LAB #: F00000-0000-0
PATIENT: Sample Patient
ID: P0000000000
SEX: Male
DOB: AGE: 78

CLIENT #: 12345
DOCTOR:
Doctor's Data, Inc.
3755 Illinois Ave.
St. Charles, IL 60174 U.S.A.

Comprehensive Stool Analysis

BACTERIOLOGY CULTURE

Expected/Beneficial flora	Commensal (Imbalanced) flora	Dysbiotic flora
3+ Bacteroides fragilis group	1+ Beta strep, group B	
3+ Bifidobacterium spp.		
4+ Escherichia coli		
4+ Lactobacillus spp.		
2+ Enterococcus spp.		
2+ Clostridium spp.		
NG = No Growth		

BACTERIA INFORMATION

Expected /Beneficial bacteria make up a significant portion of the total microflora in a healthy & balanced GI tract. These beneficial bacteria have many health-protecting effects in the GI tract including manufacturing vitamins, fermenting fibers, digesting proteins and carbohydrates, and propagating anti-tumor and anti-inflammatory factors.
Clostridia are prevalent flora in a healthy intestine. Clostridium spp. should be considered in the context of balance with other expected/beneficial flora. Absence of clostridia or over abundance relative to other expected/beneficial flora indicates bacterial imbalance. If *C. difficile* associated disease is suspected, a Comprehensive Clostridium culture or toxigenic *C. difficile* DNA test is recommended.
Commensal (Imbalanced) bacteria are usually neither pathogenic nor beneficial to the host GI tract. Imbalances can occur when there are insufficient levels of beneficial bacteria and increased levels of commensal bacteria. Certain commensal bacteria are reported as dysbiotic at higher levels.
Dysbiotic bacteria consist of known pathogenic bacteria and those that have the potential to cause disease in the GI tract. They can be present due to a number of factors including: consumption of contaminated water or food, exposure to chemicals that are toxic to beneficial bacteria; the use of antibiotics, oral contraceptives or other medications; poor fiber intake and high stress levels.

YEAST CULTURE

Normal flora	Dysbiotic flora
	2+ Candida glabrata

MICROSCOPIC YEAST

Result:	Expected:
Rare	None - Rare

The microscopic finding of yeast in the stool is helpful in identifying whether there is proliferation of yeast. Rare yeast may be normal; however, yeast observed in higher amounts (few, moderate, or many) is abnormal.

YEAST INFORMATION

Yeast normally can be found in small quantities in the skin, mouth, intestine and mucocutaneous junctions. Overgrowth of yeast can infect virtually every organ system, leading to an extensive array of clinical manifestations. Fungal diarrhea is associated with broad-spectrum antibiotics or alterations of the patient's immune status. Symptoms may include abdominal pain, cramping and irritation. When investigating the presence of yeast, disparity may exist between culturing and microscopic examination. Yeast are not uniformly dispersed throughout the stool, this may lead to undetectable or low levels of yeast identified by microscopy, despite a cultured amount of yeast. Conversely, microscopic examination may reveal a significant amount of yeast present, but no yeast cultured. Yeast does not always survive transit through the intestines rendering it unvialble.

Comments:

Date Collected: **01/28/2015**
Date Received: **01/30/2015**
Date Completed: **02/06/2015**

**** Aeromonas, Campylobacter, Plesiomonas, Salmonella, Shigella, Vibrio, Yersinia, & Edwardsiella tarda* have been specifically tested for and found absent unless reported.**

MALDI-TOF

LAB #: F000000-0000-0
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DOB: AGE: 78

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Comprehensive Stool Analysis

DIGESTION /ABSORPTION

	Within	Outside	Reference Range
Elastase	496		> 200 µg/mL
Fat Stain		Many	None - Mod
Muscle fibers	None		None - Rare
Vegetable fibers	Rare		None - Few
Carbohydrates	Neg		Neg

