

#BehindTheHeadlines

## ***“Vaginal seeding’ of babies born by C-section could pose infection risk,”***

*The Guardian reports.*

Despite the lack of studies proving cause and effect, many women in Australia and the UK are reportedly requesting the procedure after reading about it in the news.



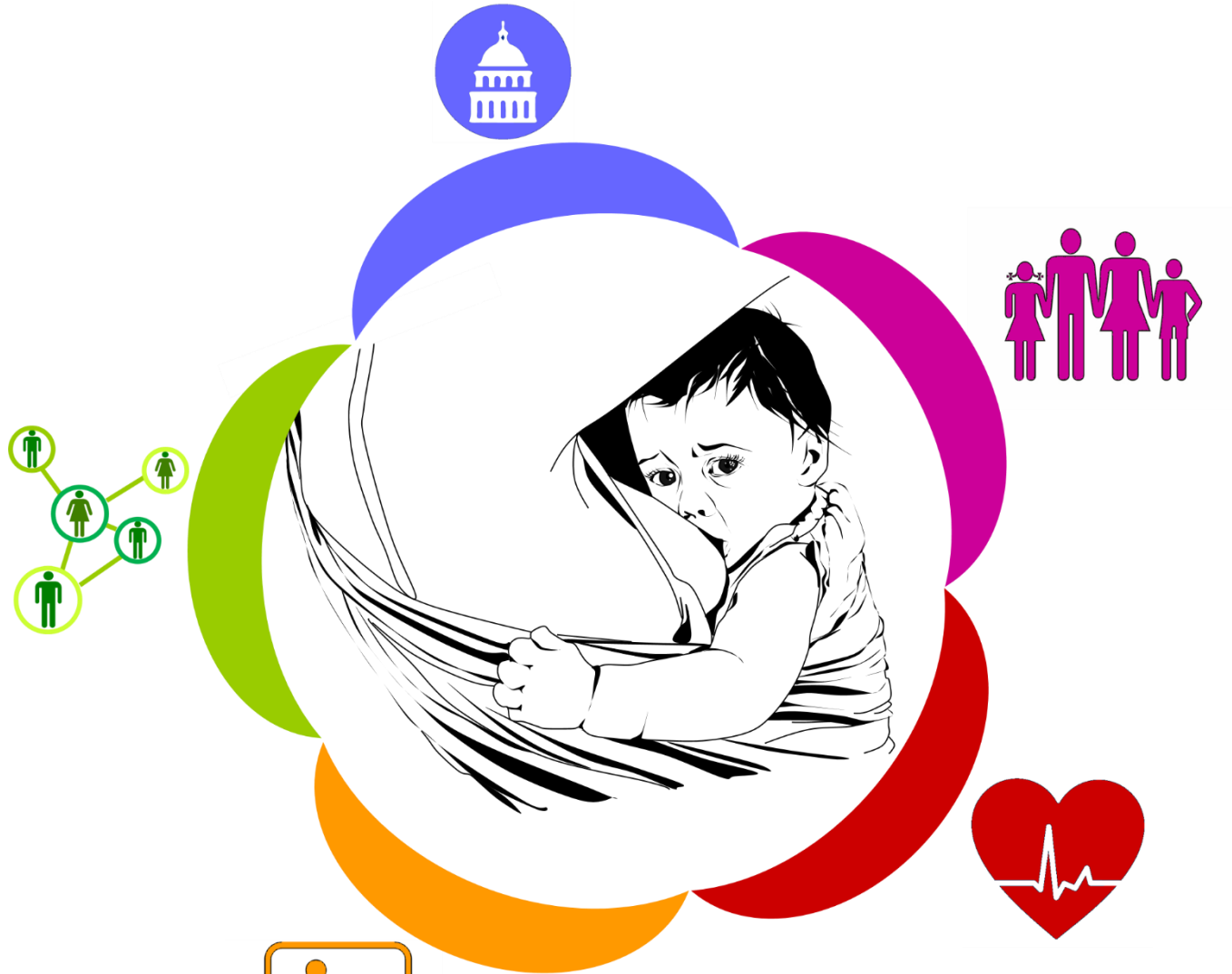
Behind The Headlines gives you the facts without the fiction

