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## To Physicians and/or Healthcare Providers:

**The ingenuity of new breakthroughs in genomic science harnesses the advent of truly personalized healthcare and offers the greatest business opportunity of the 21 century.**

Genomic profiles assess genetic variations in each person and offers a tool to reveal a person's increase disease risk. This empowers physicians and/or healthcare providers as well as patients to realize:

- **earlier, more effective preventive interventions-years before disease develops**
- **precise, customized therapies that truly address each individual's needs**
- **improved clinical insight into patients with treatment-resistant "chronic" conditions.**

SRherbs.com wants to utilize these scientific breakthroughs by working more closely with the physicians and/or healthcare providers to bring the "*START*" message to the general public.

"*START*" is a lifestyle changing program that has helped thousands of people take charge of their lives by restoring energy and vibrant health. "*START*" is a not for profit entity sponsored by Concepts For Health. Inc. d/b/a SRherbs.com.

The main purpose of "*START*" is to provide information and support to people wanting to improve their lifestyle and dietary habits. The "*START*" program since its inception advocates a similar healthcare model to that of the recently adopted and advocated genomic science program.

We are looking for physicians and/or healthcare providers who will allow us to work with them by offering the "*START*" educational program to their patients or clientele and to the general public in their area as well. Some of the professionals may want to provide these tests for "*START*" participants and monitor their progress.

Should you consider working with the "*START*" program, you can contact us through the person that provided you this package. If not please return the package and we will make this offer to the next physician or health care provider.

Thank you for taking time to look our services over.

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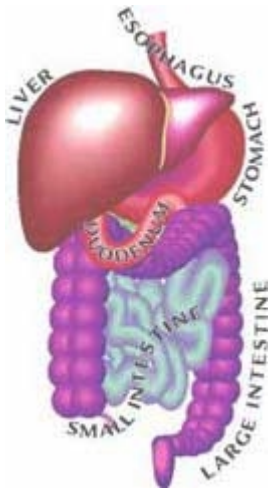
"START"
Self Treatment Arrest Reversal Therapy

# Comprehensive Digestive Stool Analysis

## Sound nutrition and digestive processes are fundamental for long term optimal health.

With changes in the modern diet, however, gastrointestinal disorders have become increasingly prevalent. One recent study reported that, over a period of three months, gastrointestinal symptoms were experienced in over 70 percent of American households. Maldigestion, malabsorption and abnormal gut flora and ecology, as well as many complex chronic illnesses and symptoms, lie at the root of most common GI complaints.




A Digestive Stool Analysis provides clinicians with a critical, non-invasive tool for evaluating the status of the digestive tract. This test helps pinpoint imbalances, provide clues about current symptoms and warn of potential problems should the imbalances progress. With an accurate assessment, custom-tailored treatment can be easily applied, greatly increasing the chances for therapeutic success.

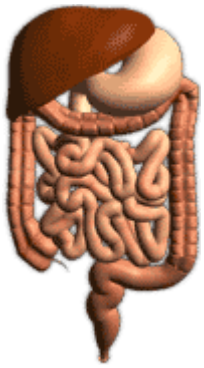
***"START" recommends using the services of a diagnostic laboratory such as Great Smokies Diagnostic Laboratory (GSDL)*** who have recently made some key enhancements to their CDSA which we believe is very beneficial and highly recommended to each START participant.

Their digestive stool analysis has been enhanced by include the addition of the Functional Physiologic Range (FPR) to the reports of all CDSA and CDSA-related tests. FPR provides healthcare providers with a clearer definition of what is "normal" for a test result. Also, the Bacterial Dysbiosis Index has been renamed Dysbiosis Risk Index. This new index will provide you with a more accurate reflection of the intestinal milieu, as well as to encourage patients to pay more attention to areas not previously represented in the index, such as maldigestion, malabsorption, and yeast overgrowth.

The CDSA is used in the evaluation of various gastrointestinal symptoms or systemic illnesses that may have started in the intestine. Because illnesses are often not discernable from symptoms, the CDSA is a valuable means of identifying critical imbalances previously unsuspected.

