

Heartburn, Excess Gas, Acid Reflux, Irritable Bowel?
Arthritis, Chronic Pain, Anxiety, Bad Skin?



GO WITH YOUR GUT

The 5-Part Plan For Healing Gastrointestinal Issues
& Preventing The Diseases That Come With Them

GO WITH YOUR GUT

The 5-Part Plan For Healing Gastrointestinal Issues & Preventing The Diseases That Come With Them

MIKE SHERIDAN

Copyright © 2016 Lean Living Inc. - All rights reserved. No part of this publication may be reproduced, distributed or transmitted in any form or by any means, including photocopying, recording, or other electronic or mechanical methods, without the prior written permission of the publisher, except in the case of brief quotations embodied in critical reviews and certain other noncommercial uses permitted by copyright law. For permission requests, email the address below with the subject line "Copyright Permission."

contact@leanlivinginc.com

Disclaimer - The ideas, concepts and opinions expressed in this book are intended to be used for educational purposes only. This book is sold with the understanding that author and publisher are not rendering medical advice of any kind, nor is this book intended to replace medical advice, nor to diagnose, prescribe or treat any disease, condition, illness or injury. It is imperative that before beginning any diet or exercise program, including any aspect of the training, nutrition, or lifestyle recommendations made in this book, you receive full medical clearance from a licensed physician. The author and publisher claim no responsibility to any person or entity for any liability, loss, or damage caused or alleged to be caused directly or indirectly as a result of the use, application or interpretation of the material in this book.

Table of Contents

<i>Introduction – “All Disease Begins In The Gut”</i>	4
<i>Part 1 - Avoid Antibiotics or Counteract With Probiotics</i>	7
<i>Part 2 - Stop Taking Antacids & Restore Stomach Acid</i>	12
<i>Part 3 - Get to Know SIBO & Consider an Intervention</i>	18
<i>Part 4 – Balance Sympathetic & Parasympathetic Stress</i>	24
<i>Part 5 – Understand What NSAIDs Do To Your Gut</i>	28
<i>Next Steps – Live It NOT Diet! Updates + \$5-off 1% Fitness</i>	32
<i>References</i>	33

INTRODUCTION

"All Disease Begins In The Gut." - Hippocrates

It's common to overlook the health of our gastrointestinal system, even though it contains 10 times more health-determining bacteria than the rest of our body. Protecting us from infection, supporting our metabolism, and promoting healthy digestion and elimination.

There are over 100 trillion organisms in the gut, that make up ¾ of our immune system.

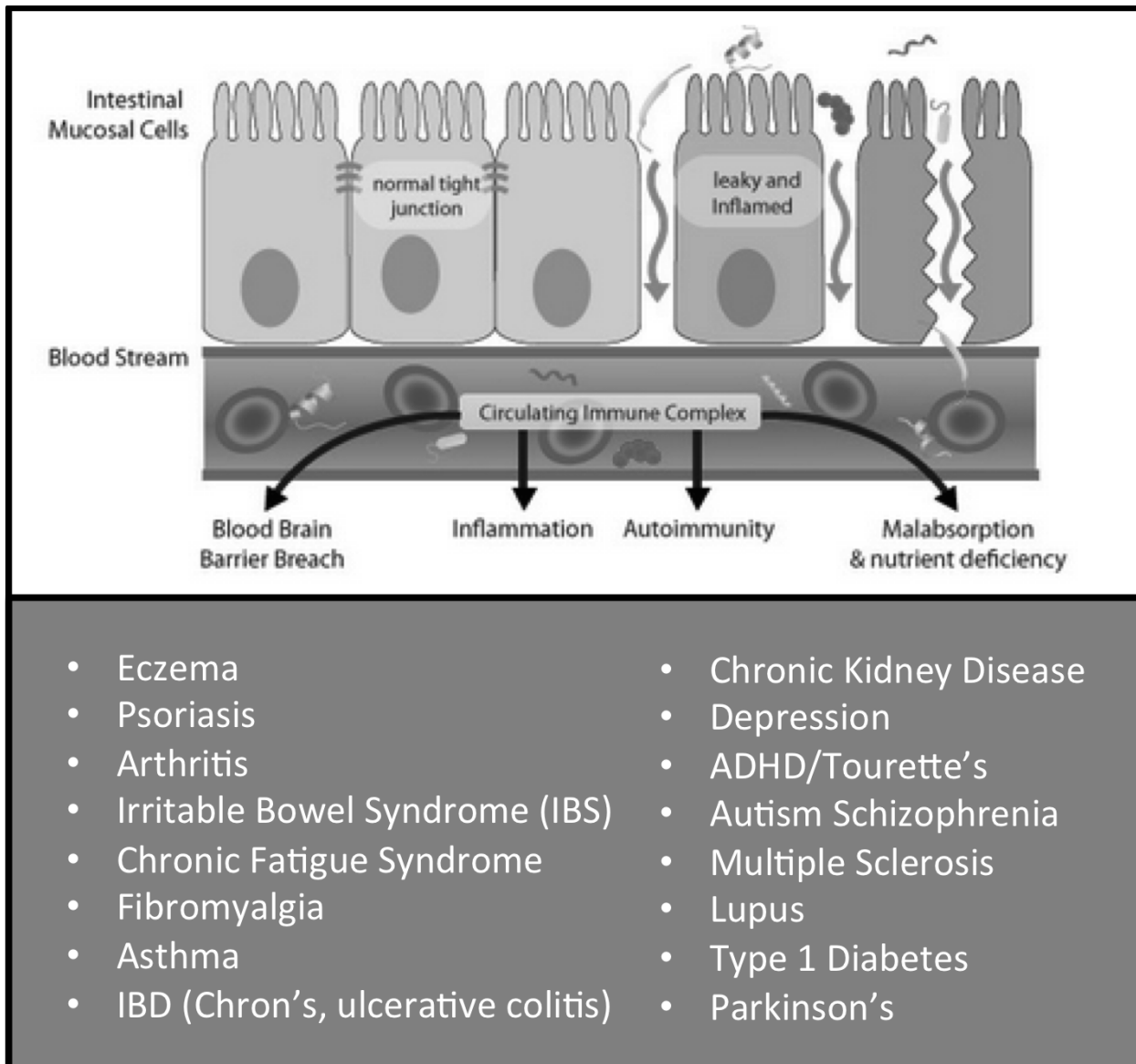
Unfortunately, the majority of the population has inadequate beneficial (good) bacteria, excessive damaging (bad) bacteria, and a lack of bacterial diversity. Largely because of a poor diet, but also because of:

- *Over-Medicating* – with antibiotics, birth control, NSAID's, anti-depressants, antacids, etc
- *Chronic Stress* – which can alter digestive secretion, gut permeability, blood flow, sensitivity, and even change bacterial composition
- *Exposure to Environmental Toxins* – notably pesticides, PCBs, herbicides, arsenic, BPA
- *Inadequate Bacterial Acquisition at Birth*
 - C-section birth
 - Parents gut health and mothers diet during pregnancy
 - Infants transition from breast milk to adult food
- *Current Health & Body Composition* – Poor overall disease status

And this is ultimately leading to unbalanced gut flora (dysbiosis), and an increased susceptibility to intestinal permeability (leaky gut). Since the same things that destroy our gut flora, can compromise our gut barrier; while the fungal infections and bad bacteria that prosper instead, can do the same.

The scary part being, that for some this presents itself as something that seems relatively harmless like bloating, heartburn, IBS, and excess gas, while for the not-so-lucky ones it can mean something more serious like a chronic inflammatory condition of the skin, joints, bowel, or brain and nervous system.

This is because, although the gut is inside our body, it's actually an external organ, that's main role is to prevent harmful substances from entering our body. So when that barrier (that's supposed to separate us from the external environment) is compromised, large foreign molecules are able to pass through to the bloodstream, and serious damage ensues. As the body launches an immune response to protect itself, but ends up damaging it's own organs and tissues. With the latest research clearly showing that a leaky gut is connected to 100's of untreatable (and often unexplainable) medical conditions.



Meaning, your best chance of finally getting it to go away, or at least getting control of it, is repairing the cause (your gut), not treating the symptom (your condition).

Unfortunately, this is usually the part where people get very angry or stop listening. Because if this information is true, which the latest research suggests it is, the underlying message is that your sub-optimal health, or critical illness is your fault. Since the damaged gut is a result of your behaviour; whether that's choosing to medicate, choosing to eat certain foods, or letting yourself get exposed to harmful things.

But here's the thing – it's not your fault!

Because all you did was listen to *the 'experts'* (doctors, government, etc) and select the options you were given; the same options everyone else has been given, and the same choices everyone else has made. But unfortunately you (or those you love) may have a little less genetic protection on your side. Or perhaps you didn't have as strong a shot from the outset (ex: vaginal birth instead of c-section, no antibiotic exposure as a child, etc).

You're not a bad person, you're not a bad parent, and the fact that you're still reading this probably means you're a good one.

What this should serve as, is hope. Because it means you have the opportunity to upgrade your health and performance. And for those dealing with (or helping a loved one deal with) an unfortunate gastrointestinal problem (Chron's, Colitis, celiac, etc), heartbreaking mental disorder (ADHD, depression, autism) or devastating neurological condition (epilepsy, Parkinson's, multiple sclerosis), potentially turn your life around!

So, what's the harm in trying?

There's obviously no guarantee, but the evidence leads us to believe that these problems originate in the gut, and healing the gut (with *Live It NOT Diet!* and the protocol below) can lead to dramatic improvements; whether we're talking about allergies and skin conditions or mental and physical performance.

And sure, not every disease is the result of a leaky gut, and not everyone will develop a leaky gut. But with the evidence we have to-date (over 10,000+ papers) proving that this 100-year old hypothesis is no longer quackery, there's no reason why you shouldn't be doing everything you can to improve the integrity of your gut lining, encourage a positive bacterial environment, and optimize the overall function of your gastrointestinal system (digestion, absorption, elimination, etc) going forward.



PART 1

Avoid Antibiotics or Counteract With Probiotics

Gone are the days of believing that our gut microbiota remains stable once it's established as an infant. As research clearly shows dramatic changes after drug treatment, and considerable differences from dietary input. Meaning, our day-to-day behaviour can cause long-term alterations to our gut microbiota that positively or negatively affect our health and body composition. Whether we're a growing-child or a fully-developed adult!

The microbiota is a tenant in our gut - with 100-150 times more genes than the human genome - that has the ability to turn our genes on or off based on what we feed it, and how we treat it.

Or simply put, we should be doing our best to keep this creature (our neighbour) happy. Since the healthier it is, the healthier we are; whether we're talking about immune protection and disease prevention, obesity and insulin resistance, or physical and cognitive performance.

Unfortunately, as discussed in the introduction, most of us *haven't* been taking care of our microbiota; especially over the last 50 years. As we've disrupted it's home, cut off it's food supply, and attacked it directly with antibiotics, crappy foods, and chronic stress. Leaving behind a gastrointestinal system that's overthrown by unfriendly bacteria (dysbiosis), chronically exposed to inflammation, and ready to break – if it hasn't already.



Arguably, one of the biggest contributors to this destruction is the introduction and rampant use of antibiotics. Since one of these treatments alone can wipe out beneficial gut bugs for years to come – and potentially never restored.

A study from 2008 in the journal PLoS Biology tested a single 7-day course of antibiotics, and saw no signs of the bacteria that was lost for over 2 years!

Without beneficial bacteria (healthy flora) in our gut we're more susceptible to damage and infection. As we lose our natural protection from 'bad' bacteria, that start setting up shop (colonizing) in our gastrointestinal tract.

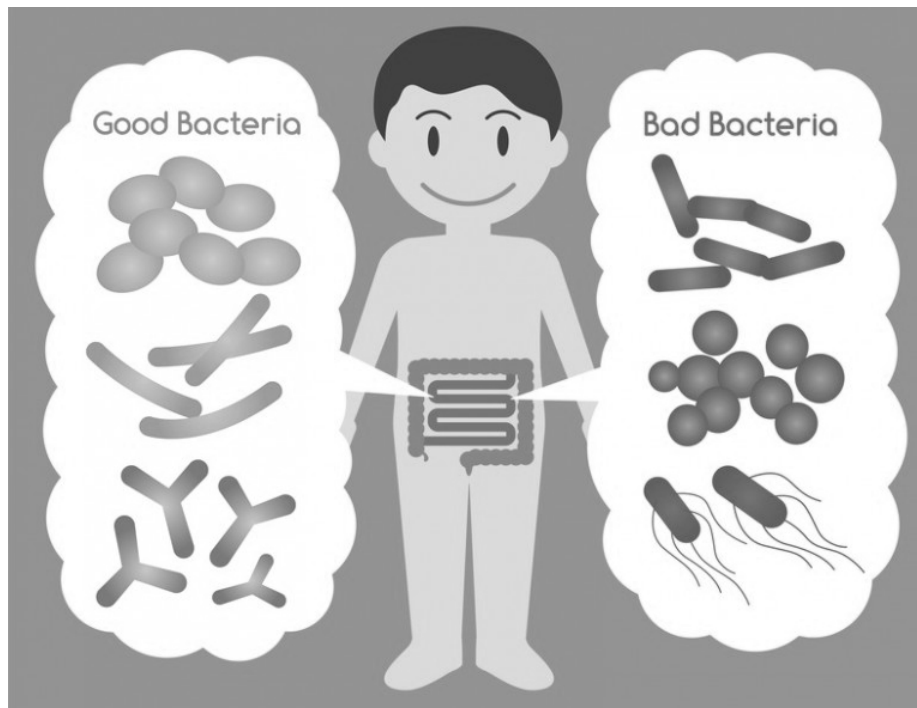
For instance:

Research has demonstrated that individuals with Irritable Bowel Disease (Chron's, colitis) have lower levels of protective bacteria (bifidobacteria, lactobacillus) and have found similar imbalances with other (non-gut-related) auto-immune conditions.

