GI Effects in Clinical Practice:
Focusing on the Gut

Rebecca Hunton, MD
August 23, 2017

The views and opinions expressed herein are solely those of the presenter and do not necessarily represent those of Genova Diagnostics. Thus, Genova Diagnostics does not accept liability for consequences of any actions taken on the basis of the information provided.
Lahnor Powell, ND, MPH
Medical Education Specialist - Asheville
Need More Resources?

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Objectives

• Discuss novel patient populations where the GI Effects test may be indicated

• Discuss recent published studies that support the use of GI Effects in these populations

• Discuss 3 interesting case studies that demonstrate utilization of GI Effects testing made real clinical differences
New Patient Pathways

- Autoimmune/CFS/Fibromyalgia
- IBS/IBD/GERD/EE
- Thyroid/Hormone
- Proactive/Preventative
- Metabolic Dysfunction/Weight
- Mood and Neurologic Issues/Diseases
- Cancer/Serious Diseases and Conditions
- Engineers
New Patient Pathways

- Autoimmune/CFS/Fibromyalgia/Skin Conditions
- IBS/IBD/GERD/EE
- Thyroid/Hormone
- Proactive/Preventative
- Metabolic Dysfunction/Weight
- Mood and Neurologic Issues/Diseases
- Cancer/Serious Diseases and Conditions
- Engineers
New Patient Pathways

- Autoimmune/CFS/Fibromyalgia
- IBS/IBD/GERD/EE
- Thyroid/Hormone
- Proactive/Preventative
- Metabolic Dysfunction/Weight
- Mood and Neurologic Issues/Diseases
- Cancer/Serious Diseases and Conditions
- Engineers
You Want Me to Do What???
TED Template

If you haven't watched the following TED talks, I highly recommend them: (TED is a nonprofit devoted to spreading ideas, usually in the form of short, powerful talks (18 minutes or less). TED began in 1984 as a conference where Technology, Entertainment and Design converged, and today covers almost all topics — from science to business to global issues — in more than 100 languages.

• Food for thought: How gut microbes change your mind
  http://www.tedmed.com/talks/show?id=293045

• How our microbes make us who we are:
  https://www.ted.com/talks/rob_knight_how_our_microbes_make_us_who_we_are?language=en

• Type 1 Diabetes and the Microbiome
  http://www.ted.com/talks/jonathan_eisen_meet_your_microbes?language=en

• Mind Altering Microbes: TEDxCalTech: https://www.youtube.com/watch?v=FWT_BLVOASI

• The gut flora: You and your 100 trillion friends TEDxBrussels
  https://www.youtube.com/watch?v=Af5qUxl1ktI

• Jessica Green: We’re covered in germs. Let’s design for that
  http://www.ted.com/talks/jessica_green_good_germs_make_healthy_buildings
News Briefs and Articles

MEN ELASTIC Casual Flat Shoes
$33.89

Health

Parkinson's disease 'may start in gut'
By James Gallagher
Health and science reporter, BBC News website

Can gut bacteria improve your health?
Initial research suggests certain bacteria in your gut can prevent and treat many common diseases.

Harvard Men's Health Watch

COPENHAGEN, Denmark — Probiotics may be effective in reducing depression symptoms in treatment-naive patients with a history of clinical depression, results of a new comprehensive study suggest.

Investigations led by Caroline Williams, PhD candidate, Queen's University, Kingston, Ontario, Canada, found that symptoms of mood, anxiety, and

Harvard Health Publications
Trusted advice for a healthier life

HEART HEALTH MIND & MOOD PAIN STAYING HEALTHY CANCER

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Can gut bacteria improve your health?
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Why Autoimmune?

Putative gut microbiome and virome interactions with an autoimmune-prone host immune system

Why Neurologic Disease?

Another Alzheimer's drug flops in pivotal clinical trial

By John Carroll, Endpoints News | Feb. 15, 2017, 11:00 AM
Objective: We determined whether Gram-negative bacterial molecules are associated with Alzheimer disease (AD) neuropathology given that previous studies demonstrate Gram-negative Escherichia coli bacteria can form extracellular amyloid and Gram-negative bacteria have been reported as the predominant bacteria found in normal human brains.

