INSIDER’S GUIDE

STOOL PATHOGENS: INTERPRETATION AND TREATMENT

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Harmful Gut Environment

The Big Picture

Our body’s have been amazingly put together and strive to achieve a level of optimal health against external and internal forces.

The focus of this lesson is to provide a detailed overview of the internal forces known to play havoc with our body’s ability to seek this desired level of health and well-being.

These internal forces make up three enemies commonly referred to as the bacterial pathogen, parasitic pathogen and the fungal pathogen, aka. Candida.

Although our body’s possess (or least should possess) a rich level of healthy bacteria, it is more common than not for this beneficial immune fighting bacteria to be reduced to a level unable to match the above enemy’s war plans of destruction.

As physicians and health care professionals, it is important to know your enemy and more important, to know how to circumvent their goals of GI domination.

What Happens When the GI Terrain is Overrun with Nasty Bugs?

If the beneficial flora succumbs to the onslaught of heavy antibiotic use and/or the consumption of the wrong foods, the harmful bugs can overgrow.

This take over of the bad guys may results in one of more of the following:

- Depletion of vitamin B12 and some amino acids
- Short-circuits digestive enzymes
- Converts essential fatty acids into damaging fats
- Increases the potential of GI infection
- Encourages GI inflammatory diseases
- Interferes with the breakdown of bile acids and estrogens creating a fertile environment for cancer

Dybiosis – The Battle Term of Imbalanced Flora

Intestinal dysbiosis may be defined as a state of disordered microbial ecology that causes disease.' Specifically, the concept of dysbiosis rests on the theory that patterns of intestinal flora, specifically overgrowth of some microorganisms found commonly in intestinal flora, have an impact on human health. Symptoms and conditions thought to
be caused or complicated by dysbiosis include inflammatory bowel diseases, inflammatory or autoimmune disorders, food allergy, atopic eczema, unexplained fatigue, arthritis, mental/emotional disorders in children and adults, malnutrition, and breast and colon cancer."

Unfortunately many well meaning physicians overlook the obvious and do not give a nod to the issue of dysbiosis and the impact on one’s health. Simply stated, dysbiosis is the term used to indicating that the inmates (bad bugs) have taken over the asylum!

In more medical terms, the harmful microbes have been allowed to grow over taking the GI terrain.

Now there is a more important question we need to ask ourselves and that question is:

What has happened that has enabled these bad bugs to overgrow and upset the balanced gut ecology?

Another question that may even be more significant is: What turns a good bug into a bad bug? Why did the good bug decide to hook up with the enemy?

Before we provide a list of the nine common pathways that lead to dysbosis, it behooves us to ask what determines just how pathogenic a microbial organism can become.

The answer is quite simple but rest assured it will take a doctor like “you” to solve this ecology breakdown.

The two primary factors that determine the extent an organism will go to destroy the ecology environment and ultimately one’s health is:

1. It’s ability to overgrow and produce toxins that can injure us.
2. Our resistance or the strength of our immune defenses

Unfortunately, our review and we are confident your review of hundreds and hundreds of medical records fail to show one traditional physician in a thousand focusing on ways to strengthen the immune system. Instead, any and all harmful microbial pathogens are treated with a bombardment of prescriptive antibiotics. And if the host has not recovered, more potent antibiotics are prescribed. This vicious cycle can go on and on creating a worsening of the gut ecology leading to a host of immunological health challenges.

We ask why the physiology of gut ecology is ignored and why a well trained health care professional does not make the re-inoculation of good flora part of his/her protocol.

As functional diagnostic medicine professionals you understand that this battle may indeed require the introduction of either a prescriptive or natural agent to irradiate the
harmful microbes but you of all intelligent medical detectives understand that building up the army forces is mandatory to ultimately win the war.

**Why Does Good Flora Go Bad**

Overgrowth of bad flora and the ultimate existence of dysbiosis are clear signals that something has gone awry in the GI ecology and requires our utmost attention.

There are at least nine pathways that lead to dysbiosis. They include:

1. Eating too much sweets and starchy food
2. Overuse of antibiotics
3. Stress
4. Poor digestion
5. Inflammation
6. Infection
7. Exposure to toxins
8. Lowered immune defenses
9. Unknown causes

**The Harmful Effects of Poor Food Choices on the Gut Ecology**

The beneficial flora of the gut requires a healthy diet of adequate amounts of fiber from vegetables and whole grains (ruling out gluten sensitive patients). Bacteria strive on high-fiber diets. If the right diet is provided, this species of bacteria will enjoy accelerated growth and flourish.

Unfortunately, meals devoid of unhealthy fats, poisoned meats (meats spiked with hormones and antibiotics) and low in fiber tend to contribute to **putrefactive dysbiosis**.