**NHS**  
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# The human microbiome: friend or foe?



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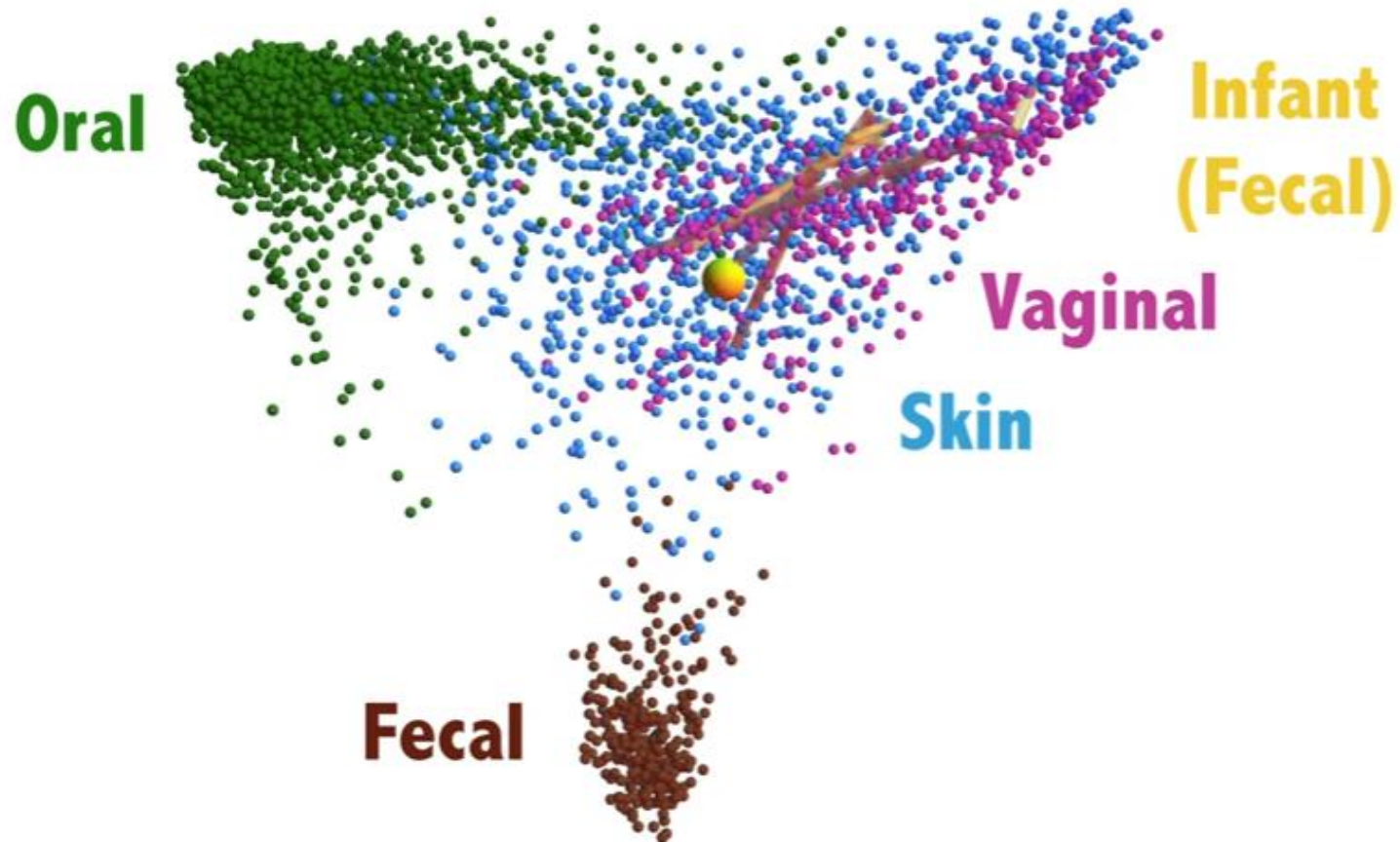
## Randomized controlled trial on the impact of early-life intervention with bifidobacteria on the healthy infant fecal microbiota and metabolome