Now, whether the poor gut flora or infection is causing the leaky gut that's driving the auto-immune condition is debatable. But we do know that a bacterial balance is necessary for a proper immune response, and antibiotics strip away the healthy gut flora that would have otherwise provided an extra layer of support.

RESTORE YOUR FLORA

Not everyone has holes in their gut, or the auto-immune conditions that develop as a result, but everyone has the potential to develop them; and probably has the unfavorable ratio of good-to-bad bacteria that precedes it. Especially if they're eating (or had been eating) anything close to a diet that resembles the Standard American Diet, they've done a round or 2 of antibiotics, and they've been exposed to a considerable dose of environmental toxins, pharmaceutical drugs, and stress.



STEP 1 – Avoid Antibiotics When Possible

In some cases, antibiotics are absolutely necessary; so we can't avoid them entirely. But it does mean looking at the risk-to-benefit ratio of taking them, and counteracting the negative effects when they are.

Antibiotics compromise gastrointestinal health, so healing the gut after (and during) treatment is critical.

Obviously, no antibiotics would be ideal, and it's important to weigh your options when the opportunity presents itself. But sometimes it's required, which makes damage control a necessity.

STEP 2 – Take Probiotics During & After Treatment

Interestingly, supplementing [a probiotic](#) during antibiotic treatment seems to be beneficial. Which may sound goofy (since antibiotics kill probiotics), but research has shown less side effects and a lower risk of infection. With one study from 2005 in the journal of International Immunopharmacology showing the superiority of a probiotic supplement 'during and after' antibiotic treatment, compared to 'after' only.

STEP 3 – Compliment With Gut-Feeding PRE-biotics

This is the step that people forget, as they don't realize that the probiotics they're supplementing need something to eat to stick around. So, make sure your *Live It NOT Diet!* plan includes plenty of gut-feeding vegetable matter, and plenty of onions and garlic. The best fruits are the ones with a high amount of polyphenols and soluble fiber, and a low glycemic response – like berries.

ONGOING GUT SUPPORT

A variety of fermented (or cultured) foods are consumed across the globe as a dietary staple. Not only because they're an excellent way to increase the shelf-life of certain foods, but because they have tremendous health benefits.

- Improving digestion and elimination
- Enhancing immune function
- Increasing nutrition absorption
- Preventing 'bad' bacterial overgrowth
- Lowering inflammation
- Improving mood and cognitive function
- Treating gastrointestinal conditions
- Reducing body fat

Unfortunately, it's a different story in North America. Where personal appliances, grocery stores (on every corner), and preservative packed foods have removed the need for fermentation. While, at the same time, making the gut-supporting, disease-preventing nutrition knowledge disappear.



Realistically, we should be consuming fermented foods on a regular basis; instead of supplementing probiotics. As once our round (or two) of restoring our flora is over, these foods provide ongoing support from a natural source, with a higher bacterial count, and more bacterial diversity. Helping you work towards a level of bacterial variation that was typical of the disease-free hunter-gatherers that came before us.

The easiest way to introduce fermented foods, is to start with coleslaw or vegetables that you're familiar with (carrots, cucumbers, peppers), and then moving on to a more foreign addition like kimchi or sauerkraut (which may not be 'foreign' to you). Fermented dairy products (yogurt, kefir, sour cream) can also be great, but not when they're from a grocery store. As this usually means 'pasteurized' dairy, and lots of insulin spiking sugar additives.

For those with access to raw milk (or grass-fed at the very least), making your own kefir at home is encouraged. Since it provides way more bacteria than you'd ever find in a probiotic supplement (>100 times), and it's way more affordable. With kefir grains being cheaper than probiotics from the outset, and providing the ability to reuse them several times before you need to purchase more.

[Note: They're called kefir grains, but they're not a grain like wheat, oats, and barley]

Essentially, you purchase a kefir starter granule, and leave it in some raw milk for approximately 12-24 hours. Once it's transitioned into kefir, you store it in the fridge and consume it as needed – a few tablespoons before bed (like your probiotic supplement) seems to do the trick.

Raw yogurt and milk can also be extremely beneficial. Although to avoid any negative impact on your physique, they're best consumed after exercise, when whey and insulin is more friend than foe.



Fermented vegetables can be consumed whenever you like (ex: kimchi, sauerkraut, etc), but starting with a small serving at dinner a few times per week is recommended at first. Mainly to get used to the taste, but also because too much at once can be quite potent.

Vegetable Fermentation Directions:

1. Choose your vegetable(s) – onions are overpowering; cabbage is a great base
2. Peel vegetables with a skin and place in mason jar
3. Add herbs and spices (optional) – garlic, ginger, basil, etc
4. Mix water or celery juice (preferred) with a starter culture - this is the brine
5. Pour brine into the mason jars to cover the vegetables completely
6. Put the lid on loosely and store in a relatively warm (approx. 72 degrees) location
7. Check on your mix after 4-7 days, and move to the refrigerator when you're happy with the flavor

To save money, you can use salt and water instead of a 'starter culture,' but the process takes a lot longer and doesn't provide as much bacterial diversity.

PART 2

Stop Taking Antacids & Restore Stomach Acid

Medical doctors and TV commercials may have you believing otherwise, but the underlying problem with heartburn or reflux is too little acid, not too much. As the malabsorption of foods increases intra-abdominal pressure that bloats the stomach, pushes the contents of the stomach up, and drives whatever acid and digestive juices that are present there into the esophagus.

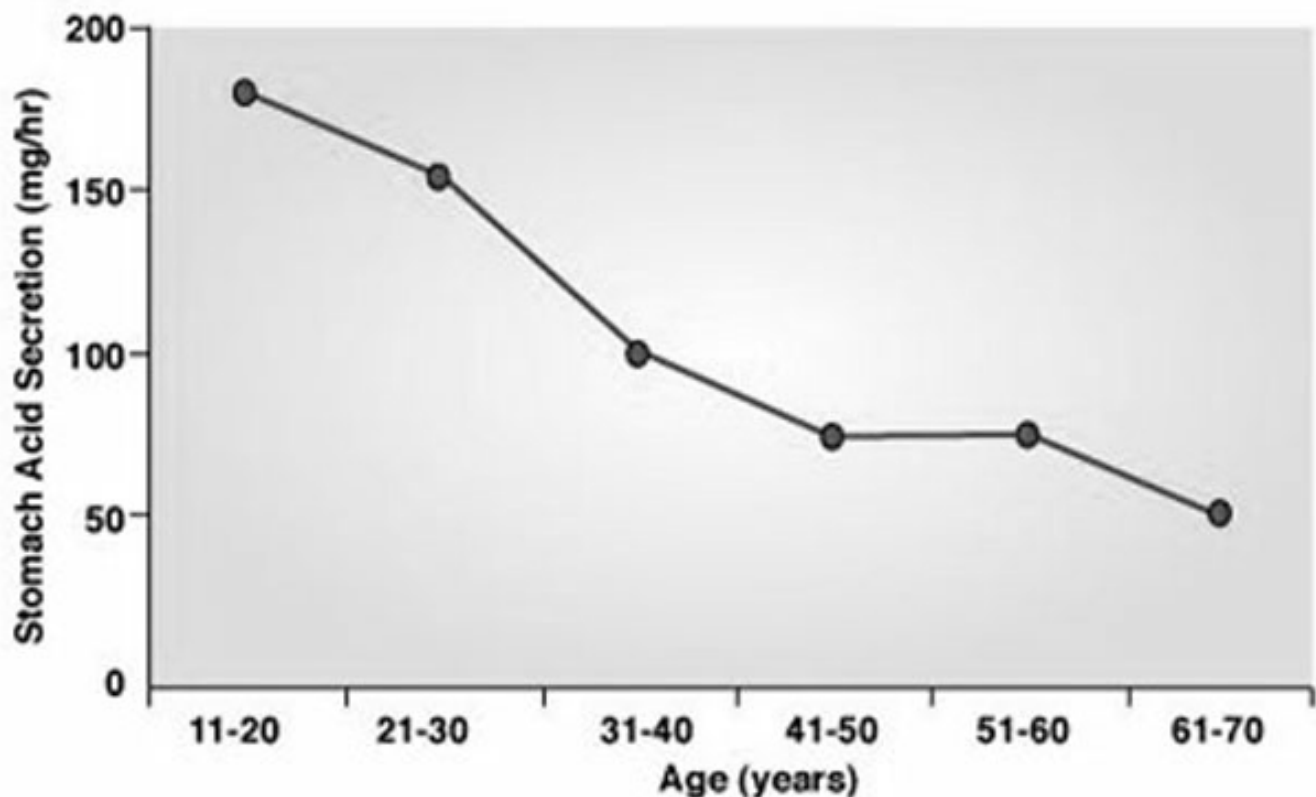


Fig. 1. Contrary to popular belief, stomach acid secretions drop with advancing age. This graph shows average decline in stomach acid secretion in humans between age 20 to age 80. (From "Why Stomach Acid is Good For You.")

Or put another way, by taking an antacid to lower stomach acid (which is already declining with age), we're only making things worse. And not just because of our hampered ability to break down and absorb the nutrients in our food, but because low stomach acid permits bad bacterial overgrowth - which may actually be what's driving the stomach acid decline in the first place.

Acid-suppressing drugs are hampering our ability to digest food properly (causing bacterial overgrowth), and decreasing the acid that suppresses bacterial overgrowth.

For instance, there's evidence to suggest that 50% of people worldwide are infected by *Helicobacter pylori* (*H. pylori*) - a bacteria that burrows itself in the mucus lining of the stomach and releases an enzyme (called urease) that reduces stomach acid (so it can survive). Yet, over 80% of those infected never exhibit any noticeable symptoms.

The disturbing part being, that the infection occurs when stomach acid is low (i.e. taking antacids), the bacteria is fueled by poorly digested starches (i.e. the western diet - fructose, wheat), and the infection rate increases with age (by 1% for every year of life). Meaning the carb-loading, antacid-popping, baby-boomers are the perfect candidates. And once their infected, the slippery slope of the acid-lowering infection, heartburn-like symptoms, and acid-lowering medication only gets worse.



One study from 1990 found that long-term use of the acid-suppressing drug Prilosec, reduced HCL secretion to zero!

But that being said, whether you have *H. pylori* or not, and whether you're a baby boomer or not, you need to assess your level of stomach acid and more than likely take action to maximize it. Given that the combination of dietary and lifestyle factors that have become common for North American's all contribute to an underactive stomach and poor digestion on their own:

- Chronic stress
- Excessive antibiotic use
- Nutrient deficient food (as enzyme production requires specific nutrients, or "co-enzymes," like animal-derived B12 and iron)

And given that failure to do so could result in inadequate enzyme production (protein, lipase, etc) and food breakdown in general. Meaning less nutrient absorption, inflammation of the gut lining (gastritis), and an increased susceptibility to infection - from H. pylori and other parasites (Cryptosporidium, Blastocystis hominis, etc), or bad bacteria overgrowth (Salmonella, E. coli, C. albicans, etc) and SIBO in general.

A study from 2002 determined that 71% of patients with GERD (Gastroesophageal Reflux Disease) also tested positive for IBS.

THE DIGESTION TEST

The following test is worthwhile for gauging where you're stomach acid is at. The goods new being, that it costs next to nothing, and you can administer it yourself. And the better news being, that if you do find out you're on the low side, you can start taking action to improve your absorption of essential nutrients (B12, iron, etc), and reduce your risk of future gastrointestinal issues (or the unfortunate conditions that accompany them).

STEP 1 - Purchase a Betaine HCL Supplement

Make sure it contains Pepsin or acid-stable protease, because, a stomach not producing enough HCL, is usually not producing enough protein digesting enzymes either. Here's an example of the contents in [one I'd recommend](#) from *Designs For Health*:

STEP 2 - Memorize These 3 Critical Dosage Points

- The test is only performed at a meal with a significant amount of protein (usually lunch and dinner)
- Each advancement in capsules (ex: 1 to 2) happens at next main meal (not the same one!)
- Stop progressing to more caps when you feel a burn OR reach 6 capsules

Supplement Facts	
Serving Size 1 capsule	
Amount Per Serving	% Daily Value
Betaine HCl	200 mg *
GastroENZ™ Proprietary Blend	180 mg *
Ox Bile Extract, Protease (DPPIV), Amylase, Pepsin, Protease SP, Glucoamylase, Lactase, Acid Protease, Invertase, Lipase	
* Daily Value not established.	

STEP 3 – Follow The 6-Step HCL Protocol

1. Eat half of the protein in your meal first (ex: half of a chicken breast)
2. Take capsule(s), starting with 1
3. Eat rest of protein + remainder of meal
4. If you feel burn (like heartburn, warm sensation) within 10-15 minutes of taking capsules, STOP THE TEST.
5. If you don't feel burn within 10-15 minutes repeat steps 1-4 AT YOUR NEXT BIG MEAL (LUNCH OR DINNER), adding 1 more capsule (up to a maximum of 6)
6. Once you've found the proper dosage for you (1 cap less than when you felt the burn). Stick with that dosage at main meals until you get the burn. Then lower dosage again by 1 cap and continue supplementing at that dosage.

