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**Association of breast milk-derived arachidonic acid- induced infant gut dysbiosis with the onset of atopic dermatitis**

Jiang S, Cai M, Li D*, et al*. Association of breast milk-derived arachidonic acid-induced infant gut dysbiosis with the onset of atopic dermatitis. *Gut*2025; 74: 45-57. doi: 10.1136/gutjnl-2024-332407

Infancy is an important period for colonisation of the gut microbiota with the sequalae of host immune development. Evidence is surmounting that the establishment of the gut microbiome in early life influences the development of atopic dermatitis (AD), a chronic, pruritic, inflammatory skin disease, which affects 15% of children. Breastfeeding can affect the composition and diversity of gut microbiota in infancy. In their longitudinal study, Jiang *et al.,* elucidated that specific breast milk-derived metabolites contribute to the onset of AD.

The observation period spanned from birth to 6 months. 250 mother-infant pairs were enrolled. Maternal and infant faecal samples were collected for metagenomics, with concurrent, breast milk samples analysed for metabolomics. Animal and cellular studies were conducted alongside for validation.

Seventy-eight infants (31.2%) were diagnosed with AD. Metabolomic analysis identified 78 differential metabolites. Anti-inflammatory, anti-allergic substrates such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) were lower in the AD group. By contrast, arachidonic acid (AA), which is metabolised into pro-inflammatory substrates were higher in concentration in the AD group. Genomic analysis identified Escherichia and Bifidobacterium as the major genera of active bacteria. *E.coli*, a gram-negative bacterium, was more prevalent than Bifidobacterium species, and its abundance increased in the AD group with high concentrations of AA, culminating in gut microbial dysbiosis. In mice models stimulated with allergen-induced dermatitis, AA increased the area of skin lesions compared to EPA.

In this cohort study, Jiang *et al.,* have postulated the potential relationship between breast milk-derived AA and the development of infant AD.