Elastase findings can be used for the diagnosis or the exclusion of exocrine pancreatic insufficiency. Correlations between low levels and chronic pancreatitis and cancer have been reported. **Fat Stain:** Microscopic determination of fecal fat using Sudan IV staining is a qualitative procedure utilized to assess fat absorption and to detect steatorrhea. **Muscle fibers** in the stool are an indicator of incomplete digestion. Bloating, flatulence, feelings of "fullness" may be associated with increase in muscle fibers. **Vegetable fibers** in the stool may be indicative of inadequate chewing, or eating "on the run". **Carbohydrates:** The presence of reducing substances in stool specimens can indicate carbohydrate malabsorption.

INFLAMMATION

	Within	Outside	Reference Range
Lactoferrin		23.6	< 7.3 µg/mL
Calprotectin*		92	10 - 50 µg/g
Lysozyme*		1400	<= 600 ng/mL
White Blood Cells	None		None - Rare
Mucus	Neg		Neg

Lactoferrin and **Calprotectin** are reliable markers for differentiating organic inflammation (IBD) from function symptoms (IBS) and for management of IBD. Monitoring levels of fecal lactoferrin and calprotectin can play an essential role in determining the effectiveness of therapy, are good predictors of IBD remission, and can indicate a low risk of relapse. **Lysozyme*** is an enzyme secreted at the site of inflammation in the GI tract and elevated levels have been identified in IBD patients. **White Blood Cells** (WBC) and **Mucus** in the stool can occur with bacterial and parasitic infections, with mucosal irritation, and inflammatory bowel diseases such as Crohn's disease or ulcerative colitis.

IMMUNOLOGY

	Within	Outside	Reference Range
Secretory IgA*		1410	51 - 204 mg/dL

Secretory IgA* (sIgA) is secreted by mucosal tissue and represents the first line of defense of the GI mucosa and is central to the normal function of the GI tract as an immune barrier. Elevated levels of sIgA have been associated with an upregulated immune response.

Comments:

Date Collected: 01/28/2015
Date Received: 01/30/2015
Date Completed: 02/06/2015

*For Research Use Only. Not for use in diagnostic procedures.
Methodology: Elisa, Microscopy, Colormetric, Gas Chromotography, ph Electrode

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Comprehensive Stool Analysis

SHORT CHAIN FATTY ACIDS

	Within	Outside	Reference Range
% Acetate	58		40 - 75 %
% Propionate	18		9 - 29 %
% Butyrate	17		9 - 37 %
% Valerate	7.0		0.5 - 7 %
Butyrate	1.2		0.8 - 4.8 mg/mL
Total SCFA's	6.7		4 - 18 mg/mL

Short chain fatty acids (SCFAs): SCFAs are the end product of the bacterial fermentation process of dietary fiber by beneficial flora in the gut and play an important role in the health of the GI as well as protecting against intestinal dysbiosis. Lactobacilli and bifidobacteria produce large amounts of short chain fatty acids, which decrease the pH of the intestines and therefore make the environment unsuitable for pathogens, including bacteria and yeast. Studies have shown that SCFAs have numerous implications in maintaining gut physiology. SCFAs decrease inflammation, stimulate healing, and contribute to normal cell metabolism and differentiation. Levels of **Butyrate** and **Total SCFA** in mg/mL are important for assessing overall SCFA production, and are reflective of beneficial flora levels and/or adequate fiber intake.

INTESTINAL HEALTH MARKERS

	Within	Outside	Reference Range
Red Blood Cells	None		None - Rare
pH	6.3		6 - 7.8
Occult Blood	Neg		Neg

Red Blood Cells (RBC) in the stool may be associated with a parasitic or bacterial infection, or an inflammatory bowel condition such as ulcerative colitis. Colorectal cancer, anal fistulas, and hemorrhoids should also be ruled out.
pH: Fecal pH is largely dependent on the fermentation of fiber by the beneficial flora of the gut.
Occult blood: A positive occult blood indicates the presence of free hemoglobin found in the stool, which is released when red blood cells are lysed.

