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hans cells. A strikingly intense fluorescent signal for FcεRI was found on all Langerhans cells, even in the absence of a clinical history of atopic dermatitis. High power microscopy showed a uniform distribution of FcεRIα throughout both the cell body and dendrites. Using a panel of monoclonal antibodies (15.1, 15A4, 22E7 and 18E4) the presence of both vacant FcεRIα and receptor occupied by IgE was confirmed. Attempts were made to investigate the possible association of the α-chain of the receptor with β- and γ-chains. Unfortunately the antibodies currently available to us gave no signal on the blisters. This is however not conclusive, since they also elicited only a very weak signal on the positive control, purified blood basophils.

In addition to FcεRI, CD1a and HLA-DR, we assessed the expression and distribution of other surface and cytoplasmic structures. While CD23 (FcεRII) expression varied considerably between skin donors, CD74 and especially CD32 (FcγRII) showed consistently strong staining, revealing even the finest of dendrites. The combined use of these antibodies suggests that the localisation of some of these markers alters during cell-maturation.

Immunohistochemical analysis of skin blisters allows the en face visualisation of Langerhans cells within a minimally disturbed tissue architecture permitting subcellular localisation of key structures as a function of health and disease.

1050 Diagnostic Test Profile of IgE Mediated Atopic Diseases in Saudi Arabia *AR Al-Frayh*, SM Hasnain†, G El-Rab*, Z Shakoor*, A Sedairy†* *College of Medicine, King Saud University †King Faisal Specialist Hospital & Research Centre

The Kingdom of Saudi Arabia is a large country with significant climatic and geographical variation. There has also been tremendous development and modernization in the Kingdom during the past two decades. These developmental factors, directly or indirectly, contribute to the growth, dissemination and or provision of sources for allergens accumulation. In Saudi Arabia, we conducted environmental and allergological studies in different areas of the Kingdom using volumetric sampling for outdoor allergens. For example, Der p 1 was prevalent in mountainous while Der f1 was prevalent in coastal region. Agricultural areas with very low composition of Der p 1 and Der f 1 did not show any variation. Fel d 1 and Per a 1 were recorded in higher composition but not clear variations were seen. Diagnostic results using SPT methods were also variable showing up to 70% reactions with commercial extract including Prosopis pollen extract. Thus, based on the aerobiological information obtained, we have prepared a diagnostic profile of allergens for screening of allergic individuals. Preparation of profiles for each region is also underway. The study resulted as a background basis for the selection of diagnostic antigen and elimination of those not directly relevant to the patient's ambient environment.

1051 Pedigree-based Linkage Analysis on Total Serum IgE Levels: Localization of Genes on Chromosomes 12 and 11 *X-Q Liu*, LR Freidhoff†, E Ehrlich†, TH Beaty*, S-K Huang†* *School of Hygiene and Public Health, Johns Hopkins University, Baltimore, MD †School of Medicine, Johns Hopkins University, Baltimore, MD

High total serum IgE levels are known to be associated with asthma and other atopic diseases which together affect about 20% of the U.S. population. Twins and family studies have shown that both genetic and environmental factors are involved in the control of total serum IgE levels. The primary objective of this study is to locate quantitative-trait-locus (QTL) controlling total serum IgE levels in humans using 12 extended Amish pedigrees and test for interaction between loci. Markers on chromosomes 5, 11 or 12 were used. Linkage analyses under variance-components model were conducted using the SOLAR program. In the one-locus analyses, markers on chromosome 12 showed modest evidence for linkage (highest LOD score is 2.5) to an unobserved QTL controlling total serum IgE levels, while markers on chromosomes 11 and 5 showed no evidence of linkage. In the two-locus analyses, when involving locations on both chromosomes 12 and 11 in a single model along with their interaction, the LOD score increased significantly to 3.7. In this model, the most informative location on chromosome 12 accounted for about 21% of the total variance of total serum IgE levels, while the interaction of this region with the most informative region on chromosome 11 accounted for about an additional 42% of the total variance. These analyses suggest that chromosome 12 is likely to contain a gene (or genes) that controls total serum IgE levels. Furthermore, this gene may interact with another QTL on chromosome 11 to influence total serum IgE levels.

1052 Relationship Between the Clinical Efficacy of rhuMAb-E25 (E25) and Serum Free IgE in Seasonal Allergic Rhinitis *TB Casale*, A Racine†, W Sallas‡, A Fowler Taylor‡, N Gupta‡, PW Rohane‡* *Nebraska Medical Research Institute, Papillion, NE †Novartis Pharmaceuticals, Basel, Switzerland ‡Novartis Pharmaceuticals, E Hanover, NJ

We evaluated the relationship between SAR severity, serum free IgE and the clinical efficacy of E25, a recombinant humanized monoclonal antibody to IgE. Five hundred thirty-six patients of either sex, ages 12 to 75 years, with a history of moderate to severe ragweed-induced SAR were enrolled in a randomized, double-blind, placebo-controlled study. Starting approximately 2 weeks prior to start of the pollen season, patients were treated with E25 300 mg (n=129), 150 mg (n=134), 50 mg (n=137) or placebo (n=136), administered subcutaneously every 3 or 4 weeks based on total serum IgE levels (151-700 and 30-150 IU/ml, respectively), for a total of 12 weeks. An analysis of covariance showed significant correlation between trough (predose) serum free IgE group and daily nasal symptoms (sneezing, itchy nose, runny nose and stuffy nose) severity (DNSS, 0-3 scale) and rescue medication use during the pollen season.

VARIABLE	GROUP: IgE NG/ML	N	MEAN	EST. DIFF. VS. GRP 4	P-VALUE VS. GRP 4
DNSS	1≤25	112	0.82	-0.19	0.007
	2>25-50	119	0.86	-0.14	0.031
	3>50-150	148	0.87	-0.13	0.039
	4>150	139	0.99	—	—
RESCUE TABS/DAY	1≤25	112	0.18	-0.22	<0.001
	2>25-50	120	0.18	-0.22	<0.001
	3>50-150	150	0.21	-0.18	<0.001
	4>150	141	0.39	—	—

E25 produced a dose-dependent decrease in serum free IgE 63%, 33%, 4% and 3% of patients treated with E25 300 mg, 150 mg, 50 mg and placebo, respectively, achieved serum free IgE concentrations of <25 μg/ml. Thus, improvement in SAR symptom severity is related to free IgE and the efficacy of E25 in SAR is related to free IgE and the efficacy of E25 in SAR is related to its ability to decrease serum free IgE levels.

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