Unfortunately, there's usually some confusion with this. So, here's an example scenario:

- Tuesday Dinner – Take 1 capsule. No burn.
- Wednesday Lunch – Take 2 capsules. No burn.
- Wednesday Dinner – Take 3 capsules. No burn.
- Thursday Lunch – Take 4 capsules. Feel burn.
- Thursday Dinner – Dose 3 capsules. No burn
- Friday Lunch – Dose 3 capsules. No burn

...continue dosing 3 capsules at main 'protein-containing' meals

- Lunch 2 weeks later – Dose 3 capsules. Feel burn.
- Dinner – Dose 2 capsules. No burn

...continue dosing 2 capsules at main 'protein-containing' meals

- Etc, etc

Once you feel a burn at 1 capsule, you no longer have to take the HCL capsules; as it means we've kick-started your acid production. Although, you may want to try the test again in the future if you start experiencing any of the symptoms listed above. Or, may want to take the odd capsule after a very large or heavy meal (all-you-can-eat sushi on a Feast Day perhaps!?).



An important point to keep in mind, is that this isn't an overnight process. With most middle-aged individuals taking anywhere from 3-6 months (with consistent HCL supplementation), and a small minority having to supplement for years without ever making any progress (because of over-medication in the past).

Research suggests that 30% of women over 60 have inadequate gastric secretion, while 40% of women over 80 have no gastric secretion at all.

If that's the case for you (no improvement after 6 months), you can either come to the grips with the fact that HCL supplementation may be required for the rest of your life, and perhaps switch to a more concentrated [Betaine HCL supplement](#) (so you're not taking 6 capsules), OR try step 4.

STEP 4 – Consider Herbal Supplement to Eradicate H. Pylori (like [this one](#))

But whatever you decide, it's essential that you create an environment where you have enough stomach acid to digest your food, absorb the nutrients in it, and prevent infection. While incorporating some diet- and lifestyle-related best practices, so you're primed for a promising digestive future.

ONGOING SUPPORT – VINEGAR

Simply put, vinegar is to digestion, as fermented foods are to gut flora - *The 'real food' supplement that came well before pills and capsules.*

Initially, vinegar was used as a food preservative, until it's long-list of medicinal benefits were discovered. With some cultures (like the Chinese) using it as a sanitizer over 1000 years ago, and others using it as a go-to for treating wounds and ailments.

Sadly, we've lost sight of many of these traditional uses; especially when it comes to improving the strength and health of our gastrointestinal system. With the majority of us opting to visit the local pharmacy to choose from 1 of 10 digestive aids, or make an appointment with their over-prescribing doctor to grab another round of reflux medication.



As discussed, antacids are a counterproductive approach to treating indigestion, and antibiotics may kill pathogens, but they also kill "healthy" gut bugs. Leaving us prone to 'bad' bacterial overgrowth, by inhibiting the digestive juices needed to properly break down food, and destroying the beneficial flora that's supposed to prevent it.

Vinegar, on the other hand, kills 'harmful' bacteria (like E. coli and Staphylococcus) and helps create the acidic environment we need to properly digest our food. Ensuring we can access the essential nutrients (in big hunks of meat), and preventing stalls that can lead to gastrointestinal discomfort and an increased risk of infection and overgrowth.

“Vinegar has a positive impact on gut health because it’s anti-microbial. It helps to break down the bad bacteria and feed the good.”

Interestingly, vinegar also has a long list of other benefits – including being anti-glycemic (lowers the blood sugar response after meals), and supplying a hefty dose of health-boosting antioxidants (catechin, chlorogenic, epicatechin, gallic, and caffeic acids, etc) – but to stay on topic, we’ll just say that it should be consumed daily for digestion. And not just the minimal amounts you get from salad dressings, pickled foods, and condiments (ketchup, mustard, hot sauce, etc).

Similar to fermented foods, you should be incorporating a daily dose of vinegar into your routine. With a little swig of Apple Cider Vinegar after non-vinegar containing meals being the easiest way to accomplish this. Starting with a small sip after your biggest meal of the day (usually dinner) and adding it to other meals as you see fit.

I’m a big fan of the Bragg’s brand, because it’s ‘raw, unfiltered, organic, and unpasteurized’ and comes in a large 1L bottle.



PART 3

Get To Know SIBO & Consider An Intervention

Aside from reducing inflammation, indigestion, and irritation in the gut, one of the reasons carbohydrate restriction alone gives people so much digestive relief, is because most of the bad bacteria that overgrows in the gastrointestinal tract thrives on glucose. Meaning, bacteria that may be present has nothing to eat, and goes dormant.

The bad news is, in many cases that's not enough for the bacteria to go away for good. Leaving you with bad bugs sitting in your gut, and waiting to feed. Whether you're adding beneficial bacteria and stomach acid to try and wipe them out, or not.

And again, this isn't to say that everyone has an overgrowth of damaging flora in their gut that's going to damage their gut lining. But it is to say that these issues are more common now than ever, and it's a direct result of the foods most of us have been eating, stressful lifestyle most of us are following, environmental toxins most of us are exposed to, and medications most of us have been advised to take.



Fortunately, when it's overgrowth from a specific pathogen, or strain of bacteria, the HCL and probiotics protocols mentioned above can often take care of it. Unfortunately, when it's Small Intestinal Bacterial Overgrowth, or SIBO, they can't. Since SIBO isn't one specific type of bad bacteria causing a problem, as much as it's too much of the wrong types of bacteria causing a problem. With good and bad bacteria from your large intestine living in your small intestine, feeding on undigested food particles (mostly starches), producing excessive hydrogen (or methane) gas, and leading to a chronically inflamed gut that's susceptible to becoming permeable.

Although, this is provided you don't pay attention to the early warning signs. Which tend to revolve around IBS and digestive distress symptoms like:

- Bloating or cramps after eating
- Excessive burping and gas
- Bad breath
- Low hunger or energy
- Constipation or undigested food in stool
- Diarrhea or loose stools
- Heartburn or Reflux
- Chronic nutrient deficiencies (despite good nutrition)

But could also present themselves as a chronic conditions related to the skin (acne, rosacea, psoriasis, eczema), brain (depression, anxiety, brain fog, etc), and joints (arthritis, fibromyalgia, etc). Since the gastrointestinal system is directly connected to these areas (gut-skin, gut-brain, and gut-joint axis), and an inflamed, permeable gut is typically seen in individuals with these conditions.

A review paper from 2007 in the journal Gastroenterology & Hepatology suggests that SIBO affects 6-15% of otherwise healthy (asymptomatic) individuals, and 80% of those suffering from irritable bowel syndrome.

Thus, if you're still exhibiting any of these symptoms after implementing the *Live It NOT Diet!* principles and Part 1 and Part 2 of this Gut Healing Protocol, you'll want to take a look at the SIBO intervention below. Especially if the symptoms flare-up after higher-carbohydrate, starch-heavy meals.

THE SIBO INTERVENTION

Before getting into a serious intervention, it's essential that you exhaust all of gut-supporting, digestion-enhancing points already mentioned. In addition to following the nutrition and lifestyle plan laid out in *Live It NOT Diet!*.

Once you've established what would be a considered a solid baseline of health, and you're confident that your current eating habits are as good as they can be, give yourself an honest assessment (or have a professional do it) and decide if the intervention is necessary.

STEP 1 – Try 1 Round of Caprylic Acid

There's the potential that your suspected SIBO is the result of a yeast or fungus overgrowth (like candida), or specific pathogen (like Streptococcus and E. coli), not an overgrowth in your small intestine. So a round of caprylic acid is recommended to rule these out, and potentially fix your issues, without having to resort to (or spend the money on) something more invasive.

Interestingly, caprylic acid is one of the main components in coconut oil that's responsible for its antibacterial, antiviral, antifungal and anti-inflammatory properties. The difference being, a caprylic acid supplement (like [this one from Biotics](#)) provides a more concentrated dose.

Start with a 500mg capsule once per day (in capsule form) and progress yourself 500mg every 3-4 days until you reach 2000mg. The capsules are best taken spread throughout the day on an empty stomach, or 30-60min before meals.



STEP 2 – Get Tested For ‘Both Types’ of SIBO

As mentioned earlier, SIBO creates excessive gas by fermenting carbohydrates. And this gas can be hydrogen, methane, or both, depending on the bacterial composition. So, it's essential that you go beyond the standard hydrogen testing; especially if it comes back negative.

Traditional SIBO breath tests only look for excessive hydrogen gas, even though anywhere from 35%-75% of those with SIBO are excessive methane producers.

Basically, a methane producing SIBO (associated with constipation) has an overgrowth of archaea – which feeds on the hydrogen and produces a methane byproduct. While non-archaea SIBO, or your standard hydrogen-producing SIBO (associated with diarrhea), doesn't. Although, keep in mind that it's also possible to see both.

Breath tests can be taken at home, or at a lab (recommended). Which usually requires 12 hours of fasting, and no fermentable foods for 2-3 days leading up to it. Look for the lactulose test instead of the glucose test - as this paints a better picture of the entire small intestine (not just the first few feet where glucose is absorbed) – and the 3-hour breath test instead of the 60-min one.

STEP 3 – Do a Herbal Intervention (if Test is Positive)

If you can believe it, the current treatment for SIBO is antibiotics. Even though, as discussed earlier, they wipe out just as much good bacteria as bad, and tend to result in SIBO resurfacing in the future. Largely because of good bacteria (probiotics) to provide ongoing protection, but also because of antibiotic resistance. Which is when bacteria outsmart the medication previously used to treat them, through natural adaptation, genetic mutation, or acquisition (from other bacteria).

The effectiveness of antibiotics against SIBO can vary between 40 and 90%, but that's before looking at the fact that 50% of all patients have a recurrence within one year.

Fortunately, this doesn't happen with the herbal options – as they include an enormous number of individual compounds the bacteria can't develop a resistance to - and new evidence (2014) suggests that they can be more effective at wiping out SIBO than antibiotics.

For instance, Rifaximin is the most commonly prescribed antibiotic for SIBO. Yet, a recent paper in the journal *Global Advances in Health and Medicine* showed that 2 herbal therapies (from nutraceutical companies – Biotics & Metagenics) were 12% more effective (46% vs 34%) at normalizing breath hydrogen tests in patients with SIBO.



The first herbal therapy, (and [the one I recommend](#)) was from Biotics - 2 capsules, twice daily of Dysbiocide & FC Cidal for 4 weeks. And the second, that was equally effective, from Metagenics – Candibactin-AR & Candibactin-BR at the same dosage – is also an option.

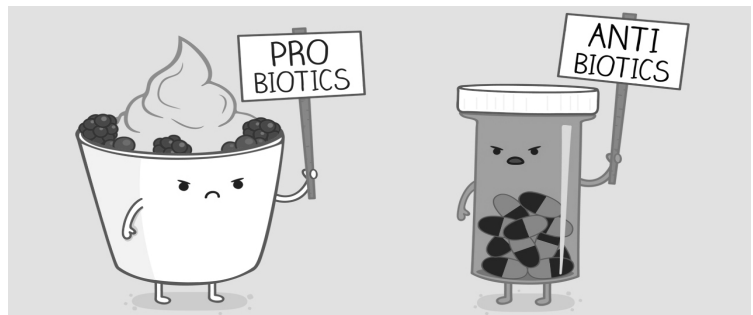
STEP 4 - Test Again & Try a New Herbal (if Necessary)

Your best bet is to consult with a Naturopathic Doctor, or Alternative Medicine Practitioner if the first round of SIBO treatment is unsuccessful. Since they will be able to provide more insight into herbal therapies that may work better in your situation.

STEP 5 - Test Again & Try Antibiotic (as Last Resort)

Fortunately, the antibiotics typically prescribed for SIBO – Rifaximin (for hydroge-type) and Neomycin (for methane-dominant) – are designed to stay in and kill bacteria in the intestine, not get absorbed into the bloodstream. Unfortunately, the 10-14 days most doctors prescribe them for aren't enough to cure the SIBO, and they generally add more antibiotics on top of the original, or a different more invasive one (like Metronidazole), if the first round is unsuccessful.

Put another way, going the antibiotic isn't favorable. But provided you've exhausted all of your other options, you don't really have a choice. And as discussed in Part 1, you can counteract a lot of the damage by committing to probiotic supplementation during and immediately after the treatment.



ONGOING SUPPORT – COCONUT OIL + HERBS

Once you've re-established your healthy gut flora, brought your stomach acid back up to a reasonable level, and removed any unwanted bacteria from your gastrointestinal tract, the only thing left to do is maintain it. And other than the fermented foods and vinegar mentioned already, the best way to do that is with a regular intake of coconut oil and some specific herbs and spices. Since they effectively disinfect the gut, and create an environment where the bad bacteria won't want to live.

Fortunately, we won't go through all of the potential gut-boosting herbs and spices, or the long-list of side benefits that go with each one. However, we will touch on 10 of the biggest players, and give a brief rundown on what you can expect.

Interestingly, many of the herbs and spices on the list (like garlic, thyme, and ginger) are found in the herbal therapies used to treat SIBO.

