

GUT MICROBIOTA & DIABETES

A selection of content from
the Gut Microbiota for Health
Experts Exchange 2015

January 2016

Diabetes

GUT MICROBIOTA
FOR HEALTH
Experts Exchange

SUMMARY

Diabetes is a major health concern in all corners of the globe. An estimated 9% of the worldwide adult population suffers from this chronic disease, which can significantly reduce quality of life and often has serious consequences, with an increased risk of heart disease, stroke, kidney failure, and overall mortality. Scientific advances in this area could lead to better outcomes for millions of people.

The complex relationship between obesity, diabetes, and gut microbiota is being uncovered, little by little, with emerging evidence. Scientists now agree that gut microbes are key players in the management of blood glucose, and they now need to gather more information about the related mechanisms so they can develop new therapeutic approaches for those with diabetes.

This brief «Best of Gut Microbiota and Diabetes» document features an editorial written by Patrice D. Cani, a researcher credited with some of the most important discoveries in this area. Dr. Cani summarizes what we know so far about the role of gut microbiota in the onset and progression of diabetes, highlighting ten recent literature selections for optional further reading. At the end of the document, Gut Microbiota for Health has provided you with a list of top ten Twitter accounts related to diabetes.

The Gut Microbiota for Health initiative is dedicated to sharing ongoing scientific work in the field of gut microbiota. Thank you for being a part of our community -- with over 11,000 members, we are stronger than ever. Stay tuned for how we will be evolving in 2016!

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TABLE OF CONTENTS

GUT MICROBIOTA & DIABETES **A selection of content from the Gut Microbiota for Health Experts Exchange 2015**

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Summary	P - 2
Table of contents	P - 3
Edito: Gut Microbiota & Diabetes	P - 4
References	P - 9
Selected content from gutmicrobiotaforhealth.com	P - 10
1/ GUT MICROBIOTA & DIABETES: Interview	P - 10
2/ GUT MICROBIOTA & DIABETES: Selections	P - 14
Diabetes: The 10 best Twitter accounts	P - 16



Patrice D. Cani

Management of blood glucose is one of the key activities sustaining human life and health. Obesity and type 2 diabetes come with serious health risks, yet treatment options for individuals with these conditions are currently limited by our incomplete understanding of the factors that interact in diabetes onset and progression.

Research in the past several years has advanced our understanding of how insulin resistance and diabetes arise. Gut microbiota has emerged as a key player in diabetes, as researchers gather evidence on the mechanisms by which gut microbiota may influence blood glucose.

Linking diabetes with gut microbiota composition and function

Composition

The first point of interest is that researchers have observed differences in gut microbiota composition between those with diabetes and healthy controls (see Delzenne, *et al.* and van Olden, *et al.*). The bacteria that differ between the groups appear to be microbes that primarily influence inflammation and energy homeostasis. Although there is no single feature of gut microbiota composition that signals diabetes, it is clear that generally those with type 2 diabetes show a reduction of butyrate-producing species and an increase in opportunistic pathogens.

Function

Gut microbiome functions are also different in those with type 2 diabetes. Some observed differences include increased membrane transport of sugars or branched amino acids, changed activity of enzymes involved in xenobiotic or carbohydrate metabolism, reduced butyrate synthesis, and decreased cofactor and vitamin metabolism.

Evidence suggests that these differences in gut microbiome composition and function are not irrelevant to the course of the disease. Gut bacterial composition is associated with response to dietary intervention, as shown in a recent paper by Dao, *et al.* Here, the teams of Karine Clément and Patrice D. Cani demonstrated for the first time that not all obese subjects have equal potential for weight loss-induced improvement of metabolic disorders when they adopt a calorie restriction diet. These researchers linked the presence of *Akkermansia muciniphila* with a more beneficial

response to the dietary intervention – that is, those with more *A. muciniphila* in their guts responded better to the dietary restriction in terms of reduced visceral fat, less severe insulin resistance, and reduced cholesterol and inflammatory markers.

Mechanisms of action

Low-grade systemic inflammation may be the driver of metabolic changes that are linked to insulin resistance and obesity. The trigger of this inflammation is not known, although we are beginning to understand more about the complex cascade of effects that leads to an inflammatory state. The gut microbiota and certain microbial metabolites seem to be intimately involved in these effects.