Methods: Brain samples from gray and white matter were studied from patients with AD (n = 24) and age-matched controls (n = 18). Lipopolysaccharide (LPS) and E. coli K99 pili protein were evaluated by Western blots and immunocytochemistry. Human brain samples were assessed for E. coli DNA followed by DNA sequencing.

Results: LPS and E. coli K99 were detected immunocytochemically in brain parenchyma and vessels in all AD and control brains. K99 levels measured using Western blots were greater in AD compared to control brains (p < 0.01) and K99 was localized to neuron-like cells in AD but not control brains. LPS levels were also greater in AD compared to control brains. LPS colocalized with Aβ in amyloid plaques and with Aβ around vessels in AD brain. DNA sequencing confirmed E. coli DNA in human control and AD brains.

Conclusions: E. coli K99 and LPS levels were greater in AD compared to control brains. LPS colocalized with Aβ in amyloid plaques and around vessels in AD brain. The data show that Gram-negative bacterial molecules are associated with AD neuropathology. They are consistent with our LPS-ischemia-hypoxia rat model that produces myelin aggregates that colocalize with Aβ and resemble amyloid-like plaques.
IMPORTANCE: Medications that influence the risk of dementia in the elderly can be relevant for dementia prevention. Proton pump inhibitors (PPIs) are widely used for the treatment of gastrointestinal diseases but have also been shown to be potentially involved in cognitive decline.

OBJECTIVE: To examine the association between the use of PPIs and the risk of incident dementia in the elderly.

DESIGN, SETTING, AND PARTICIPANTS: We conducted a prospective cohort study using observational data from 2004 to 2011, derived from the largest German statutory health insurer, Allgemeine Ortskrankenkassen (AOK). Data on inpatient and outpatient diagnoses (coded by the German modification of the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision) and drug prescriptions (categorized according to the Anatomical Therapeutic Chemical Classification System) were available on a quarterly basis. Data analysis was performed from August to November 2015.

EXPOSURES: Prescription of omeprazole, pantoprazole, lansoprazole, esomeprazole, or rabeprazole.

MAIN OUTCOMES AND MEASURES: The main outcome was a diagnosis of incident dementia coded by the German modification of the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision. The association between was analyzed using time-dependent Cox regression. The model was adjusted for potential confounding factors, including age, sex, comorbidities, and polypharmacy.

RESULTS: A total of 73,679 participants 75 years of age or older and free of dementia at baseline were analyzed. The patients receiving regular PPI medication (n = 2950; mean [SD] age, 83.8 [5.4] years; 77.9% female) had a significantly increased risk of incident dementia compared with the patients not receiving PPI medication (n = 70,729; mean [SD] age, 83.0 [5.6] years; 73.6% female) (hazard ratio, 1.44 [95% CI, 1.36-1.52]; P < .001).

CONCLUSIONS AND RELEVANCE: The avoidance of PPI medication may prevent the development of dementia. This finding is supported by recent pharmacoepidemiological analyses on primary data and is in line with mouse models in which the use of PPIs increased the levels of β-amyloid in the brains of mice. Randomized, prospective clinical trials are needed to examine this connection in more detail.
The chicken or the egg dilemma: intestinal dysbiosis in multiple sclerosis

Javier Cebolla-Reparaz1, Krişitan Magers2, Lloyd H. Kasper2

1Department of Biology, Eastern Washington University, Cheney, WA, USA; 2Department of Microbiology and Immunology, Creteil School of Medicine at Université Paris 13, Creteil, France.

Correspondence to: Javier Cebolla-Reparaz, PhD; Department of Biology, Eastern Washington University, Cheney, WA, USA.

Abstract: Recent findings suggest that the intestinal microbiota of patients suffering from multiple sclerosis (MS) show changes in the relative abundances of fixed and facultative microbes and overall structure of the microbiota may be similar compared to the intestinal microbiota of healthy controls. New and reduced frequencies support a dysbiotic microbiota in MS. Over the evidence obtained in mouse models of the disease highlighted the importance of repletion of the immune system and in the severity of the disease. More recent findings with derived from human MS patients suggest the initial observations that changes in the immunological pathways that could exacerbate disease. However, important questions remain: in what way do these processes occur? If so, the answers to these questions can help to guide the research. In this brief discussion, we speculate about the role of the gut microbiota in the onset of multiple sclerosis. Further human studies are needed to answer the different immunological pathways that could have an impact on the therapeutic approaches to MS.

