



LAB#: F000000-0000-0
PATIENT: Sample Patient
ID: PATIENT-S-00010
SEX: Female
AGE: 39

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

Comprehensive Stool Analysis / Parasitology x1

MICROBIOLOGY

Bacteriology Culture

Beneficial flora		Imbalances	Dysbiotic flora	
Bifidobacter	4+		Citrobacter freundii	1+
E. coli spp.	4+		Enterobacter cloacae	4+
Lactobacillus spp.	4+			
Enterococcus spp.	0+			

Mycology (Yeast) Culture

Normal flora	Dysbiotic flora
	Candida parapsilosis 2+

PARASITOLOGY / MICROSCOPY (TRICHROME STAIN & CONCENTRATION)

Sample 1
 No Ova or Parasites
 Mod Yeast
 Rare RBC

	Within	Outside	Ref. Range		Within	Outside	Ref. Range
Giardia Lamblia (EIA)	Neg		Neg	Cryptosporidium (EIA)	Neg		Neg

Beneficial flora: In a healthy balanced state of intestinal flora, the beneficial bacteria make up a significant proportion of the total microflora. The beneficial flora have many health-protecting effects in the gut including manufacturing vitamins, fermenting fibers, digesting proteins and the disaccharide lactose, and propagating anti-tumor and anti-inflammatory factors. Acidophilus, Bifidus, and Enterococcus produce lactic acid and short-chain fatty acids. The fermentation of fibers by beneficial bacteria and subsequent production of short chain fatty acids is crucial in lowering colonic pH and preventing the proliferation of microbial pathogens, including bacteria and yeast. Enterococcus has antibacterial activity against methicillin-resistant *S. aureus* (MRSA) and food-borne pathogens.

Parasitology: Intestinal parasites are abnormal inhabitants of the GI tract. Factors such as contaminated food and water supplies, day care centers, international travel, pets, carriers such as mosquitoes and fleas, and sexual transmission have contributed to an increased prevalence of intestinal parasites in the American population.

Date Collected: **7/3/2007** Comments:
 Date Received: **7/6/2007**
 Date Completed: **7/16/2007**



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CAMPYLOBACTER CULTURE

	Within	Outside	Ref. Range	
Campylobacter jejuni	Neg		Neg	<p>Campylobacter jejuni is a pathogenic bacteria and a common cause of diarrheal disease, often accompanied by abdominal cramping, fever, and vomiting. Campylobacter infection is often associated with raw or undercooked poultry, unpasteurized milk, or contaminated water.</p>

DIGESTION / ABSORPTION

	Within	Outside	Ref. Range	
Elastase	> 500		> 200 µg/mL	<p>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. Meat/Vegetable fibers: The presence of meat and/or vegetable fibers in the stool may be due to a number of factors including, improper mastication, excessive protein intake, a reduction of gastric HCL secretion, or insufficient output of pancreatic enzymes. Carbohydrates: The presence of reducing substances in stool specimens can indicate carbohydrate malabsorption.</p>
Fat stain	None		None - Mod	
Muscle fibers	None		None - Rare	
Vegetable fibers	Few		None - Few	
Carbohydrates	Neg		Neg	

INFLAMMATION

	Within	Outside	Ref. Range	
Lysozyme*		957	<= 600 ng/mL	<p>Lysozyme is an enzyme secreted at the site of inflammation in the GI tract and elevated levels have been identified in IBD patients. Lactoferrin is a quantitative GI specific marker of inflammation used to diagnose and differentiate IBD from IBS and to monitor patient inflammation levels during active and remission phases of IBD. WBCs: Elevated stool levels of white blood cells occur following an infiltration of leukocytes within the intestinal lumen during an inflammatory process. Mucus in the stool may result from prolonged mucosal irritation or in response to parasympathetic excitability such as spastic constipation or mucous colitis.</p>
Lactoferrin	1.9		< 7.3 µg/mL	
WBC	None		None - Rare	
Mucus	Neg		Neg	

IMMUNOLOGY

	Within	Outside	Ref. Range	
slgA*		339	51 - 204mg/dL	<p>slgA: Secretory IgA is secreted by mucosal-associated lymphoid tissue and represents the first line of defense of the GI mucosa and is central to the normal function of the GI as an immune barrier. Elevated levels of slgA have been associated with an upregulated immune response.</p>

*For Research Use Only. Not for use in diagnostic procedures.



