

## GI Map Results

**For:** Heather Witts

**Date:** 13/12/24

### **Most relevant findings:**

#### **1. Helicobacter pylori**

**High (1.32e3\*)** - reduces stomach acid with multiple downstream effects, nutritional implications, compromised digestion, opportunistic overgrowths and dysbiosis. Associated with many skin conditions including psoriasis, and recent studies showing links to migraine.

\*1.32e3 = this is equivalent to 1,320 microorganisms per gram of stool

#### **What does stomach acid do?**

- Kills microbes that come into our digestive tract - like a guardian of our stomach
- Partially denatures protein in food - begins the process
- Breaks down amino acids into smaller fragments - the building blocks that are essential for hormone and neurotransmitter production, energy, growth (skin, hair etc), repair, healing
- Stimulates/triggers release and flow of pancreatic enzymes and bile > both of these have huge antimicrobial properties which further inhibit bacterial overgrowths

#### **What causes low stomach acid?**

- Sympathetic dominant state (ie. stress, rushing) affects the release of stomach acid, as opposed to a parasympathetic state which is known as 'rest and digest'
- Age
- *H.pylori*: the presence of this in the stomach neutralises stomach acid
- Medications: PPI, antacids

#### **2. Commensal/keystone bacteria**

These are the representative members of the normal microbiome. They can provide key insights into the overall health and function of the microbiome, as well as the abundance and diversity. Also known as the 'good bacteria' these play vital roles in supporting our digestion, hormone and neurotransmitter production.

- High *Enterococcus spp*: linked with reduced digestive capacity, constipation, SIBO
- *Akkermansia muciniphila*, below detected level (<dl) this species resides in the mucus layer of large intestine, is very protective to the gut and has been shown to help maintain intestinal integrity (by down-regulating the leaky gut protein, zonulin). It also acts as a 'biofilm buster' organism. It's known to modulate the metabolic rate, low levels are associated with weight gain, obesity, insulin resistance and metabolic dysfunction.
- Studies have linked a lack of this microbe to psoriasis and GI diseases (Crohn's, Ulcerative colitis) and Type 2 diabetes.

### **Bacterial phyla**

This provides a high level view of the microbiome based on the fact that these phyla are the top of the taxonomy hierarchy. When represented as either high or low, they indicate a significant imbalance in the microbiome.

- Low Bacteroidetes - this needs an acidic colonic environment to thrive, *H.pylori* will therefore affect this and look at mucin layer health to support its growth
- Low Bacteroidetes: Firmicutes ratio

### **3. Opportunist/overgrowth microbes**

- High *Staphylococcus aureus* - will overgrow when there is low stomach acid, inflammation, constipation will also contribute to overgrowth. A mast-cell activating microbe, links with inflammatory skin conditions.
- High *Strep spp* - low stomach acid, reduced digestive capacity, constipation, inflammation, skin link, and oral microbiome link.
- Moderate presence *Bacillus spp* - linked to poor digestive function, constipation
- Moderate presence of *Enterococcus faecalis* & *Enterococcus faecium* - linked to reduced stomach acid, compromised digestion, constipation and high intake of refined carbohydrates and sugars. May change stool formation and alternate diarrhoea/constipation (IBS) type symptoms.

### **4. Intestinal health markers**

- **Raised Steatocrit (8)** - reduced digestion/absorption of fats, related to pancreatic enzymes and bile salts.

Bile has significant antimicrobial effects in the gut so bile secretion (involving liver & gallbladder) is impaired bacteria can overgrow, creating gut dysbiosis.

Often see raised steatocrit in combination with low elastase as these are interlinked.

Also often see it raised when there's low stomach acid. This then effects the pH of the gut, which has many downstream affects, particularly on microbiome.

Indicates a low 'digestive fire' which has downstream affects to both the gut environment and absorption of nutrients.

There's a relationship with oestrogen as it also affects bile production and secretion; both high and low oestrogen can cause decreased bile flow.