Includes: Digestive Function Analysis, Microbiology Analysis, Bacteriology Culture, Fecal Fat Analysis, Yeast Culture.

## Nutrition and digestion are undeniably important to good health.



We are, essentially, what we eat and then absorb. Over the long haul, excellent health is impossible without good nutrition. However, without adequate breakdown and assimilation, even the best diet offers little help. Additionally, incomplete or faulty digestive processes may lead to a variety of chronic disorders.

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Gastrointestinal disorders have a major impact on health. One recent study found that during a three-month period nearly 70% of American households experienced one or more gastrointestinal symptom.[1]

Maldigestion, malabsorption and abnormal gut flora and ecology, as well as many complex chronic illnesses and symptoms, lie at the root of most common GI complaints. Thus, nutrition and digestive processes are central to long-term health. Great Smokies' Comprehensive Digestive Stool Analysis (CDSA) provides clinicians with a critical tool for evaluating the status of the GI tract.

This assay helps pinpoint imbalances, provide clues about current symptoms and warns of potential problems should the imbalances progress. With an accurate assessment, custom-tailored treatment can be easily applied, greatly increasing the chances for therapeutic success.

The CDSA is used in the evaluation of various gastrointestinal symptoms or systemic illnesses that may have started in the intestine.

Because illnesses are often not discernable from symptoms, the CDSA is a valuable means of identifying critical imbalances previously unsuspected.

## Role of the Gastrointestinal Tract

As most food molecules can't be absorbed or utilized in their native state, a primary function of the gastrointestinal system is to break down molecules and absorb nutrients. This is a complex process taking place primarily in the gastrointestinal mucosa, where the battle for health—to absorb nutrients and exclude toxins—is fought. The gastrointestinal mucosa does this through a combination of physical barriers to diffusion, mucosal fluids and active immune processes. [2]

### Mouth

Teeth break up food and mix it with saliva. Saliva in turn helps form a bolus and protects the pharyngeal and esophageal mucosa, primarily with secretory IgA antibodies. Saliva also helps remineralize the teeth with calcium salts. The enzymes lingual lipase, salivary amylase and ptyalin initiate fat and starch digestion [3]

**Symptoms of low gastric acidity**

- Bloating, belching, burning and flatulence immediately after meals
- Sense of fullness after eating
- Indigestion, diarrhea or constipation
- Systemic reactions after eating
- Nausea after taking supplements
- Rectal itching
- Weak, peeling or cracked fingernails
- Dilated capillaries in cheeks and nose (in nonalcoholics)
- Postadolescent acne
- Iron deficiency
- Chronic intestinal infections — parasites, yeast, bacteria
- Undigested food in stool

Table 1 (ref. 6,7)

### Stomach

The stomach mechanically churns food, breaks up and emulsifies fats and exposes molecules to additional enzymes. In doing this, it produces one to two liters of gastric juices per day. [4]

### Gastric juice has several components:

- Hydrochloric acid is secreted by the parietal cells. It activates pepsinogens to convert to pepsin and renders some minerals (e.g. calcium and iron) more absorbable. Stomach acid prevents

bacterial overgrowth by creating an essentially sterile environment.
- Mucus forms an acid- and pepsin-resistant coating of the stomach lining.
- Gastric lipase begins the hydrolysis of fats.

## Small Intestine

Most digestion and absorption takes place in the small intestine and is mediated by pancreatic enzymes and bile. [4]

**The process involves several steps:**

**1.** Secretion of pancreatic juices (about 2.5 L/ day) is controlled by the vagus nerve and the duodenal hormones secretin and cholecystokinin. Hormone production, in turn, is stimulated by the presence of fat, protein and acid chyme.
**2.** Bicarbonate begins the process of neutralizing stomach acid.
**3.** The proteases trypsinogen, chymotrypsinogen and procarboxypeptidase are activated to trypsin, chymotrypsin and carboxypeptidase. These enzymes digest proteins to oligopeptides and amino acids.
**4.** Amylase splits starch to maltose.
**5.** Lipase hydrolyzes diglycerides and triglycerides, producing long chain fatty acids.
**6.** Bile secreted by the liver (about 700 ml/ daily) is stored in the gall bladder. Bile salts solubilize and emulsify fats, enabling enzymatic hydrolysis.