*Monika Bazanella,<sup>1</sup> Tanja V Maier,<sup>4</sup> Thomas Clavel,<sup>2</sup> Ilias Lagkouravdos,<sup>2</sup> Marianna Lucio,<sup>4</sup> Maria X Maldonado-Gómez,<sup>5</sup> Chloe Autran,<sup>7</sup> Jens Walter,<sup>6</sup> Lars Bode,<sup>7</sup> Philippe Schmitt-Kopplin,<sup>3,4</sup> and Dirk Haller<sup>1,2</sup>*

<sup>1</sup>Chair of Nutrition and Immunology, <sup>2</sup>ZIEL – Institute for Food & Health, and <sup>3</sup>Chair of Analytical Food Chemistry, Technical University of Munich, Freising, Germany; <sup>4</sup>Research Unit Analytical Biogeochemistry, Helmholtz Center Munich, Oberschleißheim, Germany; <sup>5</sup>Department of Food Science and Technology, University of Nebraska, Lincoln, NE; <sup>6</sup>Chair for Nutrition, Microbes and Gastrointestinal Health, University of Alberta, Edmonton, Alberta, Canada; and <sup>7</sup>Divisions of Neonatology and Gastroenterology, Hepatology, and Nutrition, Department of Pediatrics, and Larsson-Rosenquist Foundation Mother-Milk-Infant Center of Research Excellence (MoMICoRE), University of California, San Diego, La Jolla, CA

