

## Airway-gut Axis: Persistent Allergic Responses in Mouse Airway Affects Intestinal Microbial Ecosystems

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출처 (Source)	<a href="#">한국미생물학회 학술대회논문집</a> , 2019.4, 120-120(1 pages)
발행처 (Publisher)	<a href="#">한국미생물학회</a> The Microbiological Society of Korea
URL	<a href="http://www.dbpia.co.kr/journal/articleDetail?nodeId=NODE08756797">http://www.dbpia.co.kr/journal/articleDetail?nodeId=NODE08756797</a>
APA Style	Chan Min Jung, Sang Sun Yoon (2019). Airway-gut Axis: Persistent Allergic Responses in Mouse Airway Affects Intestinal Microbial Ecosystems. 한국미생물학회 학술대회논문집, 120-120
이용정보 (Accessed)	이화여자대학교 203.255.***.68 2020/05/18 04:01 (KST)

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D035

### ***Drosophila melanogaster*-based Identification of the *Pseudomonas aeruginosa* Genes for Polymicrobial Interaction with *Staphylococcus aureus***

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Microorganisms are widespread in natural habitats and human tissues where they coexist and constitute polymicrobial communities. It remains unclear how such polymicrobial interactions are orchestrated to survive the dynamic host environments. Here, we established a *Drosophila melanogaster*-based infection model to assess the key aspects of polymicrobial interaction between the two important opportunistic pathogens, *Pseudomonas aeruginosa* and *Staphylococcus aureus* that commonly coexist in the mucosal layers. This system revealed that the polymicrobial infection enhanced the virulence of *P. aeruginosa*, not that of *S. aureus*. This virulence enhancement was still evident in the interaction between the *P. aeruginosa lasRmvfR* mutant and a virulence-attenuated *S. aureus* mutant (*m6*), suggesting that the virulence enhancement did not require the LasR-MvfR quorum-sensing circuitry. To identify the genes required for the virulence enhancement in *P. aeruginosa* by *S. aureus*, we have screened ~1,000 transposon clones of the *lasRmvfR* mutant and isolated three mutants (11E3, 11G10, and 16E12), whose virulence was not enhanced at all by *m6* in the *Drosophila* polymicrobial infection. Their transposon insertion sites were determined: a hypothetical gene (PA14\_24300) under the control of MvfR, *dgt* for the dGTPase, and *fleQ* for the regulator of flagellar synthesis. These results suggest unexpected interspecies interactions during the polymicrobial infection caused by *P. aeruginosa* and *S. aureus*.

D036

### **Airway-gut Axis: Persistent Allergic Responses in Mouse Airway Affects Intestinal Microbial Ecosystems**

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Asthma and allergic airway diseases are common in Westernized country and rapidly increasing their patient population in developing country. A hallmark of allergic airway disease is airway hyperresponsiveness, which is characterized by eosinophilic inflammation and airway remodeling. The host-microbiome interactions have been considered to modulate host immune mechanisms, especially in the mucosal surfaces. Microbiome analysis by next-generation sequencing allows us to gain more information between asthma and commensal microbes. Airway, once believed to be the sterile site, have an estimated number of 10-100 bacteria per 1,000 human cells. However there is little known about the correlation between allergic airway diseases and mucosal microbiome. Herein, we asked whether persistently applied allergic stimulations to the respiratory system would affect intestinal microbial ecosystems. In mouse model, HDM was treated intranasally for 8 consecutive weeks and samples were collected from cecum, feces, lung and airway secretion of each mice. We focused on microbiome change from chronic asthma mouse models induced by house dust mite(HDM), using 16S rRNA gene sequencing. There was an increase of Proteobacteria ratio in airway secretion, however, no significant change was seen in fecal samples. Addressing this question would help us provide clues to understand how an airway-gut axis operates.

[Supported by a grant from the National Research Foundation (NRF-2017M3A9F3041233).]