



Can We Follow Koch's Postulates for Hunting Down Human Obesity Bugs in Gut Microbiota?

Professor Liping ZHAO

Director for Laboratory of Molecular Microbial Ecology and Ecogenomics
School of Life Sciences and Biotechnology, Shanghai Jiao Tong University

DATE	:	11 November 2014, Tuesday <i>(including Q & A session)</i>
TIME	:	16:30 – 18:00
VENUE	:	SCM809
LANGUAGE	:	English
FACILITATOR	:	Prof. Aiping LU, Dean of SCM

Abstract of the Seminar

The gut microbiota has been linked with chronic diseases in humans such as obesity and diabetes. Accumulating evidence indicates that gut microbiota may play a pivotal role in onset and progression of adiposity and insulin resistance, the core conditions of metabolic syndrome (MetS) via two different but complementary pathways, i.e. regulation of energy metabolism and provocation of chronic inflammation. However, these mechanistic findings are obtained almost exclusively with rodent models. Their relevance to humans still remains a question. It is also controversial whether the obesity-associated changes of gut microbiota happen at broad taxonomic-level or are more relevant with specific phylotypes. Thus, the demonstration of causality between constituents of the microbiota and specific diseases remains an important challenge in the field. In this presentation, using Koch's postulates as a conceptual framework, I explore the chain of causation from alterations in the gut microbiota, particularly the endotoxin-producing members, to the development of obesity in both rodents and humans. Three components are essential for identifying the causative agents of obesity in the human microbiota: 1) microbiome-wide association studies; 2) isolation of the putative agents and disease reproduction in gnotobiotic animals; 3) mechanistic analysis of host responses to establish the molecular chain of causation.

We have employed this strategy in dietary therapy of morbid obesity/diabetes in humans to show that specific bacterial phylotypes, which are more relevant with MetS, can be identified, isolated and demonstrated in gnotobiotic models to be causatively contributing to MetS development in humans.

****Welcome****