Role of bacteria

Bacteria themselves may play a role in inflammation. Previous work showed that if short-chain fatty acid (SCFA) -producing bacteria are reduced, the intestinal barrier might be impaired in a way that facilitates bacterial translocation. This would increase plasma lipopolysaccharides (LPS), resulting in a condition called metabolic endotoxemia. This endotoxemia predicts the development of metabolic syndrome and type 2 diabetes in obese subjects.

The efficacy of the gut barrier depends on many pathways and cell types, including mucus-producing goblet cells, tight junction proteins, the endocannabinoid system and immune responses. A recent comprehensive review (Cani, *et al.*) investigated the impact of gut microbes on the host endocannabinoid system in the context of obesity and diabetes. Authors highlighted the importance of gut barrier function and also discussed the role of specific factors involved in intestinal permeability and their role in the gut microbiota–endocannabinoid system axis. They also discussed novel mechanisms of action and crosstalk between adipose tissue and microbes, along with beiging/browning of the adipose tissue.

The intestinal immune system seems to play an essential role in the onset of obesity and type 2 diabetes. Bacterial LPS can stimulate Toll-like receptor (TLR) 4, resulting in an inflammatory response, cytokine production and chemokine-mediated recruitment of acute inflammatory cells. Everard, *et al.* showed that in mice, inactivating the protein MyD88 – a part of the innate immune system involved in the signalling of most TLRs – limited the development of adipose

tissue, slowed down the progression of diabetes, reduced harmful inflammation associated with obesity, and reinforced gut barrier function in order to prevent the inappropriate translocation of bacterial compounds. Thus, even in the context of a high-fat diet, disabling MyD88 specifically in intestinal epithelial cells had beneficial effects. This work shows an unexpected mechanism for the control of energy metabolism in obesity and type 2 diabetes – this protein of the immune system is able to shape the composition of the gut microbiota residing in the host intestine under a high-fat diet.

Role of metabolites

In addition to bacteria themselves, bacterial metabolites and components play a role in controlling the host immune system and glucose metabolism. Metabolites appear to influence the intestinal cells involved in endocrine and gut barrier functions. The ability of SCFAs (e.g. acetate, butyrate, and propionate) to affect insulin sensitivity and energy metabolism is now well supported by the data. Fermentation of dietary polysaccharides produces SCFAs (the most important one being butyrate), which signal to host genes and affect lipid and glucose metabolism through the liver. Hartstra, *et al.* provides a summary of mechanisms by which gut bacterial metabolites such as SCFA may contribute to changes in glucose metabolism.

Recently, researchers have been investigating the role of other metabolites such as indole (produced by gut bacteria from tryptophan) in reinforcing gut barrier function. Also, bile acids may be important in the digestion of dietary lipids, and they may act as signaling molecules in the context of energy, glucose and lipid metabolism.

Toward novel diabetes treatments

New data from Forslund, *et al.* showed that the gut microbiota of individuals with type 2 diabetes is shaped by metformin, a common anti-diabetic drug. *Akkermansia muciniphila* was among the specific microbes affected by metformin in this study. This serves as a reminder that investigating the interactions between microbes and host in the context of diabetes requires taking into account the subjects' context, including their existing treatment regimens.

A number of novel therapies related to the gut microbiota show promise for the treatment of diabetes. Researchers have attempted transfer of the gut microbial community (fecal microbiota transplantation, or FMT) from lean donors to recipients with metabolic syndrome; the recipients increased their levels of butyrate-producing bacteria and insulin sensitivity. This kind of therapy, first described by Max Nieuwdorp and colleagues, and discussed in a review by Hartstra, *et al.* may not become widely adopted because the risks of FMT are unknown at present.

Probiotics

A 2015 meta-analysis (Ruan, *et al.*) demonstrated that the consumption of probiotics can modestly benefit glycemic control. Although the specific strains need more study, the evidence supports modification of gut microbiota through probiotic supplementation as a safe method to help to control blood glucose in clinical practice. A review by Le Barz, *et al.* summarized the effects of probiotics on metabolic disorders in rodents, as well as the mechanisms of action. Strains of Lactobacilli have so far shown the most promise for positively affecting glucose metabolism in humans. Other 'next generation' probiotic strains also show promise – for example, *A. muciniphila* MucT showed a beneficial effect on glucose metabolism in a diet-induced mouse model of type 2 diabetes (Cani, *et al.*).

