[BIOINFO 2024]

Differences In Gastric Neoplasms Induced By *Helicobacter* and Non-*Helicobacter* Microbiomes

Youngjin Shin[1], Donghoon Kang[2], Yeun-Jun Chung[1,3*] and Seung-Hyun Jung[3,4,5*]

[1] *Basic Medical Science Facilitation Program, Catholic Medical Center*
[2] *Catholic Photomedicine Research Institute, Seoul St.Mary's Hospital, The Catholic university of Korea*
[3] *Integrated Research Center for Genomic Ploymorphism, College of Medicine, The Catholic university of Korea*
[4] *Department of Biochemistry, College of Medicine, The Catholic university of Korea*
[5] *Department of Biomedicine & Health Sciences, College of Medicine, The Catholic university of Korea*
**Corresponding author: yejun@cathlic.ac.kr, hyun@catholic.ac.kr*

Gastric neoplasms are linked to various microbial agents, including *Helicobacter* and non-*Helicobacter* genera *like Streptococcus, Neisseria, Haemophilus, Rothia*, and *Gemella*. While the role of H. pylori in gastric cancer is well-established, the contribution of other genera and the relationship between *Helicobacter* and non-*Helicobacter* bacteria in gastric tumorigenesis remains underexplored. Gastric tissue samples underwent 16S ribosomal DNA sequencing to identify bacterial genera, followed by functional profiling to determine pathways associated with each group. Correlation analysis assessed the coexistence of *Helicobacter* and non-*Helicobacter* genera, revealing distinct roles. While *Helicobacter* was predominantly found in carcinoma samples, other genera like *Streptococcus, Neisseria*, and *Gemella* were also prevalent. These genera were divided into *Helicobacter* and non-*Helicobacter* groups, each contributing independently to neoplasm development. Correlation analysis confirmed that these groups did not significantly coexist. Further, *Helicobacter* did not correlate with gastric atrophy, but *Streptococcus* was more common in samples with higher atrophy, suggesting a preference for such environments. Functional analysis of the non-*Helicobacter* group showed involvement of the PTS, MAPK and PPAR pathways, indicating that carbohydrate metabolism supports energy production, and both MAPK and PPAR activation may drive inflammation. This study emphasizes the distinct roles of *Helicobacter* and non-*Helicobacter* bacteria in gastric cancer progression, suggesting they promote neoplasm development through separate mechanisms. The findings highlight how non-*Helicobacter* genera, particularly *Streptococcus*, contribute to gastric atrophy and utilize pathways driving inflammation, stressing the need for genus-specific strategies for gastric cancer prevention and treatment.