1. *Thyme* – often used in tea format to treat everything from athlete's foot to yeast infections
2. *Oregano* – has proven just as effective in killing E.Coli and staph as penicillin
3. *Basil* - directly disarms a long list of infections (including listeria) with its various volatile oils (estragole, myrcene cineole, eugenol, limonene, etc)
4. *Garlic* – keeps things balanced in your gut, by killing yeast and pathogenic bacteria, and feeding the beneficial microbes that help keep us lean and healthy
5. *Mint* – relaxes the gut muscles, stimulates enzyme production, and kills bad bacteria overgrowth
6. *Ginger* – known for soothing or calming the muscles of the gastrointestinal system to support healthy digestion and elimination
7. *Cloves* – encourage the body to secrete hydrochloric acid (HCL), and supplies a powerful shot of antimicrobial, antifungal, and antiviral oil
8. *Tarragon* – improves digestion by stimulating enzyme production, relaxing the gastrointestinal muscles, and going to work on any bacterial infections
9. *Cardamom* – regarded as one of the top spices for aiding digestion, and its powerful oil kills pathogenic bacteria (*Streptococcus mutans* and *Candida albicans*)
10. *Fennel seeds* – provide relief to those with a stressed-out, irritated gastrointestinal tract, and protects against bacterial overgrowth and infection.

A study from 2007 in the journal Digestive and Liver Disease, had more than 75% of the 57 participants experience significant relief from their irritable bowel syndrome symptoms (abdominal bloating and pain, excess gas, diarrhoea, constipation, etc) after 4 weeks of supplemental peppermint oil.



As discussed earlier, coconut oil's caprylic acid content makes it a potent yeast and fungus killer. Though it's also a rich source of lauric acid, and has a similar anti-inflammatory and anti-oxidant effect as the herbs and spices just mentioned. Meaning it not only prevents the development of SIBO and dysbiosis, but helps protect the gut lining from any unnecessary oxidative and inflammatory damage.

The best way to get coconut oil is to cook with it. And the same goes for the 10 herbs and spices above. Add them to your meals, put them in your soups (and teas), and find a way to consume them regularly.

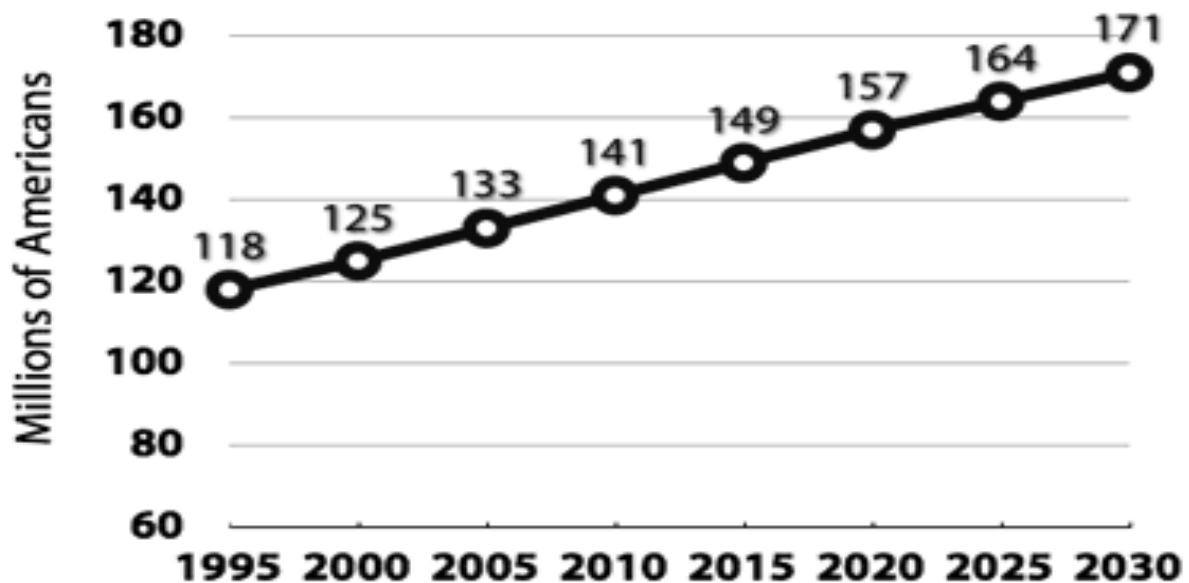
PART 4

Balance Sympathetic & Parasympathetic Stress

Most of us recognize that excess stress is bad for our health and body composition, but not many of us understand why. Because if we did, we'd probably start paying a little more attention to the techniques for reducing it; instead of treating it like hippy mumbo-jumbo or a curfew from Mom and Dad, and purposely burning the candle at both ends in spite of it.

Well maybe not *purposely*, but the North American population is more stressed than ever. And not surprisingly, we're seeing a steady decline in health and increase in degenerative disease as a result.

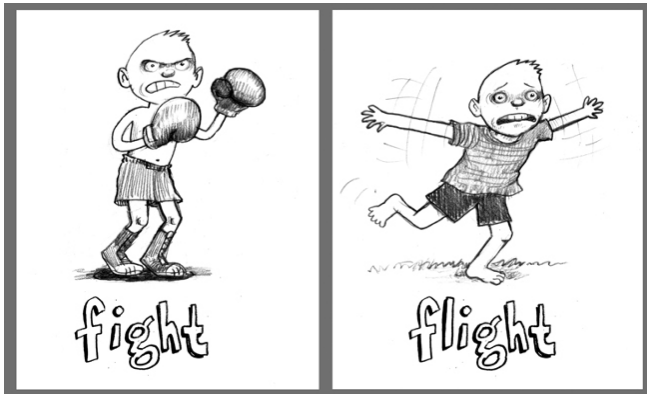
Prevalence of Chronic Disease in the U.S.



Source: Wu, Shin-Yi *et al.* 2000. Projection of Chronic Illness Prevalence and Cost Inflation. RAND Corporation.

Obviously there's more at play here, and I'll be the first to tell you it's largely diet related. But the impact of stress can't be ignored. Especially when we know it's directly related to how much we work, how much we rest, and how much we play.

Without going into too much detail, the stress response is a primal instinct designed to increase our chances of survival. Our blood pressure increases, energy floods to our muscles and brain, and we're ready to think, move, and react quickly.



This 'fight or flight' process, coined 'the alarm reaction phase' by the great Hans Selye involves the adrenal medulla (found in the adrenal glands) releasing a massive amount of hormones (called catecholamines), after getting a direct message from the brain (the two are directly connected) that *someh'n' ain't right*. Followed closely by the adrenal cortex (also found in the adrenal glands) releasing steroid hormones (corticoids), which help increase blood flow and blood sugar.

The problem is, we're biologically designed to follow up this 'sympathetic' response with a 'parasympathetic' reaction shortly after. Where the adrenal glands stop secreting extra hormones (adrenaline, aldosterone, cortisol), our kidney's stop hanging onto water and salt, blood glucose returns to normal, and energy and resources are redistributed to non-urgent, non-life threatening systems that were sacrificed during the emergency – digestive, reproductive, immune.

Parasympathetic = Rest & Digest, Feed & Breed

In other words, stress is supposed to be acute not continuous, and occasional not consistent. And unfortunately, without regular recovery we get into Selye's 'resistance' and 'exhaustion' phases, where excess glucocorticoids (like cortisol) and mineralocorticoids (like aldosterone), negatively affect our carbohydrate tolerance, nutrient metabolism, inflammatory balance, gut health and immune system.

Basically, the over-stressed professional is fight-or-flight from the time they get up in the morning to the time they go to bed. Whether that's thinking about what they have to do, doing what they have to do, worrying about what they just did, or stressing about tomorrow. Since the brain controls the response, and 'perceived' danger is equivalent to *actual* danger.



Thus, without even getting into what Stock Brocker Sam is eating, if Stock Broker Sam is sleeping, and whether Stock Broker Sam is exercising, we know aldosterone, cortisol, and epinephrine are chronically pumping out of his adrenal glands, which elevates his heart rate, increases his blood sugar, and tells his digestion, reproductive hormones, and immune system to take a backseat.

“And we wonder why high-stress people get sick?”

Sadly, high-stress people also tend to carry excess body fat, and have trouble gaining and maintaining muscle mass too. Which isn't surprising, given what we know about chronically elevated blood sugar and insulin, and given that chronically cortisol makes us store fat, burn muscle, lose our insulin sensitivity and act as a pre-cursor for the metabolic syndrome, and the diabetes and heart disease that comes with it.

But what's worse, is that the systemic release of corticoids increases inflammation. Meaning circulating cytokines on a chronic and consistent basis, and an increased risk of the chronic epithelial damage, and chronic inflammatory conditions that occur as a result.

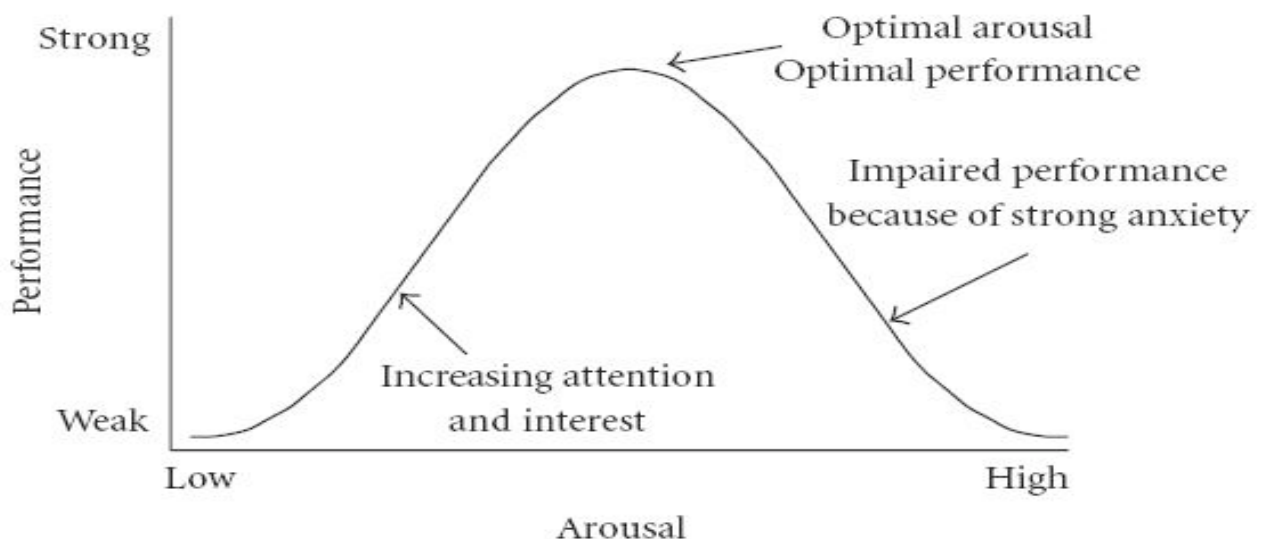
Or simply put, just like the gut can negatively affect the brain, our brain can negatively affect our gut - shutting down the gastrointestinal system (poor digestion and elimination) and promoting inflammation in times of stress, and making us more susceptible to infection, overgrowth, and permeability.

FIND YOUR ALLOSTATIC BALANCE

The good news is, just like inflammation, stress can be controlled. And the better news is, you have more control over it than inflammation. Because aside from avoiding behavior's that promote it (lack of sleep, worrying) you have the ability to control your personal response to it.

“Stress is not what happens to you, but how you react to it”
- Hans Selye, Stress of My Life, 1977

Moreover, acute stress applied to the right person at the right time, results in favorable adaptations (eustress). With exercise being the perfect example.



In all cases, the key is striking a balance between your sympathetic (work) and parasympathetic (rest) nervous system – also referred to as your ‘allostatic balance.’ Which ultimately comes down to controlling the cumulative total of anything and everything in your life that causes mental, physical and emotional stress (allostatic load).

In today’s world, this usually means increasing our parasympathetic time away from technology. Since being connected is stressful enough as it is, and worrying about the news or getting worked up on reality TV triggers the alarm phase we don’t want (especially in the evening, when it’s wind-down time).

Getting longer, high-quality sleep should be priority #1; followed by exploring other ways to recharge your batteries (like meditation).

Though your stress-reducing plan should also include turning distress (the bad stuff) into eustress (the good stuff), by doing your best to **create a simple one-task, distraction-free environment.** Because regardless of the size, task accomplishment builds self-efficacy, while a giant list of half-completed To-Do items, or getting side-tracked by the ADHD that is the internet, promotes the opposite.



Learning to say “no” and be selfish of your free time is also an extremely helpful practice. As if it’s not work or family related, and you’re not going to enjoy it, there should be no reason to do it – especially if it’s some one or some thing that adds distress.

And lastly, it’s about **matching the quantity of good stressors (eustress), like exercise, to your current allostatic load.** Which could mean doing a little bit less (sets, weight, time), opting for a low-intensity stress-reducing activity (like walking) instead, or skipping the workout all together so you can get to bed early.



This may sound backwards for improving your health and body composition, but with the way people are operating these days it’s necessary. Chronic stress changes the way your body responds to meals, changes the way your body responds to exercise, and raises biomarkers for disease whether your regimen is perfect or not.