MACROSCOPIC APPEARANCE

	Appearance	Expected
Color	Brown	Brown
Consistency	Loose/Watery	Formed/Soft

Color: Stool is normally brown because of pigments formed by bacteria acting on bile introduced into the digestive system from the liver. While certain conditions can cause changes in stool color, many changes are harmless and are caused by pigments in foods or dietary supplements. **Consistency:** Stool normally contains about 75% water and ideally should be formed and soft. Stool consistency can vary based upon transit time and water absorption.

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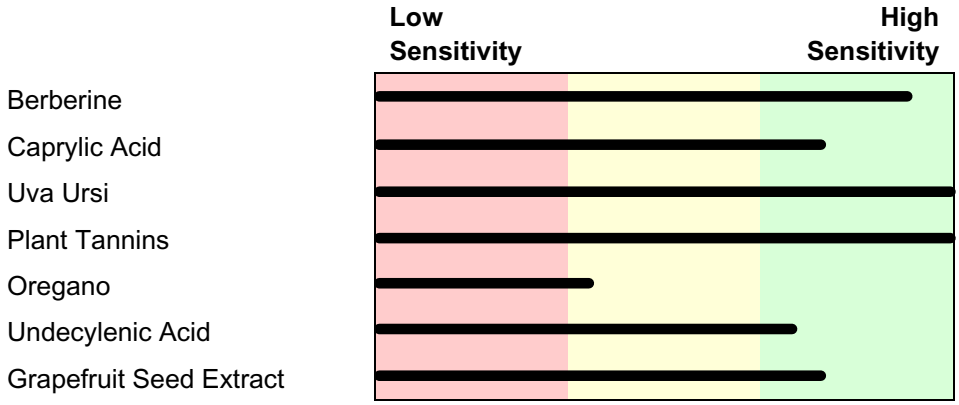
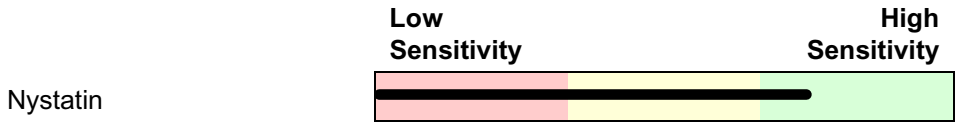
Yeast Susceptibilities: Candida glabrata

NATURAL ANTIFUNGALS



Natural antifungal agents may be useful for treatment of patients when organisms display in-vitro sensitivity to these agents. The test is performed by using standardized techniques and filter paper disks impregnated with the listed agent. Relative sensitivity is reported for each natural agent based upon the diameter of the zone of inhibition surrounding the disk. Data based on over 5000 individual observations were used to relate the zone size to the activity level of the agent. A scale of relative sensitivity is defined for the natural agents tested.

NON-ABSORBED ANTIFUNGALS



Non-absorbed antifungals may be useful for treatment of patients when organisms display in-vitro sensitivity to these agents. The test is performed using standardized commercially prepared disks impregnated with Nystatin. Relative sensitivity is reported based upon the diameter of the zone of inhibition surrounding the disk.

AZOLE ANTIFUNGALS

	Resistant	S-DD	Susceptible
Fluconazole		S-DD	
Itraconazole			S
Ketoconazole			S

Susceptible results imply that an infection due to the fungus may be appropriately treated when the recommended dosage of the tested antifungal agent is used.
Susceptible - Dose Dependent (S-DD) results imply that an infection due to the fungus may be treated when the highest recommended dosage of the tested antifungal agent is used.
Resistant results imply that the fungus will not be inhibited by normal dosage levels of the tested antifungal agent.

Standardized test interpretive categories established for Candida spp. are used for all yeast isolates.