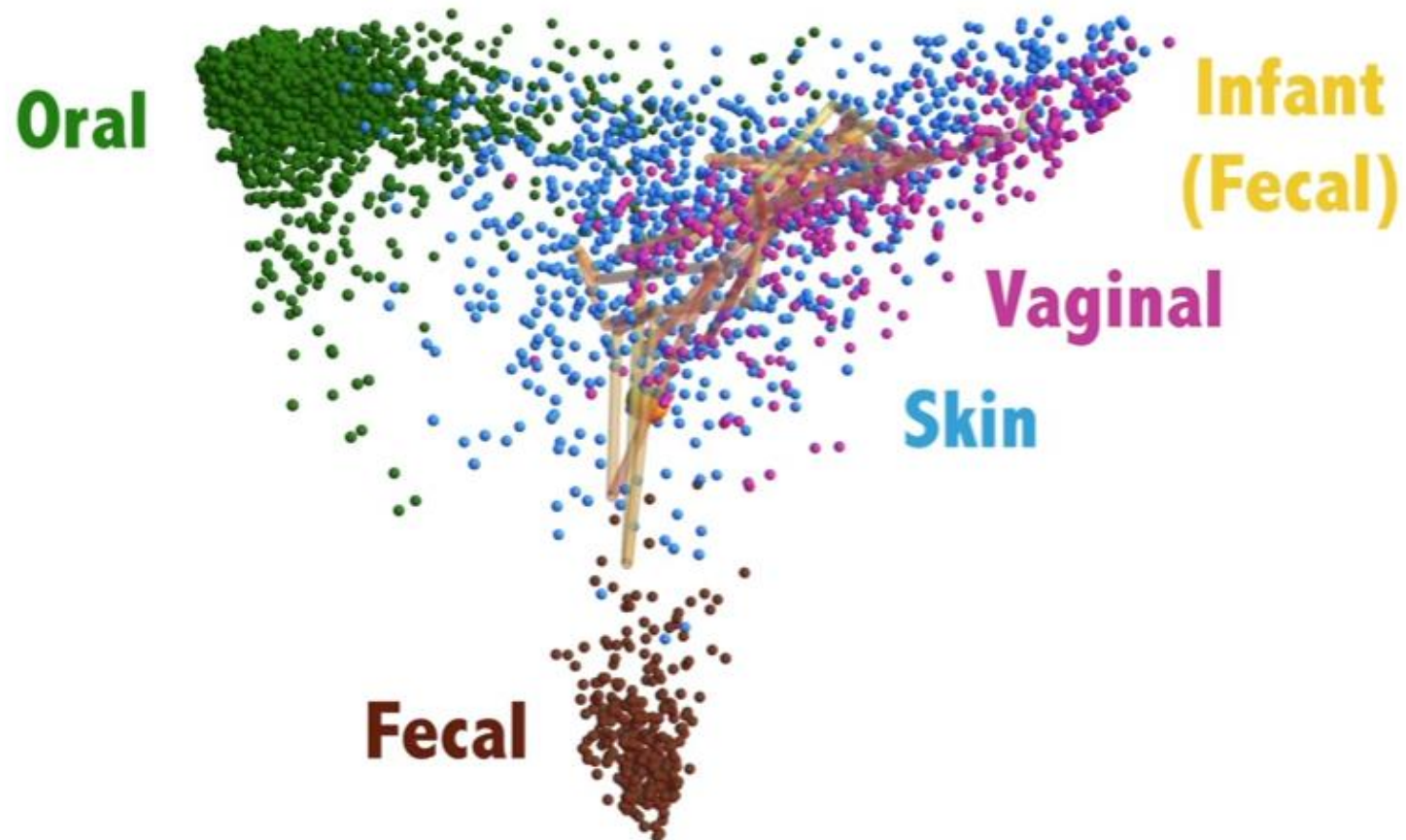


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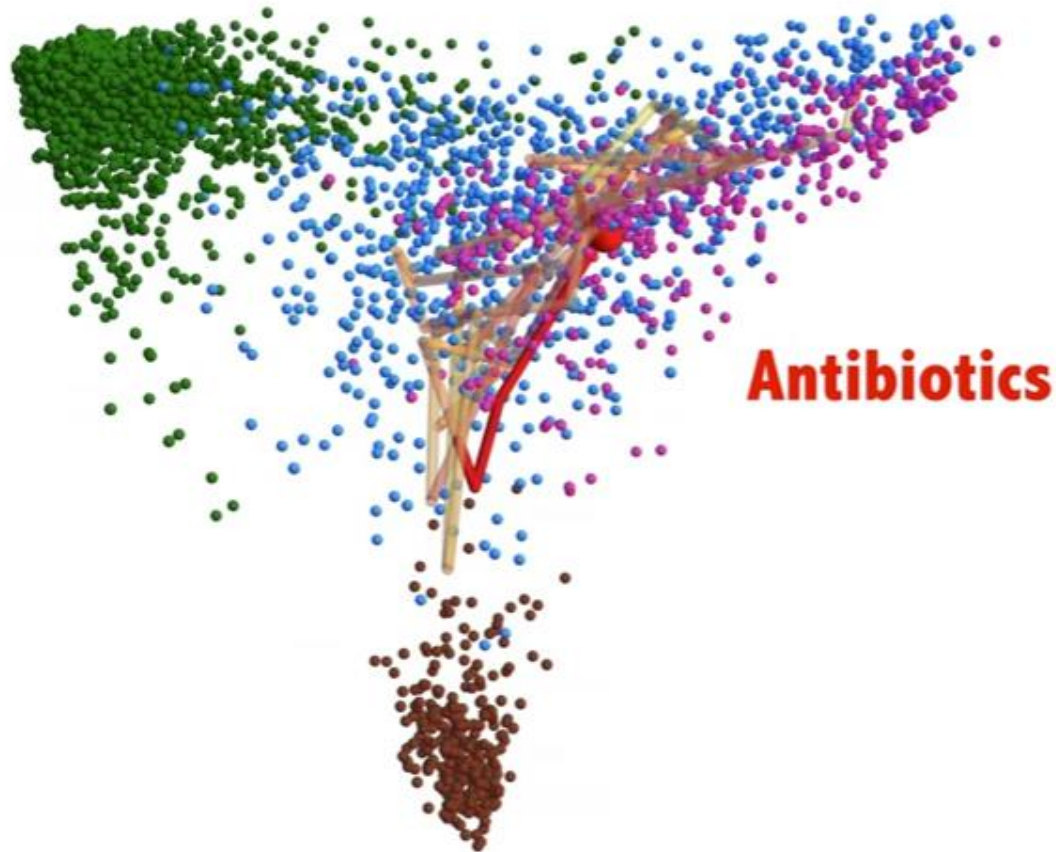
[https://www.ted.com/talks/rob\\_knight\\_how\\_our\\_microbes\\_make\\_us\\_who\\_we\\_are?language=en#t-617423](https://www.ted.com/talks/rob_knight_how_our_microbes_make_us_who_we_are?language=en#t-617423)