The Crypts of Lieberkuhn of the intestinal mucosa also produce immunoglobulins and small amounts of digestive enzymes such as peptidase and disaccharidases.

## Large Intestine

A primary role of the large intestine is absorption of water—about one liter daily. The large intestine also provides an environment for microbial fermentation of soluble fiber, starch and undigested carbohydrates.

Anaerobic colonic fermentation results in production of short chain fatty acids, the main energy source for colonic epithelial cells. It is largely these SCFAs, in combination with amines derived from protein degradation, that provide buffering and create the slightly acidic pH of fecal matter.

## Absorption of Specific Nutrients [5-7]

Carbohydrate Digestion: Salivary amylase initiates starch digestion in the mouth. However, this activity is short-lived as the enzyme is denatured by low gastric pH. In the duodenum, oligosac-charides and starch polymers undergo hydrolysis by pancreatic amylase. Specific disaccharides are hydrolyzed by brush border enzymes (lactase, maltase, sucrase) located on the enterocyte microvilli. Resulting onosaccharides are absorbed by specific sodium-dependent transport carrier mechanisms.

**Diseases linked to low gastric acidity**

- Addison's disease
- Asthma
- Celiac disease
- Chronic autoimmune disorders
- Dermatitis herpetiformis
- Diabetes mellitus
- Eczema
- Food allergies
- Gallbladder disease
- Gastric carcinoma
- Gastritis
- Grave's disease
- Hepatitis
- Lupus erythematosus
- Osteoporosis
- Pernicious anemia
- Psoriasis
- Rosacea
- Thyrotoxicosis
- Urticaria
- Vitiligo Table 2 (ref. 14-24)

## Protein Digestion

Gastric acid and pepsin initiate the digestion of dietary protein. This is followed in the duodenum by hydrolysis into oligopeptides and amino acids by proteolytic pancreatic enzymes. Final protein digestion is accomplished by intestinal brush border peptidases. Dipeptides, tripeptides, free amino acids, and probably other short-chain peptides are then absorbed.

## Fat Digestion

Processing of dietary fat is the most complex of the digestive and absorptive processes. Fat is water insoluble so the GI tract must transform large water-insoluble particles into a soluble, absorbable form.

Digestion begins in the mouth with secretion of lingual lipases. The stomach disperses fat globules into an evenly divided phase, called chyme. Pancreatic enzymes then split triglycerides into fatty acids and monoglycerides, which then combine with bile acids and phospholipids to form micelles. This process transforms water insoluble lipids into a water-soluble form absorbed in the proximal small intestine.

After absorption, fatty acids and other lipids are re-esterified in the intestinal cell to form chylomicrons, which are then secreted into the lymphatic system. Medium-chain triglycerides can be absorbed directly in the jejunum without forming chylomicrons.

## Digestive Abnormalities [8-10]

Maldigestion: Gastric acid secretion is a fundamental step in digestion and assimilation. Many clinical conditions originate with decreased gastric acidity. Acid secretion decreases with age, and low stomach acidity is found in more than half of patients over age 60.[11,12] Researchers speculate that malabsorption of nutrients in the elderly is due to atrophy of various digestive organs because of hypochlorhydria. [13]

Gastric acid has a fundamental role in activating pancreatic proenzymes and converting them from inactive precursors (chymotrypsinogen, trypsinogen, etc.) to their active forms (chymotrypsin, trypsin). Intestinal peristalsis and gastric acid secretion normally prevent excessive growth of bacteria in the small intestine. It has been suggested that bacterial overgrowth might interfere with fat digestion and irritate the intestinal mucosa.

## Pancreatic Exocrine Insufficiency

Inadequate delivery of pancreatic lipases and proteases to the small intestine can lead to inadequate breakdown of fats and protein. The net effect is a failure to obtain nourishment from protein, carbohydrate and fiber foods and an unhealthy environment for the flora of the large colon. It has been argued that even small decreases in pancreatic output can contribute substantially to maldigestion and have far-reaching effects in chronically ill patients.

