



LAB #: F150217-0014-1
 PATIENT: Af'HYgh
 ID: P150480013
 SEX: Male
 DOB: 02/13/1988

CLIENT#: 24510

AGE: 27

Comprehensive Stool Analysis / Parasitology x2

BACTERIOLOGY CULTURE

Expected/Beneficial flora	Commensal (Imbalanced) flora	Dysbiotic flora
4+ Bacteroides fragilis group 4+ Bifidobacterium spp. 1+ Escherichia coli 2+ Lactobacillus spp. 3+ Enterococcus spp. 3+ Clostridium spp. NG = No Growth	3+ Alpha hemolytic strep 1+ Enterobacter cloacae complex	4+ Citrobacter freundii complex 3+ Citrobacter freundii complex, isolate 2

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
1+ Trichosporon mucoides	

MICROSCOPIC YEAST

Result:	Expected:
None	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 unviable.

Comments:

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



CLIENT#: 24510

Comprehensive Stool Analysis / Parasitology x2

PARASITOLGY/MICROSCOPY *
<p>Sample 1 None Ova or Parasites</p>
<p>Sample 2 None Ova or Parasites</p>
<p>*A trichrome stain and concentrated iodine wet mount slide is read for each sample submitted.</p>

PARASITOLGY INFORMATION
<p>Intestinal parasites are abnormal inhabitants of the gastrointestinal tract that have the potential to cause damage to their host. The presence of any parasite within the intestine generally confirms that the patient has acquired the organism through fecal-oral contamination. Damage to the host includes parasitic burden, migration, blockage and pressure. Immunologic inflammation, hypersensitivity reactions and cytotoxicity also play a large role in the morbidity of these diseases. The infective dose often relates to severity of the disease and repeat encounters can be additive.</p> <p>There are two main classes of intestinal parasites, they include protozoa and helminths. The protozoa typically have two stages; the trophozoite stage that is the metabolically active, invasive stage and the cyst stage, which is the vegetative inactive form resistant to unfavorable environmental conditions outside the human host. Helminths are large, multicellular organisms. Like protozoa, helminths can be either free-living or parasitic in nature. In their adult form, helminths cannot multiply in humans.</p> <p>In general, acute manifestations of parasitic infection may involve diarrhea with or without mucus and or blood, fever, nausea, or abdominal pain. However these symptoms do not always occur. Consequently, parasitic infections may not be diagnosed or eradicated. If left untreated, chronic parasitic infections can cause damage to the intestinal lining and can be an unsuspected cause of illness and fatigue. Chronic parasitic infections can also be associated with increased intestinal permeability, irritable bowel syndrome, irregular bowel movements, malabsorption, gastritis or indigestion, skin disorders, joint pain, allergic reactions, and decreased immune function.</p> <p>In some instances, parasites may enter the circulation and travel to various organs causing severe organ diseases such as liver abscesses and cysticercosis. In addition, some larval migration can cause pneumonia and in rare cases hyper infection syndrome with large numbers of larvae being produced and found in every tissue of the body.</p> <p>One negative parasitology x1 specimen does not rule out the possibility of parasitic disease, parasitology x3 is recommended. This exam is not designed to detect <i>Cryptosporidium</i> spp, <i>Cyclospora cayetanensis</i> or <i>Microsporidia</i> spp.</p>

GIARDIA/CRYPTOSPORIDIUM IMMUNOASSAY			
	Within	Outside	Reference Range
Giardia intestinalis	Neg		Neg
Cryptosporidium	Neg		Neg

Giardia intestinalis (lamblia) is a protozoan that infects the small intestine and is passed in stool and spread by the fecal-oral route. Waterborne transmission is the major source of giardiasis.

Cryptosporidium is a coccidian protozoa that can be spread from direct person-to-person contact or waterborne transmission.

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DIGESTION / ABSORPTION

	Within	Outside	Reference Range
Elastase	439		> 200 µg/mL
Fat Stain	Few		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	1.3		< 7.3 µg/mL
Calprotectin*	19		10 - 50 µg/g
Lysozyme*	518		range 12 ng/mL
White Blood Cells	None		None - Rare
Mucus	Neg		Neg

Lactoferrin and **Calprotectin** are reliable markers for differentiating organic inflammation (IBD) from functional 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*		572	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.