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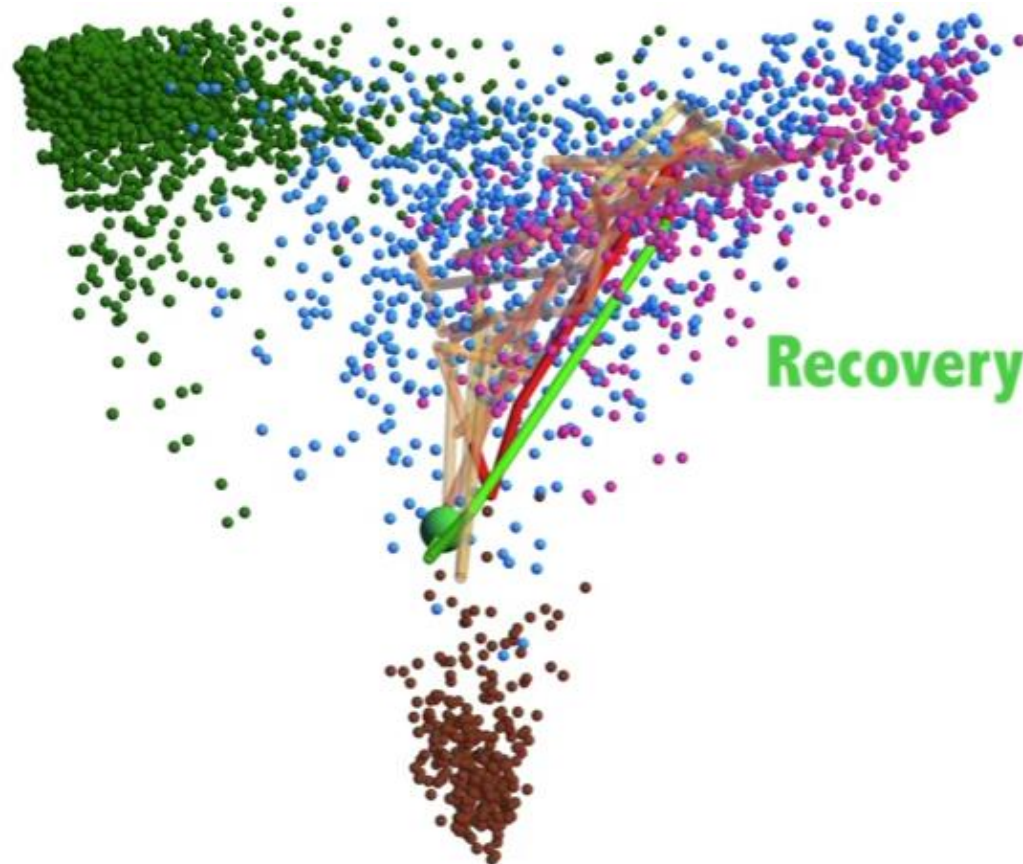
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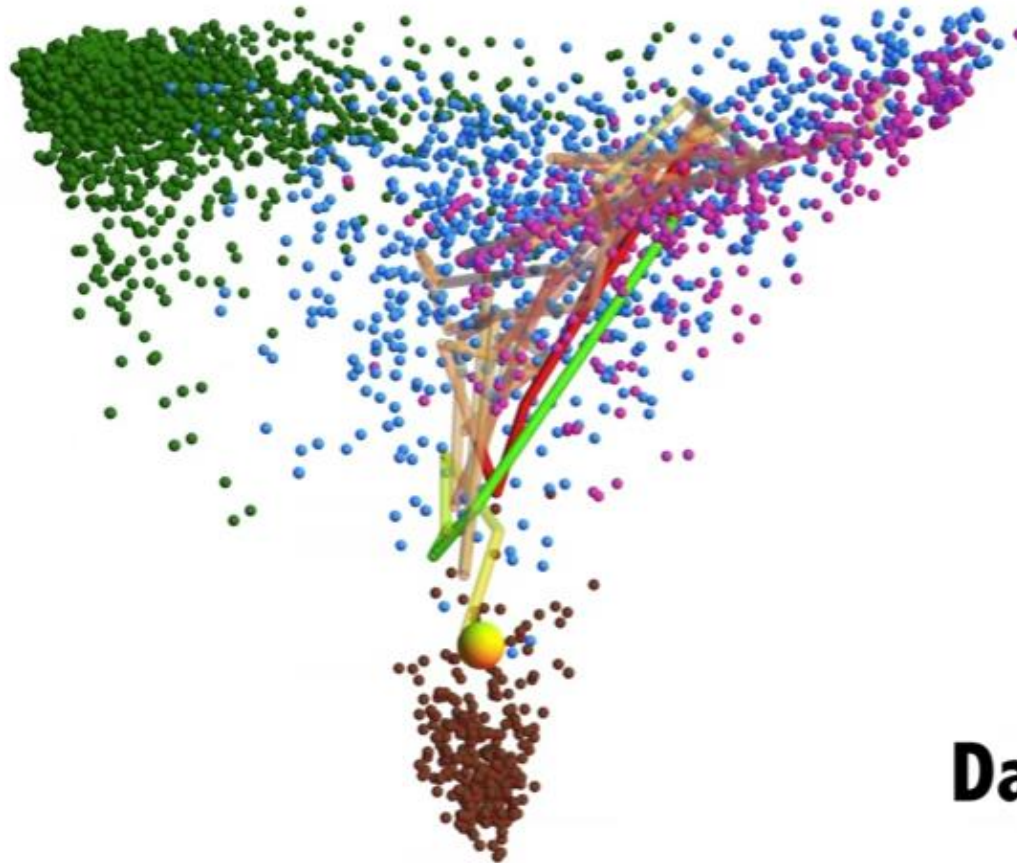
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**Day 838**