Often linked with low stomach acid, because having adequate stomach acid plays a role in signalling to and stimulating the production and release of these enzymes.

- **Low Elastase (182)** - poor pancreatic enzyme output, possible low stomach acid (HCL) optimal 500

This is just one of the several pancreatic (digestive) enzymes; it's been found that if elastase is low then the others are often low too, so it's a good marker for pancreatic function.

When low, both digestion and nutrient absorption will be affected.

Often see as low alongside low stomach acid; this contributes to certain organisms overgrowing, often the opportunistic/overgrowth microbes.

Anything < 200 is considered pancreatic insufficiency, ideally like to see it at 500.

Often related to mineral deficiencies (eg. iron, B12) as critical steps start up the top of the digestive tract.

Commonly also seen when there is *Helicobacter pylori*, either moderate presence or flagged as high.

Common causes of low pancreatic output: low stomach acid (hypochlorhydria), vegetarian diet, gallstones, suppressed pancreatic function.

- **Very high Beta-glucuronidase (3997)** optimal 500-1000

This is an enzyme produced by certain species of intestinal bacteria and also cells of our body (liver, kidneys, the gut wall, reproductive organs).

This result is a marker of the *activity* of the enzyme, and not the quantitative *amount* of the the enzyme.

This enzyme essentially 'undoes' what the liver did to the various toxins, hormones (our own and environmental xeno-oestrogens), xenobiotics, mould mycotoxins, medications when they when through the liver detox pathways. The liver binds up these toxins (through a process called glucuronidation) in preparation for their excretion from the body via bowel. This enzyme breaks this bond, undoing the liver's work and essentially frees up these toxins, allowing them to be reabsorbed and go back into circulation. High levels seen with bacterial overgrowth, lack of beneficial bacteria, antibiotics use, dietary factors, liver stress, lifestyle factors.

This enzyme is produced a a subset of bacteria, which major producers in your case being *Staph*, *Strep* and other overgrowths.

Having a higher colonic pH (ie. less acidic) is associated with higher Beta-glucuronidase activity.

Liver detox stress: 40-70% of medications are detoxified through the glucuronidation pathway.

High meat, high fat, low fibre diets and alcohol have been associated with higher activity levels.

BPA or BPS lined cans/bottles will contribute.

A higher level of activity:

- is associated with hormonal imbalance when >1000
  - is associated with an imbalance of oestrogen to progesterone, and creates a back-log in the clearance of oestrogen metabolites through your liver > a leading cause of hormonal migraines. Oestrogen also increases histamine which also contributes to hormonal migraines.
  - may lead to high oestrogen/excess circulating oestrogen > increases symptoms of PMS, heavy/painful periods, breast tenderness, fibroids. Excess oestrogen may lead to specific breast cancers.
  - *may* increase cancer risk as hormones and toxins re-enter bloodstream instead of being eliminated.
- Associations with breast and colon cancer.

- **Occult blood (1)**

A measurement of Hb found in the stool. Low levels (ie. 4-5-6) may be due to possible haemorrhoids, anal fissures, on period with sample collection and/or dysbiosis or pathogenic overgrowth.

- **Very high Secretory IgA (3308)** - elevated immune response, optimal 1200

This is our primary immunoglobulin in the intestinal mucosa. It's involved in immunological surveillance, and is our first line of defence for antigens and pathogens. It also monitors and balances the microbiome. When elevated, it's basically an 'over active immune response' to an antigen of some sorts; could be a food antigen/sensitivities or pathogenic in nature.

Can also be linked to autoimmunity, acute stress and *H. pylori* can also contribute.

- **Anti-gliadin IgA (81)** - a marker to look for non-coeliac gluten sensitivity (NCGS), optimal 40-50

Gliadin (from gluten) can stimulate intestinal immunity and increase levels of this immunoglobulin, AG-IgA. When 50-100 consider pulling out gluten/hidden gluten to give the gut a break, especially if inflammation is reflected with calprotectin so remove gluten and hidden gluten.