Comments:
Date Collected: **01/28/2015**
Date Received: **01/30/2015**
Date Completed: **02/06/2015**

Yeast antifungal susceptibility testing is intended for research use only.
Not for use in diagnostic procedures.

v10.11

INTRODUCTION

This analysis of the stool specimen provides fundamental information about the overall gastrointestinal health of the patient. When abnormal microflora or significant aberrations in intestinal health markers are detected, specific interpretive paragraphs are presented. If no significant abnormalities are found, interpretive paragraphs are not presented.

Clostridium spp

Clostridia are expected inhabitants of the human intestine. Although most clostridia in the intestine are not virulent, certain species have been associated with disease. Clostridium perfringens is a major cause of food poisoning and is also one cause of antibiotic-associated diarrhea. Clostridium difficile is a causative agent in antibiotic-associated diarrhea and pseudomembranous colitis. Other species reported to be prevalent in high amounts in patients with Autistic Spectrum Disorder include Clostridium histolyticum group, Clostridium cluster I, Clostridium bolteae, and Clostridium tetani.

If these disease associations are a concern further testing may be necessary.

Washington W, Allen S, Janda W, Koneman E, Procop G, Schreckenberger P, Woods, G. Koneman's Color Atlas and Textbook of Diagnostic Microbiology, 6th edition. Lippincott Williams and Wilkins; 2006. pg 931-939

Song Y, Liu C, Finegold SM. Real-Time PCR Quantitation of Clostridia in Feces of Autistic Children. Applied and Environmental Microbiology. Nov. 2004, 6459-6465.

Parracho H, Bingham MO, Gibson GR, McCartney AL. Differences Between the Gut Microflora of Children with Autistic Spectrum Disorders and That of Healthy Children. Journal of Medical Microbiology. 2005;54, 987-991.

Imbalanced flora

Imbalanced flora are those bacteria that reside in the host gastrointestinal tract and neither injure nor benefit the host. Certain dysbiotic bacteria may appear under the imbalances category if found at low levels because they are not likely pathogenic at the levels detected. When imbalanced flora appear, it is not uncommon to find inadequate levels of one or more of the beneficial bacteria and/or a fecal pH which is more towards the alkaline end of the reference range (6 - 7.8). It is also not uncommon to find hemolytic or mucoid E. coli with a concomitant deficiency of beneficial E. coli and alkaline pH, secondary to a mutation of beneficial E. coli in alkaline conditions (DDI observations). Treatment with antimicrobial agents is unnecessary unless bacteria appear under the dysbiotic category.

Mackowiak PA. The normal microbial flora. N Engl J Med. 1982;307(2):83-93.

Dysbiotic Yeast

Yeast was cultured from this stool specimen and the amount is considered to be dysbiotic. A positive yeast culture and sensitivity to prescriptive and natural agents is helpful in determining which anti-fungal agents to use as part of a therapeutic plan for chronic yeast syndrome. When investigating the presence of yeast, disparity may exist between culturing and microscopic examination. Yeast grows in colonies and is typically not uniformly dispersed throughout the stool. This may lead to undetectable or low levels of yeast identified by microscopy, despite a significant amount of yeast cultured.

Microscopic yeast

Microscopic examination has revealed yeast in this stool sample. The microscopic finding of yeast in the stool is helpful in identifying whether the proliferation of fungi, such as Candida albicans, is present. Yeast is normally found in very small amounts in a healthy intestinal tract. While small quantities of yeast (reported as none or rare) may be normal, yeast observed in higher amounts (few, moderate to many) is considered abnormal.

An overgrowth of intestinal yeast is prohibited by beneficial flora, intestinal immune defense (secretory IgA), and intestinal pH. Beneficial bacteria, such as Lactobacillus colonize in the intestines and create an environment unsuitable for yeast by producing acids, such as lactic acid, which lowers intestinal pH. Also, lactobacillus is capable of releasing antagonistic substances such as hydrogen peroxide, lactocidin, lactobacillin, and acidolin.