Under these circumstances, there is an overgrowth of a normal bacteria called **bacteroides**. Unfortunately this bacteria has a nasty ability to release a toxic by product. The by product is **ammonia**. Increased ammonia makes the gut ecology too alkaline resulting in a decreased production of the essential gut fuel, **butyrate**.

With less gut fuel, there is a widespread shortage of nutrition able to feed the digestive tract. In addition, increased levels of bile acids and estrogen go up raising the risk for colon and breast cancer.

And if that was not bad enough, when the destructive flora overgrows, their increased concentration becomes toxic to the gut potentially causing a host of health challenges including inflammatory GI disorders.

This eventually becomes a vicious cycle that is **self-perpetuating**.
The Best Kept Secret: Immunity

There is a secret out there and many health professionals have forgotten it. Sit down and read any medical physiology textbook and you will be enlightened to the secret of the ages. This secret is the gastrointestinal tract is the largest immune organ in our body. The intestinal lining has an enormous surface area that separates the outer world from the internal environment of our body.

This is a tactical location for our body's defense structure. Remember this is the site of entry for potentially dangerous organisms or chemicals. It doesn't take an Einstein to see that when this large, strategically placed immune system isn't working well, our defenses are lowered.

Once our barriers are down, it may not be possible to keep the intestinal terrain in balance making it more difficult to defend against harmful pathogens.

The Diagnosis of Dysbiosis

Although dysbiosis is not a disease per se, it serves as a warning for the potential of a detectable disease.

A patient may have no GI symptoms but the presence of dysbiosis (disturbed gut ecology) can create a vague sense of not feeling well.

The diagnosis of dysbiosis centers around three approaches

- A careful history
- A specific test called a comprehensive diagnostic stool analysis (CDSA)
- Organic Acids/Dysbiosis (MetaMetrix) Will be discussed in another lesson
- Testing for yeast, bacteria and parasites

It is quite common for other illnesses to develop in the presence of dysbiosis. It is because of this that we believe a CDSA is of value and should be done whenever there is an unresolved illness.

The Next Step in Dealing With Dysbiosis

In the management of dysbiosis, identifying the underlying cause is of utmost importance.

The goals in managing dysbiosis are to restore the normal microflora, provide nutrients that will heal the intestinal mucosa, reduce toxic exposures and increase antibodies in the GI tract (SigA).
Facing Our Enemy and Taking Up Arms

Gastrointestinal (GI) tract infections are common and can be either clinical (symptomatic) or sub-clinical (without symptoms).

Some have active GI symptoms, others present with general complaints: fatigue, body pain, headaches, cognitive problems, light headedness, brain fog and/or general malaise.

Currently the two most common infections are Helicobacter pylori, a bacterium that primarily inhabits the stomach, esophagus and upper duodenum, and Cryptosporidium parvum, a parasite that primarily inhabits the small intestine and regularly cycles from intracellular to extracellular.

Helicobacter pylori infections are well documented to be an underlying cause of ulcers, acid reflux, burping and belching and general upper GI distress as well as stomach cancer.

Due to its ability to embed itself into the deep mucosa of the stomach, Helicobacter are often difficult to eradicate.

Cryptosporidium parvum is a common and aggressive parasite found to invade the intestinal epithelial cells damaging the topography of the small intestine. This in turn will inhibit absorption and assimilation of nutrients and compromise intestinal mucosal barrier defenses, further weakening the body's defense against other opportunistic infectious agents.

Although Cryptosporidium parvum can cause high fevers, severe diarrhea and even death, it most frequently exhibits very mild or no GI symptoms making it a silent but harmful pathogen.

Other commonly encountered parasite infections include Entamoeba histolytica, Giardia lamblia, Blastocystis hominis and Ascaris (round worm).

Even more menacing is the fact that primary GI pathogens have the unique ability to metamorphosize into various stages allowing them to migrate to tissues and organs sometimes distant from the GI tract.

For example Cryptosporidium parvum can sometimes be found in the lungs and conjunctiva of the eyes and Helicobacter pylori has been located in the oral cavity and even the prostate gland. Such stages, including cysts, can remain dormant within tissues, and can be extremely difficult to detect. That is why it is extremely important to test for GI infections using both microbiology and immunological assays.
Now that you have an understanding of the issue of compromised gut ecology, we will now discuss the topic of harmful pathogens and show you what actions steps to take to win the battles and ultimately the war.

**The Parasitic Pathogen**

We have been led to believe that our modern sanitation and water treatment has eradicated the existence of parasites from our present society. But research shows that parasitic infection is common, and the incidence is increasing. In many cases these infections underlie familiar digestive illness and other conditions as well.

With increase of overseas travel poorly controlled immigration exposure to and transmission of parasitic infection is a reality we need to take very serious.