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Methodology: Elisa, Microscopy, Colormetric, Gas Chromatography, pH Electrode

Comprehensive Stool Analysis / Parasitology x2

SHORT CHAIN FATTY ACIDS

	Within	Outside	Reference Range
% Acetate	68		40 - 75 %
% Propionate	9.8		9 - 29 %
% Butyrate	21		9 - 37 %
% Valerate	1.3		0.5 - 7 %
Butyrate	2.6		0.8 - 4.8 mg/mL
Total SCFA's	12		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	6.0		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.

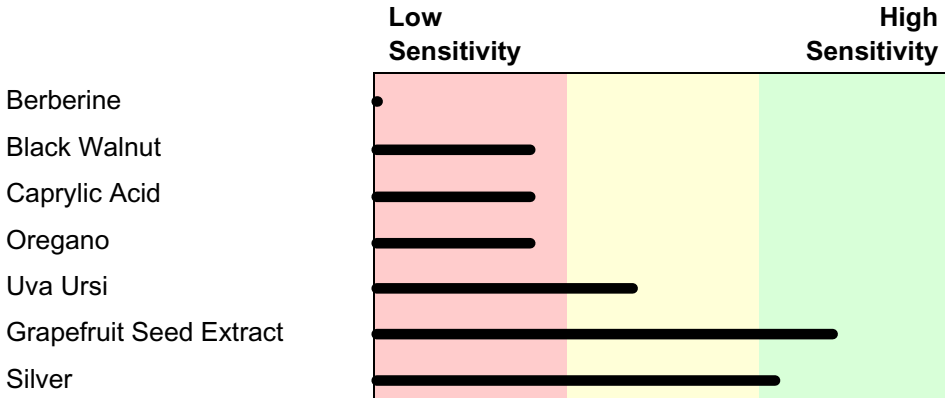
MACROSCOPIC APPEARANCE

	Appearance	Expected
Color	Brown	Brown
Consistency	Soft	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.

Bacterial Susceptibilities: Citrobacter freundii complex

NATURAL ANTIBACTERIALS



Natural antibacterial 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 activity is reported for each natural agent based upon the diameter of the zone of inhibition or no growth zone 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 activity is defined for the natural agents tested.

PRESCRIPTIVE AGENTS

	Resistant	Intermediate	Susceptible
Amoxicillin-Clavulanic Acid	R		
Ampicillin	R		
Cefazolin	R		
Ceftazidime			S
Ciprofloxacin			S
Trimeth-sulfa			S

Susceptible results imply that an infection due to the bacteria may be appropriately treated when the recommended dosage of the tested antimicrobial agent is used.

Intermediate results imply that response rates may be lower than for susceptible bacteria when the tested antimicrobial agent is used.

Resistant results imply that the bacteria will not be inhibited by normal dosage levels of the tested antimicrobial agent.

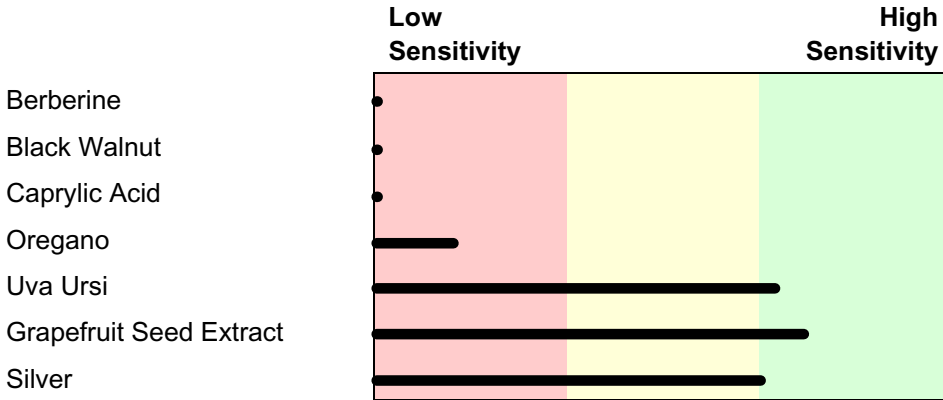
Comments:

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Natural antibacterial agent susceptibility testing is intended for research use only.
 Not for use in diagnostic procedures.

Bacterial Susceptibilities: Citrobacter freundii complex, isolate 2

NATURAL ANTIBACTERIALS



Natural antibacterial 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 activity is reported for each natural agent based upon the diameter of the zone of inhibition or no growth zone 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 activity is defined for the natural agents tested.

