ID der Kurzfassung : **119**

Microbiota dysbiosis in aging weakens gut–lung axis defense against influenza A virus

Inhalt

Aging alters the intestinal microbiota, reducing diversity, metabolic capacity, and antiviral defense along the gut–lung axis. This study investigated whether aging-associated microbial dysbiosis affects susceptibility to influenza A virus (IAV) and whether short-chain fatty acid (SCFA) supplementation can restore antiviral responses during cellular senescence.

Using young and aged mice, we analyzed microbiota composition and metabolic function before and after IAV infection. Shotgun metagenomics, RNAseq, flow cytometry, and plaque assays were used to assess microbial, transcriptomic, cytokine, and viral changes. *In vitro*, senescent primary human lung fibroblasts were infected with IAV and treated with acetate.

Aging caused microbial shifts, including reduced *Akkermansia muciniphila* and *Faecalibaculum rodentium*, reduced carbohydrate-fermenting enzymes, and impaired SCFA synthesis. IAV infection further worsened these changes. Senescent lung fibroblasts were more susceptible to IAV infection, while acetate reduced viral replication and inflammatory cytokine expression via GPR41/43 activation and histone acetylation.

Our findings show that aging-associated microbial dysbiosis weakens antiviral defense, whereas acetate restores responses in senescent cells.

Keywords

Registration ID

INF26- 67

Professional status of the speaker

Postdoc

Junior scientist status

No, I am not a junior scientist.

Track Klassifizierung: Viral Pathogenesis

Typ des Beitrags: Oral presentation