It’s also one of the hardest changes to make, because people who want to ‘do better’ have trouble doing what they perceive as *nothing* to get there.

But it isn’t nothing! Because perfect balance is only achieved by touching extremes at both ends (ex: acute stress with exercise, recovery with sleep), and doing your best to avoid living a life that’s dominated by one or the other (ex: high stress work, zero activity). Not only so you can thrive and be happy, but because *realistically* your life (and your gut health) depends on it.

PART 5

Understand What NSAIDs Do To Your Gut

Hate to generalize, but if you're a Baby Boomer you're probably used to popping Advil or Aspirin when something hurts.

Headache? No problem.

Back ache? Handled.

Arthritis? Taken care of.

Someone else is in pain? "Did you take an ibuprofen?"

What's worse, is that many have been advised to take a Baby Aspirin each day as a preventative measure. Since your trusty doctor believes (or has been taught to believe) it's the best way to prevent a heart attack.



Meanwhile, these NSAIDs (Non-Steroidal Anti-Inflammatory Drugs) are damaging your gut, and doing nothing for the underlying issue:

CHRONIC INFLAMMATION!

Sure, the inflammatory symptoms can be masked, or temporarily reduced with pills and potions, but the problem is never properly addressed. Giving you the false reassurance that you feel good (or better), forcing you to depend-on (and likely increase) your regular dosage, and doing consistent damage to your gut lining.

Sadly, this is the same area that's already taking a beating from the Western diet and lifestyle. Meaning, your NSAID popping behavior is poking holes in your gut that's already inflamed, and will continue to be inflamed because you (and your doctor) have made no effort to get to the root of what's causing this inflammation in the first place.

The ultimate irony being, that in many cases it's a leaky gut driving the chronic inflammatory condition. So, people prescribed NSAIDs are taking medication that's worsening the very root of the problem - a busted gut!

For example:

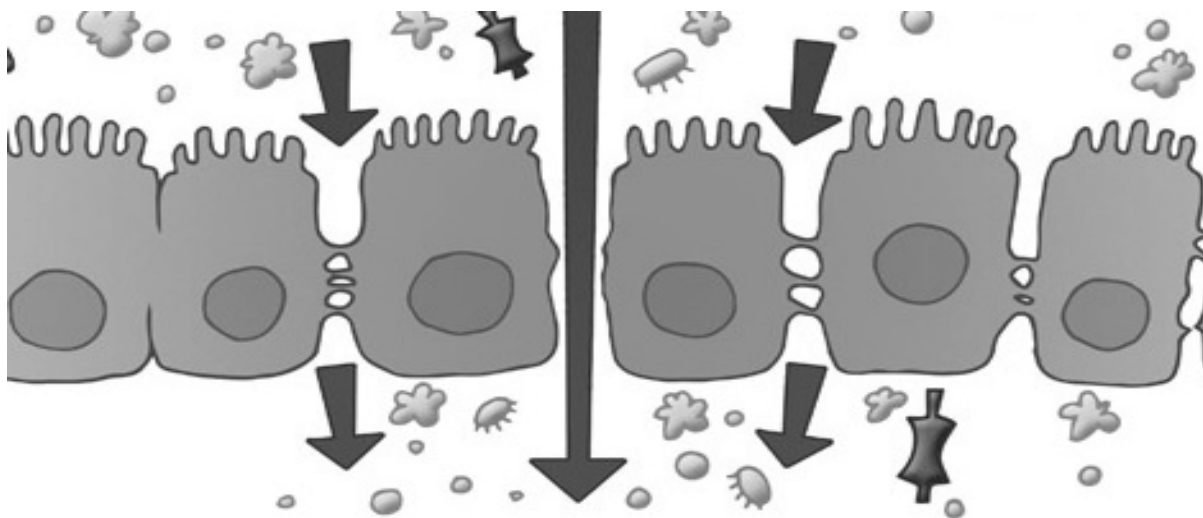
“NSAIDs are thus shown to disrupt intestinal integrity and long term treatment leads to inflammation of the small intestine.”

“Aspirin acts on the colon to unmask a susceptibility to gut leakiness.”

“Ibuprofen aggravates exercise-induced small intestinal injury and induces gut barrier dysfunction in healthy individuals.”

“A very low dose of aspirin (10 mg daily) decreases the gastric mucosal prostaglandin levels and causes significant gastric mucosal damage.”

The reason this happens is because NSAIDs are designed to inhibit COX enzymes (cyclooxygenase), which normally help the body produce prostaglandins that fight pain and inflammation. And unfortunately, preventing the action of one of those enzymes (COX-1) has a negative impact on the gut; since the prostaglandins that should have been secreted to protect the gut from damage are not. Making you more susceptible to intestinal permeability, increasing your sensitivity to inflammatory foods, and even changing the microbiota makeup of your gut.



To which one might say:

“Then let’s make a drug that only inhibits one enzyme...?”

Although that’s been done, and it was a disaster. Largely, because these enzymes work synergistically – if you block one, the other is upregulated. And when scientists developed a drug to block COX-2 only (ex: Celebrex), they realized it increased heart disease risk.

In other words, NSAIDs are a lose/lose. Take ibuprofen, which reduces both COX-1 and COX-2, and deal with gut issues. Take aspirin, which preferentially reduces COX-1 and deal with BIG TIME gut issues. Or take a prescription COX-2 inhibitor, and increase your risk of a heart attack.

WHAT ABOUT TYLENOL?

Also known as paracetamol or acetaminophen, Tylenol is technically not an anti-inflammatory drug, as it doesn’t prevent blood from clotting (block COX enzymes). And thus, unlike aspirin and ibuprofen it doesn’t irritate the gut, or cause any of the COX enzyme issues discussed above. But it does increase your risk of liver failure significantly with chronic or ‘above daily dosage’ use and there’s plenty of evidence suggesting it’s not much better than placebo for pain.



Frankly, if you’re taking Tylenol for chronic headaches or migraines, it’s likely because of a similar inflammatory condition. So, just like Advil and Aspirin, the true remedy is fixing your inflammation problem (NOT signing up for a Tylenol pez dispenser).

This means eliminating inflammatory foods, balancing your omega 6:3 ratio, embracing the anti-inflammatory lifestyle practices (walking, sleeping) discussed in *Live It NOT Diet!*. and following the gut-healing protocol we just covered:

- Restoring your gut flora and bacterial balance
- Improving the digestive environment with greater HCL production
- Removing SIBO and other potential pathogens
- Reducing stress and finding an allostatic balance
- Avoiding unnecessary medications – antibiotics, antacids, NSAIDs

Along with that, you can look to incorporate some of the secondary tips listed below. Which are all discussed in greater detail at <http://coachmikeblogs.com>.

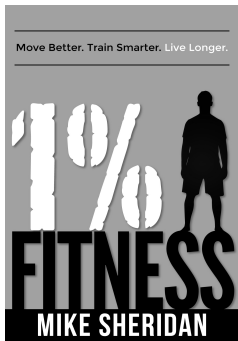
1. *Keep taking Zinc, Magnesium and Fish Oil* - to strengthen the gut lining, lower stress and relax the gastrointestinal muscles, and reduce inflammation.
<http://coachmikeblogs.com/what-to-supplement-and-why/>
2. *Drink Bone Broth* – to heal the gastrointestinal tract with collagen and glucosamine.
<http://coachmikeblogs.com/bone-broth-make-it-a-staple-this-winter/>

3. *Get Blue Blocking Glasses and Black Out Blinds* – to prevent blue light (from technology) and artificial light from messing with your sleep. <http://coachmikeblogs.com/cant-sleep-blame-technology/>
4. *Consider Going Wheat-Free (even on Feast Days)* – to avoid the gliadin proteins in wheat (and other gluten containing grains) that activate zonulin, and permeate the gut (in everyone). <http://coachmikeblogs.com/the-wheat-free-feast/>
5. *Avoid Water With Meals & Chew Your Food* – to let the digestive fire burn (during and after meals), and encourage easier breakdown and smoother passage through the GI tract (with smaller food particles). <http://coachmikeblogs.com/5-small-nutrition-tweaks-that-make-a-big-difference/>
6. *Try Fasting With Herbal Tea* – to give the gastrointestinal tract the opportunity to relax, while the beneficial compounds in tea do some house cleaning. <http://coachmikeblogs.com/why-you-should-fast-occasionally/>
7. *Consider Adding a Blended Beverage to Breakfast (Super Shake)* – to get some much needed pre- and probiotic support (from berries, greens, coconut oil, kefir and spices), that you may have trouble incorporating into your daily diet otherwise.
8. *Experiment With “Bitters” (in VERY small doses)* – to help stimulate HCL and other digestive juices from herbs you wouldn’t otherwise use when cooking (Dandelion, Gentian root, Beet root, Goldenseal root, Milk thistle, Wormwood, Yellow dock, etc).

NEXT STEPS

Live It NOT Diet! Updates + \$5-off *1% Fitness*

Your next step is *1% Fitness*, but I urge you to hold back on pursuing it until you've been comfortably following *Live It NOT Diet!* for a month or two. As nutrition and healthy living is going to give you 80-90% of the results, and I'd hate to see you compromise any of that because you're caught up with having to "workout."



When you are finally ready, make sure you head over to <http://liveitbonus.com> and grab the \$5 off coupon I'm offering as an incentive to leave a *Live It NOT Diet!* review (on Amazon). Keeping in mind that there's no pressure for it to be a positive 5-star one (if that's how you feel), and it doesn't have to be long or extravagant either.

All I ask for is your honest opinion of the book, as it helps people find my work, and helps me understand where and how I can improve it.

Outside of that, look for an email from me every week (or so) where I'll be sharing some FAQs and best practices for *Live It NOT Diet!*. And if you haven't already, you can check-out the latest updates from me on Twitter and Facebook, or by subscribing to my Monthly Newsletter at: <http://coachmikeblogs.com>.

Stay Lean!
Coach Mike

REFERENCES

Introduction

- Gill HS and Guarner F. 2003. Probiotics and human health: a clinical perspective. *BMJ-PMJ* 80(947).
- Backhed F, et al. 2005. Host-bacterial mutualism in the human intestine. *Science* 307(5717):1915-1920.
- Bested AC, et al. 2013. Intestinal microbiota, probiotics and mental health: from Metchnikoff to modern advances: Part II – contemporary contextual research. *Gut Pathogens* 5:3.
- Blaser M. 2011. Antibiotic overuse: Stop the killing of beneficial bacteria. *Nature* 476:393-394.
- Sighthorsson G, et al. 1998. Intestinal permeability and inflammation in patients on NSAIDs. *Gut* 43(4):506-11.
- Konturek PC, et al. 2011. Stress and the gut: Pathophysiology, clinical consequences, diagnostic approach and treatment options. *J Physiol Pharmacol* 62(6):591-99.
- Lyte M, et al. 2011. Stress at the intestinal surface: catecholamines and mucosa-bacteria interactions. *Cell Tissue Res* 343(1):23-32.
- Codru N, et al. 2007. Diabetes in relation to serum levels of polychlorinated biphenyls and chlorinated pesticides in adult Native Americans. *Environ Health Perspect* 115(10):1442-1447.
- Navas-Acien A, et al. 2008. Arsenic exposure and prevalence of type 2 diabetes in US adults. *JAMA* 300(7):814-822.
- Gronlund MM, et al. 1999. Fecal microflora in healthy infants born by different methods of delivery: permanent changes in intestinal flora after caesarean delivery. *J Pediatr Gastroenterol Nutr* 28(1):19-25.
- Dominguez-Bello MG, et al. 2010. Delivery mode shapes the acquisition and structure of the initial microbiota across multiple body habitats in newborns. *Proc Natl Acad Sci USA* 107(26):11971-5.
- Centers for Disease Control and Prevention. 2006. Community-associated methicillin-resistant *Staphylococcus aureus* infection among healthy newborns—Chicago and Los Angeles County, 2004. *MMWR Morb Mortal Wkly Rep* 55(12):329-32.
- Bager P, et al. 2008. Caesarean delivery and risk of atopy and allergic disease: meta-analyses. *Clin Exp Allergy* 38(4):634-42.
- Ravelli AC, et al. 1998. Glucose tolerance in adults after prenatal exposure to famine. *Lancet* 351(9097):173-7.
- Koenig JE, et al. 2011. Succession of microbial consortia in the developing infant gut microbiome. *Proc Natl Acad Sci USA* 108(1):4578-85.
- Turnbaugh PJ, et al. 2006. An obesity-associated gut microbiome with increased capacity for energy harvest. *Nature* 444:1027-1031.
- Dicksved J, et al. 2008. Molecular analysis of the gut microbiota of identical twins with Crohn's disease. *ISME J* 2:716-727.
- Wen L, et al. 2008. Innate immunity and intestinal microbiota in the development of type 1 diabetes. *Nature* 455:1109-1113.
- Visser J, et al. 2009. Tight junctions, intestinal permeability, and autoimmunity: celiac disease and type 1 diabetes paradigms. *Ann NY Acad Sci* 1165:195-205.
- Fasano A. 2012. Leaky gut and autoimmune diseases. *Clin Rev Allergy Immunol* 42(1):71-8.
- Rosenfeldt V, et al. 2004. Effect of probiotics on gastrointestinal symptoms and small intestinal permeability in children with atopic dermatitis. *J Pediatr* 145(5):612-6.
- Humbert P, et al. 1991. Intestinal permeability in patients with psoriasis. *J Dermatol Sci* 2(4):324-6.
- Smith MD, et al. 1985. Abnormal bowel permeability in ankylosing spondylitis and rheumatoid arthritis. *J Rheumatol* 12(2):299-305.
- Dunlop SP, et al. 2006. Abnormal intestinal permeability in subgroups of diarrhea-predominant irritable bowel symptoms. *Am J Gastroenterol* 101(6):1288-94.
- Maes M, et al. 2007. Increased serum IgA and IgM against LPS of enterobacteria in chronic fatigue syndrome (CFS): indication for the involvement of gram-negative enterobacteria in the etiology of CFS and for the presence of an increased gut-intestinal permeability. *J Affect Disord* 99(1-3):237-40.
- Maes M and Leunis JC. 2008. Normalization of leaky gut in chronic fatigue syndrome (CFS) is accompanied by a clinical improvement: effects of age, duration of illness and the translocation of LPS from gram-negative bacteria. *Neuro Endocrinol Lett* 29(6):902-10.
- Hijazi Z, et al. 2004. Intestinal permeability is increased in bronchial asthma. *Arch Dis Child* 89(3):227-9.
- Benard A, et al. 1996. Increased intestinal permeability in bronchial asthma. *J Allergy Clin Immunol* 97(6):1173-8.
- Hollander D. 2013. Intestinal permeability barrier in chron's disease: the difficulty in shifting the paradigm. *Dig Dis Sci* 58(7):1827-1829.