Keywords: Microbiota, intestinal dysbiosis, multiple sclerosis (MS)

Introduction: MS is an autoimmune disease characterized by a progressive and relapsing inflammatory disease of the central nervous system (CNS) that results in irreversible damage to the brain and spinal cord. The exact cause of MS is not known, but it is believed to be a complex interaction between genetic and environmental factors, and the immune system.

The microbiota is the collection of microorganisms, such as bacteria, fungi, and viruses, that live on and inside the body. The gut microbiota plays a crucial role in the regulation of the immune system and in the maintenance of human health. Changes in the gut microbiota have been associated with various diseases, including multiple sclerosis.

Recent advances in the field of microbe-microbiome interactions have led to the development of new therapeutic strategies for MS. The gut microbiota is involved in the regulation of the immune system, and alterations in the gut microbiota can contribute to the development of MS.

Figure 1: The bidirectional nature of the gut/disease axis in the context of MS. To date, the studies performed using samples obtained from MS patients and healthy individuals suggest that relative abundances of specific gut microbes are significantly altered (Table 1). Experimental evidence suggests that changes in the microbiota might affect immune, endocrine and neuronal function (a). However, it is not known whether the intestinal disruption precedes the onset of disease or the changes occur once the immunological dysfunction that results in disease is already ongoing. Therapies that target the immune system and modulate the function of key immune cells (b) could affect their interaction with the gut microbiota (c). Disease-associated immunological responses could also affect the intestinal ecosystem, by alterations in the intestinal barrier permeability for example (d). That could also result in concomitant effects on the function of immune cells. Additionally, experimental data suggest a direct effect of the microbiota on the endocrine and the neuronal system. On the other hand, changes in mood, stress, depression and other behavioral factors that occur in MS could ultimately affect the composition of the microbiota (e). MS, multiple sclerosis; IMS, immunomodulators.
Case 1: 54 Year Old Male

- A 54 year old practicing general dentist who presented with the main complaint of fatigue that has slowly worsened over last 2 years
- His MD did blood work, told him he was fine and wanted to prescribe Zoloft for depression
  - “We aren’t young bucks anymore”
Case #1: 54 Year Old Male

- **History:**
  - Sleeping 6-8 hours, up once to urinate
  - Eats a healthier version of the SAD (makes sandwiches at home, brings to work, etc.)
  - Up 20 pounds since his 20’s
  - Forces himself to exercise, recovery slow (personal trainer 2x a week)
  - Sexually active 1 time a week, would like more
  - Hobbies include surfing (too tired) and carpentry (too tired)
  - Denies anhedonia, change in appetite or sleep, inertia
Case #1: 54 Year Old Male

From HH

Medications/Supplements

- Dexilant
- Flomax
- Fish oil
- Curcumin
- Prostate Blend
Case #1: 54 Year Old Male

Notable Labs (Primary MD)

- Testosterone 566
- TSH 1.02, total T4 8.1
- CBC/CMP normal and healthy
- B12 282
- PSA 1.4
- Chol: 212 HDL 64 LDL 110 Trigs 68

Testing Options

- Adrenals
- Heavy metals
- Nutreval
- Autoimmune
- Mitochondria
- MTHFR
Case #1: 54 Year Old Male
Fatigue

**INFECTION**
- EPX ▲
- Fecal secretory IgA ▲

**INFLAMMATION**

**INSUFFICIENCY**
- Protein Products (Total) ▲

**IMBALANCE**
- Beneficial Bacteria ▼

**DIVERSITY ASSOCIATION**

**RELATIVE ABUNDANCE**
- Patient Results
- Healthy Cohort

-Verrucomicrobia Phylum
-Fusobacteria Phylum
-Euryarchaeota Phylum
- Proteobacteria Phylum
- Actinobacteria Phylum
- Firmicutes Phylum
- Bacteroidetes Phylum
- Deinococcus-Thermus Phylum

*Genova Diagnostics*
Case #1: 54 Year Old Male
Fatigue
Case #1: 54 Year Old Male

His yard...
Case #1: 54 Year Old Male
Fatigue

Gastrointestinal Microbiome

**Bacteriology (Culture)**
- Lactobacillus spp.
  - NG
- Escherichia coli
  - 4+ NP
- Bifidobacterium
  - NG

**Additional Bacteria**
- alpha haemolytic Streptococcus
  - 3+ NP
- Haemolytic Escherichia coli
  - 4+ NP
- gamma haemolytic Streptococcus
  - 2+ NP
- Enterobacter cloaceae
  - 3+ NP