Many factors can lead to an overgrowth of yeast including frequent use of antibiotics (leading to insufficient beneficial bacteria), synthetic corticosteroids, oral contraceptives, and diets high in sugar. Although there is a wide range of symptoms which can result from intestinal yeast overgrowth, some of the most common include brain fog, fatigue, reccurring vaginal or bladder infections, sensitivity to smells (perfumes, chemicals, environment), mood swings/depression, sugar and carbohydrate cravings, gas/bloating, and constipation or loose stools.

A positive yeast culture (mycology) and sensitivity to prescriptive and natural agents is helpful in determining which anti-fungal agents to use as part of a therapeutic treatment plan for chronic colonic yeast. However, yeast are colonizers and do not appear to be dispersed uniformly throughout the stool. Yeast may therefore be observed microscopically, but not grow out on culture even when collected from the same bowel movement.

Lysozyme

The level of lysozyme, a biomarker of inflammation, is elevated in this specimen. Lysozyme is an enzyme that catalyzes the hydrolysis of specific glycosidic bonds in mucopolysaccharides that constitute the cell wall of gram-positive bacteria. Lysozyme is an antibacterial defense present in the

G.I. tract and is secreted by granulocytes, macrophages, Paneth cells, and Brunner's Glands as well as normal colonic crypt cells [1]. The main source for fecal lysozyme is the intestinal granulocytes.

Moderate elevations in fecal lysozyme are commonly associated with significant overgrowth of enteropathogens such as yeast or dysbiotic bacteria. Markedly elevated levels of fecal lysozyme have been identified in colonic inflammatory bowel disease (IBD), such as Crohn's disease and ulcerative colitis as well as other non-IBD G.I. diseases with diarrhea, compared to healthy controls [2,3]. In Crohn's disease, excess lysozyme may be a result of active secretions of macrophages in the lamina propria, and monocytic cells in the granulomas (sites of G.I. inflammation) [4]. In ulcerative colitis, it has been postulated that elevations in fecal lysozyme may be secondary to intestinal loss of granulocytes and their secretory granules [5]. Additionally, Paneth cell metaplasia, a phenomenon that occurs with various inflammatory conditions of the large intestine, may be a minor contributor to fecal lysozyme elevations [5]. Paneth cells are part of the intestinal epithelial lining found in the deepest part of intestinal cryptwhich are the crypts of Lieberkühn. Paneth cells contain lysozyme in their secretory granules, and combined with their phagocytic capability, help to regulate intestinal microbial flora [5].

Lysozyme is helpful in the determination of colonic inflammatory activity rather than small bowel disease [2]. Slightly elevated levels of lysozyme may be treated with anti-inflammatory agents or by removing the antagonist, such as enteroinvasive microorganisms or allergens. Moderate to high levels of lysozyme (>2,000) may indicate an active inflammatory bowel condition which often requires further testing such as colonoscopy. To rule out IBD, check fecal lactoferrin levels (elevated with IBD).

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2. Van der Sluys Veer A, Brouwer J, Biemond I, et al. Fecal lysozyme in assessment of disease activity in inflammatory bowel disease. Dig Dis & Sci. 1998;43(3):590-5.
3. Klass HJ, Neale G. Serum and faecal lysozyme in inflammatory bowel disease. Gut 1978;19:233-9.
4. Geboes K, Van den Oord JJ, Rutgeerts P, et al. Immunohistochemical identification of lysozyme in pseudopyloric gland metaplasia in Crohn's disease. Hepatogastroenterology 1986;90:1121-8.
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Fecal Lactoferrin

The level of fecal lactoferrin, a biomarker of serious gastrointestinal inflammation, is abnormally high in this fecal sample. Fecal lactoferrin is elevated in association with Inflammatory Bowel Disease (IBD) such as Ulcerative Colitis (UC) or Crohn's Disease (CD)[1,2], but NOT Irritable Bowel Syndrome (IBS)[1,3]. Therefore, assessment of fecal lactoferrin levels enables distinction between IBD and non-inflammatory IBS. Such distinction

is critical because, although both IBD and IBS may share some common symptoms such as diarrhea, abdominal cramping and weight loss, the diseases are treated quite differently. IBD may become life threatening, requires life long treatment and possibly surgery. In contrast, IBS is often effectively treated with dietary restrictions, stress reduction and medication.

