Type IV-sensitisation to local anaesthetics in more than 1% of the respective group was only seen in patients with anal involvement. Thirteen patients were sensitised to cincochaine, which is 9.3% of those tested ($n = 140$). Of 142 patients tested with lidocaine, seven were sensitised (4.9%), and five out of 169 patients (3.0%) reacted to benzocaine.

Conclusion: Summed up, patients with dermatitis of the anal area are more frequently sensitised to ingredients of hygiene products and topically applied drugs than patients with genital dermatoses.

98
Molecular basis of nickel allergy

Yin, L; Crawford, F; Marrack, P; Kappler, J; Dai, S
Immunology, National Jewish Health, Denver, CO, United States

Background: Metal-induced allergic contact dermatitis results in a wide range of cutaneous reactions following dermal and systemic exposure. Ni^{2+} allergy is the most common cause of the contact dermatitis, and afflicts around 10–15% of the human population. Apart from the significance of Ni^{2+} in developing contact dermatitis, Ni^{2+} hypersensitivity in patients undergoing joint replacement and tooth implants is a severe problem. Ni^{2+} allergy is mediated by T cells, but how the T cell receptor (TCR) recognises Ni^{2+} is unknown.

Method: We are studying a TCR, ANi2.3, from an allergic patient that recognises Ni^{2+} bound to the MHCII molecule, DR52c, containing an unknown self-peptide. We have identified Ni^{2+}-independent peptide mimotopes for the ANi2.3 TCR by using baculovirus peptide/DR52c libraries. These peptides satisfy the requirements of the TCR both for peptide and Ni^{2+}. We determined the crystal structures of the DR52c/Ni^{2+} independent mimotopes, and ANi2.3 TCR bound to DR52c and one of these Ni^{2+}-independent mimotope peptide.

Results: We identified mimotope peptides that can replace both the self-peptide and Ni^{2+} in this ligand. They share a p7 lysine, whose εNH2 group is surface exposed when bound to DR52c. While the TCR uses germline CDR1/2 amino acids to dock in the conventional diagonal mode on the mimotope-DR52c complex, the interface is dominated by the TCR VβCDR3 interaction wth the p7 lysine. Mutations in the TCR CDR loops have similar effects on the T cell response to either the mimotope or Ni^{2+} ligand. The pocket of p7K of the mimotope bound to DR52c lays in a wide space between the arch of the β1 helix and the side chains of p4 and p7 of the peptide. We have previously shown that this is the functional site of Be^{2+} binding to DP2.

Conclusion: We suggest the mimotope p7 lysine mimics Ni^{2+} in the natural TCR ligand and that MHCII β chain flexibility in the area around the peptide p7 position forms a common site for cation binding in metal allergies.

99
Innate lymphoid cells (ILC) in allergic contact dermatitis

van de Poel, S[1,2]; Kunz, S[1]; Martin, SF[1]; Jakob, T[1]
[1]Department of Dermatology, Allergy Research Group, Medical Center – University of Freiburg, Freiburg, Germany; [2]Faculty of Medicine and Biology, University of Freiburg, Freiburg, Germany

Innate lymphoid cells (ILC) are a recently identified family of heterogeneous innate immune cells belonging to the lymphoid lineage. In analogy to T helper cell subsets, ILC can be classified into three groups based on the developmental dependency on transcription factors and expression of cytokines. Prototypical ILC are NK cells (ILC1), natural helper cells (ILC2) and lymphoid tissue-inducer (ILC3) cells. ILC are known to play a role in lymphoid organogenesis, tissue remodeling and inflammation including allergic asthma and reside especially in the intestine and the lung. Recent studies showed that ILC are also present in skin and mediate pathology in a mouse model of atopic dermatitis as well as in psoriatic plaque formation.