**Mycology (Culture)**
- Rhodotorula species
  - 1+ NP
- Yeast, not Candida albicans
  - 1+ NP
Case #1: 54 Year Old Male - Treatment

• Have to know your patient
• Allow them to participate in choices
• Give them nutritional guidelines:
  – The bacteria within eat what we eat, if we don’t change nutrition, we will typically have a relapse!
• Always order DEXA with long term PPI
Case #1: 54 Year Old Male - Treatment

• Nutrition:
  – Fiber, fiber, fiber and fermented foods
  – Avoid the simple carbs that feed yeast

• PPI: slow wean off utilizing Mastic Gum and OTC H2 blockers (8 weeks!)

• B12 and other nutrients known to deplete when on a PPI for a long time

• GI revive (DFH product)

• UF IB and UF Spectrum (Metagenics products)

• Digestive Enzymes: DFH x 3 months
Case #2: 20 Year Old Female

- A 20 year old college student with 5 year history of linea morphea
- She had followed with several different rheumatologists at tertiary centers, had taken several different biologics, did horribly on methotrexate
- As a student, started researching and wanted to try alternative approach
Case #2:
20 Year Old Female
Case #2: 20 Year Old Female

**Inflammation and Immunology**
- Calprotectin†*: <16
- Eosinophil Protein X (EPX)†: 2.3
- Fecal secretory IgA**: 280

**Gastrointestinal Microbiome**
- Short-Chain Fatty Acids (SCFA) (Total*)
  - (Acetate, n-Butyrate, Propionate): 28.9
- n-Butyrate Concentration: 3.6
- n-Butyrate %: 12.5
- Acetate %: 53.4
- Propionate %: 34.2
- Beta-glucuronidase: 745

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*Note: Values outside the normal range are highlighted in red.**
Case #2: 20 Year Old Female

<table>
<thead>
<tr>
<th>Phylum</th>
<th>Result (CFU/g stool)</th>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
<th>5th</th>
<th>Reference Range (CFU/g stool)</th>
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<td><strong>Bacteroidetes</strong></td>
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<td>3.4E6-1.5E9</td>
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<td>Bacteroides spp.</td>
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<td><strong>Firmicutes</strong></td>
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<td>Butyricicoccus coeliclus</td>
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<td>5.5E3-5.9E5</td>
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<td>1.7E6-1.5E10</td>
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<td>Faecalibacterium prausnitzii</td>
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<td>Lactobacillus spp.</td>
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<td>8.3E6-5.2E9</td>
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<td>4.2E5-1.3E8</td>
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<td>9.5E7-1.6E9</td>
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<td><strong>Fusobacteria</strong></td>
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<td><strong>Firmicutes/Fusobacteria</strong> Ratio</td>
<td>3.8L</td>
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<td>12-620</td>
</tr>
</tbody>
</table>

**Gastrointestinal Microbiome**
Case #2: 20 Year Old Female

Gastrointestinal Microbiome **

Bacteriology (Culture)

- Lactobacillus spp.: 1+ NG, 2+ NP, 3+ NP, 4+ NP
- Escherichia coli:
  - 3+ NP
- Bifidobacterium:
  - 4+ NP

Additional Bacteria

- alpha haemolytic Streptococcus: 3+ NP
- Klebsiella ornithinolytica: 2+ NP
- Citrobacter freundii: 1+ NP
- Haemolytic Escherichia coli:
  - 2+ NP
- Pseudomonas species: 2+ NP
- Bacillus species:
  - 2+ NP
- gamma haemolytic Streptococcus: 3+ NP

Mycology (Culture)

- Yeast, not Candida albicans: 1+ NP
Case #2:
20 Year Old Female
Case #2: 20 Year Old Female