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RESEARCH ARTICLE  
Host-Microbe Biology



## Antibiotic-Induced Alterations of the Gut Microbiota Alter Secondary Bile Acid Production and Allow for *Clostridium difficile* Spore Germination and Outgrowth in the Large Intestine

Casey M. Theriot,<sup>a,b</sup> Alison A. Bowman,<sup>b</sup> Vincent B. Young<sup>b,c</sup>



## Fecal microbiota transplantation: in perspective

**Shaan Gupta, Emma Allen-Vercoe and Elaine O. Petrof**

*Ther Adv Gastroenterol*

2016, Vol. 9[2] 229–239

DOI: 10.1177/  
1756283X15607414

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**Abstract:** There has been increasing interest in understanding the role of the human gut microbiome to elucidate the therapeutic potential of its manipulation. Fecal microbiota transplantation (FMT) is the administration of a solution of fecal matter from a donor into the intestinal tract of a recipient in order to directly change the recipient's gut microbial composition and confer a health benefit. FMT has been used to successfully treat recurrent *Clostridium difficile* infection. There are preliminary indications to suggest that it may also carry therapeutic potential for other conditions such as inflammatory bowel disease, obesity, metabolic syndrome, and functional gastrointestinal disorders.

## Creating crapsules: is faeces in a pill the cure for our ills?



<https://www.smh.com.au/lifestyle/health-and-wellness/creating-crapsules-is-faeces-in-a-pill-the-cure-for-our-ills-20180319-p4z53z.html>



# The human microbiome: friend or foe



Minute  
Earth

Created by:  
**Henry Reich**

Production Team:  
**Alex Reich**  
**Emily Eiert**  
**Ever Salazar**  
**Henry Reich**  
**Peter Reich**

Music by:  
**Nathaniel Schroeder**  
[soundcloud.com/drschroeder](https://soundcloud.com/drschroeder)