There can also be an issue with cross-reactive foods, because proteins can appear very similar - eg dairy, wheat, oats, corn, millet. Until gut repair is done, best to take them out then wait to retest, then may be able to reintroduce them.

- **High Calprotectin (50)** - low grade inflammation, optimal <50

This is the gold standard marker for GI inflammation, a great indicator for inflammation in the colon.

Also consider the gut-brain axis and inflammatory status associated with migraine.

Sometimes low levels of calprotectin can be associated with non-coeliac gluten sensitivity because of the irritation occurring to the gut lining.

Taking NSAIDS can also affect it.

When calprotectin is high, it shows the mucin layer is compromised - and as the *Bacteroides* phyla lives here (reported as LOW), plus *Akkermansia* (reported as <dl) feed on and regenerate the mucin layer, this indicates it needs support also.

Consider the gut-brain axis with inflammation.

- **Zonulin (151)** - 'leaky gut' marker, optimal 40-50

This is the protein that opens up the intercellular tight junctions in the mucous membranes, so it's reflective of the "degree of permeability" occurring in the mucosa of the gut wall.

Zonulin production is stimulated by high levels of certain overgrown bacteria.

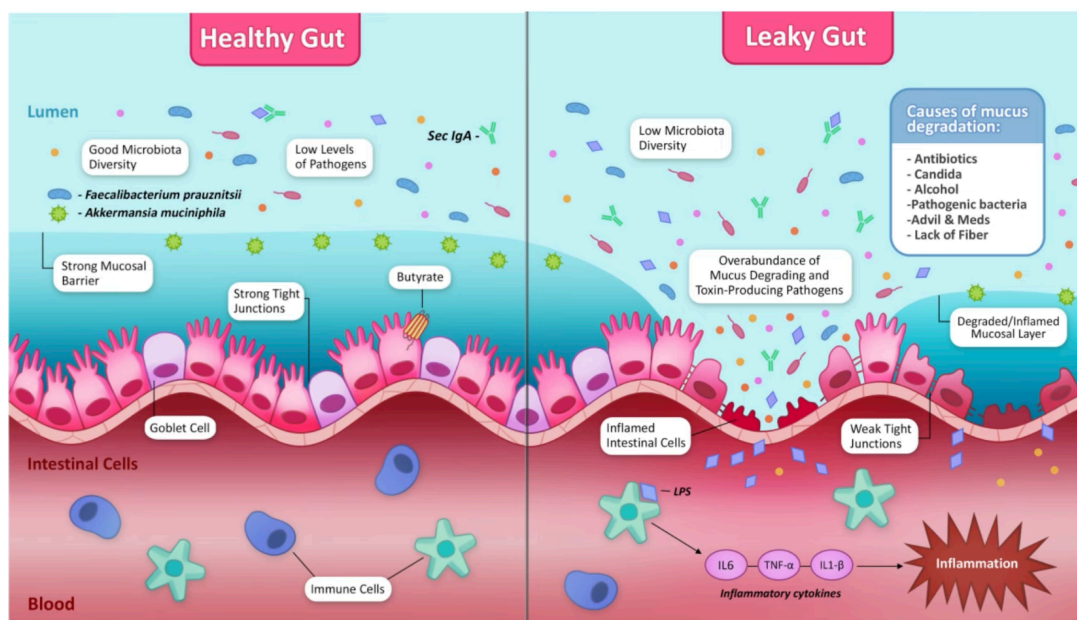
When it's elevated, especially when combined with bacterial overgrowths, it allows not only the bacteria - but the bacterial metabolites (eg. LPS, endotoxins) and any other antigens that might be from food - to translocate outside of that lumen surface/lumen area and have free access to other more systemic parts of the body too > skin issues, brain fog, systemic inflammation.