PRESCRIPTIVE AGENTS

	Resistant	Intermediate	Susceptible
Amoxicillin-Clavulanic Acid	R		
Ampicillin	R		
Cefazolin	R		
Ceftazidime			S
Ciprofloxacin			S
Trimeth-sulfa			S

Susceptible results imply that an infection due to the bacteria may be appropriately treated when the recommended dosage of the tested antimicrobial agent is used.
Intermediate results imply that response rates may be lower than for susceptible bacteria when the tested antimicrobial agent is used.
Resistant results imply that the bacteria will not be inhibited by normal dosage levels of the tested antimicrobial agent.

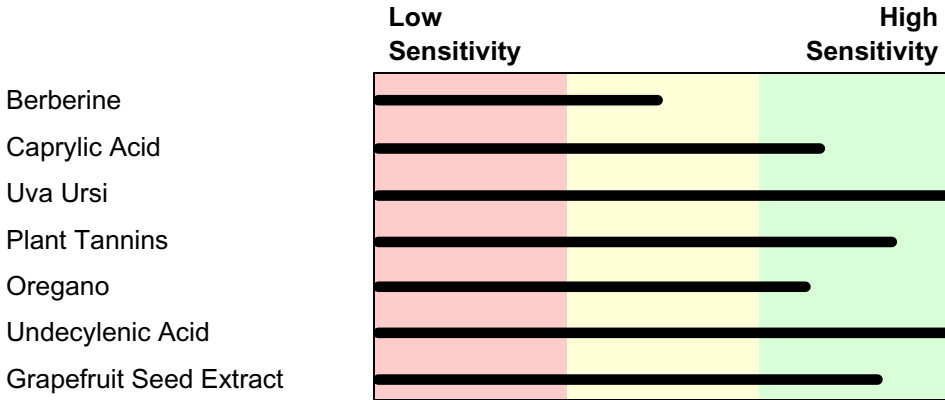
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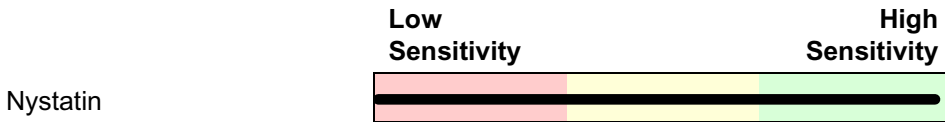
Yeast Susceptibilities: *Trichosporon mucoides*

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 activity is reported for each natural agent based upon the diameter of the zone of inhibition or no growth zone 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 activity 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 activity is reported based upon the diameter of the zone of inhibition or no growth zone surrounding the disk.

Comments:

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 Date Completed: 03/02/2015

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

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 Flora

In a healthy balanced state of intestinal flora, the beneficial bacteria make up a significant proportion of the total microflora. However, in many individuals there is an imbalance or deficiency of beneficial flora and an overgrowth of non-beneficial (imbalance) or even pathogenic microorganisms (dysbiosis). This can be due to a number of factors including: consumption of contaminated water or food; daily exposure of chemicals that are toxic to beneficial bacteria; the use of antibiotics, oral contraceptives or other medications; poor fiber intake and high stress levels.

A number of toxic substances can be produced by the dysbiotic bacteria including amines, ammonia, hydrogen sulfide, phenols, and secondary bile acids which may cause inflammation or damage to the brush border of the intestinal lining. If left unchecked, long-term damage to the intestinal lining may result in leaky gut syndrome, allergies, autoimmune disease (e.g. rheumatoid arthritis), irritable bowel syndrome, fatigue, chronic headaches, and sensitivities to a variety of foods. In addition, pathogenic bacteria can cause acute symptoms such as abdominal pain, nausea, diarrhea, vomiting, and fever in cases of food poisoning.