Giardia, for instance, is often waterborne, and these infections are on the rise. In 1997, *The Wall Street Journal* reported an average of 2 million cases annually in the United States. Giardia is also a problem worldwide, even in some modern cities (via the public water systems).

Cyclospora, a parasite in the news, is tracked as a new or emerging pathogen; sometimes it is transmitted on imported fruit. In 1996 it was found on Guatemalan strawberries and raspberries. However, it is also domestic and common in the United States; like all infectious agents, it can be transferred in stool, on human hands, and as contaminants in food, especially fresh vegetables and fruit, and water.

Cryptosporidium, another waterborne parasite, caused illness in more than 400,000 people in Milwaukee in 1993. More than 4,000 were hospitalized, and more than 100 died. Cryptosporidium is found in the public water systems and reservoirs of many American cities. In some places, such as the San Francisco Bay Area, it is known to be transmitted by the runoff from hill-sides where cattle graze, upstream from unprotected reservoirs.
Most of us live crowded together in big cities, many of us travel overseas, we frequently have contact with people from all over the world, and we have many opportunities for exposure.

**Common Parasitic Pathogens**

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Digestive Symptoms &amp;/or Comments</th>
<th>General Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blastocystis Hominis</td>
<td>Flatulence, bloating, diarrhea, cramps, constipation, poor digestion/poor absorption</td>
<td>Fatigue, nervous and skin disorders, pain, skin conditions, nausea, allergies, muscle problems</td>
<td>Treatment is controversial. Metronidazole or iodoquinole are reportedly effective.</td>
</tr>
<tr>
<td>Dientamoeba fragilis</td>
<td>Primary symptoms are diarrhea, fatigue and abdominal bloating, although may be asymptomatic. In children, symptoms include abdominal pain, fatigue, nausea, intermittent diarrhea, poor weight gain.</td>
<td>Iodoquinol is the drug of choice. Tetracycline is effective, but considered investigational for this use by the FDA.</td>
<td></td>
</tr>
<tr>
<td>Giardia lamblia</td>
<td>Clinical features range from asymptomatic to severe diarrhea and malabsorption.</td>
<td></td>
<td>Treat with metronidazole and tinidazole. Nitazoxanide is somewhat effective in children.</td>
</tr>
<tr>
<td>Ascaris lumbricoides</td>
<td>Infections may cause stunted growth in children, but adult worms usually cause no acute symptoms. High worm burdens may cause abdominal pain and intestinal obstruction. Migrating adult worms may cause symptomatic occlusion of the biliary tract or oral expulsion.</td>
<td>Mebendazole, (albendazole and pyrantel pamoate are FDA-approved but this use is investigational). In the US, ascariasis is generally treated for 1-3 days.</td>
<td></td>
</tr>
<tr>
<td>Clonorchis sinensis</td>
<td>Clonorchis sinensis is transmitted via ingestion of pickled, smoked, salted, imported or undercooked freshwater fish.</td>
<td>Symptoms arise as inflammation and intermittent obstruction of the biliary ducts. Acute, abdominal pain, nausea, diarrhea, and eosinophilia can occur. In long-standing infections, cholangitis, cholelithiasis, pancreatitis, and cholangiocarcinoma can develop.</td>
<td>Praziquantel or albendazole are the drugs of choice, although albendazole is considered investigational by the FDA.</td>
</tr>
<tr>
<td>Enterobius vermicularis (Pinworm)</td>
<td>Pinworm is transmitted by scratching perianal area, then putting hand to mouth, thus passing on eggs. Bed linens and</td>
<td>Typical symptoms include nocturnal perianal pruritus, which can lead to skin bacterial infection, and</td>
<td>The drug of choice is pyrantel pamoate. The best preventive measures are good personal and household hygiene.</td>
</tr>
<tr>
<td>Stool Pathogens</td>
<td>Interpretation and Treatment</td>
<td>Treatment</td>
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<tr>
<td><strong>Hookworms</strong></td>
<td>Symptoms include itching and a rash at the site of where the larvae penetrated the skin. While a light infection may cause no symptoms, heavy infection can cause anemia, abdominal pain, diarrhea, loss of appetite, and weight loss.</td>
<td>Hookworm is generally treated with the drug mebendazole. It is highly effective if given twice per day for three days. It kills both the worms and the eggs.</td>
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</tr>
<tr>
<td><strong>Cryptosporidium</strong></td>
<td>Watery diarrhea is the most frequent symptom, and can be accompanied by dehydration, weight loss, abdominal pain, fever, nausea and vomiting. Cryptosporidium can be fatal.</td>
<td>Oral nitazoxanide is recommended for immunocompetent patients</td>
<td></td>
</tr>
<tr>
<td><strong>Entamoeba histolytica</strong></td>
<td>Diarrhea, constipation, cramps, bloating, flatulence</td>
<td>Fatigue, nausea, pain, weight loss, insomnia</td>
<td></td>
</tr>
<tr>
<td><strong>For asymptomatic infections</strong>, iodoquinol, paromomycin, or diloxanide furoate (not commercially available in the U.S.) are drugs of choice. For symptomatic intestinal disease, or extraintestinal infections (e.g., hepatic abscess), drugs of choice are metronidazole or tinidazole, immediately followed by iodoquinol, paromomycin, or diloxanide furoate.</td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>Taenia sp. (Tapeworm)</strong></td>
<td>Tapeworms are long, segmented worms. Adult worms survive inside their human hosts, where they are limited to the intestinal tract.</td>
<td>The most common treatment for tapeworm infection involves oral medications that are toxic to the adult tapeworm, such as praziquantel and albendazole. The medication prescribed depends on the species of organism involved.</td>
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</tr>
<tr>
<td><strong>Trichuris trichiura</strong></td>
<td>Also called whipworm infection, T. trichiura, is a common worldwide infection that mainly</td>
<td>Symptoms range from mild to severe, sometimes, and whipworm can even be asymptomatic. A severe</td>
<td></td>
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<td></td>
<td></td>
<td>Mebendazole taken by mouth for 3 days is commonly prescribed when the infection causes</td>
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</table>
affects children. When the eggs hatch the whipworm attaches to the wall of the large intestine. Whipworm is found primarily in warm, humid climates. The main risk factor for infection is swallowing soil contaminated with feces. Some outbreaks have been traced to contaminated vegetables (due to presumed soil contamination).