<table>
<thead>
<tr>
<th>Inflammation and Immunology</th>
<th>Gastrointestinal Microbiome</th>
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<tbody>
<tr>
<td>Calprotectin † •</td>
<td>Metabolic</td>
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<tr>
<td>&lt;16</td>
<td>Short-Chain Fatty Acids (SCFA) (Total*) (Acetate, n-Butyrate, Propionate)</td>
</tr>
<tr>
<td></td>
<td>n-Butyrate Concentration</td>
</tr>
<tr>
<td></td>
<td>16.9</td>
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<tr>
<td></td>
<td>n-Butyrate %</td>
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<tr>
<td></td>
<td>21.1</td>
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<tr>
<td></td>
<td>Acetate %</td>
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<td></td>
<td>69.3</td>
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<td></td>
<td>Propionate %</td>
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<td></td>
<td>9.7</td>
</tr>
<tr>
<td></td>
<td>Beta-glucuronidase</td>
</tr>
<tr>
<td></td>
<td>321 L</td>
</tr>
</tbody>
</table>

**Value Ranges:**
- Inflammation and Immunology:
  - Calprotectin: <16 ≤50 mcg/g ≤120 mcg/g
  - Eosinophil Protein X (EPX) †: 0.3 ≤4.6 mcg/g
  - Fecal secretory IgA: 39 · ≤885 mcg/g

**Gastrointestinal Microbiome:**
- Metabolic:
  - Short-Chain Fatty Acids (SCFA) (Total*) (Acetate, n-Butyrate, Propionate):
    - 80.1 · ≥23.3 micromol/g
    - n-Butyrate Concentration: ≥3.6 micromol/g
  - n-Butyrate %: 21.1 · 11.8-33.3 %
  - Acetate %: 69.3 H · 48.1-69.2 %
  - Propionate %: 9.7 · ≤29.3 %
  - Beta-glucuronidase: 321 L · 368-6,266 U/g
Case #2: 20 Year Old Female Follow-up
Case #2: 20 Year Old Female – Follow-up
Case #3: 55 Year Old Female

• Long term patient >10 years that had not been seen in awhile

• Diagnosed Celiac as a child, eating it because “no symptoms”

• Diagnosed MS in 2005, came to me and we took her back off gluten and surprise, the lesions resolved! (also had positive celiac panel at that time)

• Diagnosed breast cancer in 2014, she was back to eating gluten (No symptoms with exposure)

• Also now with Hashimoto’s
Case #3: 55 Year Old Female
Case #3: 55 Year Old Female

<table>
<thead>
<tr>
<th>Bacteroidetes Phylum</th>
<th>Result CFU/g stool</th>
<th>Q1</th>
<th>Q2</th>
<th>Q3</th>
<th>Q4</th>
<th>Q5</th>
<th>Reference Range CFU/g stool</th>
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<tr>
<td>Bacteroides-Prevotella group</td>
<td>1.5E6</td>
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<td></td>
<td>3.4E6-1.5E9</td>
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<td>6.4E8</td>
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<td>&lt;=2.2E9</td>
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<td>Barnesiella spp.</td>
<td>9.3E6</td>
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<td>Cotorbacter spp.</td>
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<td>Firmicutes Phylum</td>
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<tr>
<td>Anaerotruncus colitomis</td>
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<td>2.6E8</td>
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<td>1.7E8-1.5E10</td>
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<td>Coprococcus建国</td>
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<td>Proteobacteria Phylum</td>
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<td>Fasobacteria Phylum</td>
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<td>Fusobacterium spp.</td>
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<td>Akkermansia mucicarpa</td>
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<td>&gt;=1.2E6</td>
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</tbody>
</table>

| Firmicutes/Bacteroidetes Ratio | 13   |    |    |    |    |     | 12-620                      |
Case #3: 55 Year Old Female
Case #3:
55 Year Old Female
After Radiation, 
7 months after first test
Case #3: 55 Year Old Female Follow-up