## Malabsorption

Malabsorption is characterized by abnormal fecal excretion of fat (steatorrhea) and variable malabsorption of fats, fat-soluble vitamins, other vitamins, proteins, carbohydrates, minerals and water. Common causes include:

- Defective protein, fat or carbohydrate breakdown
- Inadequate solubility of fatty acids (inadequate bile salts)
- Rapid transit (e.g. diarrhea), which doesn't

allow sufficient time for absorption
• Mucosal cell abnormality and inadequate surface area
• Intestinal infection

A number of important clinical diseases are strongly associated with and may cause mucosal malabsorption. They include sprue, Whipple's disease, Crohn's disease, Giardiasis, Cryptosporidiosis, lactose intolerance and eosinophilic gastroenteritis.

Clinical Considerations of Malabsorption: The signs and symptoms of malabsorption are varied. Interestingly, malabsorption increases with age. [25] Amino acids, carbohydrates, fats, vitamins and trace elements may be absorbed by different processes so an individual may suffer malabsorption for one nutrient but not for others. In fat malabsorption, essential fatty acid deficiency may result in addition to the loss of the highest dietary source of calories.

## Helicobacter Pylori Antibody Assay

More than 12 million Americans suffer from conditions such as duodenal ulcers, peptic ulcers, and chronic gastritis–conditions directly related to the presence of Helicobacter pylori. [1] What's more, H. pylori has been linked to increased risk of gastric cancer (as much as six-fold) and chronic abdominal pain in children. [2,3] Great Smokies' Helicobacter pylori test utilizes the most efficient and economical detection methods available to identify this hidden cause of gastritis and ulcers, allowing physicians to effectively eradicate this potentially damaging gastrointestinal bacteria. The test also serves as a powerful tool for monitoring the efficacy of antimicrobial treatment over time, thereby safeguarding against a possible relapse of symptoms.

## Helicobacter pylori

H. pylori is the only known bacteria to live in the very acidic environment of the stomach. H. pylori cells adhere to the stomach's mucus-secreting epithelial cells. A gram negative curved rod, it possesses multiple unipolar sheathed flagella with terminal bulbs and a smooth cell wall.

H. pylori produces three enzymes in large amounts: urease, superoxide dismutase and catalase. These help it adapt and survive in the stomach environment.

Urease acts upon the urea, generating ammonia and bicarbonate that increase local pH, allowing the bacteria to live in a layer of bicarbonate-rich cells. H. pylori adheres to and disrupts epithelial cells, depletes microvilli, and decreases mucus production.

## Gastritis and Ulcers

Gastritis and duodenal/peptic ulcers are major conditions affecting more than 12 million people in the United States. Gastritis includes a group of inflammatory states of the stomach, differentiated according to the area affected, type of inflammatory cells, and extent of damage.

Peptic ulcers are thought to be related to the resistance of the mucus lining covering the gastric epithelium. Type A chronic gastritis, associated with pernicious anemia, is characterized by diffusely atrophic mucosa of the gastric body (but with a normal antrum); diminished secretion of acid, pepsinogen, intrinsic factor and elevated gastrin levels.

In type-B gastritis, the antrum (the area just before the pyloric valve) is always affected, while the fundus may also be involved, and is frequently characterized by infiltration of

neutrophils into gastric mucosa. The antral G-cell mass is reduced and gastrin levels are normal. This is the type of gastritis associated with H. pylori infection.

## Theory of Excess Gastric Acidity

Until ten years ago, physicians agreed that excess gastric acidity caused ulcers and gastritis. According to this hypothesis, excess acid secretion damages the stomach mucosa, causing inflammation and if unchecked, eventual perforation. This process triggers the familiar symptoms of chronic pain and discomfort.

However, while the process of how acid is formed in the stomach is well understood, there is no fully-accepted explanation as to how and why excess gastric secretion develops. Speculation includes genetic predisposition, diet, smoking, heavy use of non-steroidal anti-inflammatory drugs, physical trauma, and stress.