- Keita AV and Soderholm JD. 2010. The intestinal barrier and its regulation by neuroimmune factors. *Neurogastroenterol Motil* 22(7):718-33.
- Anders HJ, et al. 2013. The intestinal microbiota, a leaky gut, and abnormal immunity in kidney disease. *Kidney Int* 83(6):1010-6.
- Maes M, et al. 2008. The gut-brain barrier in major depression: intestinal mucosal dysfunction with an increased translocation of LPS from gram negative enterobacteria (leaky gut) plays a role in the inflammatory pathophysiology of depression. *Neuro Endocrinol Lett* 29(1):117-24.
- Mayer EA, et al. 2014. Altered brain-gut axis in autism: comorbidity or causative mechanisms? *Bioessays* 36(10):933-9.
- Cryan JF and Dinan TG. 2012. Mind-altering microorganisms: the impact of the gut microbiota on brain and behaviour. *Nat Rev Neurosci* 13(10):701-12.
- Joscelyn J and Kasper LH. 2014. Digesting the emerging role for the gut microbiome in central nervous system demyelination. *Mult Scler* 20(12):1553-9.
- Fasano A and Shea-Donohue T. 2005. Mechanisms of disease: the role of intestinal barrier function in the pathogenesis of gastrointestinal autoimmune diseases. *Gastro Hepat* 2(9):416-422.
- Secondulfo M, et al. 2004. Ultrastructural mucosal alterations and increased intestinal permeability in non-celiac, type 1 diabetic patients. *Dig Liver Dis* 36(1):35-45.
- Scheperjans F, et al. 2015. Gut microbiota are related to Parkinson's disease and clinical phenotype. *Mov Disord* 30(3):350-8.
- Fasano A. 2011. Zonulin and its regulation of intestinal barrier function: the biological door to inflammation, autoimmunity, and cancer. *Physiol Rev* 91(1):151-175.
- Gluck U and Gebber J. 2003. Ingested probiotics reduce nasal colonization with pathogenic bacteria (*Staphylococcus aureus*, *Streptococcus pneumoniae*, and *B-hemolytic streptococci*). *Am J Clin Nutr* 77(2):517-520.
- Wassenberg J, et al. 2011. Effect of *Lactobacillus paracasei* ST11 on a nasal provocation test with grass pollen in allergic rhinitis. *Clin Exp Allergy* 41(4):565-73.
- Ouwehand AC, et al. 2009. Specific probiotics alleviate allergic rhinitis during the birch pollen season. *World J Gastroenterol* 15(26):3261-8.
- Bowe WP, et al. 2010. Diet and acne. *J Am Acad Dermatol* 63(1):124-41.
- Simmering R and Breves R. 2009. Pre- and probiotic cosmetics. *Hautarzt* 60(10):809-14.
- Muizzuddin N, et al. 2012. Physiological effect of a probiotic on skin. *J Cosmet Sci* 63(6):385-95.
- Bowe WP and Logan AC. 2011. Acne vulgaris, probiotics and the gut-brain-skin axis – back to the future? *Gut Pathog* 3(1):1.
- Messaoudi M, et al. 2011. Assessment of psychotropic-like properties of a probiotic formulation (*Lactobacillus helveticus* R0052 and *Bifidobacterium longum* R0175) in rats and human subjects. *Br J Nutr* 105(5):755-64.
- Dinan TG and Quigley EM. 2011. Probiotics in the treatment of depression: science or science fiction? *Aust NZ J Psychiatry* 45(12):1023-5.
- Li W, et al. 2009. Memory and learning behavior in mice is temporarily associated with diet-induced alterations in gut bacteria. *Physiol Behav* 96(4-5):557-67.
- Liu X, et al. 2015. Modulation of gut microbiota-brain axis by probiotics, prebiotics, and diet. *J Agric Food Chem* 63(36):7885-95.
- Tillisch K, et al. 2013. Consumption of fermented milk product with probiotic modulates brain activity. *Gastroenterology* 144(7).
- Messaoudi M, et al. 2011. Beneficial psychological effects of a probiotic formulation (*Lactobacillus helveticus* R0052 and *Bifidobacterium longum* R0175) in healthy volunteers. *Gut Microbes* 2(4):256-61.
- Cordain L, et al. 2000. Modulation of immune function by dietary lectins in rheumatoid arthritis. *Br J Nutr* 83(3):207-17.
- Naruszewicz M, et al. 2002. Effect of *Lactobacillus plantarum* 299v on cardiovascular disease risk factors in smokers. *Am J Clin Nutr* 76(6):1249-55.
- Rao AV, et al. 2009. A randomized, double-blind, placebo-controlled pilot study of a probiotic in emotional symptoms of chronic fatigue syndrome. *Gut Pathog* 1(1):6.
- Bested AC, et al. 2013. Intestinal microbiota, probiotics and mental health: from Metchnikoff to modern advances: Part 1 – auto-intoxication revisited. *Gut Pathog* 5:5.
- Part 1**
- Palmer C, et al. 2007. Development of the human infant intestinal microbiota. *PLoS Biol* 5(7):e177.
- Mackie RI, et al. 1999. Development microbial ecology of the neonatal gastrointestinal tract. *Am J Clin Nutr* 69(5):1035S-1045S.

- Costello EK, et al. 2009. Bacterial community variation in human body habitats across space and time. *Science* 326(5960):1694-7.
- Ouwehand A, et al. 2002. The role of the intestinal microflora for the development of the immune system in early childhood. *Eur J Nutr* 41(1):13207.
- Ley RE, et al. 2006. Microbial ecology: human gut microbes associated with obesity. *Nature* 444(7122):1022-3.
- Dethlefsen L, et al. 2008. The pervasive effects of an antibiotic on the human gut microbiota, as revealed by deep 16S rRNA sequencing. *PLoS Biol* 6(11):e280.
- Dethlefsen L and Relman DA. 2011. Incomplete recovery and individualized responses of the human distal gut microbiota to repeated antibiotic perturbation. *Proc Natl Acad Sci USA* 108(1):4554-61.
- Jumpertz R, et al. 2011. Energy-balance studies reveal associations between gut microbes, caloric load, and nutrient absorption in humans. *Am J Clin Nutr* 94(1):58-65.
- Milena Marques T, et al. 2010. Programming infant gut microbiota: influence of dietary and environmental factors. *Curr Opin Biotech* 21(2):149-56.
- Koenig JE, et al. 2011. Succession of microbial consortia in the developing infant gut microbiome. *Proc Natl Acad Sci USA* 108(1):4578-4585.
- Louis P, et al. 2007. Understanding the effects of diet on bacterial metabolism in the large intestine. *J Appl Microbiol* 102(5):1197-208.
- Muegge BD, et al. 2011. Diet drives convergence in gut microbiome functions across mammalian phylogeny and within humans. *Science* 332(6032):970-4.
- Wu GD, et al. 2011. Linking long-term dietary patterns with gut microbial enterotypes. *Science* 334(6052):105-8.
- Xu J and Gordon JI. 2003. Honor thy symbionts. *Proc Natl Acad Sci USA* 100(18):10452-9.
- Heselmans M, et al. 2005. Gut flora in health and disease: potential role of probiotics. *Curr Issues Intest Microbiol* 6(1):1-7.
- Perry GH, et al. 2007. Diet and the evolution of human amylase gene copy number variation. *Nat Genet* 39(10):1256-60.
- Iebba V, et al. 2012. Gut microbiota and the immune system: an intimate partnership in health and disease. *Int J Immunopathol Pharmacol* 25(4):832-33.
- Simren M, et al. 2013. Intestinal microbiota in functional bowel disorders: a Rome foundation report. *Gut* 62(1):159-76.
- Turnbaugh PJ, et al. 2009. A core gut microbiome in obese and lean twins. *Nature* 457(7228):480-4.
- Moreno-Navarrete JM, et al. 2012. Circulation lipopolysaccharide-binding protein (LBP) as a marker of obesity-related insulin resistance. *Int J Obes* 36:1442-1449.
- Kemp DM. 2013. Does chronic low-grade endotoxemia define susceptibility of obese humans to insulin resistance via dietary effects of gut microbiota? *Adipocyte* 2(3):188-90.
- Dhiman RK. 2013. Gut microbiota and hepatic encephalopathy. *Metab Brain Dis* 28(2):321-6.
- Chen X, et al. 2013. The role of gut microbiota in the gut-brain axis: current challenges and perspectives. *Protein Cell* 4(6):403-14.
- Dinan TG and Cryan JF. 2013. Melancholic microbes: a link between gut microbiota and depression? *Neurogastroenterol Motil* 25(9):713-9.
- Saulnier DM, et al. 2013. The intestinal microbiome, probiotics and prebiotics in neurogastroenterology. *Gut Microbes* 4(1):17-27.
- Jernberg C, et al. 2013. Long-term ecological impacts of antibiotic administration on the human intestinal microbiota. *ISME J* 7(456).
- Manichanh C, et al. 2010. Reshaping the gut microbiome with bacterial transplantation and antibiotic intake. *Genome Res* 20(10):1411-9.
- Hawrelak JA and Myers SP. 2004. The causes of intestinal dysbiosis: a review. *Alt Med Rev* 9(2):180-197.
- Stecher B, et al. 2013. 'Blooming' in the gut: how dysbiosis might contribute to pathogen evolution. *Nature Rev Microb* 11:277-284.
- Trasande L, et al. 2013. Infant antibiotic exposures and early-life body mass. *Int J Obes* 37:16-23.
- Hernandez E, et al. 2013. Functional consequences of microbial shifts in the human gastrointestinal tract linked to antibiotic treatment and obesity. *Gut Microbes* 4(4):306-15.
- Dethlefsen L, et al. 2008. The pervasive effects of an antibiotic on the human gut microbiota, as revealed by deep 16S rRNA sequencing. *PLoS Biol* 6(11):e280.
- Ambrose NS, et al. 1985. The influence of single dose intravenous antibiotics on faecal flora and emergence of *Clostridium difficile*. *J Antimicrob Chemother* 15(3):319-326.

