Via Immune Pathways Specific Foods Linked to Autism Risk

A group of researchers determined whether specific dietary patterns are associated with autism spectrum disorder (ASD) risk in ways consistent with potential causal pathways, particularly through genetic and immune-related mechanisms.

Study

A two-sample Mendelian randomization design was used to explore potential causal relationships between diet-related exposures and ASD. This approach uses genetic variants as proxies for long-term dietary tendencies, reducing bias from confounding and reverse causation. Genome-wide association study data were examined for 199 dietary factors, two ketone bodies, and food allergy. ASD served as the outcome.

Single-nucleotide polymorphisms were selected using predefined statistical thresholds and filtered for independence through linkage disequilibrium clumping. Instrument strength was confirmed using F-statistics. Inverse variance weighting was the primary analytical method, supported by weighted median estimation, Mendelian randomization-Egger regression, and Mendelian randomization pleiotropy residual sum and outlier testing. Multiple sensitivity analyses assessed heterogeneity and horizontal pleiotropy.

To better understand potential biological pathways, inflammatory proteins, immune cell phenotypes, gut microbiota, and antibody responses were analyzed. Researchers also conducted a retrospective, non-randomized clinical analysis of 78 children between ages 2 and 7 years, all of whom had a diagnosis of ASD based on the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition.

Participants followed either a gluten-free casein-free diet or a standard diet, with outcomes measured using the Autism Diagnostic Observation Schedule, Second Edition, and the Childhood Autism Rating Scale, alongside food-specific immunoglobulin G levels.

Findings

Genetic analyses revealed that not all foods carry equal relevance for ASD risk. Among the many dietary factors examined, wholemeal pasta intake and cheese spread intake stood out. Genetic analyses suggested that a higher genetically predicted intake of wholemeal pasta, a gluten-

containing food, was associated with a significantly increased risk of ASD. A similar pattern was observed for cheese spread, a concentrated source of casein.

These findings do not suggest that a child's occasional meal causes autism. Instead, they reflect long-term, genetically influenced dietary patterns across a lifetime. However, the estimates were imprecise, with wide confidence intervals. The authors therefore interpreted the results as evidence of association consistent with potential causal effects, rather than definitive proof. Sensitivity analyses consistently supported the direction of the associations, but not their precision.

In contrast, banana intake showed a protective association. People with a genetic tendency to eat more bananas were less likely to have autism. While bananas are nutritionally dense and rich in fiber and bioactive compounds, this finding should be interpreted cautiously and is best viewed as a signal rather than proof of protection. The authors note that banana intake may also act as a proxy for broader dietary patterns or overall diet quality, rather than a food-specific effect.

Ketone bodies (3-hydroxybutyrate and acetoacetate) did not have a statistically significant link to ASD, despite showing potential protective trends. Similarly, food allergy showed a non-significant trend toward increased ASD risk.

Mediation analyses provided insight into how dietary factors may influence the relationship. Cheese spread intake appeared to affect autism risk partly by altering immune function. Specifically, reduced expression of Cluster of Differentiation 45 (CD45) on human leukocyte antigen-DR-positive T cells. This, along with changes in anti-Epstein-Barr virus immunoglobulin G seropositivity, explained a meaningful proportion of the association. The authors note that these immune features may normally play inhibitory or protective roles.

Clinical findings partially aligned with the genetic evidence. Gluten- and casein-free diets did not significantly improve autism symptom scores compared to a regular diet, and group differences were not statistically significant. However, they did show greater numerical improvements over time.

Notably, the diet resulted in significant reductions in milk- and wheat-specific immunoglobulin G levels, with pronounced interactions between the diet and time. In practical terms, immune-related markers responded more clearly than behavioral measures, which the authors caution may reflect the limited sample size and observational design.

Conclusion

Together, these findings suggest that specific gluten- and casein-rich foods, specifically wholemeal pasta and cheese spread, may be associated with ASD risk through immune-related mechanisms. While diet changes are unlikely to prevent or cure autism, they may modulate biological pathways related to immune function.

This is important because diet is one of the few areas where families can actively make changes. Still, dietary strategies should be viewed as supportive, not curative, and integrated alongside established therapies.

Given the observational and genetic nature of the evidence, the restriction of genetic data largely to individuals of European ancestry, and sample-size limitations, larger, long-term studies across diverse populations are essential before firm recommendations can be made.

Source:

https://www.news-medical.net/news/20260114/Specific-foods-linked-to-autism-risk-via-immune-pathways.aspx