Nutrition

Nutritional interventions are also being explored for the treatment of diabetes. A review by Delzenne *et al.* focuses on the role of specific nutrients on the gut microbiota composition: prebiotics (inulin-type fructans), arabinoxylans, and polyphenols. These dietary components appear to impact gut microbiota composition as well as modulate gut peptide production (e.g. GLP-1 and PYY), conferring a beneficial physiological effect on the host. Some colleagues are testing compounds used in traditional Chinese medicine; for example, a recent review (Pang, *et al.*) outlined evidence that berberine – a polyphenol – can influence gut microbiota composition and contribute to modulation of glucose metabolism. Authors of this paper outlined numerous mechanisms of action by which this compound can indeed influence molecular pathways, ranging from AMPK and FOXO to nuclear receptor activity modulation.

EDITO

As new data emerges at a rapid pace, we are filling in the enormously complex picture of how blood glucose homeostasis is maintained in the human body. With research on the gut microbiota contributing to our increased understanding, new therapies for diabetes and obesity may be just around the corner.

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DELETION OF MYD88 IN INTESTINAL EPITHELIAL CELLS PARTIALLY PROTECTS AGAINST DIET-INDUCED OBESITY

Published in IMMUNE FUNCTION / METABOLIC CONDITIONS / RESEARCH TOOLS | Written on February 26, 2015 by [Patrice D. Cani](#)

In this paper, the first aim we had with Dr. Amandine Everard, was to investigate whether some key molecules involved in the innate immune system, mainly MyD88 myeloid differentiation primary response gene 88, may contribute to the development of obesity, diabetes and low grade inflammation.

This is not something novel, because we knew that MyD88 or Toll-like receptors are involved in the crosstalk between microbes and the host in the context of obesity resistance, but here, more specifically what we did was to delete more precisely MyD88 only in the intestinal epithelial cells, and via a system which was inducible. In other words, we were able to have the normal mice with a normal immune system, a normal gut microbiota, and then we can induce the deletion by using tamoxifen.

The power of the model was this key inducible system. Some people have demonstrated that, in absence of MyD88 in the whole body, mice developed obesity. Some others said they are protected. Some people said that in absence of MyD88 you have a different gut microbiota, and some others have shown that deleting MyD88 on the epithelial cells but via a non-inducible system is associated with changes in gut microbes and colitis. So there was no clear explanation on what the real contribution of MyD88 was in this crosstalk between microbes and host in the intestinal epithelial cells.

After we prepared and generated this kind of mice, we first validated that MyD88 can be deleted on the epithelial cells... and we saw indeed that we can delete MyD88 from the epithelial cells without affecting the different other organs. Then we took mice who were born normally and developed a normal microbiota, normal immune system; we fed the mice with a high-fat diet after having induced the deletion in adult mice. We found that they were gaining less weight. The

body weight gain was lower in absence of MyD88 in the epithelial cells of the intestine, and the fat mass development was also lower.

We were surprised by the fact that we didn't find any changes in food intake. So mice were not eating less food and there was no fat malabsorption or no energy loss in the feces. So we put the mice in the metabolic cages, and we did find an increased energy expenditure. So that was the first discovery of the paper: that in absence of this specific innate immune protein MyD88, mice were able to increase their energy expenditure, although they were fed with a high-fat diet. Because we know that a high-fat diet feeding decreases energy expenditure, and that may also contribute to body weight gain, of course.

Following this finding, we also decided to analyze and investigate glucose metabolism. We found here, again, that besides the lower body fat mass, mice exhibit a lower inflammatory tone and a lower plasma glucose. So they had lower insulin resistance and an improved profile of inflammatory markers, suggesting indeed that there are probably impacts of intestinal MyD88 upon all these different parameters that we know are involved in the development of insulin resistance.