Conventional treatment to reduce acid flow has utilized antacids and H-2 receptor antagonist drugs. These treatments to alter acid output, however, have high relapse rates and apparently do not cure the disease they appear to treat just the symptom.

## Research Turns to H. pylori

Within the last decade, researchers began to consider a new hypothesis: that gastritis and ulcers are caused by infection by H. pylori Led by Barry Marshall, M.D., numerous clinical trials investigating the link have revealed a strong association between H. pylori and gastritis or duodenal/peptic ulcers. Proof that H. pylori was the major cause of gastritis has clarified many previously unexplained gastritis associations. [4]

In some studies, more than 90% of duodenal ulcer patients and 70% of gastric ulcer patients were colonized with H. pylori.[5] A number of treatment studies have also confirmed this link, by showing the resolution of inflammation and or/ulcers when H. pylori infection was treated and eliminated. [6,7]

Other studies have shown that when healthy volunteers ingest live cultures of H. pylori, many develop a mild case of gastroenteritis, discomfort, bloating and nausea. Some begin vomiting mucus, with halitosis becoming noticeable. **As bacteria works its way underneath the mucosal layer of the stomach lining, it appears to digest the mucosal layer, causing it to become thin and runny.** In some cases, the entire protective lining becomes eroded, allowing gastric acid and pepsin to come into direct contact with the unprotected epithelial cells.

## Detection of Helicobacter pylori using ELISA

There are a number of ways to detect the presence of H. pylori–endoscopy followed by CLO test, $C^{13}$ and $C^{14}$ breath test, silver stain, etc. Measuring the presence of antibodies in serum specific to H. pylori is the most efficient and economical method of detection. A high immunoglobulin titer indicates host immune response against H. pylori.

Culture with specific immunoglobulin titer response shows a strong correlation with biopsy, as well as a high correlation with clinical findings. These antigens have been ultra-purified and are used in a micro-ELISA procedure to measure specific immunoglobulins to H. pylori.

Host immune response may precede clinical symptoms, and may be used as an early indication of H. pylori infection. The presence

of high immunoglobulin titers to H. pylori may also alert the clinician as to the possibility of asymptomatic type B gastritis.

By gauging changes in specific IgG titer levels**, ELISA testing can also be used to monitor the effectiveness of therapy**: an increase may show recurrence of gastritis and ulcers; a steady decrease may show successful intervention. Monitoring treatment effectiveness with a serum test has been recommended as a simple tool for follow-up of H. pylori infection in elderly patients.[8]

## Methods of Treatment

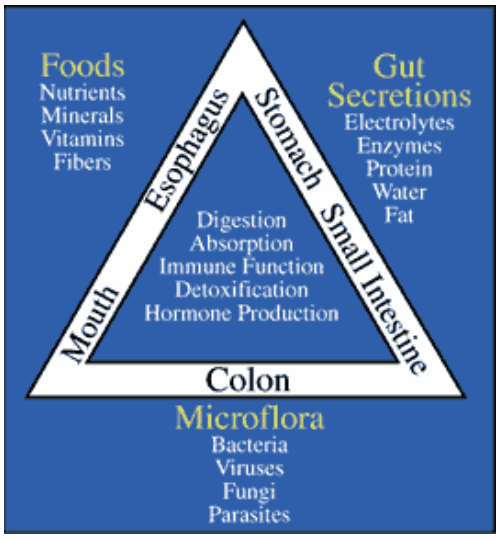
## "START" has an Intestinal Cleansing program using whole foods that has proven to be very effective in over 30 countries through out the world.

## Microbiology: Pathogens Bacteria:

Because the oxygen content of the colon is low, the vast majority of bacteria are anaerobes. There are, however, hundreds of varieties of anaerobic flora in vastly different concentrations, all growing very slowly. The significance of most of these flora remains largely unknown. Most researchers, therefore, utilize the aerobic flora as an indication of bacterial health.

Three frequently identified organisms, Lactobacilli, Bifidobacteria, and Escherichia coli, are employed as indicators of eubiosis or healthy overall flora. Lactobacilli and Bifidobacteria are well established as offering intrinsic benefit and aiding digestion while helping to prevent overgrowth of abnormal flora.