<table>
<thead>
<tr>
<th>Bacteroides Phylum</th>
<th>Result (CFU/mL)</th>
<th>Reference Range (CFU/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteroides-Prevotella group</td>
<td>≥7.4E8</td>
<td>≥3.4E6-1.5E9</td>
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<tr>
<td>Bacteroides vulgatus</td>
<td>≥2.3E9 H</td>
<td>≤&lt;2.2E9</td>
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<tr>
<td>Bacteroides spp.</td>
<td>&lt;DL</td>
<td>≤&lt;1.6E8</td>
</tr>
<tr>
<td>Odoribacter spp.</td>
<td>&lt;DL</td>
<td>≤&lt;8.0E7</td>
</tr>
<tr>
<td>Prevotella spp.</td>
<td>≥1.8E6</td>
<td>1.4E5-1.6E7</td>
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<tr>
<td>Firmicutes Phylum</td>
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<tr>
<td>Anaerotrophs colitomina</td>
<td>≥2.5E7</td>
<td>≤&lt;3.2E7</td>
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<tr>
<td>Butyricobacterium crosstotus</td>
<td>≥1.9E4</td>
<td>5.5E3-5.9E5</td>
</tr>
<tr>
<td>Clostridium spp.</td>
<td>≥2.5E9</td>
<td>1.7E8-1.5E10</td>
</tr>
<tr>
<td>Coprococcus eutactus</td>
<td>≥6.9E6</td>
<td>≤&lt;1.2E8</td>
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<tr>
<td>Faecalibacterium prausnitzii</td>
<td>≥1.7E8</td>
<td>5.8E7-4.7E9</td>
</tr>
<tr>
<td>Lactobacillus spp.</td>
<td>≥3.2E9</td>
<td>8.3E6-5.2E9</td>
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<tr>
<td>Peptostreptococcus spp.</td>
<td>≥2.6E8 H</td>
<td>4.2E5-1.3E8</td>
</tr>
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<td>Roseburia spp.</td>
<td>≥6.5E8</td>
<td>1.3E8-1.2E10</td>
</tr>
<tr>
<td>Ruminococcus spp.</td>
<td>≥1.4E8</td>
<td>9.5E7-1.6E9</td>
</tr>
<tr>
<td>Veillonella spp.</td>
<td>≥4.8E7</td>
<td>1.2E5-5.5E7</td>
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<tr>
<td>Actinobacteria Phylum</td>
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<tr>
<td>Bifidobacterium spp.</td>
<td>≥2.1E9</td>
<td>≤&lt;6.4E9</td>
</tr>
<tr>
<td>Bifidobacterium longum</td>
<td>≥3.5E7</td>
<td>≤&lt;7.2E8</td>
</tr>
<tr>
<td>Clostridium perfringens</td>
<td>≥8.6E6 L</td>
<td>1.4E7-7.1E9</td>
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<tr>
<td>Proteobacteria Phylum</td>
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<tr>
<td>Desulfovibrio piger</td>
<td>&lt;DL</td>
<td>≤&lt;1.8E7</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>≥5.2E7 H</td>
<td>9.6E4-4.6E7</td>
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<tr>
<td>Oscillobacter formigenesi</td>
<td>≥3.0E6</td>
<td>≤&lt;1.5E7</td>
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<tr>
<td>Eurarchaeota Phylum</td>
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<tr>
<td>Methanoreabacter smithii</td>
<td>&lt;DL</td>
<td>≤&lt;8.6E7</td>
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<tr>
<td>Fusobacteria Phylum</td>
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<tr>
<td>Fusobacterium spp.</td>
<td>≥2.6E4</td>
<td>≤&lt;2.4E5</td>
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<td>Verrucomicrobiia Phylum</td>
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<tr>
<td>Akkermansia muciniphila</td>
<td>≥9.0E5 L</td>
<td>1.2E6</td>
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<tr>
<td>Firmicutes/Bacteroidetes Ratio</td>
<td>≥9.0 L</td>
<td>12.620</td>
</tr>
</tbody>
</table>
Case #3: 55 Year Old Female
– Follow-up

Gastrointestinal Microbiome

Bacteriology (Culture)
- *Lactobacillus spp.*
  - 2+ NP
- *Escherichia coli*
  - 4+ NP
- *Bifidobacterium*
  - 3+ NP

Additional Bacteria
- *alpha haemolytic Streptococcus*
  - 3+ NP
- *Klebsiella pneumoniae*
  - 4+ PP
- *Proteus mirabilis*
  - 4+ PP

Mycology (Culture)
- NG
Please schedule a complimentary appointment with one of our Medical Education Specialists for questions related to:

- Diagnostic profiles featured in this webinar
- How Genova’s profiles might support patients in your clinical practice
- Review a profile that has already been completed on one of your patients

We look forward to hearing from you!
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September 27, 2017 – Thomas Guiliams, PhD Presents:

**Supporting Gut Barrier Function**

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GI Effects in Clinical Practice:
Focusing on the Gut

Rebecca Hunton, MD
August 23, 2017

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