Bacterial sensitivities to a variety of prescriptive and natural agents have been provided for the pathogenic bacteria that were cultured from this patient's specimen. This provides the practitioner with useful information to help plan an appropriate treatment regimen. Supplementation with probiotics or consumption of foods (yogurt, kefir, miso, tempeh, tamari sauce) containing strains of lactobacilli, bifidobacteria, and enterococci can help restore healthy flora levels. Polyphenols in green and ginseng tea have been found to increase the numbers of beneficial bacteria. Hypochlorhydria may also predispose an individual to bacterial overgrowth, particularly in the small intestine. Nutritional anti-inflammatories can aid in reversing irritation to the GI lining. These include quercetin, vitamin C, curcumin, gamma-linoleic acid, omega-3 fatty acids (EPA, DHA), and aloe vera. Other nutrients such as zinc, beta-carotene, pantothenic acid, and L-glutamine provide support for regeneration of the GI mucosa. A comprehensive program may be helpful in individuals in whom a dysbiotic condition has caused extensive GI damage.

Lispi E. Digestive Wellness. New Canaan,CT: Keats Publishing;1996.

Mitsuoka T. Intestinal Flora and Aging. Nutr Rev 1992;50(12):438-446.

Weisburger JH. Tea and Health: The Underlying Mechanisms. Proc Soc Exp Biol Med 1999;220(4):271-275.4.

Pereira SP, Gainsborough N, Dowling RH. Drug-induced Hypochlorhydria Causes High Duodenal Bacterial Counts in the Elderly. Ailment Pharmacol Ther 1998;12(1)99-104.

Murray MT. Stomach Ailments and Digestive Disturbances. Rocklin, CA: Prima Publishing; 1997.

Citrobacter species

Citrobacter species, a gram-negative bacterium and member of the Enterobacteriaceae family, is considered dysbiotic at 3+ or greater.

Citrobacter freundii complex, including Citrobacter freundii, Citrobacter braakii, Citrobacter gullenii, Citrobacter murlinae, Citrobacter rodentium, Citrobacter werkmanii, Citrobacter oungae, and less commonly, Citrobacter koseri and Citrobacter farmeri, can cause diarrheal disease. Symptoms due to Citrobacter freundii complex seem to be a result of the elaboration of an E. coli-like heat-stable enterotoxin and hydrogen sulfide. Citrobacter freundii complex has been implicated as a cause of gastrointestinal infection and inflammation, acute dysentery, and dyspepsia. Acute symptoms can include profuse, watery diarrhea which is often unaccompanied by abdominal pain, fecal blood, or white blood cells.

Citrobacter species thrive on Fructooligosaccharides (FOS), a common ingredient in artificial or alternative sweetener.

Antibiotics may be indicated if symptoms are prolonged. Refer to the bacterial sensitivities to identify the most appropriate agent.

Guarino, A, et al. Production of Escherichia coli Sta-Like Heat-Stable Enterotoxin by Citrobacter freundii Isolated from Humans. Journal of Clinical Microbiology. January 1987;110-114.

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 686-689.

Murray PR, Baron EJ, Jorgensen JH, Pfaller MA, Tenover FC, Tenover FC. Manual of Clinical Microbiology, 8th edition. Washington, DC: ASM Press; 2003. pg 701-704.

Cultured Yeast

Yeast, such as Candida are normally present in the GI tract in very small amounts. Many species of yeast exist and are commensal; however, they are always poised to create opportunistic infections and have detrimental effects throughout the body. Factors that contribute to a proliferation of yeast include frequent use of wide-spread antibiotics/low levels of beneficial flora, oral contraceptives, pregnancy, cortisone and other immunosuppressant drugs, weak immune system/low levels of sIgA, high-sugar diet, and high stress levels.

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 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 unviable for culturing. Therefore, both microscopic examination and culture are helpful in determining if abnormally high levels of yeast are present.

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.
2. Roberts JA. Factors predisposing to urinary tract infections in children. *Ped Neph* 1996;10:517-522.
3. Carins J, Booth C. Salivary immunoglobulin-A as a marker of stress during strenuous physical training. *Aviat Space Environ Med* 2002;73(12)1203-7.
4. Teodosio MR, Oliveira ECM. Urinary secretory IgA after nutritional rehabilitation. *Braz J Med Biolog Res* 1999;32-421-426
5. Alverdy J. Effects of glutamine-supplemented diets on immunology of the gut. *J Parent Enteral Nutr* 1990;14(4):1095-1135.
6. Burke DJ, et al. Glutamine-supplemented total parenteral nutrition improves gut function. *Arch Surg* 1989;24:2396-2399.
7. Alverdy JA. The effect of total parenteral nutrition on gut lamina propria cells. *J Parent. Enteral Nutr* 1990;14(suppl).
8. Qamar A, Aboudola S, Warny M, et al. *Saccharomyces boulardii* stimulates intestinal immunoglobulin A immune response to clostridium difficile toxin A in mice. *Infect Immun* 2001;69(4):2762-5.
9. Buts JP, Bernasconi P, Vaerman JP, et al. Stimulation of secretory IgA and secretory component of immunoglobulins in small intestine of rats treated with *Saccharomyces boulardii*. *Dig Dis Sci* 1990;35(2):251-6.