<table>
<thead>
<tr>
<th>Entamoeba coli (E. coli)</th>
<th>Diarrhea, cramps, flatulence, bloating, constipation, irritable bowel</th>
<th>Fatigue, allergies, headaches, nausea, depression/irritability, joint/back pain, skin problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyclospora</td>
<td>Symptoms that come and go, bloating, flatulence, diarrhea, cramps</td>
<td>Fatigue, itching, depression nausea, muscle problems, anemia</td>
</tr>
<tr>
<td>Entamoeba hartmanni</td>
<td>Diarrhea, bloating, cramps, flatulence, irritable bowel</td>
<td>Nervous system, respiratory, and skin disorders, allergies, nausea</td>
</tr>
</tbody>
</table>

Risk Factors You Need to Know

- Foreign travel
- Having a partner or someone in the household with a parasite problem
- Previous parasitic infection
- Not washing fresh vegetables
- Drinking tap water
- Poor hygiene
- Dining out often
- Frequenting salad bars
- Having pets
- Going camping (or drinking water from streams or even fountains)
- Working at an infant-care center
- Living in an institutional setting or group home
How Parasites Damage the Body

The parasitic pathogen is a survivalist and will compromise the human immune system to promote and ensure its existence.

Although the simple presence of a parasitic pathogen can play havoc with the gut, liver and other organs, it is in effect the toxic by-products emitted by the bug that proves to be most destructive. This in turn can disrupt digestive activity causing malabsorption and interfering with the action of digestive enzymes and nutrients.

The Challenge of Parasitic Detection

Parasites have intricate life cycles and are often not shed at regular intervals. In fact, three of the major parasites in the United States and worldwide (amoebas, giardia, and cyclospora) tend to be shed at irregular intervals. This means that the parasite may be present in the stool for two, three, or four days a week, but not the rest of the week.

Entamoeba histolytica is active for one or two days, and then is not typically active or detectable the next day or two. When E. histolytica migrates to the liver it disappears from the gut and becomes undetectable in fecal specimens. If the stool sample is collected from a patient with one of these cyclical parasites on a day when the pathogen is not active, it won't be in the stool and obviously won't be detected by testing.

However, this doesn't mean that there's no infection present. At the current time this is a limitation for which no modern technology can compensate. Consequently repeated samples are very important. Generally, to make testing practicable, we recommend at least two or three samples be taken on different days.

Novel Methods of Detection

The gold standard and still considered the most effective method of detecting parasites is the stool analysis. The optimal approach involves taking samples every other day, a minimum of 48 hours apart, collecting at least two or three samples.

Although some microbes such as E. histolytica reside in the large intestine, many are harbored in the small intestine. Pathogens such as giardia reside primarily in the small intestine, where they hang on for there life to the intestinal folds and cannot usually be detected in samples from stool further down the digestive tract.

In order to increase the odds that a parasite can be found, it is necessary to have one of the samples done as a purge inducing a diarrheal episode.
Other Methods of Testing

Elevated white blood count (eosinophil level >3 and monocytes >7) may be used as a screening tool to indicate the need for further testing.