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SHORT CHAIN FATTY ACIDS

	Within	Outside	Ref. Range	
Acetate	55		36 - 74	%
Propionate	10		9 - 32	%
Butyrate	32		16 - 39	%
Valerate	3		1 - 8	%
Butyrate	2.5		0.8 - 3.8	mg/mL
Total SCFA's	7.8		4 - 14	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. Lactobacillus and Bifidus 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/g are important for assessing overall SCFA production, and are reflective of beneficial flora levels and/or adequate fiber intake.

INTESTINAL HEALTH MARKERS

	Within	Outside	Ref. Range	
RBC	Rare		None - Rare	
pH	6.1		6 - 7.8	
Occult Blood	Neg		Neg	
Yeast		Mod	None - Rare	

RBC: Red blood cells 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. **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. **pH:** Fecal pH is largely dependent on the fermentation of fiber by the beneficial flora of the gut. **Yeast:** A positive microscopic yeast level indicates the presence of fungi such as Candida albicans in the stool.

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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BACTERIAL SUSCEPTIBILITIES

Citrobacter freundii

Prescriptive agents

Sensitive

Intermediate

Resistant

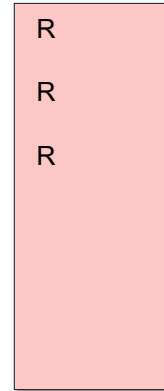
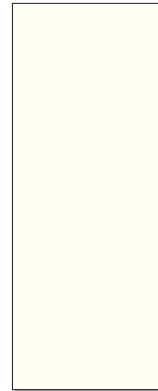
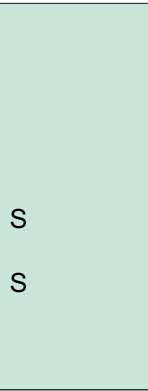
Amoxicillin

Ampicillin

Augmentin

Ciprofloxacin

Trimeth-sulfa



Natural agents

Sensitive

Resistant

Berberine

Black Walnut

Caprylic Acid

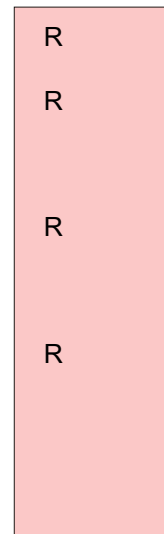
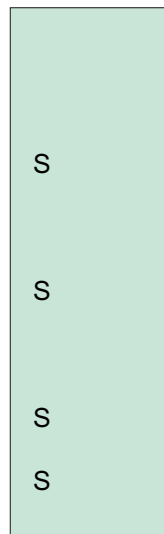
Cats Claw

Citrus Seed Extract

Goldenseal

Oregano

Uva Ursi





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BACTERIAL SUSCEPTIBILITIES

Enterobacter cloacae

Prescriptive agents

Sensitive

Intermediate

Resistant

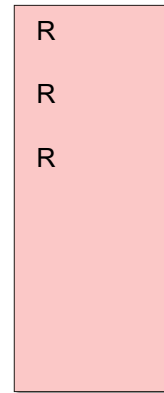
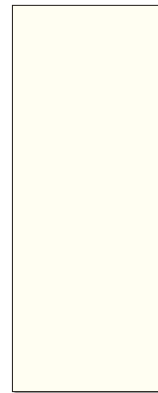
Amoxicillin

Ampicillin

Augmentin

Ciprofloxacin

Trimeth-sulfa



Natural agents

Sensitive

Resistant

Berberine

Black Walnut

Caprylic Acid

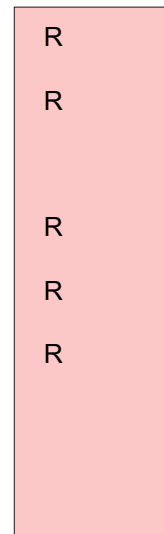
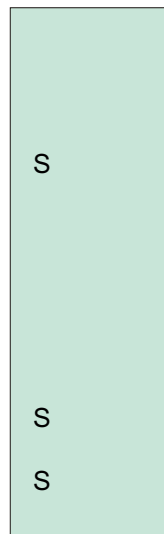
Cats Claw

Citrus Seed Extract

Goldenseal

Oregano

Uva Ursi





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YEAST SUSCEPTIBILITIES

Candida parapsilosis

Prescriptive agents

Sensitive

Intermediate

Resistant

Fluconazole

S

Itraconazole

S

Ketoconazole

S

Nystatin

S

Natural agents

Sensitive

Resistant

Berberine

S

Caprylic Acid

S

Goldenseal

S

Oregano

S

Tanalbit

S

Undecylenic Acid

S

Uva Ursi

S

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.