- Mason KL, et al. 2012. *Candida albicans* and bacterial microbiota interactions in the cecum during re-colonization following broad spectrum antibiotic therapy. *Infect Immun*.
- Linskens RK, et al. 2001. The bacterial flora in inflammatory bowel disease: current insights in pathogenesis and the influence of antibiotics and probiotics. *Scan J Gastroenterol Suppl* (234):29-40.
- Traskalova-Hogenova H, et al. 2004. Commensal bacteria (normal microflora), mucosal immunity and chronic inflammatory and autoimmune diseases. *Immunol Lett* 93(2-3):97-108.
- Visser J, et al. Tight junctions, intestinal permeability, and autoimmunity: celiac disease and type 1 diabetes paradigms. *Ann NY Acad Sci* 1165:195-205.
- Fasano A and Shea-Donohue T. 2005. Mechanisms of disease: the role of intestinal barrier function in the pathogenesis of gastrointestinal autoimmune diseases. *Gastro Hepat* 2(9):416-422.
- Rook GAW and Brunet LR. 2005. Microbes, immunoregulation, and the gut. *Gut* 54(3):317-20.
- Sazawal S, et al. 2006. Efficacy of probiotics in prevention of acute diarrhoea: a meta-analysis of masked, randomised, placebo-controlled trials. *Lancet Infect Dis* 6(6):374-382.
- Szajewska H and Mrukowicz J. 2005. Meta-analysis: non-pathogenic yeast *Saccharomyces boulardii* in the prevention of antibiotic-associated diarrhoea. *Aliment Pharmacol Therap* 22(5):365-372.
- Cremonini F, et al. 2002. Meta-analysis: the effect of probiotic administration on antibiotic-associated diarrhoea. *Aliment Pharmacol Therap* 16(8):1461-1467.
- D-Souza AL, et al. 2002. Probiotics in prevention of antibiotic associated diarrhoea: meta-analysis. *BMJ* 324:1361.
- Hickson M, et al. 2007. Use of probiotic *Lactobacillus* preparation to prevent diarrhoea associated with antibiotics: randomised double blind placebo controlled trial. *BMJ* 335:80.
- Armuzzi A, et al. 2001. The effect of oral administration of *Lactobacillus* GG on antibiotic-associated gastrointestinal side-effects during *Helicobacter pylori* eradication therapy. *Aliment Pharmacol Therap* 15(2):163-169.
- Czerucka D, et al. 2007. Review article: yeast as probiotics – *Saccharomyces boulardii*. *Aliment Pharmacol Therap* 26(6):767-778.
- Madden JAJ, et al. 2005. Effect of probiotics on preventing disruption of the intestinal microflora following antibiotic therapy: A double-blind, placebo-controlled pilot study. *Inter Immunopharmacol* 5(6):1091-1097.
- Guyonnet D, et al. 2009. Fermented milk containing *Bifidobacterium lactis* DN-173 010 improves gastrointestinal well-being and digestive symptoms in women reporting minor digestive symptoms: a randomised, double-blind, parallel, controlled study. *Br J Nutr* 102(11):1654-62.
- Guyonnet D, et al. 2007. Effect of a fermented milk containing *Bifidobacterium animalis* DN-173 010 on the health-related quality of life and symptoms in irritable bowel syndrome in adults in primary care: a multicentre, randomized, double-blind, controlled trial. *Aliment Pharmacol Therap* 26(3):475-86.
- He T, et al. 2008. Effects of yogurt and bifidobacteria supplementation on the colonic microbiota in lactose-intolerance subjects. *J Appl Microbiol* 104(2):595-604.
- Agrawal A, et al. 2009. Clinical trial: the effect of a fermented milk product containing *Bifidobacterium lactis* DN-173 010 on abdominal distension and gastrointestinal transit in irritable bowel syndrome with constipation. *Aliment Pharmacol Therap* 29(1):104-14.
- Tabbers MM, et al. 2009. Effect of the consumption of a fermented dairy product containing *Bifidobacterium lactis* DN-173 010 on constipation in childhood: a multicentre randomised controlled trial (NTRTC:1571). *BMC Pediatrics* 9:22.
- Marteau P, et al. 2002. *Bifidobacterium animalis* strain DN-173 010 shortens the colonic transit time in healthy women: a double-blind, randomized, controlled study. *Aliment Pharmacol Therap* 16(3):587-93.
- Schiffirin EJ, et al. 1997. Immune modulation of blood leukocytes in humans by lactic acid bacteria: criteria for strain selection. *Am J Clin Nutr* 66(2):515S-520S
- Klein A, et al. 2008. *Lactobacillus acidophilus* 74-2 and *Bifidobacterium animalis* subsp *lactis* DGCC 420 modulate unspecific cellular immune response in healthy adults. *Eur J Clin Nutr* 62(5):584-93.
- Hatakka K, et al. 2001. Effect of long term consumption of probiotic milk on infections in children attending day care centres: double blind randomised trial. *BMJ* 322(7298):1327.
- Prasanna PHP, et al. 2014. *Bifidobacterium* in milk products: An overview of physiological and biochemical properties, exopolysaccharide production, selection of criteria of milk products and health benefits. *Food Res Inter* 55:247-262.
- Barrett JS, et al. 2008. Probiotic effects on intestinal fermentation patterns in patients with irritable bowel syndrome. *World J Gastroenterol* 14(32):5020-4.

- Furuno T and Nakanishi M. 2012. Kefiran suppresses antigen-induced mast cell activation. *Biol Pharm Bull* 35(2):178-83.
- Jones SE and Versalovic J. 2009. Probiotic *Lactobacillus reuteri* biofilms produce antimicrobial and anti-inflammatory factors. *BMC Microbiol* 9:35.
- Seth A, et al. 2008. Probiotics ameliorate the hydrogen peroxide-induced epithelial barrier disruption by a PKC- and MAP kinase-dependent mechanism. *Am J Physiol Gastrointest Liver Physiol* 294(4):G1060-9.
- Yan F, et al. 2007. Soluble proteins produced by probiotic bacteria regulate intestinal epithelial cell survival and growth. *Gastroenterology* 132(2):562-75.
- Bested AC, et al. 2013. Intestinal microbiota, probiotics and mental health: from Metchnikoff to modern advances: Part I – auto-intoxication revisited. *Gut Pathog* 5(1):5.
- Messaoudi M, et al. 2011. Assessment of psychotropic-like properties of a probiotic formulation (*Lactobacillus helveticus* R0052 and *Bifidobacterium longum* R0175) in rats and human subjects. *Br J Nutr* 105(5):755-64.
- Tillisch K, et al. 2013. Consumption of fermented milk product with probiotic modulates brain activity. *Gastroenterology* 144(7):1394-401.
- Culligan EP, et al. 2009. Probiotics and gastrointestinal disease: successes, problems and future prospects. *Gut Pathog* 1(1):19.
- Dajani AI, et al. 2013. Do probiotics improve eradication response to *Helicobacter pylori* on standard triple or sequential therapy? *Saud J Gastroenterol* 19(3):113-20.
- Sachdeva A and Nagpal J. 2009. Effect of fermented milk-based probiotic preparations on *Helicobacter pylori* eradication: a systematic review and meta-analysis of randomized-controlled trials. *Eur J Gastroenterol Hepatol* 21(1):45-53.
- Kadooka Y, et al. 2010. Regulation of abdominal adiposity of probiotics (*Lactobacillus gasseri* SBT2055) in adults with obese tendencies in a randomized controlled trial. *Eur J Clin Nutr* 64(6):636-43.
- Kalliomaki M, et al. 2008. Early differences in fecal microbiota composition in children may predict overweight. *Am J Clin Nutr* 87(3):534-538.
- Turnbaugh PJ, et al. 2006. An obesity-associated gut microbiome with increased capacity for energy harvest. *Nature* 444(7122):1027-31.
- Ley RE, et al. 2006. Microbial ecology: human gut microbes associated with obesity. *Nature* 444(7122):1022-3.
- Chen J, et al. 2012. *Bifidobacterium adolescentis* supplementation ameliorates visceral fat accumulation and insulin sensitivity in an experimental model of the metabolic syndrome. *Br J Nutr* 107(10):1429-1434.

Part 2

- Sugerman HJ. 2007. Increased Intra-abdominal pressure and GERD/Barrett's Esophagus. *Gastroenterol* 133(6):2075.
- English J. 2013. Gastric balance: heartburn not always caused by excess acid. *Nutrition Review* <<https://nutritionreview.org/2013/04/gastric-balance-heartburn-caused-excess-acid/>>
- Greenwald DA. 2004. Aging, the gastrointestinal tract, and risk of acid-related disease. *Am J Med* 117(5A):8S-13S.
- Krasinski SD, et al. 1986. Fundic atrophic gastritis in an elderly population. Effect on haemoglobin and several serum nutritional indicators. *J Am Geriatr Soc* 34(11):800-6.
- Jacobs A, et al. 1966. Gastric acid secretion in chronic iron-deficiency anaemia. *Lancet* 2(7456):190-2.
- El-Omar EM, et al. 1997. *Helicobacter pylori* infection and chronic gastric acid hyposecretion. *Gastroenterology* 113(1):15-24.
- Pounder RE and Ng D. 1995. The prevalence of *Helicobacter pylori* infection in different countries. *Aliment Pharmacol Therap* 9(2):33-9.
- Amieva MR and El-Omar EM. 2008. Host-bacterial interactions in *Helicobacter pylori* infection. *Gastroenterol* 134(1):306-23.
- Mobley HLT, et al. 2001. *Helicobacter pylori*: Physiology and Genetics. ASM Press, Washington DC. Ch 16.
- Bytzer P, et al. 2011. Diagnosis and treatment of *Helicobacter pylori* infection. *Dan Med Bull* 58(4):C4271.
- Marshall BJ, et al. 1985. Pyloric *Campylobacter* infection and gastroduodenal disease. *Med J Austr* 142(8):439-44.
1990. Omeprazole: blocks gastric acid secretion completely. *Drug Ther Bull* 28(13):49-52.
- Lee HR and Pimentel M. 2006. Bacteria and irritable bowel syndrome: the evidence for small intestinal bacterial overgrowth. *Curr Gastroenterol Rep* 8(4):305-311.
- Jacobs A, et al. 1966. Gastric acid secretion in chronic iron-deficiency anaemia. *Lancet* 2(7456):190-2.

- Henderson LM, et al. 1995. Effect of intragastric pH on the absorption of oral zinc acetate and zinc oxide in young healthy volunteers. *J Parent Enteral Nutr* 19(5).
- Budak NH, et al. 2014. Functional properties of vinegar. *J Food Sci* 79(5):R757-R764.
- Rutala WA, et al. 2000. Antimicrobial activity of home disinfectants and natural products against potential human pathogens. *Infec Control Hosp Epidemiol* 21(1):33-8.
- Leeman M, et al. 2005. Vinegar dressing and cold storage of potatoes lowers postprandial glycaemic and insulinaemic responses in healthy subjects. *Eur J Clin Nutr* 59(11):1266-71.
- Johnston CS, et al. 2004. Vinegar improves insulin sensitivity to a high-carbohydrate meal in subjects with insulin resistance or type 2 diabetes. *Diabetes Care* 27(1):281-282.
- Brighenti F, et al. 1995. Effect of neutralized and native vinegar on blood glucose and acetate responses to a mixed meal in healthy subjects. *Eur J Clin Nutr* 49(4):242-7.
- White AM and Johnston CS. 2007. Vinegar ingestion at bedtime moderates waking glucose concentrations in adults with well-controlled type 2 diabetes. *Diabetes Care* 30(11):2814-2815.
- Iizuka M, et al. Inhibitory effects of balsamic vinegar on LDL oxidation and lipid accumulation in THP-1 macrophages. *J Nutr Sci Vitaminol* 56(6):421-7.
- Laranjinha JA, et al. 1994. Reactivity of dietary phenolic acids with peroxy radicals: antioxidant activity upon low density lipoprotein peroxidation. *Biochem Pharmacol* 48(3):487-94.
- Gordon MH and Wishart K. 2010. Effects of chlorogenic acid and bovine serum albumin on the oxidative stability of low density lipoproteins in vitro. *J Agric Food Chem* 58(9):5828-33.

Part 3

- Yancy WS, et al. 2001. Improvement of gastroesophageal reflux disease after initiation of a low-carbohydrate diet: five brief case reports. *Altern Ther Health Med* 7(6):120.
- Austin GL, et al. 2006. A very low-carbohydrate diet improves gastroesophageal reflux and its symptoms. *Dig Dis Sci* 51(8):1307-12.
- Pimentel M and Lezcano S. 2007. Irritable bowel syndrome: bacterial overgrowth – what’s known and what to do. *Curr Treat Options Gastro* 10(4):328-337.
- Bures J, et al. 2010. Small intestinal bacterial overgrowth syndrome. *World J Gastroenterol* 16(24):2978-2990.
- Fasano A. 2012. Leaky gut and autoimmune diseases. *Clin Rev Allergy Immunol* 42(1):71-8.
- Lee HR and Pimentel M. 2006. Bacteria and irritable bowel syndrome: the evidence for small intestinal bacterial overgrowth. *Curr Gastroenterol Rep* 8(4):305-311.
- Pimentel M, et al. 2002. Increased prevalence of irritable bowel syndrome in patients with gastroesophageal Reflux. *J Clin Gastroenterol* 34(3):221-224.
- Bowe WP and Logan AC. 2011. Acne vulgaris, probiotics and the gut-brain-skin axis – back to the future? *Gut Pathog* 3(1):1.
- De Palma G, et al. 2014. The microbiota-gut-brain axis in gastrointestinal disorders: stressed bugs, stressed brain, or both? *J Physiol* 592(14):2989-2997.
- Hvatum M, et al. 2006. The gut-joint axis: cross reactive food antibodies in rheumatoid arthritis. *Gut* 55(9):1240-1247.
- Dukowicz AC, et al. 2007. Small intestinal bacterial overgrowth: a comprehensive review. *Gastroenterol Hepatol* 3(2):112-122.
- Valipe SR, et al. 2011. In vitro antimicrobial properties of caprylic acid, monocaprylin, and sodium caprylate against *Dermatophilus congolensis*. *Am J Vet Res* 72(3):331-5.
- Omura Y, et al. 2011. Caprylic acid in the effective treatment of intractable medical problems of frequent urination, incontinence, chronic upper respiratory infection, root canal tooth infection, ALS, etc., caused by asbestos & mixed infections of *Candida albicans*, *Helicobacter pylori* & cytomegalovirus with or without other microorganisms & mercury. *Acupunct Electrother Res* 36(1-2):19-64.
- Hoshimoto A, et al. 2002. Caprylic acid and medium-chain triglycerides inhibit IL-8 gene transcription in Caco-2 cells: comparison with the potent histone deacetylase inhibitor trichostatin A. *Br J Pharmacol* 136(2):280-286.
- Nair MK, et al. 2005. Antibacterial effect of caprylic acid and monocaprylin on major bacterial mastitis pathogens. *J Dairy Sci* 88(10):3488-95.
- Ghoshal UC. 2011. How to interpret hydrogen breath tests. *J Neurogastroenterol Motil* 17(3):312-317.
- Pimentel M, et al. 2012. Methanogens in human health and disease. *Am J Gastroenterol Supp* 1:28-33.