Antibody testing is also available. Antibody levels for immunoglobulin G (IgG) can indicate infection, but not whether the infection is current or previous. Repeated testing for IgM levels will show if the infection is currently active.
Samples of blood serum can be evaluated to detect parasites found in the blood. However, this method is useful only for parasites of the circulatory system, not those most typically found in the GI tract.

**The Parasite Antibodies Profile (BioHealth Diagnostics)**

**Overview**

The Parasite Antibodies Profile II uses serum to assess the presence of IgG and IgM antibodies to the common parasites Entamoeba histolytica and Giardia lamblia, Entamoeba histolytica, Giardia lamblia and Toxoplasma gondii.

**Clinical Aspects**

The IgM antibody is the early responder. IgM is elevated during the early stages of an exposure to a parasite. There are two possible outcomes, either the immune system will eradicate the parasite or the parasite will begin to colonize and infect the body. If IgM is the only elevated antibody to a parasite, it must be correlated with active symptoms to warrant treatment. If asymptomatic, it is quite possible that the patient's immune system has won the battle with the parasite on its own. If treatment is not chosen at this time, it would be prudent to retest in 2-3 months to make sure that the patients’ immune system has in fact eradicated the infection.

The IgG antibody is the long term responder. IgG elevations mean that either a long term chronic infection exists that was either treated or untreated, or it is indicative of a protective antibody from a past infection that was resolved. The patient must be asked about current symptoms and past parasite infections and treatments in order to distinguish clinically relevant results. If necessary, other lab tests may be indicated to clinically correlate all data to a final accurate diagnosis.

**The importance of secretory IgA in the diagnosis of and protection against parasitic infections**

The detection of antibodies can be a very useful indicator that an individual has been infected with a specific parasite. A positive result for antibodies in a person with no exposure to the parasite prior to recent travel in an area where disease is endemic indicates recent infection. However, detection of specific antibodies in a person native to an area where the parasite is endemic may reflect only a past infection unrelated to current clinical status.
In general, the detection of antibodies to parasitic diseases indicates infection only at some in determinate time, and not necessarily acute or current infection. Levels of antibodies to parasites slowly decline after the patient is cured of the infection but generally last for at least 6 month sup to many years, depending on the infecting parasite.

Since the measurement of salivary antibodies may represent mucosal immunity, and the test is simple to perform and is noninvasive, we developed ELISA tests for determining salivary anti-parasite IgA antibodies and their target receptors or antigens. This includes, but is not limited to, the following:

- Entamoeba histolytica
- Giardia lamblia
- Toxoplasma gondii
- Blastocystis hominis
- Cryptosporidium parvum
- Naegleria
- Trichomonas vaginalis
- Ascaris
- Taenia pisiformis
- Trichuris trichiura
- Lectin Adherence Receptor
- Colonic Epithelial Cells
- Transglutaminase
- Tropomyosin

Successful induction of secretory antibodies is dependent on a number of variables, including the nature of the antigen, duration of antigenic challenge, and prior immune status. Local or central challenge of secretory tissues may also have an effect on the systemic immune system. The sequence of events characteristic of mucosal immune reaction following epithelial tissue (gut, oral, nasal, skin, genitourinary) exposure to parasitic antigens:

a. First event: Production of secretory IgA in saliva against the parasite.

b. Secondary events occur only in cases of impaired epithelial tissue integrity, which result in the production of IgA, IgM and IgG antibodies in the blood against the parasite.

Initially, all these antibodies are developed specifically against parasitic antigens. But due to colonic epithelial cell damage caused by proteases produced by the parasite or lectin-like receptors, or due to molecular mimicry, antibodies may be produced against the host tissue antigens, which may in turn initiate autoimmune reaction.

The simultaneous performance of tests for IgA, IgM, IgG in the blood and IgA in saliva against parasitic antigens and target tissue antigens make it possible to distinguish between pathogenic versus protective antibodies produced against parasitic antigens.
Therefore, for the early and comprehensive detection of these parasitic infections as the cause of autoimmune disease, both saliva (IgA) and blood (IgA, IgG, IgM) antibody levels should be measured. Moreover, since impaired immunity, as reflected by a low total secretory IgA, may result in an equally low level of saliva IgA against the offending agent, a false negative result may occur. It is therefore highly recommended that total Secretory IgA always be evaluated along with the specific antibody to parasitic antigens.