Interestingly, when we measured the hepatic lipids - so steatosis - in absence of MyD88 in the intestine, mice were completely protected against the diet-induced hepatic steatosis... maybe due to the increase in energy expenditure or an increased lipid oxidation, but they were completely protected against this hepatic lipid accumulation. We also found that plasma LPS lipopolysaccharide (the metabolic endotoxemia) was completely reversed. We know that a high-fat diet increases plasma LPS, and in absence of MyD88 in the intestinal epithelial cells, mice were completely resistant, again, against this diet-induced metabolic endotoxemia.

If we take into account all these data, we can suggest that MyD88 at the level of the intestinal epithelial cells contributes to a reinforcement of the gut barrier, thereby reducing metabolic endotoxemia, although the mice are fed with the high-fat diet. This effect may also contribute to the reduction in hepatic steatosis and an improved insulin resistance. That's one of the key points.

Then of course, knowing that we are changing the immune system at the level of the epithelial cells, we investigated if the abundance of immune cells, and immune markers, were modified in the intestine. We did find a decrease in Treg cells regulatory T cells following a high-fat diet. But the decreased Treg cells was not observed in the knockout mice. So again, if we delete MyD88 in the intestinal epithelial cells, mice were protected against this high-fat-diet-induced modification of the immunity at the level of the intestine, suggesting that there are crosstalks between the fats and the intestinal epithelial cells, or maybe the gut microbiota, because we know that the gut microbiota can change the situation of immune cells in the intestinal epithelial layer.

This is one of the key reasons why we decided to investigate the gut microbiota composition. First, because we found a decrease in plasma LPS, and second, because we found that markers of antimicrobial peptide production were changed, were improved, in the knockout, and also the immune cells.

When we analyzed the gut microbiota composition, we indeed found modification of the gut microbiota that was induced by the deletion. And 72 different OTUs operational taxonomic units were different between high-fat diet knockouts and high-fat control mice. So deleting the MyD88 in the intestinal epithelial cells, although it was performed in adult mice, changed the gut microbiota composition.

We transferred this microbiota into germ-free mice just to see if the modification of the gut microbiota contributes to the improvement of the gut barrier and also the fat mass development, and

when we transferred the gut microbes into non-knockout germ-free mice and we fed the germ-free with a high-fat diet we found that transferring the gut microbiota partially protects against diet-induced obesity and also reduces low-grade inflammatory tone in the germ-free mice, suggesting that there are changes in gut microbes, or specific gut microbes, that contribute to this phenotype. Thus, the reduction in obesity and inflammation is likely due to modification of the gut microbiota.

Finally, one of the questions that we had also was: You have a specific model where you can induce the deletion of MyD88 under high-fat diet conditions. You observed a reduction in body weight gain and fat mass development. But is there any place for a putative therapeutic role of this deletion? So what we did was to put the mice on a high-fat diet for a couple of weeks, for six weeks exactly. They gained weight, they became obese. And then thanks to this inducible model we were able to induce the deletion in obese mice. Then, if we induced the deletion in obese mice, they lost weight and they had improved glucose tolerance and a reduction in inflammatory tone also, suggesting and showing indeed that, if we delete MyD88 in obese mice, we can observe a decrease in body weight and fat mass, and improved glucose tolerance also.

Below, I answer some questions from the GMFH editors:

I How does this new research fit in with your previous research on prebiotics?

We found that the prebiotics were able to change the immune markers at the level of the intestine. So I had in mind that microbes are modified when using prebiotics. We found, for instance, a change in REG3 γ regenerating islet-derived protein 3 gamma and different markers of the antimicrobial peptide production at the level of the gut, which means that microbes dialogue with the host, and we know that microbes dialogue with the host via for instance the Toll-like receptors. We knew that the microbes were modified by the prebiotics; we also have different data in hand suggesting that indeed the prebiotics

increase gut hormones... but the link with the innate immune system has never been, to my knowledge, investigated; only at the level of the epithelial cells in the context of obesity.

By using prebiotics in our previous studies we found these different links. So the parallel with the prebiotics study was, first, when we change gut microbes, we can change the epithelial cells and the gut barrier function. But why, and via which mechanism? The idea here was to verify the effect of deleting specific proteins of this crosstalk, because we know that almost all the Toll-like receptors will need MyD88 to transmit the signal. In other words, by using prebiotics to change gut microbiota, and then by changing gut microbiota, we change host metabolism; here we were able to change the crosstalks the another way around: I mean, by changing the host.