Bacterial cultures also identify and show potential pathogens. We utilize the term "potential pathogens" because individuals may harbor traditional pathogens and appear healthy, while others harbor weak or questionable pathogens and have gastrointestinal complaints.




While they are sometimes found linked to GI tract disturbances, some intestinal bacteria may also be involvedin the etiology of various chronic or systemic problems seemingly unrelated to GI function. These include Klebsiella, Proteus, Pseudomonas, and Citrobacter. These organisms may be involved, through molecular mimicry, in various autoimmune diseases. This has been reported in diabetes mellitus, meningitis, thyroid disease, ulcerative colitis, arthritis, ankylosing spondylitis and systemic lupus.[26,27]

Some potential pathogens may cause clinical and subclinical malabsorption of nutrients and increase bowel permeability to large macromolecules. A number of clinicians speculate that this is directly related to the etiology of food and chemical sensitivity and intolerance.

Whipple's disease, although rare, presents an interesting model of the interaction of bacterial infection, absorptive processes and

systemic health. This disease is known to be caused by an unusual bacteria which resists attempts to culture it in vitro. Symptoms include severe alterations in intestinal permeability and chronic fatigue. [28] There is strong scientific support for the profound relation between GI tract flora, malabsorption, permeability changes and overall health.

## Yeast

In the last few years, colonic yeast infections have attracted attention and controversy as a possible cause of chronic complex illness. [29] Many investigators suggest that an intestinal overgrowth of Candida albicans (and other intestinal yeast) may be involved in food allergy, migraine, irritable bowel, asthma, indigestion and gas, depression related to PMS, vaginitis and chronic fatigue. [30-35]

Although others have dismissed these claims as speculation, we suggest that part of the problem is focusing on the terms “pathogen” and “commensal.” It may be more accurate to use the terms “strong pathogen” and “weak pathogen.” A significant and surprising amount of peer-reviewed literature supports yeast as a weak pathogen. [36-38]

While the normal GI tract harbors small amounts of yeast, overgrowth as a consequence of the wide use of antibiotics, corticosteroids, birth control pills and increased dietary carbohydrates may be abnormal. [39] Odds’ text on Candida summarized more than 20 papers that found patients had a frequency of C. albicans in their feces more than twice as often as normal controls. [40] One study reported that chronic diarrhea and abdominal cramps may be caused by large numbers of dead or damaged yeast, as found in feces. [41] Other research indicates Candida as a cause of colitis in patients with AIDS, neoplastic disease and renal transplants. [42-44]

While the yeast pathogenicity debate continues, high-quality lab work is essential. Yeast may be observed directly via a microscope or indirectly through a culture. Both are necessary for proper analysis.

GI Tract and Arthritis: Researchers increasingly acknowledge that there is a link between digestive processes and arthritis. In patients with altered bowel anatomy, chronic bacterial overgrowth can lead to the formation of circulating immune complexes and synovitis. [45] Changes in bowel permeability due to local gut inflammation may expose the host immune system to microbial or food antigens and even bacterial translocation. [46,47] In some cases, toxins derived from enteric organisms (e.g., Clostridium difficile) may play a direct role in the induction of arthritis.

## Microbiology: Dysbiosis

Dysbiosis is the state of disordered microbial ecology that causes disease. It may exist in the oral cavity, gastrointestinal tract or vaginal cavity. In dysbiosis, organisms of low intrinsic virulence, including bacteria, yeasts and protozoa, induce disease by altering the nutrition or immune responses of their host.[48]

The concept of intestinal flora having a major impact on human health has increasingly gained support, particularly as the widespread use of antibiotics has been observed to disrupt the normal flora.

Published research has implicated intestinal dysbiosis as contributing to vitamin B12 deficiency, steatorrhea, irritable bowel syndrome, inflammatory bowel disease, autoimmune arthropathies, colon and breast cancer, psoriasis, eczema, cystic acne and chronic fatigue. [49-54]

## Normal Intestinal Microflora

The microflora of the GI tract constitute a complex ecosystem of aerobic and anaerobic microorganisms. [49] There are more bacteria in the gut than human cells in the body, and the flora possess more metabolic activity than the host itself.

