENO, height, weight and BMI was 16ppb (P25-75 = 12.8–64.3 ppb), 153 cm (P25-75 = 136–166 cm), 49 Kg (P25-75 = 34–61 Kg) and 20.5 (P25-75 = 17.5–23.5). Statistical significant correlations were found between FENO and height (0.648), weight (0.602), age (0.582) and BMI (0.424).

Discussion and conclusion: As in healthy subjects, a correlation between FENO and age, weight, height and BMI was found in our asthmatic population. This could be due to the same reasons pointed for healthy people.

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The effects of emotions and stress on pulmonary function in asthmatic patients

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Objectives: Clinical observations suggest that both positive and negative stress can precipitate asthmatic symptoms, but there isn't enough experimental evidence regarding the link between the specificity of emotions (inducing either active or passive coping) and bronchial diameter (constrictive and dilatatory modifications). In this paper we studied the impact of different emotional states and types of stress on respiratory resistance in asthmatic and nonasthmatic individuals.

Methods: Participants (25 asthmatic and 25 nonasthmatic patients) viewed short film sequences selected to induce anxiety, anger,

sadness, surprise, amusement, and completed two stressful tasks: mental arithmetic to induce active coping efforts and viewing of medical films to induce passive coping efforts. Peak Expiratory Flow (PEF), heart rate, and self-reported affective states were measured throughout the experimental session.

Results: Decreases (between 4–12%) in PEF were found in all emotional states compared with the neutral state, asthmatic patients showing stronger reactions to the films than healthy control subjects. The experimental studies inducing passive coping have generated PEF decreases in most subjects who viewed a medical (surgery operations) film and violent (drama) short film sequences (both in 88% asthmatics). Even in the case of positive emotions (using a funny short movie with Mr. Bean) PEF decreased with 4–9% in 76% of the subjects. The active coping induced by mental arithmetic have decreased PEF in 75% subjects but only with 4–7%. Also, significant increases in heart rate and self-reported affective states, sadness, amusement and dyspnea were detected.

Conclusions: Various emotional states and types of stress increase respiratory resistance(measured by PEF) but may be possible that the intensity of these modifications could decisively depend on the degree of emotional involvement of the subjects and the severity of bronchial inflammations during the experiment as shown by our own previous study (Iamandescu-1980).

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Influence of obesity to osteoporosis in postmenopausal asthmatic patients

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Both incidences of asthma and obesity are increasing worldwide. Age, usage of inhaled and oral corticosteroids are risk factors for osteoporosis for asthmatic patients in postmenopausal stage. In our study we aimed to find out the incidence of osteoporosis in our asthmatic patients and compare them with control groups. Then we investigated the effect of asthma related parameters and influence of obesity to osteoporosis. Forty-six patients (diagnosed as asthma according to GINA criteria) and 75 age matched postmenopausal women included to the study. Weight and height were measured and body mass index (BMI) was calculated. Bone mineral density (BMD) of total body, lumbar spine and femoral neck was measured by dual-energy X-ray absorptiometry.