References, detailed credits  
and more in the description



# The human microbiome: friend or foe

Neptune Studios  
presents

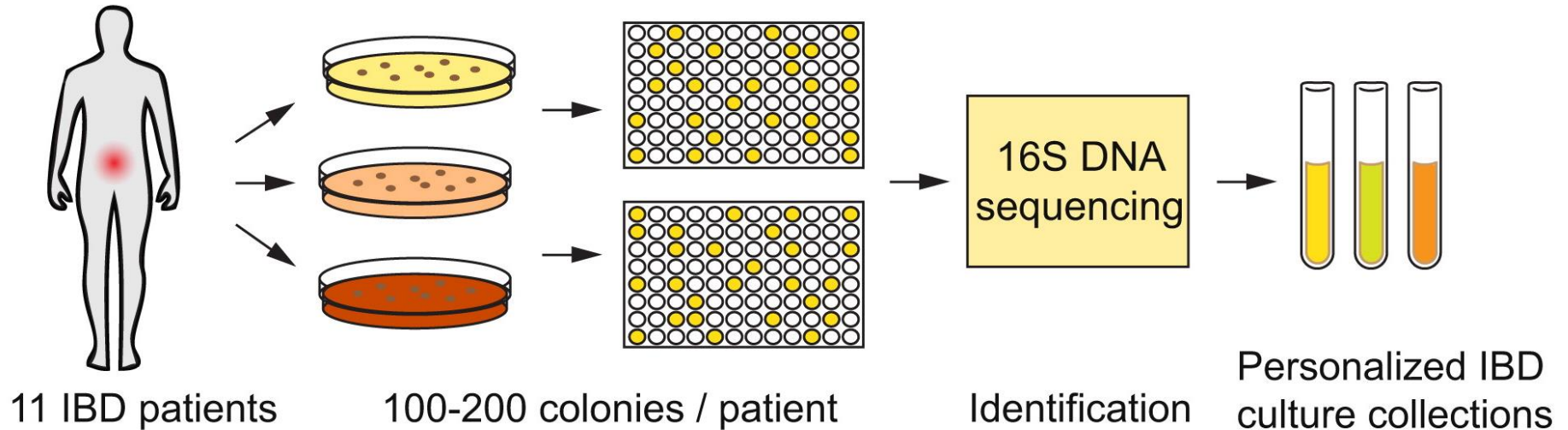
# The human microbiome: friend or foe?

**Table 1.** Role of Pathogenic Gut Microbiota in Gastrointestinal Diseases

| Risk factor                                | Microbial change                                                           | Possible mechanisms                                            | Ref.       |
|--------------------------------------------|----------------------------------------------------------------------------|----------------------------------------------------------------|------------|
| <b>IBD</b>                                 |                                                                            |                                                                |            |
| Genetics (Nlrp6 deficient)                 | <i>Prevotellaceae</i> ↑, TM7 ↑                                             | IL-18↓, CCL5↑, and innate and adaptive immune cell recruitment | 14, 15, 16 |
| Genetics (IL-10, IL-2 deficient)           | <i>Escherichia coli</i> or <i>Enterococcus faecalis</i> (monocolonization) | IL-12, IFN-γ ↑                                                 | 23         |
| Genetics (HLA-B27)                         | <i>Bacteroides fragilis</i> (monocolonization)                             | Unknown                                                        | 24         |
| Diet (high fat derived from milk)          | Firmicutes ↓, <i>Bifidobacterium wadsworthia</i> ↑                         | Immune system (Th1) disruption                                 | 26, 27     |
| Diet (high protein)                        | <i>Desulfovibrio</i> spp. ↑, <i>Desulfuromonas</i> spp. ↑                  | Genotoxic ↑, DNA damage ↑, inflammation ↑                      | 28, 29     |
| Diet (high fat, high beef)                 | <i>Erysipelotrichaceae</i> ↑, <i>Bacteroides fragilis</i> ↑                | Unknown                                                        | 30, 31     |
| Smoking                                    | <i>Anaerostipes</i> ↓                                                      | Butyrate ↓                                                     | 35         |
| Antibiotics (ciprofloxacin, metronidazole) | <i>Dorea</i> ↓, <i>Butyrivibrio</i> ↓, <i>Coriobacteriaceae</i> ↓          | Organic acid ↓ (e.g., formic acid, butyrate)                   | 40, 41     |
| Antibiotics                                | <i>Clostridium scindens</i> ↓, <i>Clostridium difficile</i> ↑              | DCA ↓                                                          | 47         |
| Unknown                                    | <i>Faecalibacterium prausnitzii</i> ↓                                      | Anti-inflammatory effect ↓                                     | 50, 51     |
| Unknown                                    | <i>pks+</i> <i>Escherichia coli</i> ↑                                      | Colibactin ↑, DNA damage ↑                                     | 64         |

Nagao-Kitamoto et al. (2016) *Intest Res.* 14: 127–138

## Irritable bowel disease: confirmed using mouse models



Selected individual bacterial isolates comprising of IgA+ and IgA– bacteria and colonized germ free mice.