**Common Bacterial Pathogens**

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Digestive Symptoms &amp;/or Comments</th>
<th>General Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Helicobacter pylori</strong></td>
<td>H. pylori causes peptic ulcer disease and has been associated with increased gastric cancer risk. H. pylori is a Type 1 carcinogen. It is estimated that 50% of the world’s population is infected with H. pylori.</td>
<td>The main symptoms are acute gastritis with abdominal pain, nausea and vomiting, usually within two weeks of infection. Recurrent abdominal symptoms without ulcer disease (non-ulcer dyspepsia) are common.</td>
<td>The most common treatment is the “triple therapy” of colloidal bismuth, tetracycline and metronidazole, which has resulted in 60-95% eradication. The NIH also recommends antisecretory drugs.</td>
</tr>
<tr>
<td><strong>Clostridium difficile</strong></td>
<td>C. difficile is found in chronic diarrheal diseases such as pseudomembranous colitis. It increases when the antibiotic-sensitive flora are suppressed. Overuse of antibiotics such as clindamycin, ampicillin, tetracyclines, chloramphenicol has been implicated in causing antibiotic</td>
<td>The main symptoms are cramping, lower abdominal pain, fever and diarrhea. Symptoms usually decrease once antibiotics are stopped, though they can continue for up to 4 weeks.</td>
<td>Animal studies have found vancomycin to aid in decreasing diarrhea. Nonantibiotic treatments include: Saccharomyces boulardii (inhibits toxin from ulcerating mucosa), berberine, oregano oil or curcumin.</td>
</tr>
<tr>
<td><strong>Campylobacter sp.</strong></td>
<td>Contaminated animal food sources are the primary cause, especially poultry and red meat. Puppies may also become infected from rodents and birds.</td>
<td>Symptom onset is generally abrupt. Influenza-like symptoms are common, including headache and malaise. GI symptoms include abdominal pain, nausea and vomiting. The degree of diarrhea varies. Campylobacter has been associated with reactive arthritis. The infection is generally self-limiting.</td>
<td>Erythromycin is the best choice, though tetracycline and nalidixic acid are also effective. Grapefruit seed extract may also be helpful.</td>
</tr>
<tr>
<td><strong>Entero-hemorrhagic Escherichia coli (EHEC).</strong></td>
<td>Also referred to as; Shiga toxin-producing E. coli (STEC). This strain is one of the four recognized categories of diarrheagenic E. coli.</td>
<td>Symptoms include bloody diarrhea, cramping, little or no fever.</td>
<td>Since the 1990’s strain 0157 and other STEC strains have demonstrated resistance to streptomycin, sulfanamides and tetracycline.</td>
</tr>
</tbody>
</table>
EHEC includes serotypes that produce Shiga toxins, including E. coli 0157:H7. 50% of Shiga toxin strains are not E. coli 0157. Overall prevalence varies. Incidence is highest in summer months.

**Testing for Bacterial Anaerobes**

There are currently a number of ways to indirectly gain information on anaerobes:

- Antibody testing—Immunosciences Laboratory.
- Organic acids testing—Metametrix and Great Plains Lab. (Will be discussed in another lesson)
- Monitoring metabolic by-products—A comprehensive digestive analysis measures various biochemicals in the stool that reflect the health of the digestive tract and possible bacterial overgrowth.

### Pathogens (E4)

<table>
<thead>
<tr>
<th>Pathogenic Bacteria</th>
<th>Value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>H. pylori</td>
<td>&lt; 0.001</td>
<td>&lt;= 1.0</td>
</tr>
<tr>
<td>C. difficile</td>
<td>&lt; 0.001</td>
<td>&lt;= 1.0</td>
</tr>
<tr>
<td>Campylobacter</td>
<td>&lt; 0.001</td>
<td>&lt;= 1.0</td>
</tr>
<tr>
<td>E. H.E. coli</td>
<td>&lt; 0.001</td>
<td>&lt;= 1.0</td>
</tr>
</tbody>
</table>
Candida/Fungal Pathogens

The overgrowth of Candida albicans is well known to practitioners of nutritional medicine. What is less well known is the fact that candida infections often accompany parasitic infections. The energy drain on the system by the parasite tends to provide an opportunity for the candida.

An overgrowth of candida can cause physical symptoms. It can also decrease the beneficial bacteria. Researchers have found a definite relationship between the higher levels of fungal spores and lower levels of the desirable bacteria. This means that the microorganisms are competing for the same limited space and finite supply of nourishment, in the closed ecosystem of the digestive tract. If there are factors that promote the growth of candida, it consumes the resources and the space that would have originally been allotted to the beneficial bacteria (lactobacillus and bifidus). These flora then decrease, limiting their positive role in the digestive process. Lab testing frequently demonstrates this change in balance.

Microbial Sensitivity Profile

Rhodotorula

<table>
<thead>
<tr>
<th>Pharmaceuticals</th>
<th>Sensitive</th>
<th>Resistant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluconazole</td>
<td>S</td>
<td></td>
</tr>
<tr>
<td>Amphotericin B</td>
<td>S</td>
<td></td>
</tr>
<tr>
<td>Ketoconazole</td>
<td>S</td>
<td></td>
</tr>
<tr>
<td>Nystatin</td>
<td>S</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Botanicals</th>
<th>Sensitive</th>
<th>Resistant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Berberine</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>Black walnut</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>Caprylic acid</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>Cats Claw</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>Goldenseal</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>Oregano</td>
<td>R</td>
<td></td>
</tr>
</tbody>
</table>

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Which came first—the decrease in the friendly flora or the overgrowth of candida?

