Commentary

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Smoking, Inflammation and Gut Microbiota

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Description

Always population-based study the effect of smoking on intestinal flora, found that smokers have diversity than non-smokers, an increase in the abundance of pathogenic or opportunistic pathogens and a decrease in the abundance of beneficial bacteria. However, the research for population screening is not rigorous; there may be bias of disease and living habit, etc. In our study, people with chronic diseases and sub-health were excluded, and personal lifestyle information was collected to explore the effects of smoking on intestinal microflora in healthy people, and to eliminate the bias in exercise and diet.

By comparing the diversity between the two groups, this study found that the diversity of smokers was higher than that of non-smokers, which is contrary to previous studies. We hypothesized that the previous decrease in diversity may be related to chronic diseases in the enrolled population. Studies have shown that chronic diseases (diabetes, hypertension, etc.) can reduce the diversity of intestinal flora, the increase of diversity may be caused by the increase of the abundance of pathogenic bacteria or opportunistic pathogens. Using metagenomic sequencing, we found that the changes at the species level in the smoking group were concentrated in a few key functional bacteria, *Ruminococcus gnavus, Bacteroides vulgatus, Faecalibacterium prausnitzii,* and *Akkermansia muciniphila*, which were identified as being associated with the severity of inflammation. *Ruminococcus gnavus* contains specific genes that encode superantigens to induce and bind IgA antibody *in vivo*. Bacteroides vulgatus produces LPS, which activates T cells and promotes immune cells to produce cytokines such as IL-6, IL-8, and TNF-α. Faecalibacterium prausnitzii inhibits inflammation by producing MAM to inhibit NF-κB activation in the intestinal microbiome. Akkermansia muciniphila secretes the peptide, Amuc_1100, which interacts with Toll-like receptor 2, improves the gut barrier and decreases inflammation. What the current studies have in common is that the changes in gut microbiota caused by smoking are like those found in diseases such as IBD and obesity, suggesting that the mechanism by which smoking increases inflammation in the body is common to both IBD and obesity.

Further functional examination showed that the smoking group had a higher concentration of pathways responsible for amino acid biosynthesis. Our hypothesis is that accumulation of amino acids, the basic structures of proteins, may be due to the increased demand for amino acids caused by changes in the bacterial abundances due to smoking, thereby increasing the amino acid abundances.

For the discovery and interpretation of microbial biomarkers, changes in the intestinal microbiome can also be analyzed by Linear Discriminant Analysis effect Size (LEfSe), which will emphasize statistical significance and biological correlation. LEfSe can identify not only the differences of rich bacteria, but also the differences of non-rich bacteria. This shows the statistical science of our study.

Our study has outstanding advantages. Firstly, the population is all from healthy people, which avoids the impact of disease. Secondly, we used metagenomes to define the differential flora at the species level. Thirdly, we analyzed the influence of lifestyle and other factors on the microflora, and determined that smoking was the main factor driving the change of microflora. Finally, we clearly found that smoking causes an increase in inflammation, possibly through changes in the microbiome.

Conclusion

In conclusion, our study provides new insights. However, the accuracy of microbiome assessments is still limited. We need a more comprehensive understanding of the overall structure and composition of the gut microbiome and the mechanisms of action of key functional bacteria.