Beneficial Flora

One or more of the expected or beneficial bacteria are low in this specimen. Normally abundant include lactobacilli, bifidobacteria, clostridia, *Bacteroides fragilis* group, enterococci, and some strains of *Escherichia coli*. The beneficial flora have many health-protecting effects in the gut, and as a consequence, are crucial to the health of the whole organism. Some of the roles of the beneficial flora include digestion of proteins and carbohydrates, manufacture of vitamins and essential fatty acids, increase in the number of immune system cells, break down of bacterial toxins and the conversion of flavinoids into anti-tumor and anti-inflammatory factors. Lactobacilli, bifidobacteria, clostridia, and enterococci secrete lactic acid as well as other acids including acetate, propionate, butyrate, and valerate. This secretion causes a subsequent decrease in intestinal pH, which is crucial in preventing an enteric proliferation of microbial pathogens, including bacteria and yeast. Many GI pathogens thrive in alkaline environments. Lactobacilli also secrete the antifungal and antimicrobial agents lactocidin, lactobacillin, acidolin, and hydrogen peroxide. The beneficial flora of the GI have thus been found useful in the inhibition of microbial pathogens, prevention and treatment of antibiotic associated diarrhea, prevention of traveler's diarrhea, enhancement of immune function, and inhibition of the proliferation of yeast.

In a healthy balanced state of intestinal flora, the beneficial flora make up a significant proportion of the total microflora. Healthy levels of each of the beneficial bacteria are indicated by either a 3+ or 4+ (0 to 4 scale). However, some individuals have low levels of beneficial bacteria and an overgrowth of nonbeneficial (imbalances) or even pathogenic microorganisms (dysbiosis). Often attributed to the use of antibiotics, individuals with low beneficial bacteria may present with chronic symptoms such as irregular transit time, irritable bowel syndrome, bloating, gas, chronic fatigue, headaches, autoimmune diseases (e.g., rheumatoid arthritis), and sensitivities to a variety of foods. Treatment may include the use of probiotic supplements containing various strains of lactobacillus and bifidobacterium species and consumption of cultured or fermented foods including yogurt, kefir, miso, tempeh and tamari sauce. Polyphenols in green and ginseng tea have been found to increase the numbers of beneficial bacteria. If dysbiosis is present, treatment may also include the removal of pathogenic bacteria, yeast, or parasites.

Percival M. Intestinal Health. *Clin Nutr In.* 1997;5(5):1-6.

Fuller R. Probiotics in Human Medicine. *Gut.* 1991;32: 439-442.

Siitonen S, Vapaatalo H, Salminen S, et al. Effect of Lactobacilli GG Yoghurt in Prevention of Antibiotic Associated Diarrhea. *Ann Med.* 1990; 22:57-59.

Oksanen P, Salminen S, Saxelin M, et al. Prevention of Travelers' Diarrhea by Lactobacillus GG. *Ann Med.* 1990; 22:53-56.

Perdigon G, Alvarez M, et al. The Oral Administration of Lactic Acid Bacteria Increases the Mucosal Intestinal Immunity in Response to Enteropathogens. *J Food Prot.* 1990;53:404-410.

Valeur, N, et al. Colonization and Immunomodulation by Lactobacillus reuteri ATCC 55730

in the Human Gastrointestinal Tract. Appl Environ. Microbiol. 2004 Feb; 70(2):1176-81.

Elmer G, Surawicz C, and McFarland L. Biotherapeutic agents - a Neglected Modality for the Treatment and Prevention of Intestinal and Vaginal Infections. JAMA. 1996; 275(11):870-876.

Fitzsimmons N and Berry D. Inhibition of *Candida albicans* by *Lactobacillus acidophilus*: Evidence for Involvement of a Peroxidase System. Microbio. 1994; 80:125-133

Weisburger JH. Proc Soc Exp Biol Med 1999;220(4):271-5.