Gastrointestinal inflammation associated with IBD is associated with increased infiltration of activated neutrophils into the mucosa and increased release of lactoferrin into the gut[1,4,5]. Patients with inflammation of the GI tract, such as IBD (but not IBS), exhibit elevated lactoferrin concentrations in the feces[1].

Clinical studies have shown that fecal lactoferrin levels of healthy persons are similar to IBS patients, but markedly increased in patients with active IBD[1,3]. Patients with IBD oscillate between active and inactive disease states, and fecal lactoferrin levels increase 2-3 weeks prior to onset of clinical symptoms[6]. During remission and effective treatment, fecal lactoferrin decreases significantly. Therefore disease activity, and efficacy of treatment can be monitored by following fecal lactoferrin levels. The test can be ordered separately to track disease activity in patients with IBD.

Moderately elevated levels of fecal lactoferrin can occur, with fecal red blood cells and leukcytes, in association with invasive enterpathogens [7,8]. Such levels would be expected to be much lower than those associated with the active phase of IBD. Therefore, with moderately elevated levels of fecal lactoferrin, one should check for the presence of enteropathogens (eg. Shigella, Campylobacter, Clostridium difficile).

Guidelines for interpreting the results of this test are provided by the results of a large multi-center clinical study which evaluated fecal lactoferrin levels in 180 patients suffering with IBS and IBD (UC and CD) compared to 56 healthy controls.

GROUP	# of SPECIMENS	FECAL LACTOFERRIN mean mcg/ml +/- SE
Inactive UC	41	67 +/- 24
Active UC	31	815 +/- 789
Inactive CD	26	239 +/- 83
Active CD	51	672 +/- 242
IBS	31	1.3 +/- 0.3
Healthy Controls	55	1.6 +/- 0.4

1. Kane, S.V., Sandborn, W.J., Rufo, P.A., et al. Am. J. Gastroenterol. (2003)98(6):1309-14.
2. Sugi, K., Saitoh, O., Hirata, I., et al. Am. J. Gastroenterol.

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8. Guerrant, R.L., Araujo, V., Soares, E. et al. J. Clin. Microbiol. (1992)30(5):1238-42.

Secretory IgA (sIgA)

The concentration of sIgA is abnormally high in this fecal specimen. Immunological activity in the gastrointestinal tract can be assessed using secretory immunoglobulin A (sIgA). Secretory IgA is the predominant antibody or immune protein the body manufactures and releases in external secretions such as saliva, tears, and milk [1]. It is also transported through the epithelial cells that line the intestines out into the lumen. Secretory IgA represents the first line of defense of the GI mucosa and is central to the normal function of the GI tract as an immune barrier [1]. As the principal immunoglobulin isotype present in mucosal secretions, sIgA plays an important role in controlling intestinal milieu which is constantly presented with potentially harmful antigens such as pathogenic bacteria, parasites, yeast, viruses, abnormal cell antigens, and allergenic proteins [1]. Secretory IgA antibodies exert their function by binding to antigenic epitopes on the invading microorganism limiting their mobility and adhesion to the epithelium of the mucus membrane [2]. This prevents the antigens from reaching systemic circulation allowing them to be excreted directly in the feces.

Elevated fecal sIgA is an appropriate response to an antigenic presence. Microbial and microscopic studies of the stool are useful in identifying if bacteria, yeast, or parasites are present. Eradication of the pathogenic microorganisms will bring sIgA back down into the normal range. Elevated sIgA levels have been observed in the absence of bacteria, yeast or parasites, in individuals with atopic conditions such as food allergies, urticaria, and dermatitis.

