Microbiota and Metabolic Diseases

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Metabolic syndrome is defined with at least 3 of the following issues: visceral obesity, low LDL-cholesterol, high triglyceride, hypertension and insulin resistance. Metabolic syndrome affects 40% of people over 60 years old. Microbiota plays important roles in metabolic syndrome via increasing LPS and bacterial lipopeptides, inducing metabolic endotoxemia to activate MyD88, TLR4, NF-kB, and enhancing inflammatory cytokines, etc. Increased energy harvest by gut microbiota is also an alternative explanation.

Several factors have been reported to induce dysbiosis and increase risk of metabolic syndrome, such as exposure to antibiotics, proton pump inhibitor, or H2 receptor antagonist in early life. Taking high fat diet reduces microbiota diversity, which cannot be recovered after shifting back to taking normal diet. The dysbiosis status can be transmitted into the next generations. Exercise can increase gut microbiota, especially in colon. Our animal experiments demonstrated that fecal microbiota transplantation (FMT) can transfer the beneficial effects of diet control and exercise.

In human studies, oligofructose-enriched inulin was found to improve appetite control and to reduce total body fat and trunk body fat. Allogenic FMT was found to improve peripheral insulin sensitivity, but not hepatic insulin sensitivity. However, FMT in obese patients with BMI>35 did not improve microbiota diversity and did not change BMI.

In conclusion, metabolic syndrome is closely related with gut microbiota. FMT is a potential to improve metabolic syndrome. Prebiotics are another promising method. Specific bacteria or their metabolites are the targets for future studies.