Title

Gene Expression Analysis of Influenza Infection and Its Association with Autism-related Genes

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Abstract

Influenza infection during pregnancy has been widely studied due to its potential impact on fetal neurodevelopment. Epidemiological studies report no increased risk of autism spectrum disorder (ASD) associated with either maternal influenza infection or vaccination; however, these analyses are based on population-level data and do not investigate underlying molecular mechanisms. Influenza infection induces strong immune responses and widespread transcriptional changes, while autism has been associated with immune dysregulation and altered neurodevelopmental pathways. The molecular relationship between influenza-induced gene expression changes and autism-associated genes remains unclear.

In this study, a bioinformatics-based transcriptomic analysis was performed to evaluate whether genes differentially expressed during influenza infection and vaccination overlap with autism-associated genes. Publicly available datasets, including GSE17156 (infection) and GSE59654 (vaccination), were analyzed using the limma package in R with thresholds of adjusted false discovery rate (FDR < 0.05) and fold-change cutoffs. Autism-associated genes were obtained from the SFARI Gene database, and overlap analysis, gene ontology enrichment, and visualization approaches were applied.

Results indicate that influenza infection produces widespread transcriptional changes and shows substantial overlap with autism-associated genes, particularly in pathways related to neurodevelopment. In contrast, influenza vaccination induces fewer and smaller gene expression changes with minimal overlap. Comparative analysis suggests that infection and vaccination affect largely distinct gene sets with weak correlation between their expression patterns.

Overall, these findings provide a molecular perspective that is consistent with epidemiological evidence showing no increased ASD risk, as the observed gene expression changes are limited, weakly correlated, and not coordinated across conditions. This study is hypothesis-generating and does not imply causation but may inform future investigations into immune-related molecular pathways relevant to neurodevelopment.