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Immune-mediated differentiation of autism subtypes: evidence from pediatric Neurocytokine profiling in central Asia

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Abstract: This study investigates immunological distinctions between clinical subtypes of autism spectrum disorder (ASD), focusing on pediatric populations diagnosed with Kanner's and Asperger's syndromes. Utilizing quantitative analysis of serum cytokines—IL-6, TNF-α, IL-10, and IL-1β—we evaluated immune deviations in relation to behavioral severity and neurocognitive function. The data revealed subtype-specific inflammatory patterns, with heightened IL-6 and TNF-α in Kanner's syndrome and selective IL-1β elevation in Asperger's syndrome. A deficiency in IL-10 was common across both groups, suggesting impaired anti-inflammatory modulation. These results underscore the role of immune biomarkers in refining ASD subtypes and tailoring early interventions.

Keywords: Autism spectrum disorder, Kanner syndrome, Asperger syndrome, IL-6, TNF-α, IL-10, IL-1β, pediatric neuroinflammation.

Introduction: Autism spectrum disorder (ASD) encompasses a heterogeneous group of neurodevelopmental conditions, clinically marked by deficits in communication, social interaction, and behavioral flexibility. Among its clinical phenotypes, Kanner's and Asperger's syndromes represent distinct constellations of symptoms that may reflect underlying biological divergence.

Emerging research has implicated immune system dysfunction, particularly cytokine imbalance, in ASD pathophysiology [1, 2]. Elevated pro-inflammatory cytokines (e.g., IL-6, TNF-α) and reduced anti-

inflammatory agents (e.g., IL-10) are recurrent findings in ASD studies, suggesting neuroimmune involvement in cognitive and behavioral anomalies [3–5]. Moreover, IL-1β has been shown to modulate synaptic plasticity and neuronal excitability—factors relevant to ASD-specific behaviors [6].

However, the regional specificity of these immune patterns remains poorly characterized, particularly in Central Asia. This study examines these immune markers in Uzbek children, aiming to clarify whether distinct inflammatory signatures align with clinical subtypes of ASD.

Purpose of the Research

The purpose of this study was to: Identify differential serum levels of IL-6, IL-10, IL-1β, and TNF-α in children with Kanner's and Asperger's syndromes; Correlate cytokine profiles with behavioral scales (CARS, AQ-Child) and cognitive performance; Explore the prognostic utility of immune markers in guiding subtype-specific therapeutic strategies; Highlight regional cytokine trends in a Central Asian pediatric cohort for translational applicability.

METHODS

Participants: 180 children aged 3–11 were recruited and categorized into three groups: 70 with Kanner's syndrome, 70 with Asperger's syndrome, and 40 neurotypical controls. ASD diagnosis was confirmed using DSM-5 criteria, ADOS-2, and CARS evaluations conducted by pediatric neurologists and child psychologists.

Cytokine Assays: Morning fasting venous blood samples were collected under aseptic conditions. Serum levels of IL-6, TNF-α, IL-10, and IL-1β were determined using ELISA kits (BioLegend, USA) with sensitivity thresholds suitable for pediatric immune studies.

Behavioral and Cognitive Evaluation: Autism Spectrum Quotient – Child Version (AQ-Child); Childhood Autism Rating Scale (CARS); Leiter International Performance Scale – Third Edition (Leiter-3).

Statistical Analysis: Data were analyzed using SPSS v27. ANOVA was used for intergroup comparison. Correlations between cytokines and behavioral outcomes were tested using Pearson's coefficient. Logistic regression was used to model predictive relationships between cytokine profiles and ASD subtypes. (Table 1)

RESULTS

Table 1

shows the cytokine concentrations (pg/mL) across all study groups.

Group	IL-6 (pg/mL)	TNF-α (pg/mL)	IL-10 (pg/mL)	IL-1β (pg/mL)
Kanner's (n=70)	19.7 ± 2.3*	25.8 ± 3.0*	4.1 ± 1.6*	5.1 ± 1.2
Asperger's (n=70)	13.4 ± 1.9	16.5 ± 2.5	5.7 ± 1.9*	7.5 ± 1.3*
Controls (n=40)	6.1 ± 1.0	8.6 ± 1.2	9.8 ± 2.4	4.0 ± 0.9

*Statistically significant compared to controls ($p<0.001$)

Strong positive correlations were found between IL-6 and CARS scores ($r = 0.65$), indicating greater inflammatory load in children with severe symptoms [7]. An inverse relationship was noted between IL-10 and repetitive behavior scores ($r = –0.48$, $p<0.01$) [8].

Regression analysis showed IL-6 and TNF-α as independent predictors of Kanner syndrome ($R^2 = 0.44$, $p<0.001$), whereas IL-1β was predictive of Asperger's subtype ($p<0.05$). Boys showed higher cytokine levels across all categories [9].

DISCUSSION

This study corroborates the role of immune dysregulation in ASD and highlights subtype-specific cytokine patterns. IL-6 and TNF-α elevations in Kanner syndrome reflect systemic inflammation potentially driven by microglial activation, consistent with imaging studies showing increased glial markers in children with classic autism [10].

Conversely, elevated IL-1β in Asperger syndrome aligns with enhanced neuroexcitation and local cortical hyperconnectivity described in this subtype. IL-1β is known to influence glutamate transmission, which may underpin the high-functioning yet rigid behavioral features of Asperger's presentations [11].

The deficiency in IL-10 across ASD subtypes indicates compromised anti-inflammatory regulation, which may perpetuate chronic immune activation. Such imbalances support a shift toward immunopsychiatric models of ASD, where biomarkers inform diagnostic, prognostic,

and therapeutic decisions [12].

Importantly, the geographic context of Uzbekistan introduces a new perspective, supporting the global relevance of cytokine-based ASD stratification. Local environmental and genetic factors may modulate cytokine expression, necessitating region-specific standards [13].

CONCLUSION

Distinct immune patterns in Kanner and Asperger syndromes validate cytokine profiling as a tool for clinical differentiation and targeted management. Elevated IL-6 and TNF-α in Kanner's suggest intense neuroinflammation, while IL-1β dominance in Asperger's implicates alternate immunological pathways. A shared IL-10 deficiency underscores the need for anti-inflammatory therapeutic adjuncts. Future strategies in ASD care should integrate immune diagnostics to personalize interventions, particularly in early developmental windows. Expanding cytokine panels and combining immunological data with genetic and neuroimaging findings will enhance subtype precision.

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