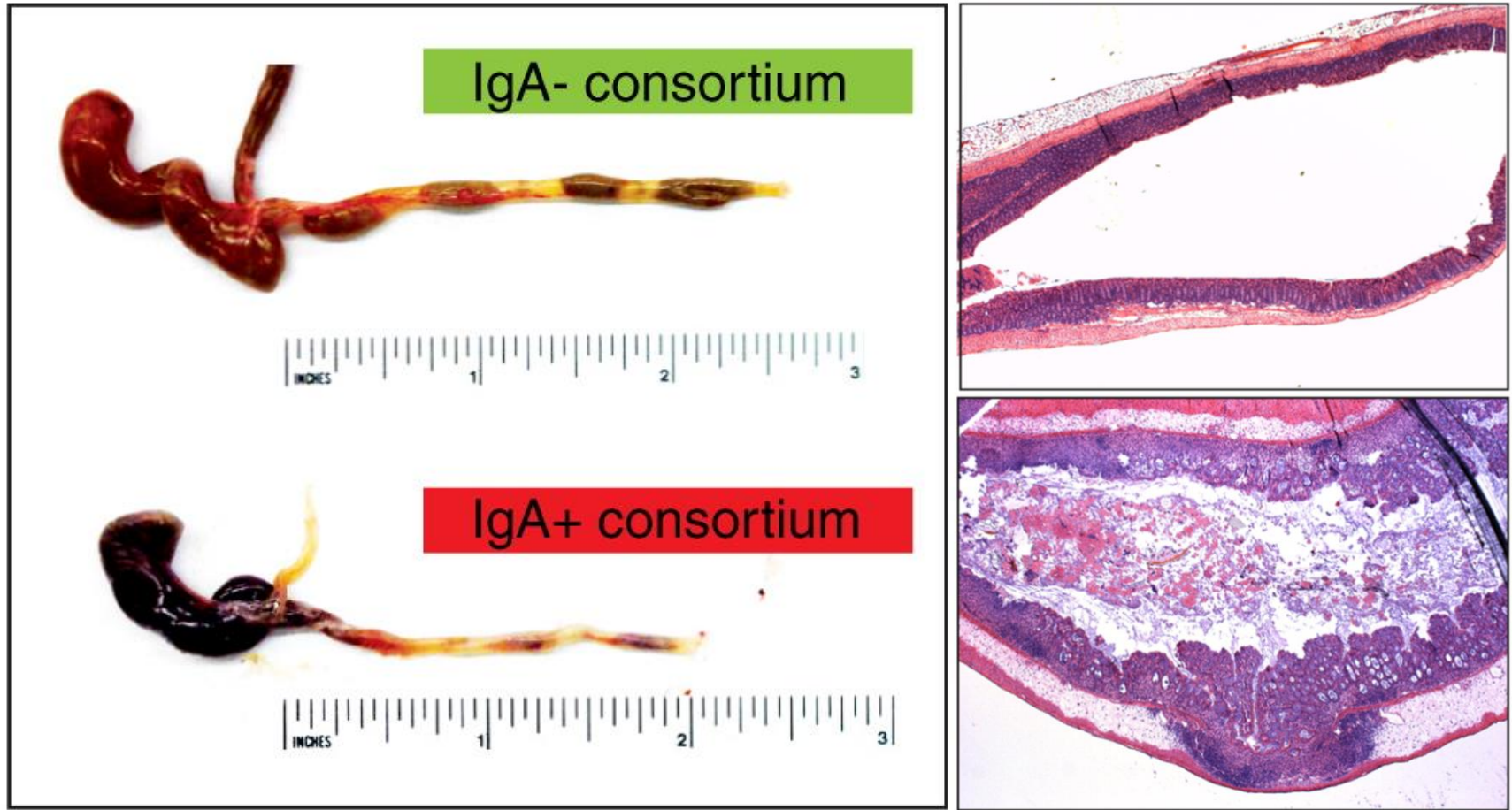
*High IgA coating are thought to mark colitogenic bacteria in inflammatory bowel disease*

Palm et al. (2014) *Cell* 158: 1000-1010



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## Irritable bowel disease: confirmed using mouse models



Palm et al. (2014) *Cell* 158: 1000-1010

## In summary:

- Our GIT microbiome is a “plastic entity” which is modulated by a number of exposures throughout our lives.
- A large number of 16S studies have contributed to our current knowledge of the GIT microbiome – which has led to a number of potential interventions for disease states.
- To date, the majority of 16S studies have focussed on the GIT microbiome.
- This research, however, is still very new and more well designed studies are needed to better understand not only “what’s there”, but also “what they’re doing”.