- Quigley EMM and Quera R. 2006. Small intestinal bacterial overgrowth: role of antibiotics, prebiotics, and probiotics. *Gastroenterol* 130:S78-S90.
- Chedid V, et al. 2014. Herbal therapy is equivalent to rifaximin for the treatment of small intestinal bacterial overgrowth. *Glob Adv Health Med* 3(3):16-24.
- Li Z, et al. 2010. Antioxidant-rich spice added to hamburger meat during cooking results in reduced meat, plasma, and urine malondialdehyde concentrations. *Am J Clin Nutr* 91(5):1180-1184.
- Opalchenova G and Obreshkova D. 2003. Comparative studies on the activity of basil—an essential oil from *Ocimum basilicum* L.—against multidrug resistant clinical isolates of the genera *Staphylococcus*, *Enterococcus* and *Pseudomonas* by using different test methods. *J Microbiol Methods* 54(1):105-10.
- Siddiqui BS, et al. 2012. Evaluation of the antimycobacterium activity of the constituents from *Ocimum basilicum* against *Mycobacterium tuberculosis*. *J Ethnopharmacol* 144(1):220-2.
- Sienkiewicz M, et al. 2013. The potential use of basil and rosemary essential oils as effective antibacterial agents. *Molecules* 18(8):9334-51.
- Grigoleit HG and Grigoleit P. 2005. Peppermint oil in irritable bowel syndrome. *Phytomedicine* 12(8):601-6.
- Rahimi R and Abdollahi M. 2012. Herbal medicine for the management of irritable bowel syndrome: a comprehensive review. *World J Gastroenterol* 18(7):589-600.
- Cappello G, et al. 2007. Peppermint oil (Mintoil) in the treatment of irritable bowel syndrome: a prospective double blind placebo-controlled randomized trial. *Dig Liver Dis* 39(6):530-6.
- Ford AC, et al. 2008. Effect of fibre, antispasmodics, and peppermint oil in the treatment of irritable bowel syndrome: systematic review and meta-analysis. *BMJ* 337:a2313.
- Raeisi M, et al. 2012. Essential oil tarragon (*Artemisia dracunculus*) antibacterial activity on *Staphylococcus aureus* and *Escherichia coli* in culture media and Iranian white cheese. *Iran J Microbiol* 4(1):30-34.
- Josling P. 2001. Preventing the common cold with a garlic supplement: a double-blind, placebo-controlled survey. *Adv Ther* 18(4):189-93.
- Nantz MP, et al. 2012. Supplementation with aged garlic extract improves both NK and γ -T cell function and reduces the severity of cold and flu symptoms: a randomized, double-blind, placebo-controlled nutrition intervention. *Clin Nutr* 31(3):337-44.
- Rivlin RS. 2001. Historical perspective on the use of garlic. *J Nutr* 131(3):951S-954S.
- De M, et al. 1999. Antimicrobial screening of some Indian spices. *Phytother Res* 13(7):616-8.
- Arora DS and Kaur J. 1999. Antimicrobial activity of spices. *Int J Antimicrob Agents* 12(3):257-62.
- Sivam GP, et al. 1997. *Helicobacter pylori*—in vitro susceptibility to garlic (*Allium sativum*) extract. *Nutr Cancer* 27(2):118-21.
- Ozaki Y, et al. 1991. Anti-inflammatory effect of *Zingiber cassumunar* Roxb. and its active principles. *Chem Pharm Bull* 39(9):2353-6.
- Zick SM, et al. 2011. Phase II study of the effects of ginger root extract on eicosanoids in colon mucosa in people at normal risk for colorectal cancer. *Cancer Prev Res* 4(11):1929-37.
- Ernst E and Pittler MH. 2000. Efficacy of ginger for nausea and vomiting: a systematic review of randomized clinical trials. *Br J Anaesth* 84(3):367-71.
- Chaiyakunapruk N, et al. 2006. The efficacy of ginger for the prevention of postoperative nausea and vomiting: a meta-analysis. *Am J Obstet Gynecol* 194(1):95-9.
- Vutyavanich T, et al. 2001. Ginger for nausea and vomiting in pregnancy: randomized, double-masked, placebo-controlled trial. *Obstet Gynecol* 97(4):577-82.
- Anti-emetic effect of ginger powder versus placebo as an add-on therapy in children and young adults receiving high emetogenic chemotherapy. *Pediatr Blood Cancer* 56(2):234-8.
- Chaieb K, et al. 2007. The chemical composition and biological activity of clove essential oil, *Eugenia caryophyllata* (*Syzygium aromaticum* L. Myrtaceae): a short review. *Phytother Res* 21(6):501-506.
- Aneja KR and Joshi R. 2009. Antimicrobial activity of *amomum subulatum* and *elettaria cadamomum* against dental caries causing microorganisms.
- Jamal A, et al. 2006. Gastroprotective effect of cardamom, *Elettaria cardamomum maton*. Fruits in rats. *J Ethnopharmacol* 103(2):149-153.
- Verma SK, et al. 2009. Blood pressure lowering, fibrinolysis enhancing and antioxidant activities of cardamom (*Elettaria cardamomum*). *Indian J Biochem Biophys* 46(6):503-6.
- Sharma R. 2012. Cardamom comfort. *Dent Res J* 9(2):237.

Fratini F, et al. 2014. Antibacterial activity of essential oils, their blends and mixtures of their main constituents against some strains supporting livestock mastitis. *Filoterapia* 96:1-7.

Alexandrovich I, et al. 2003. The effect of fennel (*Foeniculum Vulgare*) seed oil emulsion in infantile colic: a randomized, placebo-controlled study. *Altern Ther Health Med* 9(4):58-61.

Part 4

Selye H. 1976. *Stress Without Distress*. Psychopathy of Human Adaptation. Springer. Pg 137-146.

Selye H. 1946. Relation of the adrenal cortex to arthritis. *Lancet* 247(6408):942.

Selye H. 1951. Role of the adrenals in the production of renal and cardiovascular damage by anterior pituitary preparations. *Lancet* 257(6653):483-487.

Selye H and Pentz EI. 1943. Pathogenetical correlations between periarteritis nodosa, renal hypertension and rheumatic lesions. *Can Med Assoc* 49(4):264-272.

Mayer EA, et al. 2000. The evolving neurobiology of gut feelings. *Prog Brain Res* 122:195-206.

Konturek PC, et al. 2011. Stress and the gut: Pathophysiology, clinical consequences, diagnostic approach and treatment options. *J Physiol Pharmacol* 62(6):591-99.

Lyte M, et al. 2011. Stress at the intestinal surface: catecholamines and mucosa-bacteria interactions. *Cell Tissue Res* 343(1):23-32.

Cohen S, et al. 2007. Psychological stress and disease. *JAMA* 298(14):1685-1687.

Salleh MR. 2008. Life event, stress and illness. *Malays J Med Sci* 15(4):9-18.

Peeke PM and Chrousos GP. 1995. Hypercortisolism and obesity. *Ann NY Acad Sci* 771:665-76.

Adam TC, et al. 2010. Cortisol is negatively associated with insulin sensitivity in overweight Latino youth. *J Clin Endocrinol Metab* 95(10):4729-35.

Black PH. 2006. The inflammatory consequences of psychologic stress: relationship to insulin resistance, obesity, atherosclerosis and diabetes mellitus, type II. *Med Hypotheses* 67(4):879-91.

O'Donovan A, et al. 2012. Lifetime exposure to traumatic psychological stress is associated with elevated inflammation in the Heart and Soul Study. *Brain Behav Immun* 26(4):642-649.

Cohen S, et al. 2012. Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. *PNAS* 109(16):5995-5999.

Elenkov IJ and Chrousos GP. 1999. Stress hormones, Th1/Th2 patterns, pro-anti-inflammatory cytokines and susceptibility to disease. *Trends Endocrinol Metab* 10(9):359-368.

Bailey MT, et al. 2010. Stressor exposure disrupts commensal microbial populations in the intestines and leads to increased colonization by *Citrobacter rodentium*. *Infect Immun* 78(4):1509-1519.

Bowe WP and Logan AC. 2011. Acne vulgaris, probiotics and the gut-brain-skin axis – back to the future? *Gut Pathog* 3(1):1.

Bailey MT, et al. 2011. Exposure to a social stressor alters the structure of the intestinal microbiota: implications for stressor-induced immunomodulation. *Brain Behav Immun* 25(3):397-407.

Saunders PR, et al. 1994. Acute stressors stimulate ion secretion and increase epithelial permeability in rat intestine. *Am J Physiol* 267(5 pt 1):G794-9.

Spitz JC, et al. 1996. Characteristics of the intestinal epithelial barrier during dietary manipulation and glucocorticoid stress. *Crit Care Med* 24(4):635-41.

Le Fevre M, et al. 2006. Eustress, distress, and their interpretation in primary and secondary occupational stress management interventions: which way first? *J Manag Psychol* 21(6):547-565.

Howard F. 2008. Managing stress or enhancing wellbeing? Positive psychology's contributions to clinical supervision. *Austral Psychol* 43(2):105-113.

Ambriz MGJ, et al. 2012. Psychological and social factors that promote positive adaptation to stress and adversity in the adult life cycle. *J Happin Studies* 13(5):833-848.

Liefooghe B, et al. 2008. Working memory costs of switching. *J Exp Psychol* 34(3):478-494.

Russ TC, et al. 2012. Association between psychological distress and mortality: individual participant pooled analysis of 10 prospective cohort studies. *BMJ* 345:e4933

Part 5

Park SC, et al. 2011. Prevention and management of non-steroidal anti-inflammatory drugs-induced small intestinal injury. *World J Gastroenterol* 17(42):4647-4653.

- Galland L. 2010. Diet and inflammation. *Nutr Clin Pract* 25(6):634-40.
- Kerckhoffs AP, et al. 2010. Intestinal permeability in irritable bowel syndrome patients: effects of NSAIDs. *Dig Dis Sci* 55(3):716-23.
- Bjarnason I, et al. 1986. Effect of non-steroidal anti-inflammatory drugs on the human small intestine. *Drugs* 32(1):35-41.
- Farhadi A, et al. 2008. Susceptibility to gut leakiness: a possible mechanism for endotoxaemia in non-alcoholic steatohepatitis. *Liver Int* 28(7):1026-33.
- Val Wijck K, et al. 2012. Aggravation of exercise-induced intestinal injury by Ibuprofen in athletes. *Med Sci Sports Exerc* 44(12):2257-62.
- Iwamoto J, et al. 2013. Clinical features of gastroduodenal injury associated with long-term low-dose aspirin therapy. *World J Gastroenterol* 19(11):1673-1682.
- Winzler S and Rosenstein BD. 1998. Non-steroidal anti-inflammatory drugs. A review. *AAOHN J* 46(5):253-9.
- Ferreira SH. 1980. Peripheral analgesia: mechanism of the analgesic action of aspirin-like drugs and opiate-antagonists. *10(52):237S-245S*.
- Rao P and Knaus EE. 2008. Evolution of nonsteroidal anti-inflammatory drugs (NSAIDs): cyclooxygenase (COX) inhibition and beyond. *J Pharm Pharm Sci* 11(2):81s-110s.
- Rogers MA and Aronoff DM. 2016. The influence of non-steroidal anti-inflammatory drugs on the gut microbiome. *Clin Microbiol Infect* 22(2):178.e1-9.
- Wallace JL. 2008. Prostaglandins, NSAIDs, and gastric mucosal protection: why doesn't the stomach digest itself? *Physiol Rev* 88(4):1547-1565.
- Sachs CJ. 2005. Oral analgesics for acute nonspecific pain. *Am Fam Physician* 71(5):913-918.
- Nema H and Kato M. 2010. Investigation of gastroduodenal mucosa injuries caused by low-dose aspirin therapy in patients with cerebral infarction. *J Gastroenterol Hepatol* 25(1):S1119-21.
- Sorenson HT, et al. 2000. Risk of upper gastrointestinal bleeding associated with use of low-dose aspirin. *Am J Gastroenterol* 95:2218-2224.
- Baigent C, et al. 2009. Aspirin in the primary and secondary prevention of vascular disease: collaborative meta-analysis of individual participant data for randomised trials. *Lancet* 373(9678):1849-60.
- Aronoff DM, et al. 2006. New insights into the mechanism of action of acetaminophen: It's clinical pharmacologic characteristics reflect its inhibition of the two prostaglandin H2 synthases. *Clin Pharmacol Ther* 79(1):9-19.
- Watkins PB, et al. 2006. Aminotransferase elevations in healthy adults receiving 4 grams of acetaminophen daily. A randomized controlled trial. *JAMA* 296(1):87-93.
- Lee WM. 2004. Acetaminophen and the U.S. acute liver failure study group: lowering the risk of hepatic failure. *Hepatology* 40(1):6-9.
- Machado GC, et al. 2015. Efficacy and safety of paracetamol for spinal pain and osteoarthritis; systematic review and meta-analysis of randomised placebo controlled trials. *BMJ* 350:h1225.
- Koes BW and Enthoven WT. 2014. Do patients with acute low-back pain need paracetamol. *Lancet* 384(9954):1556-1557.
- Sturniolo GC, et al. 2001. Zinc supplementation tightens "leaky gut" in Chron's disease. *Inflamm Bowel Dis* 7(2):94-8.