In the philosophy of the lab, and what I'm trying to develop as a researcher, is this idea of crosstalks and dialogue between microbes and host, host and microbes. We want to understand what's the contribution of one or other system to obesity, diabetes and inflammation.

I What individual differences will you need to take into account when making parallels to humans in this line of research?

First, we have to keep in mind that we are not all equal in terms of response when we digest fat, in terms of body weight gain. If you have one obese patient, and you put this guy on a low-calorie diet, he will lose weight, let's say 10 or 15 kilograms. You can give the same low-calorie diet to another person, with the same physical activity, and he will lose less weight. So it means that there is something somewhere, a physiological response of the body, that is different between these two guys.

One of the ideas I have — of course we have to prove it, but I think this is something that may exist according to recent data from colleagues — is that the microbiota might be different between both subjects. They have a microbiota different from me, suggesting there are probably some signatures

that are different from one to the second. Then I think that the response of the immune system itself, and the innate immune system in our case, at the level of the gut, might be different between obese subjects also. Not only if you're slim; but also between individual obese and diabetic subjects. It means that we can argue that we are different not only because of the microbes, but also because of the way we are able to dialogue with microbes.

There are also data suggesting that fat requires Toll-like receptor 4 to induce the increase in low-grade inflammatory tone, and in this context the fact that MyD88 might be differently expressed may also suggest that the obese patients may respond differently to a high-fat diet because of these responses. So we have data in hand, and other colleagues have suggested, that the microbial response or the intestinal immune system might be different between these subjects.

I What are the potential clinical applications of this research?

I think that from a therapeutic point of view, although we have no clear tool in hand to target specifically MyD88 in the intestine and then induce body weight loss, what I think might be possible to do is to understand how the microbes are discussing with the epithelial cells, or with the immune system. Then maybe this also suggests that if we are able to reduce the overstimulation of the different Toll-like receptors related to MyD88, it might be one way to reduce the body weight gain and fat mass development and improve the gut barrier function. This is something that we will try to investigate. If we can target only in the epithelial cells the expression of MyD88 — I cannot say how so far — maybe we will be able to reproduce the same effects.

Then in the simple understanding of the physiology of obesity and diabetes, this study suggests indeed the existence of a crosstalk between host and microbes and vice versa. This also clearly demonstrates that we cannot say that all the different organs and all the different cells are equivalent in terms of response against diet-induced obesity. In other words, here we found that

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GUT MICROBIOTA & DIABETES: INTERVIEW

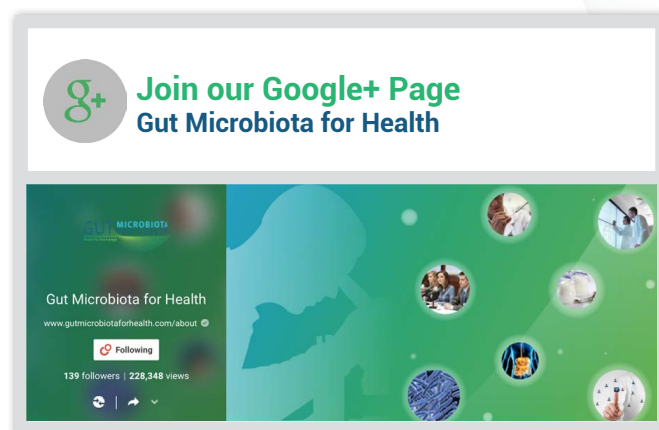
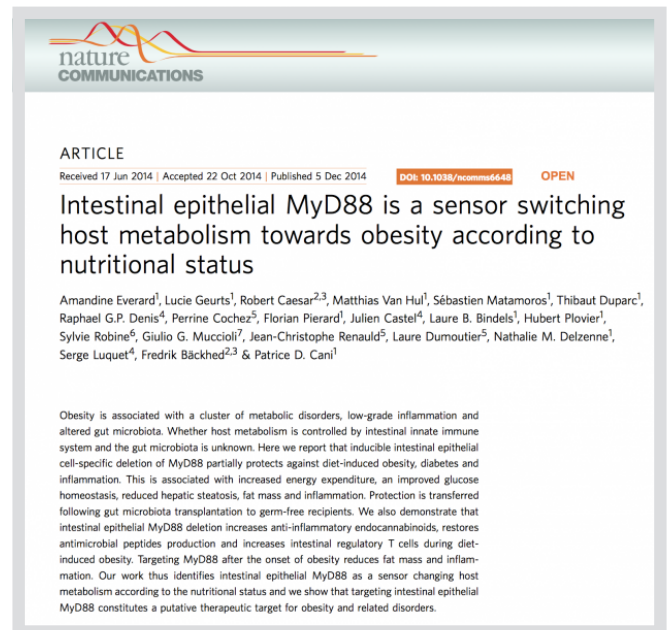
epithelial cells are really crucial to detecting the fat coming from the diet, and that the role of the epithelial cells is not only the absorption of the nutrients, but also maybe the role of sensing fat. Not only sensing fat via specific receptors, but maybe via the innate immune system. Can we find a similar relationship in humans? I have no data in hand to say yes or not. But I know that there are tools, and I hope that we will be able to demonstrate that there are some tools that are beyond this MyD88; some of these proteins are probably inhibited by specific inhibitors.

