Faecalibacterium and *Blautia*, respectively. Moreover, DNA microarray analysis of peripheral blood mononuclear cells (PBMC) detected more highly than low expressed genes in ASD infants than in healthy infants. Gene Ontology analysis revealed that differentially expressed genes between ASD and healthy infants were involved in interferon (IFN)-γ and type-I IFN signaling pathways. Finally, strong positive correlations between expression of IFN signaling-associated genes in PBMC and fecal abundance of *Faecalibacterium* were found. Our results strongly suggested that altered gut microbiota in infants resulted from ASD development and was associated with systemic immunity dysregulation, especially chronic inflammation.

Altered gut microbiota in autistic infants play a role in chronic inflammation induced by interferon signaling that may cause further inflammation in central nervous system.