

# The interaction of gut microbiota with host immune responses: friends or foes

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Soumita Das

Department of Pathology, University of California, USA

**Correspondence:** Soumita Das, Department of Pathology, University of California, USA, Tel 858-822-5523, Email sodas@ucsd.edu

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## Editorial

Human gut is associated with trillions of bacteria, ten times higher than the number of the cells in the mammalian body. So the microbiota can be considered as a separate organ. Their presence seems inert in the case of commensals or has beneficial roles in symbionts. In certain circumstances when the gut barrier is disrupted, the gut microbiota can generate immune responses detrimental to the human host. Besides that, irregular distribution of commensal bacteria is linked with the metabolic disorder, obesity, inflammatory bowel disease and cancer.<sup>1</sup> The underlying mechanism of host-gut microbe interactions are still not clear and better knowledge will help to prevent diseases and in the generation of targeted therapy.

The impact of the gut microbiota is not only limited to the mucosal surface but it also regulates distant organ sites by promoting innate immune cell development by hematopoiesis. Disruption of the gut microbiota or dysbiosis can lead to gastric ulcers, nonalcoholic fatty liver disease, obesity, metabolic syndromes and hypertension; inflammatory bowel disease, colon cancer, mood and behavioral changes through hormone signaling. The gut microbiota also controls the colonization of pathogenic microbes.

The role of gut microbiota and host interactions in metabolic and cardiovascular diseases will be highlighted in this editorial. The link between gut flora and the development of obesity is well established. In genetically obese mice or in obese human volunteers, a significant alteration in Bacteroidetes and Firmicutes was observed compared to lean controls.<sup>2</sup> The microbial production of short-chain fatty acids (SCFA), trimethylamine, acetaldehyde and inflammatory mediators has been shown to significantly impact the metabolic health of the host through pathways that influence satiety, gut permeability and immune function. N-butyrate generated from SCFA regulates leptin production in adipocytes and also controls anti-inflammatory responses by reducing cytokine-chemokine release by immune cells. So, targeting butyrate and butyrate producing gut-bacteria can be a new way of restoring host immune function and regulation of energy metabolism.<sup>3</sup>

Cardiovascular disease (CVD) is widely recognized as an inflammatory disease. Chronic inflammation is an independent risk factor for atherosclerosis by promoting plaque formation and inducing endothelial dysfunction. Coronary heart disease (CHD) occurs significantly more frequently in patients with inflammatory bowel disease (IBD). However, there is an ongoing debate whether heart disease is linked to viral or bacterial infection. A 13-year study period with 272 adults with non-typhoidal *Salmonella* NTS bacteremia showed 35% of the patients have extra-intestinal focal infections; 15% developed mycotic aneurysm exclusively after age 45years, and the rates of mycotic aneurysm (bacterial infection of the arterial wall) increased with the age of patients with NTS bacteremia.

Recent literature supports that the gut microbiota play an intermediate role in converting dietary choline from egg or red meat to trimethylamine-N-oxide (TMAO), the candidate responsible for cardiovascular disease and in the development of plaque in the arteries of mice.<sup>4</sup> Healthy people's feces are rich in *Eubacterium*, *Roseburia* and *Bacteroides* species and have more *Clostridium*. These bacteria carry genes involved in making anti-inflammatory molecules such as butyrate, lycopene and beta-carotene. A study of stroke patients showed them to have different microbiota populations with a new community characterized by *Ruminococcus* and *Collinsella* bacteria. In addition, stroke patients carried bacterial cell wall components that can set off inflammation. These studies indicate the possibility of bacteria mediated inflammation in heart diseases but the underlying mechanism is unknown. It also suggests the possibility of targeting the gut microbiota for the therapeutic benefit of cardiovascular diseases.

The interaction between the host immune system and the bacteria dictates whether the microbiota will be the friend or the enemy. Knowledge about the interaction between gut microbiota and immune response is rapidly expanding. This will help to develop personalized medicine, in chronic gut diseases and diseases beyond the gut. Other than diet, aging and infection; inflammation can change host-microbiota mutualism and force the pathogen composition by altering the health promoting bacteria. For example, colorectal cancer patients were significantly enriched in fecal *Fusobacterium*, *Enterococcaceae*, *Campylobacter*, *Erysipelotrichaceae*, *Collinsella*, *Peptostreptococcus* and *Anaerotruncus*, and depleted in members of the *Clostridium* cluster IV, such as *Faecalibacterium prausnitzii* (*F. prausnitzii*) and *Roseburia* with respect to healthy controls.<sup>5</sup>

The International Human Microbiome Consortium and the Human Microbiome Project (HMP) with the National Institutes of Health are involved with the identification of the role of microbiota in mouth, gut and vagina. These efforts will help to understand the capability of gut microbiota in human health and in the progression of disease responses. The first phase of HMP (FY2007-2012) already characterized the composition and diversity of microbial communities which inhabit major mucosal surfaces of the human body. The current

phase of HMP (FY2013-2015) will target the integrated dataset from both the microbiome and host to understand microbiome-associated diseases.

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## Conflicts of interest

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