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Impact of innate immunity in a subset of children with autism spectrum disorders: a case control study

Harumi Jyonouchi*[1], Lee Geng[1], Agnes Cushing-Ruby[1] and Huma Quraishi[2]

Address: [1]Division of Allergy/Immunology and Infectious Diseases, Department of Pediatrics, University of Medicine and Dentistry of New Jersey (UMDNJ)-New Jersey Medical School (NJMS), Newark, NJ, USA and [2]Department of Surgery, University of Medicine and Dentistry of New Jersey (UMDNJ)-New Jersey Medical School (NJMS), Newark, NJ, USA

Email: Harumi Jyonouchi* - jyanouha@umdnj.edu; Lee Geng - gengle@umdnj.edu; Agnes Cushing-Ruby - cushinag@umdnj.edu; Huma Quraishi - quraisha@umdnj.edu

* Corresponding author

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Abstract

Background: Among patients with autism spectrum disorders (ASD) evaluated in our clinic, there appears to be a subset that can be clinically distinguished from other ASD children because of frequent infections (usually viral) accompanied by worsening behavioural symptoms and/or loss/decrease in acquired skills. This study assessed whether these clinical features of this ASD subset are associated with atopy, asthma, food allergy (FA), primary immunodeficiency (PID), or innate immune responses important in viral infections.

Methods: This study included the ASD children described above (ASD test, N = 26) and the following controls: ASD controls (N = 107), non-ASD controls with FA (N = 24), non-ASD controls with chronic rhinosinusitis/recurrent otitis media (CRS/ROM; N = 38), and normal controls (N = 43). We assessed prevalence of atopy, asthma, FA, CRS/ROM, and PID. Innate immune responses were assessed by measuring production of proinflammatory and counter-regulatory cytokines by peripheral blood mononuclear cells (PBMCs) in response to agonists of Toll-like receptors (TLRs), with or without pre-treatment of lipopolysaccharide (LPS), a TLR4 agonist.

Results: Non-IgE mediated FA was equally prevalent in both ASD test and ASD control groups, occurring at higher frequency than in the non-ASD controls. Allergic rhinitis, atopic/non-atopic asthma, and atopic dermatitis were equally prevalent among the study groups except for the CRS/ROM group in which non-atopic asthma was more prevalent (52.6%). CRS/ROM and specific polysaccharide antibody deficiency (SPAD) were more prevalent in the ASD test group than in the ASD control, FA, and normal control groups: 23.1% vs. < 5% for CRS/ROS and 19.2% vs. < 1% for SPAD. However, CRS/ROM patients had the highest prevalence of SPAD (34.2%). When compared to ASD and normal case controls, PBMCs from 19 non-SPAD, ASD test group children produced: 1) less IL-1β with a TLR7/8 agonist, less IL-10 with a TLR2/6 agonist, and more IL-23 with a TLR4 agonist without LPS pre-treatment, and 2) less IL-1β with TLR4/7/8 agonists with LPS pre-treatment. These are cytokines associated with the neuro-immune network.

Conclusion: Clinical features of the ASD test group were not associated with atopy, asthma, FA, or PID in our study but may be associated with altered TLR responses mediating neuro-immune interactions.

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