References:

1. Crago SS, Tomasi TB. Mucosal Antibodies, Food Allergy and Intolerance. Bailliere Tindall/W.B. Saunders 1987;167-89.
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Fecal Fat Stain

The amount of total fat is higher than normal in this specimen. Individuals who have pancreatic insufficiency secondary to pancreatic or biliary tract disease will be unable to digest and absorb fat normally. The gold standard used to assess this type of malabsorption has been total fecal fat [1]. However, this type of test is too cumbersome (requires a 72 hr fecal collection while consuming 70 grams of fat per day) to be used as a frequent monitor of fat malabsorption [2]. Research has demonstrated that the microscopic fecal fat test is a reliable marker for fat malabsorption [1,3,4] as well as evaluating enzyme therapy in patients with pancreatic exocrine insufficiency [1,5]. In assessing mechanisms for steatorrhea, the following should be considered: gastric surgery, pancreatic disease, biliary obstruction, liver disease, mucosal integrity, and problems with chylomicron formation [6]. Supplementation with pancreatic enzymes, HCL, and/or bile salts may be indicated. Steatorrhea is associated with a particularly foul odor of the stool.

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Fecal Calprotectin

The level of fecal Calprotectin is higher than expected. Elevated fecal Calprotectin and Lactoferrin levels indicate the presence of neutrophils and inflammation in the gastrointestinal (GI) mucosa. Calprotectin and Lactoferrin facilitate differentiation between irritable bowel syndrome (IBS) and inflammatory bowel disease (IBD). IBD includes autoimmune conditions such as Crohns disease and ulcerative colitis (UC); these conditions may become life-threatening and require lifelong treatment.

For patients 4 years old to adults (Manz 2012; Fagerberg 2005):

High levels of Calprotectin (> 200 µg/gm) are associated with active IBD and gastrointestinal inflammation; elevation may also occur due to bacterial infection, colitis or sometimes, cancer. Fecal Calprotectin should be reassessed after about 4 weeks for confirmation.

Moderate calprotectin (50-200 µg/gm) is an indicator of chronic inflammation. Inflammation at this level may be due to IBD in remission or inflammation caused by non-steroidal anti-inflammatories (NSAIDs). Levels should be reassessed after about 4 weeks. Low levels of Calprotectin (< 50 µg/gm) are usually associated with viral GI infections or non-inflammatory bowel conditions such as IBS.

Multiple studies have shown fecal Calprotectin and Lactoferrin to be equivalent with respect to clinical sensitivity and specificity. Studies suggest that Calprotectin may correlate more closely with histological (cell microscopy) findings. Lactoferrin may correlate better to macroscopic (endoscopy) findings, and may be the better indicator of impending relapse, elevating 2-3 weeks prior to clinical symptoms.

Chronic inflammation of the gastrointestinal mucosa contributes to symptoms of IBD. Chronic stress is known to contribute to symptom flare-ups and increased inflammation. Liver disease or the use of aspirin or nonsteroidal anti-inflammatory (NSAID) medications may elevate Calprotectin levels. Fecal Calprotectin levels may also be increased in newborns.

References:

Carroccio, Antonio; Iacono, Giuseppe; Cottone, Mario; Di Prima, Lidia; Cartabellotta, Fabio et al. (2003) Diagnostic Accuracy of Fecal Calprotectin Assay in Distinguishing Organic Causes of Chronic Diarrhea from Irritable Bowel Syndrome: A Prospective Study in Adults and Children
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Journal of Pediatric Gastroenterology & Nutrition: April 2005 - Volume 40 - Issue 4 - pp 450-455

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Vieira, Andrea; Fang, Chia; Rolim, Ernani; Klug, Wilmar; Steinwurz, Flavio et al. (2009)
Inflammatory bowel disease activity assessed by fecal calprotectin and lactoferrin: correlation with laboratory parameters, clinical, endoscopic and histological indexes
BMC Research Notes vol. 2 (1) p. 221