

The Gut Microbiota For Health Newsletter #34  
**Metabolic conditions**  
Special edition – June 27, 2014

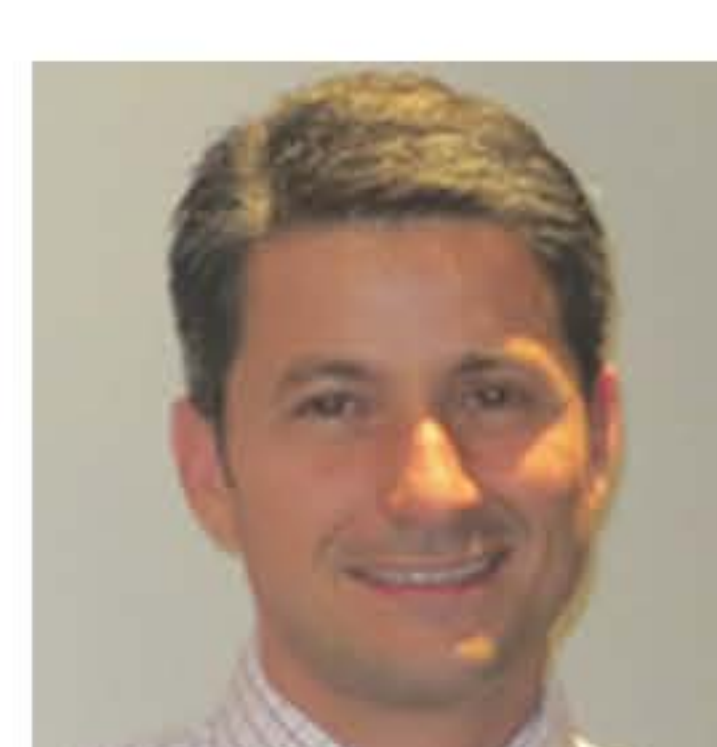
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**Edito**

Dear Friends,

In this newsletter, we are pleased to highlight one area of gut microbiota research: **metabolic conditions**. Until recently, metabolic disorders have been understood as health conditions caused primarily by genetic defects. While host genes might play a role in some metabolic diseases, new findings show that gut microbiota functions are strongly associated with host metabolism through their effects on chemical pathways.



Human metabolism is exceedingly complex. Colleagues have observed many correlations between the gut microbiota and chemical markers, yet the ongoing challenge is to translate the data from controlled rodent models into human subjects. Clinical applications are emerging over time from a more nuanced understanding of the human microbiota.

What follows is a selection of important articles that involve the gut microbiota in the field of metabolic conditions. We invite you to read them, add more selections, and participate in the dialogue with your colleagues, so this exciting field will continue to advance.

Prof. Patrice D. Cani  
Science team member, expert of the Metabolic Condition category on gutmicrobiotaforhealth.com.

[Read P. Cani's interview.](#)

**Transplanting fecal microbiota from twins discordant for obesity differentially affects metabolic activities in mice**

In this contribution article, Prof. Patrice Cani investigates the role of the gut microbiota in the development of metabolic disorders: obesity, type 2 diabetes and low-grade inflammation.

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Written by P.D. CANI



**Intestinal microbiota determines development of non-alcoholic fatty liver disease in mice**

Seen on [Gut](#)  
By T. Le Roy *et al.* - 2013

In this study, researchers use mice model to monitor the role of gut microbiota in the development of non-alcoholic fatty liver disease (NAFLD) and explain why not all subject with obesity develop the condition. Two groups of mice were selected on the basis of their response to a high fat diet. Though they displayed similar weight gain, one group developed hyperglycaemia and had a high plasma concentration of pro-inflammatory cytokines. The other group showed a lower level of systematic inflammation. They concluded that the composition of the gut microbiota can determine the response to a high fat diet and that the gut microbiota contributes to NAFLD independently of obesity.

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Selected by Y. WINOGRADSKY



**Microbiota-generated metabolites promote metabolic benefits via gut-brain neural circuits**

Seen on [Cell](#)  
By F. de Vadder, *et al.* - 2014

This selection presents the study from from Gilles Mithieux's lab, to go along with an interview from Philippe de Vadder. The study that focuses on:

- Propionate directly initiates portal-brain neural communication.
- Butyrate and propionate induce intestinal gluconeogenesis via different mechanisms.
- Intestinal gluconeogenesis provides a causal link for benefits of dietary fiber.
- Propionate and butyrate positively influence the host metabolism

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Written by J. TAP



**Dietary intervention impact on gut microbial gene richness**

Seen on [Nature](#)  
By A. Coillard - 2013

Complex gene-environment interactions are considered important in the development of obesity. The composition of the gut microbiota can determine the efficacy of energy harvest from food and changes in dietary composition have been associated with changes in the composition of gut microbial populations.

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Selected by P.D. CANI



**Replication of obesity using gut microbiota transfer**

Seen on [Diabetes](#)  
By FA Duca, *et al.* - 2014

A study published in Diabetes by Covasa lab tried to decipher causality regarding potential association between obesity and the gut microbiota. They fed rats using high fat diet and split them into two groups: obesity resistant and obese prone phenotypes. They observed that transferring microbiota from obese prone rats to others rats allowed to transfer obesity and other signs of suboptimal metabolic functioning. The main difference between obese prone compared to obese resistant rats was a diversity reduced in *Clostridiales* bacteria in their gut microbiota.

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Written by J. TAP



**How probiotics attenuate metabolic syndrome in mice on a high-fat diet**

Seen on [The ISME Journal](#)  
By J. Wang, *et al.* - 2014

In this study, a research group in China administered 3 probiotic candidates (*Lactobacillus paracasei* CNCM 1-4270, *L. rhamnosus* 1-3690, and *Bifidobacterium animalis* subsp. lactis 1-2494) to mice with metabolic syndrome induced by a high-fat diet. Each of the strains slowed the mice's weight gain and improved glucose-insulin homeostasis; the data showed a shift in the gut microbiota toward that of normal, lean mice. The different strains appeared to affect the microbiota through different chemical mechanisms.

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Selected by K. CAMPBELL



**The gut microbiota manages host metabolism**

Seen on [Nature Reviews Endocrinology](#)  
By P.D. Cani - 2014

Professor Patrice Cani summarizes studies in the area of metabolism in 2013, which reaffirm that the gut microbiota have an important role in host metabolism. He notes converging results that show how the gut microbiota influence energy homeostasis, glucose metabolism and metabolic inflammation. Cani discusses what is known about the gut microbiota and gastric bypass surgery, some recent studies on obesity, and how certain bacteria or bacterial metabolites help regulate various disease states.

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Written by K. CAMPBELL



**Role of the microbiome in energy regulation and metabolism**

Seen on [Gastroenterology](#)  
By M. Nieuwdorp *et al.* - 2014

A body of research shows that an altered microbial community potentially contributes to the development of metabolic diseases. This paper reviews the interplay between the intestinal microbiota and host metabolism, focusing on glucose and lipid metabolism, in order to shed light on the mechanisms by which the gut microbiota might execute its influence.

[Read more](#)

Selected by K. CAMPBELL