Targeting the MyD88 in the whole body is probably not the solution; it's too risky because we need the immune response. But targeting the response only in the epithelial cells is something worth pursuing.

Source: Everard A, et al. *Intestinal epithelial MyD88 is a sensor switching host metabolism towards obesity according to nutritional status.* *Nature Communications* doi:10.1038/ncomms6648

Read the original post online at:

<http://www.gutmicrobiotaforhealth.com/s/deletion-myd88-intestinal-epithelial-cells-partially-protects-diet-induced-obesity>



plus.google.com/103497649210561693858/posts

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GUT MICROBIOTA & DIABETES: SELECTIONS

META-ANALYSIS SHOWS PATIENTS WITH DIABETES BENEFIT FROM PROBIOTICS

Published in METABOLIC CONDITIONS / NUTRITION / PROBIOTICS / TRENDS & DISCOVERIES | Written on August 1, 2015 by [Kristina Campbell](#)

A meta-analysis of seventeen randomized clinical trials confirmed the efficacy of different probiotics, either in capsule form or in yogurt or drinks, to control hyperglycemia, hyperinsulinemia, and insulin resistance in patients suffering from diabetes.

Probiotics tested in the clinical trials, either alone or in combination, were: *Lactobacillus acidophilus*, *rhamnosus*, *casei*, *paracasei*, *plantarum*, *reuteri*, *gasseri*, *bulgaricus*, *salivarius*, *delbrueckii*, *sporogenes*; *Bifidobacterium longum*, *infantis*, *breve*, *lactis*, *Bb 12*, *animalis*, *animalis ssp lactis*; *Streptococcus thermophilus*.

Source: Ruan Y, Sun J, He J, Chen F, Chen R, & Chen H. (2015) *Effect of Probiotics on Glycemic Control: A Systematic Review and Meta-Analysis of Randomized, Controlled Trials*. *PLoS One*. doi: 10.1371/journal.pone.0132121. eCollection 2015.

Read the original post online at:

<http://www.gutmicrobiotaforhealth.com/s/meta-analysis-shows-patients-diabetes-benefit-probiotics>

THE ROLE OF PROBIOTICS IN TREATING METABOLIC DISORDERS

Published in GUT MICROBIOTA / IMMUNE FUNCTION / METABOLIC CONDITIONS / PROBIOTICS | Written on September 15, 2015 by [Pierre-Yves Arnoux](#)

Le Barz et al. reviewed the effects of probiotic administration on metabolic disorders, with special attention to mechanisms of action. They concluded:

- Microorganisms can fight the gut microbiota dysbiosis that is frequently associated with metabolic disorders in humans
- There is no "universal strain" of probiotics that alone provides all the benefits associated with probiotics; rather, probiotic effects are often strain-specific
- Probiotics can act by (1) restoring the disrupted intestinal barrier and therefore reducing endotoxemia, (2) producing short-chain fatty acids such as butyrate, or (3) decreasing low-grade inflammation
- Prebiotics can enhance the functionality of probiotics

