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Serum anti-nucleosome-specific antibody as a marker of autoimmunity in children with autism spectrum disorders (ASD)

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Background: Increasing evidence of autoimmune phenomena in some individuals with autism could represent the presence of altered or inappropriate immune responses in this disorder. The role of the nucleosome in the induction of antibody response in some autoimmune-mediated tissue damage may provide novel targets for treatment. Due to the paucity of studies investigating the frequency of systemic auto-antibodies in autism, we are the first to investigate the frequency of antinucleosome-specific antibodies in a group of autistic children.

Methods: Serum antinucleosome-specific antibodies were measured by ELISA in 60 autistic children, between the ages of 3 and 12 years, in comparison to 60 healthy children. Autistic severity was assessed using the Childhood Autism Rating Scale (CARS).

Results: Autistic children had significantly higher serum antinucleosome-specific antibodies than healthy children ($P < 0.001$). The seropositivity of antinucleosome-specific antibodies was found in 46.7% of autistic children. Autistic children with a family history of autoimmunity (40%) had a significantly higher frequency of serum antinucleosome-specific antibodies (83.3%) than patients without such a history (22.2%, $P < 0.001$).

Conclusions: Serum levels of antinucleosome-specific antibodies were increased in some autistic children. However, these data should be treated with caution until further investigations are performed with a larger subject population, to determine whether these antibodies have a role in the induction of autoimmunity in a subgroup of autistic children. The role of immunotherapy in children with autism should be also studied.

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The possible relationship between allergic manifestations and elevated serum levels of brain specific auto-antibodies in autistic children

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Noroviruses are positive-sense, single-stranded RNA viruses and are a major cause of gastroenteritis worldwide. Norovirus capsid protein can be subdivided into a shell (S) domain, hinge (H) region, and protruding (P) domain. The P domain contains the most genetically variable region on the capsid and binding site for histo-blood group antigens (HBGAs). Despite their discovery over 40 years ago, there are still no vaccines or antivirals. In this project we characterized binding of several VHHs (nanobodies) to human norovirus capsid. One nanobody, Nano-85, was broadly reactive, while the others, Nano-25 and Nano-27, were strain specific. All nanobodies bound to the lower region on the P domain and had nanomolar affinities. The Nano-85 binding site mainly comprised highly conserved amino acids among the genetically distinct genogroup II noroviruses. Several of the conserved residues also were recognized by a broadly reactive monoclonal antibody, which suggested this region contained a dominant epitope. Superposition of the P domain nanobody complex structures into a cryoelectron microscopy particle structure revealed that both nanobodies bound at occluded sites on the particles. The flexible hinge region likely permitted a certain degree of P domain movement on the particles in order to accommodate the nanobodies. Interestingly, the Nano-85 binding interaction with intact particles caused the particle disassembly in vitro. Altogether, these results suggested that the highly conserved Nano-85 binding epitope contained a trigger mechanism for particle disassembly.

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