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 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 [1].

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 [2]. 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 [1]. 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, tofu, tamari sauce) containing strains of *Lactobacillus* and *Bifidus* can help restore healthy flora levels [1]. Polyphenols in green and ginseng tea have been found to increase the numbers of beneficial bacteria [3]. Hypochlorhydria may also predispose an individual to bacterial overgrowth, particularly in the small intestine [4]. 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 [5]. A comprehensive program may be helpful in individuals in whom a dysbiotic condition has caused extensive GI damage.

1. Lispi E. Digestive Wellness. New Canaan,CT: Keats Publishing;1996.
2. Mitsuoka T. Intestinal flora and aging. Nutr Rev 1992;50(12):438-46.
3. Weisburger JH. Tea and health: the underlying mechanisms. Proc Soc Exp Biol Med 1999;220(4):271-5.
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.
5. Murray MT. Stomach Ailments and Digestive Disturbances.

Rocklin, CA: Prima Publishing; 1997.

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. 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.

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 rare or few) may be normal, yeast observed in higher amounts (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, recurring 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 yeast syndrome. However, culturing of yeast is not always possible, due to the fact that yeast does not always survive transit through the intestines. Additionally, yeast colonizes in groups and is not dispersed uniformly throughout the stool. Yeast may therefore appear (microscopically) to be inconsistently concentrated in some stool specimens, 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 yeasts 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 crypts which 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).

1. Saito H, Ksajima T, Masuda A, et al. Lysozyme localization in human gastric and duodenal epithelium. *Cell Tissue Res* 1988; 251:3-7-313.
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.
5. Stamp GWH, Poulsom R, Chung LP, et al. Lysozyme gene expression in inflammatory bowel disease. *Gastroenterol* 1992;103:532-538.

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:

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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.

Citrobacter species

Citrobacter freundii, a gram-negative bacterium and member of the Enterobacteriaceae family, is considered to be an enteropathogen. Other citrobacter species are less commonly identified as pathogens. *Citrobacter* is common in the environment, including food and animal feces. Incidence, per laboratory observation, seems to be higher in Spring and Summer.

C. freundii and, less commonly, *C. koseri* can cause diarrheal disease. *Citrobacter koseri* has also been isolated in rare cases of neonatal meningitis. *Citrobacter* is often asymptomatic, but has been known to cause sepsis and infection in a number of tissues. Symptoms due to *C. freundii* seem to be a result of the elaboration of an *E. coli*-like heat-stable enterotoxin and hydrogen sulfide. *Citrobacter freundii* 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.

C. freundii thrives on FOS, a common ingredient in probiotic formulas. Treatment herbs include garlic and aloe vera. Specific natural or pharmaceutical treatments should be based on susceptibility testing.

Derlet RW, Carlson JR. An analysis of human pathogens found in horse/mule manure along the John Muir Trail in Kings Canyon and Sequoia and Yosemite National Parks. *Wilderness and Environmental Medicine*, 13(2):13-118, 2004.

Morris JG Jr, Lin FY, Morrison CB, Gross RJ, Khabbaz R, Maher KO, Rowe B, Israel E, Libonati JP. Molecular epidemiology of neonatal meningitis due to *Citrobacter diversus*: a study of isolates from hospitals in Maryland. *J Infect Dis*, 154(3):409-14, 1986.

Murray PR, Baron EJ, Pfaller MA, Tenover FC, Tenover RH. *Manual of Clinical Microbiology*, 6th edition. Washington, DC: ASM Press; 1995.

Enterobacter cloacae

This gram negative bacterium is considered dysbiotic in the amount of 3 - 4+. A member of Enterobacteriaceae, *Enterobacter* spp are considered opportunistic pathogens associated with diarrhea in children, and can also be involved in extraintestinal infections including the urinary tract. *Enterobacter* is also one of the main causative organisms in nosocomial septicemia and surgical wound infections. *Enterobacter* spp. are also common causes of intraabdominal, respiratory tract, and bloodstream infections in transplant recipients.

Widely distributed in the environment, *Enterobacter* is commonly isolated from both human and animal feces. Environmental strains of *Enterobacter* are capable of growth in foods at refrigeration temperatures. It produces a heat-stable toxin similar to that of *E. coli*.

Treatment herbs include aloe vera. Refer to susceptibilities for further treatment options.

Celkan T et al. Bacteremia in childhood cancer. *J Trop Pediatr*, 48(6):373 - 7, 2002.