Source: Le Barz M, et al. *Probiotics as Complementary Treatment for Metabolic Disorders. Diabetes and Metabolism Journal*. 2015 Aug; 39(4): 291–303. Published online 2015 Aug 17. doi: 10.4093/dmj.2015.39.4.291

Read the original post online at:

<http://www.gutmicrobiotaforhealth.com/s/role-probiotics-treating-metabolic-disorders>



www.youtube.com/channel/UCQLZz5EFC7tk2YgjTvll6gA

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GUT MICROBIOTA & DIABETES: SELECTIONS

METFORMIN AFFECTS GUT MICROBIOTA OF INDIVIDUALS WITH TYPE 2 DIABETES

Published in GUT MICROBIOTA / METABOLIC CONDITIONS | Written on December 16, 2015 by [Kristina Campbell](#)

Researchers have not controlled for treatments in many previous studies that aimed to discover the gut microbial signature of various diseases.

A new study from the MetaHIT consortium examined 784 human gut metagenomes and showed that metformin, a widely-used antidiabetic medication, affects the microbiota of those with type 2 diabetes mellitus (T2D).

In untreated T2D, researchers saw a distinct signature in the gut microbiota, with a depletion of taxa that produce butyrate.

In patients taking metformin, microbes appeared to mediate the therapeutic effects of the drug through their production of short-chain fatty acids. The known adverse effects of metformin on the intestine may also be mediated by the microbiota through a relative increase in the abundance of *Escherichia* species. These metformin-induced changes partly alleviate the functional shifts in the microbiota that were associated with the disease.

This study provides an example of a medication that influences gut microbiota signature. In future studies, researchers must control for medication regimens in order to discover diagnostically relevant signals in the gut microbiota.

Source: Forslund K, *et al.* (2015) *Disentangling type 2 diabetes and metformin treatment signatures in the human gut microbiota.* *Nature* doi:10.1038/nature15766

Read the original post online at:

<http://www.gutmicrobiotaforhealth.com/s/metformin-affects-gut-microbiota-individuals-type-2-diabetes>



www.twitter.com/GMFHx



DIABETES: THE 10 BEST TWITTER ACCOUNTS



Patients associations, blogs, social networks, research foundations, here we bring you 10 Twitter accounts to follow to keep up to date with what is hot in the field of #Diabetes.

1



PATIENTS
ASSOCIATION



Amer. Dia...
@AmDiabetesAssn

Leading the fight to #StopDiabetes and its deadly consequences, and fighting for all those affected by #diabetes. En Español: @AmerDiabetesESP

Followed by Robyn Webb and 125 others

TWEETS	FOLLOWING	FOLLOWERS
16K	1,159	101K

American Diabetes Association

- ▶ <http://twitter.com/AmDiabetesAssn>
- ▶ <http://www.diabetesforecast.org>

2



BLOG



Diabetes Daily
@diabetesdaily

Leading diabetes community - we want YOU to live a healthier, happier, more hopeful life!

Followed by Peter Janiszewski and 43 others

TWEETS	FOLLOWING	FOLLOWERS
5,547	1,336	41.9K

Diabetes Daily

- ▶ <http://twitter.com/diabetesdaily>
- ▶ <http://www.diabetesdaily.com>

3



PATIENTS
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Canadian Diabe...
@DiabetesAssoc

The official Twitter account of the Canadian Diabetes Association. Leading the fight against diabetes one tweet at a time.

Followed by Keystone Symposia and 42 others

TWEETS	FOLLOWING	FOLLOWERS
7,926	6,213	22.5K

Canadian Diabetes

- ▶ <http://twitter.com/DiabetesAssoc>
- ▶ <http://www.take2minutes.ca>



DIABETES: THE 10 BEST TWITTER ACCOUNTS



Patients associations, blogs, social networks, research foundations, here we bring you 10 Twitter accounts to follow to keep up to date with what is hot in the field of #Diabetes.

