

mation in subjects with FA. However, information on normal faecal ECP values in paediatric subjects and on the efficacy of new commercial ELISA Kits to measure faecal ECP concentration are scanty.

Aims. To identify gut inflammation during CFC through measurement of faecal ECP levels in children with FA.

Methods. Patients consecutively referred for suspected FA related gastrointestinal symptoms were enrolled into the study. Healthy controls matched for age and weight were enrolled for normal data definition. Stool samples collected during office visit were stored, prepared and analysed by an ELISA assay for ECP (ECP ELISA kit, Dasit, Milan, Italy). Consecutive FA affected children receiving CFC at our Department during the last 3 month were enrolled. Faecal ECP levels were measured in pre-challenge samples, collected after 4 weeks of elimination diet period, and in post-challenge samples obtained within 24 h from oral antigen exposure.

Results. Normative data in 89 healthy children (mean age 15.29 ± 11.85 months) were defined (mean 0.88 ± 0.9 $\mu\text{g/g}$ stool). Pre-challenge ECP levels (mean 0.7 ± 0.7 $\mu\text{g/g}$ stool) were determined in 25 enrolled patients (mean age 16.2 ± 11.9 m). ECP values increased in patient with positive-CFC (n: 10, mean 1.40 ± 0.2 $\mu\text{g/g}$ stool) ($p < 0.001$). Faecal ECP values remained stable in patients with negative CFC. Faecal ECP levels resulted not different in IgE- and non-IgE FA affected patients.

Conclusions. The determination of faecal ECP through new ELISA assay is a simple method to easily detect gut inflammation in children with FA. Faecal ECP measurement could be useful for the interpretation of CFC result.

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PROPOSAL FOR A STANDARDIZED INTERPRETATION OF THE ATOPY PATCH TEST IN CHILDREN WITH GASTROINTESTINAL SYMPTOMS AND SUSPECTED FOOD ALLERGY

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Background. We have recently demonstrated that the atopy patch test (APT) is an useful diagnostic tool in children with gastrointestinal symptoms and suspected food allergy (FA) (*Allergy* 2007;62:738–743). The interpretation of the APT to foods is not yet completely standardized, it is still subjective and prone to intra- and inter-observer variations.

Aims. We aimed to test the diagnostic accuracy of APT in the diagnosis of FA-related gastrointestinal diseases and to validate the reading of the APT in terms of the diagnostic accuracy of individual skin signs.

Participants and methods. Children referred for suspected FA-related gastrointestinal symptoms were subjected to APT using fresh food (cow's milk, hen's egg, wheat). Twelve-millimetre Finn chambers were applied for 48 h, and results were read after 48 and 72 h. Skin changes were graded for erythema, induration and papule formation. Food allergy was assessed by double blind, placebo-controlled food challenges (DBPCFC). Sensitivity, specificity and predictive values were calculated for each skin signs in relation to challenge outcome.

Results. One hundred sixty-six DBPCFC were performed in 119 patients (77 boys, median age 26.7 months, range 3–48 months): 66 tested positive for cow's milk (CM), 29 for hen's egg (HE), and 7 for wheat. The results of APT with fresh food were (1) CM: sensitivity: 65.7%, specificity 78.7%, positive predictive value (PPV) 92.0% and negative predictive value (NPV) 60.6%; (2) HE: sensitivity 75.8%, specificity 100%, PPV 100% and NPV 63.1%. The combination of skin induration plus presence of papules, with or without moderate erythema, had a PPV and specificity for the challenge outcome of 100%, both in CM and in HE allergy.

Conclusions. The diagnostic properties of APT improved significantly if skin induration and presence of papules were used in combination. These skin signs were highly predictive of challenge outcome. Other skin signs, i.e. erythema, were found to be less accurate, particularly if used in isolation. Careful and standardized documentation of the severity of single APT skin signs may improve the reproducibility of APT interpretation and reduce intra- and inter observer error.

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GLIADIN PEPTIDE P31-43 PRESENTS IL15 IN TRANS TO THE NEIGHBOURING CELLS

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Background and aim. We have previously observed that gliadin peptide P31-43 can induce proliferation of NIH3T3 cells and of enterocytes of untreated celiac mucosa due to an interference of this peptide with the degradation of the ligand loaded EGF receptor [1]. We have also shown that in presence of P31-43 the EGF receptor stays longer activated as a consequence of a delay of the endocytic maturation. Aim of our study was to investigate whether proliferative effects induced by gliadin peptide P31-43 could be mediated also by activation of innate immunity. In particular we tested P31-43 effects on IL15 induction at level of transcription, translation, intracellular trafficking and role in P31-43 induced proliferation of both Caco2 cells and celiac enterocytes.