Flora content is surprisingly stable over time but is affected by diet, antibiotic use and health status. [55] In many ways, the gut flora can be viewed as an organ of the body, as these microbes profoundly influence physiologic processes of the host.

Certain normal metabolic functions and enzyme activities can be attributed to the microflora, and these play a role in metabolizing nutrients, vitamins, drugs, endogenous hormones and carcinogens; synthesizing short chain fatty acids; preventing colonization of pathogens; and stimulating maturation of the normal immune response. [56,57]

## Food allergy

Food allergy is a well documented problem, although its prevalence, testing methods and treatment modalities are controversial. J.O. Hunter proposed that food allergy is not an immunological disease but a disorder of bacterial fermentation in the colon. He theorized in The Lancet that the combined mechanisms of reduced gut enzyme concentrations, imbalanced bacterial flora and increased permeability account for many cases of food intolerance. [60]

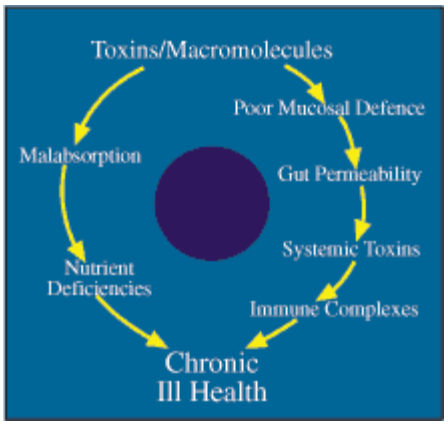
## Four Patterns of Dysbiosis

Leo Galland, M.D., has advanced the idea of four interlocking patterns of bacterial dysbiosis:

## 1. Putrefaction

**This is the Western degenerative disease pattern which results from diets high in fat and meat and low in fiber.** This type of diet produces increased concentrations of Bacteroides sp. and induces bacterial urease and beta-glucuronidase activity. These enzymes may then metabolize bile acids to tumor promoters and deconjugate excreted estrogens, raising the plasma estrogen level. The fecal pH may increase as a result of increased ammonia production.

Epidemiologic data implicates this type of dysbiosis in the pathogenesis of colon cancer and breast cancer. It is usually corrected by decreasing dietary fat and flesh, increasing fiber consumption and consuming probiotic preparations.




## 2. Fermentation Excess

This is a condition of carbohydrate intolerance induced by an excess of normal bacterial fermentation usually resulting from small bowel bacterial overgrowth. Abdominal distention, flatulence, diarrhea, constipation and feelings of malaise are commonly described. In small bowel bacterial overgrowth, degradation of intestinal brush border and pancreatic enzymes by bacterial proteases may cause maldigestion. Fecal short chain fatty acids may be elevated. Patients with fermentation excess are usually

intolerant of soluble fiber supplements and often benefit from antimicrobials and a reduction of carbohydrate consumption.

## 3. Deficiency

Exposure to antibiotics or a diet depleted of soluble fiber may create a deficiency of normal fecal flora, including Bifidobacteria, Lactobacillus and E. coli. Direct evidence of this condition is seen in stool cultures when concentrations of any of these organisms are reduced. This condition has been described in patients with irritable bowel syndrome and food intolerance. Deficiency and putrefaction dysbiosis often occur together and respond to the same treatment. Probiotic supplementation as well as fructooligosaccharides are often helpful in reestablishing a normal flora.

## 4. Sensitization

Abnormal immune responses to components of the normal indigenous intestinal microflora may contribute to the pathogenesis of inflammatory bowel disease, spondyloarthropathies and other connective tissue diseases or skin disorders such as psoriasis or acne. Endotoxins may activate the alternative complement pathway, and sensitization may complement fermentation excess. Similar treatments may benefit both conditions.

**“START” strongly encourages each of the participants to get a digestive stool analysis from their health provider and then 90 days after being on the program to once again take the test to see their progress.**



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