Here we analyzed the involvement of ILC in allergic contact dermatitis using the mouse model of TNCB-induced contact hypersensitivity. Using flow cytometry, we identified all three groups of ILC in the skin of naïve BALB/c mice. Quantification of these cells using counting beads revealed a frequency of approximately 1150 ILC2, 350 ILC3, and 700 NK cells per 50 mg ear skin. During the elicitation phase of contact hypersensitivity we observed a distinct increase of NK cells in the inflamed skin, their number peaking at around 24 h after challenge. The frequency of ILC2 cells in the skin showed a slight increase 24 h after challenge, whereas no alterations in the frequency of ILC3 cells could be observed. Analysis of the activation profile using ICOS as an activation marker revealed a higher activation of ILC2 cells in mice with contact hypersensitivity compared to control groups. Upon primary contact with the allergen or an irritant, we observed no difference in the frequency of ILC2 cells, whereas ILC3 and NK cell increased at later time points.

In conclusion, our data indicate that numbers and activation profile of dermal ILC are influenced by contact allergens and thus, ILC might be functionally involved in allergic contact dermatitis.

100
Detection of contact allergy to palladium: sodium tetrachloropalladate is better than palladium chloride

Spiewak, R[1,2]; Samochocki, Z[3]; Pasnicki, M[4]; Czarnobilska, E[5]; Grubska-Suchanek, E[6]; Bukiel, M[7]; Jedrzejewska-Jurga, K[8]; Czarnecka-Operacz, M[9]; Cisowska, A[10]; Niklasson, B[11]
[1]Department of Experimental Dermatology and Cosmetology, Jagiellonian University, Krakow, Poland; [2]Research Department, Institute of Dermatology, Krakow, Poland; [3]Dermatology Clinic, Medical University of Warsaw, Warsaw, Poland; [4]Alergicus-Dent, Zary, Poland; [5]Department of Clinical and Environmental Allergology, Jagiellonian University Medical College, Krakow, Poland; [6]Dermatology Clinic, Medical University of Gdansk, Gdansk, Poland; [7]NZOZ Alergologia-Pulmonologia, Stargard Szczecinski, Poland; [8]Lower-Silesian Centre for Occupational Medicine, Wroclaw, Poland; [9]Dermatology Clinic, Medical University of Poznan, Poznan, Poland; [10]Dermatology Practice, Kamienna Gora, Poland; [11]Chemotechnique Diagnostics, Vellinge, Sweden

Background: Contact allergy to palladium emerges as a relevant concern for public health due to the increasing environmental exposure (nickel-free jewellery gold alloys, electronic devices including mobile phones, computers and LED television sets, palladium release from car exhaust converters). Until now, patch test with palladium chloride was the common way of diagnosing palladium allergy, however, newer data suggest that sensitivity of this test may be too low. With this regard, sodium tetrachloropalladate seems a better option. This study was aimed at comparing results of patch tests to palladium chloride and sodium tetrachloropalladate in a large population of eczema patients.

Method: In phase 2 of the KRAK study, patients with chronic/recurrent eczema were patch tested to both palladium(II) chloride 2% pet. (Chemotechnique Diagnostics; cat.-No. P-001) and sodium tetrachloropalladate(II) hydrate 3% pet. (S-017). The test substances were applied on the patient's back for 2 days in IQ Ultra chambers (Chemotechnique), with subsequent readings on days 3, 5 and 8.

Results: 1026 patients were tested: 730 females and 296 males, aged 1–90 (median 40) years. Positive reaction to at least one of the compared substances was recorded in 223 patients, including 81 who reacted to both formulations, 123 who reacted to S-017 only, and 19 who reacted to P-001 only (chi2: $P < 0.001$). The overall detection rate was 19.9% for S-017 (204 positive reactions, including 50 rated as clinically relevant, and 48 as cross reactions), and 9.7% for P-001 (100 positive reactions, including 24 clinically relevant and 30 cross reactions). Testing to P-001 only would have missed 29 clinically relevant reactions detected with S-017 (2.8% of all patients), while testing to S-017 only would have missed four relevant reactions to P-001 (0.4%).

Conclusion: Sodium tetrachloropalladate (II) hydrate 3% pet. is more effective than palladium chloride 2% pet. in the detection of contact allergy to palladium.