4



BLOG

DiabetesMine
@DiabetesMine

A gold mine of straight talk and encouragement for people living with diabetes. Tweets by @AmyDBMine, @MHoskins2179 & @ProbablyRachel

Followed by Kristin K and 46 others

TWEETS	FOLLOWING	FOLLOWERS
24.9K	2,347	26.3K

Diabetes Mine

- ▶ <http://twitter.com/DiabetesMine>
- ▶ <http://www.diabetesmine.com>

5



EVENT

World Dia...
@WDD

Halt the diabetes epidemic

World Diabetes Day is marked every year on November 14. The campaign is led by the International Diabetes Federation.

Followed by Mitzi Dulan and 44 others

TWEETS	FOLLOWING	FOLLOWERS
10.2K	444	44.1K

World Diabetes Day

- ▶ <http://twitter.com/WDD>
- ▶ <http://www.worlddiabetesday.org>

6



RESEARCH
FOUNDATION

Diabetes ...
@DiabetesUK

SWIM 22
GO TO GREAT LENGTHS

We are the leading UK charity that cares, connects and campaigns for people affected by and at risk of diabetes. Tweets answered between 9-5pm Mon-Fri

Followed by GI Nursing Journal and 47 others

TWEETS	FOLLOWING	FOLLOWERS
33.8K	10.5K	135K

Diabetes UK

- ▶ <http://twitter.com/DiabetesUK>
- ▶ <http://www.diabetes.org.uk>



DIABETES: THE 10 BEST TWITTER ACCOUNTS



Patients associations, blogs, social networks, research foundations, here we bring you 10 Twitter accounts to follow to keep up to date with what is hot in the field of #Diabetes.

7



SOCIAL
NETWORK



the global diabetes community
Diabetes.co.uk

Diabetes.co.uk
@Diabetescouk

Join the UK's #1 diabetes community with 176,657 forum members and 1.4m years of cumulative experience. Raising #diabetesawareness with #BloodSugarSelfie

Followed by [Hootsuite](#) and 27 others

TWEETS	FOLLOWING	FOLLOWERS
9,295	3,808	30.2K

Diabetes.co.uk

- ▶ <http://twitter.com/Diabetescouk>
- ▶ <http://www.diabetes.co.uk>

8



MEDIA



dN **DiabetesNews...**
@alldiabetesnews

The most comprehensive diabetes news aggregator on the web. For contact info write to randolph@diabetesnews.com

Followed by [EASO](#) and 34 others

TWEETS	FOLLOWING	FOLLOWERS
19.8K	6,577	53K

Diabetes News

- ▶ <http://twitter.com/alldiabetesnews>
- ▶ <http://www.diabetesnews.com>

9



PATIENTS
ASSOCIATION



Almost 600 million of us may be living with type 2 diabetes by 2035. Healthy eating can help reduce your risk. Let's make healthy eating a habit, not a privilege. Act today to change tomorrow.

Int. Diabet...
@IntDiabetesFed

The International Diabetes Federation (IDF) is the global advocate for the 415 million people living with diabetes and the many more at risk.

Followed by [DR LEONARDO ROMANO](#) and 47 others

TWEETS	FOLLOWING	FOLLOWERS
9,413	728	31.2K

The International Diabetes Federation

- ▶ <http://twitter.com/IntDiabetesFed>
- ▶ <http://www.idf.org>



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10




JDRF
@JDRF

JDRF is the leading global funder of type 1 diabetes research.

Followed by ResearchAmerica and 27 others

TWEETS	FOLLOWING	FOLLOWERS
7,571	1,779	45.1K

Juvenile Diabetes Research Foundation

- ▶ <http://twitter.com/JDRF>
- ▶ <http://jdrf.org>



GUT MICROBIOTA FOR HEALTH
World Summit 2016

5-6 March 2016
Hilton Miami Downtown, Miami, FL, USA

A section of the

Gut Microbiota & Health

ESNM
European Society of Neurogastroenterology & Motility
Member of UEG

AGA INSTITUTE

ESPGHAN

ECCO
European Crohn's and Colitis Organisation

with the support of **DANONE DAIRY**

BIOCODEX

Read the editorial from Gail Hecht, Chair of the Scientific Committee of the 2016 Gut Microbiota for Health World Summit.

Visit the *Summit Registration website* for information and registration!

GUT MICROBIOTA & DIABETES

January 2016



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