Stable flora are observed in very young children, and it is true that candida is normally present in the gut of most people as a harmless coexisting organism (a commensal).

However, if yeast becomes the dominant gut flora, it can become a destructive pathogen and take over in the digestive ecosystem.

If the population of candida becomes too large and its strength and virulence increases, candida can cause considerable damage, especially in immune-compromised patients.

The yeast can migrate to other tissues and become systemic. Once candida becomes blood born and systemic, then every organ system is at risk. Systemic candidiasis can cause serious damage, particularly to the reproductive system in females once it develops as a chronic urogenital infection.

When candida is present at low levels, especially if there are no symptoms, the patient is not treated. However, when a parasite is present (for example, an amoeba such as E. histolytica), even a low-level yeast overgrowth is to be taken much more seriously, since it could become a moderate over-growth or worse within a brief period of time.

Candida is sometimes a factor in ADD and autism. Overgrowth of candida and other species of yeast, particularly in children, due to overuse of antibiotics and sugar in their diet, can lead to the formation in the gut of abnormal organic acids, which are neurotoxic and have been associated with both attention deficit disorders and autism.

A correlation exists between the presence of parasites and the presence of candida (and other forms of fungus as well). In addition when there is excessive candida present, the levels of beneficial bacteria tend to be lower. If there are factors present such as parasites that promote the growth of candida, it consumes the resources and the space that would have originally been allotted to the beneficial microflora (the lactobacillus and bifidus). Yeast overgrowth is also documented as a significant factor in some cases of attention deficit disorder and autism (based on the work of Dr. William Shaw and others).

Invasive Candidiasis is a serious condition that affects thousands of patients. Candida is yeast that normally inhabits a healthy colon in small numbers. However, in many cases, the yeast becomes so prolific that it escapes the confines of the intestinal tract and causes havoc throughout the rest of the body creating a condition termed invasive Candidiasis. These microorganisms produce gas and toxins that irritate and can damage tissues, glands, or organs and severely compromise the immune system. Various autoimmune conditions are linked to Candidiasis, where cross reactivity (molecular mimicry) with human tissue and Candida organisms has occurred.
Testing for Candida

D-Arabinitol (DA)

D-Arabinitol (DA) is a metabolite of most pathogenic Candida species including: Candida albicans, Candida tropicalis, Candida parapsilosis, Candida pseudotropicalis, Candida kefyr, Candida lusitaniae and Candida guilliermondii.

Positive DA results have been obtained several days to weeks before positive Candida blood cultures and the normalization of DA levels has been correlated with the therapeutic response in both humans and animals.

By looking at DA, the direct metabolite of pathogenic Candida species listed above, it is possible to assess whether a person has invasive Candidiasis.

The D-Arabinitol test differentiates between Invasive and Non-Invasive Candida.

Results may be interpreted as follows:

**SERUM**
- 1.0 - 5.0 umol/l No evidence of invasive Candida.
- 5.1 – 9.0 umol/l Equivocal – Candida problem but not Invasive
- > 9.0 umol/l Presumptive Evidence of Invasive Candida

**SALIVA**
- 3.0 - 9.0 umol/l No evidence of invasive Candida.
- 9.0 — 15.0 umol/l Equivocal – Candida problem but not Invasive
- > 15.0 umol/l Presumptive Evidence of Invasive Candida

Anti-Candida Antibody Testing

<table>
<thead>
<tr>
<th>Anti-Candida Antibody</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Candida Immune Complex (serum)</td>
<td>&lt;= 1.2</td>
</tr>
<tr>
<td>Candida Antibody, IgA (serum)</td>
<td>&lt;= 22</td>
</tr>
<tr>
<td>Candida Antibody, IgG (serum)</td>
<td>&lt;= 11</td>
</tr>
<tr>
<td>Candida Antibody, IgM (serum)</td>
<td>&lt;= 4</td>
</tr>
</tbody>
</table>

Antibody Testing
Candida immune complexes contain IgG Candida antibodies, Candida antigen and fragments of complement.

Elevations of these complexes correlate with the clinical symptoms related to chronic Candidiasis, and diminish with effective Candida treatment.

Elevated levels of IgA Candida antibody titers are indicative of local mucosal infections, including the vagina, skin, urethra, and GI tract.

Chronic candidiasis can be positively diagnosed by the determination of high serum levels of IgG and IgM antibodies against Candida albicans. High levels of IgG antibody titers can be indicative of past or ongoing Candida infection.

New Technologies in Testing

Conventional bacteriological methods such as microscopy, culture and identification are used for the analysis and/or quantification of the intestinal microbiology.