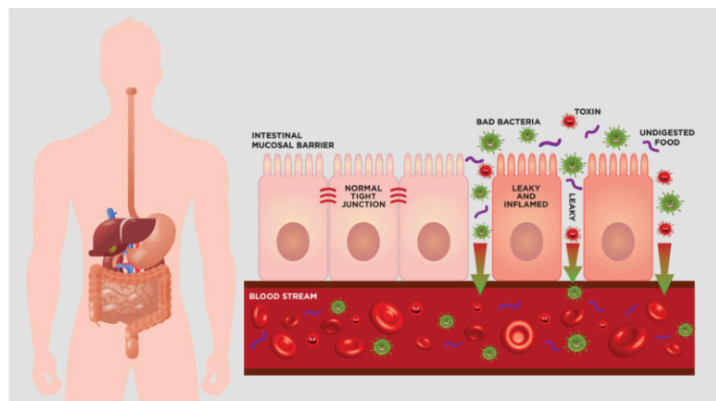
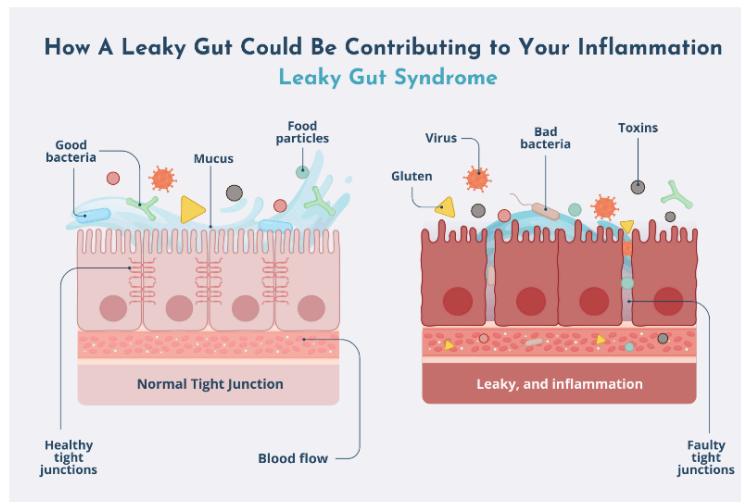
It's linked to chronic stress, presence/high *H.pylori* and low/nil *Akkermansia* as this microbe usually down-regulates zonulin, protecting against intestinal permeability.

### MOST RELEVANT FINDINGS:

1. **Infection:** *H.pylori*
2. **Opportunistic dysbiosis:** Bacterial overgrowth (Staph, Strep) and moderate presence of *Bacillus spp*, *Enterococcus faecalis* & *Enterococcus faecium*
3. **Low keystone species:** <dl *Akkermansia muciniphila*
4. **Digestive insufficiency:** raised Steatocrit, low Elastase
5. **Poor detoxification:** high beta-Glucuronidase
6. **Increased immune activity:** high Secretory IgA
7. **Inflammation:** raised Calprotectin
8. **Increased intestinal permeability:** raised Zonulin

These images I referred to from 36min onwards (at times) but they didn't show in the video!





## INTESTINAL DYSBIOSIS- PSORIASIS

Skin disease	Lower levels of beneficial gut flora	Higher levels of pathogenic bacteria	Correlated GI diseases
Psoriasis	<i>Lactobacillus</i> <i>Bifidobacteria</i> <i>Faecalibacterium prausnitzii</i> <i>Akkermansia</i>	<i>Escherichia coli</i> <b><i>Streptococcus pyogenes</i></b> <i>Klebsiella pneumoniae</i> <i>Enterococcus faecalis</i> <i>Salmonella</i> <i>Helicobacter pylori</i> <i>Campylobacter</i> <i>Mycobacterium</i> <i>Alcaligenes</i>	IBD- UC, Crohn's SIBO Celiac disease

## THE GUT-SKIN CONNECTION



## PSORIASIS STREP CONNECTION

▶ **Strep throat** can trigger guttate psoriasis

Strep throats can make plaque psoriasis **worse**

**Tonsillectomy** can help improve psoriasis

**RCT: 15 psoriasis patients** underwent tonsillectomy

**13/15 (87%)** saw improvement of their psoriasis (from 30%-90%)