Although of value and still considered the staple testing of microbial pathogens, there are some limitations of these conventional methods.

To overcome some of the limitations of traditional stool testing, techniques based on 16S ribosomal DNA (rDNA) genes have been developed:

This allows the clinician to develop the most appropriate therapy based on the patient's true gut microbiota, resulting in better clinical results.

Microbial Detection with DNA Probes and PCR

One of the most important contributions to molecular biology is the advent of the polymerase chain reaction (PCR). PCR has led to the development of DNA and RNA-based technologies, enabling the detection of a single genome of an infectious agent in any body fluid with improved accuracy and sensitivity.

Many infectious agents that are missed by routine cultures, serological assays, DNA probes and Southern blot hybridizations can be detected by PCR.

Therefore, PCR-based tests are best suited for the clinical and epidemiological investigation of pathogenic bacteria and viruses.

The introduction of PCR in the late 1980s dominated microbial research because it was superior to all previously used culture techniques and the more recently developed DNA pro and kits.

PCR-based tests are several orders of magnitude more sensitive than those based on direct hybridization with the DNA probe. PCR does not depend on the ability of an organism to grow in culture.
Furthermore, PCR is fast, sensitive and capable of copying a single DNA sequence of a viable or non-viable cell over a billion times within 3 to 5 hours.

The sensitivity of the PCR to is also based on the fact that PCR methodology requires only 1 to 5 cells for detection compared to the 1,000 to 5000 in conventional evaluations.

The following benefits are:

- Ability to detect non-viable organisms that are not retrievable by culture-based methods
- Ability to detect and identify organisms that cannot be cultured or are extremely difficult to grow (e.g., anaerobes)
- More rapid detection and identification of organisms that grow slowly (e.g., mycobacteria and fungi)
- Ability to detect previously unknown organisms directly in clinical specimens by using broad-range primers
- Ability to quantify infectious organism burden in patient specimens for better clinical responsiveness

Laboratories that make the transition to molecular diagnostics will become a more integral part of the clinical arena, as they can prove the value of their improved services.

**Functional Treatment**

Treatment involves destroying and clearing harmful bacteria or parasites. If the problem is due to yeast or bacterial overgrowth, treatment may mean minimizing the overgrowth of a microbe that is normally harmless.

These harmful microbes can take over the ecosystem of the digestive tract and crowd out beneficial bacteria.

The most destructive of the pathogens are generally the parasites.

In other cases, overgrowths can involve potentially aggressive species of bacteria, including clostridium, bacterioides, or other anaerobes that are not directly detectable on lab tests.

**Treatment Steps**

**Lab Testing**

Prescriptive and Natural Agents based on [microbial sensitivity profiling](#) are designed to target specific organisms—whether they are bacteria, parasites, or yeast—so it is important to have the most complete, accurate laboratory evaluation(s) available.
Preparing for Treatment

Someone weakened by chronic illness may need a period of strengthening before beginning pathogen elimination. Preparing for treatment involves enhanced nutrition and possibly an alternative liver evaluation to be sure the liver can handle the medication.

Treatment

Based on microbial sensitivity profiling you may decide on using prescriptive agent or a natural herbal agent to combat a bacterial and parasitic infection. The objective of treatment is to kill off the targeted microorganisms as completely as possible.

In a functional medicine approach, medications and herbs may be prescribed together as two components of a specific program. Additionally, herbs are often used in follow-up to drug therapy although there are physicians who decide on minimizing stress on the hepatic system and use natural agents.

As important as medications are in the treatment of GI infections or overgrowth, they do have their host of problems. Some are simply too harsh for the lining of the GI tract or cause nausea unless taken with food. Others may result in certain side effects.

Die-Off

The die-off reaction typically occurs when you're killing off microorganisms (yeast, fungus, protozoa) at a pace quicker than the eliminative organs (the liver and the GI tract) can competently handle. This can occur when taking drugs or potent botanicals and can be disruptive to the entire body because the eliminative resources must be utilized to minimize the ill effects of the microbes.

The toxic by-products of the microbes can circulate and recirculate through the blood and lymphatics, causing possible symptoms of die-off such as fatigue, achiness, fever, and difficulty with concentration, sometimes resembling a mild case of the flu.

Avoiding Reinfection

It is important to observe the best possible personal hygiene. Effective resolution of these problems often require treating family members or a partner to be sure the microbe in not simply being passed back and forth through food handling, intimate contact, or exposure in the household environment.
Avoiding Overgrowth

In the case of candida and some of the other organisms that tend to over-grow, we have a different situation. It's not that difficult to get rid of candida, but keeping it from overgrowing again can be a problem. It is important to remember that improving GI immune function is the real key when it comes to the overgrowth of organisms like candida.