NutriPATH

INTEGRATIVE PATHOLOGY SERVICES

CDSA STOOL TESTING

The Practitioner Hand Book

1300 688 522 | www.nutripath.com.au

NutriPATH
INTEGRATIVE PATHOLOGY SERVICES

About NutriPATH Pathology:

NutriPATH is a privately owned medical laboratory that specialises in the area of functional health and wellbeing pathology testing. NutriPATH today, is one of the largest functional testing laboratories servicing integrative medicine practitioners throughout Australasia. Our functional pathology and routine pathology tests assist general practitioners, naturopaths, nutritionists, dieticians, chiropractors, osteopaths and other complementary healthcare practitioners in identifying the underlying cause of illness as well as providing tools for the prevention of disease and premature ageing.

NutriPATH offers more tests than any other integrative medical laboratory. With more than 200 tests available, it is the anti-ageing and integrative medical practitioners' one-stop-shop. Assessments investigate the areas of endocrine, gastrointestinal, immunological, genomic, metabolic and nutritional status.

Our clinical and scientific staff:

The clinical and scientific staff at NutriPATH are pioneers in the industry of functional medicine with many years of experience and technical knowledge and are proactively involved in further training and introducing new and advancements in pathology testing within Australasia.

Our Mission Statement:

"We work with our Practitioners to provide the most efficient, effective and comprehensive range of functional pathology testing, so that our Practitioners can provide the best consultative process for the treatment of their patients."

Mary Cavaggion
Director

The Reliability of Test Results With NutriPATH Pathology

In a laboratory environment, the reliability of delivering high quality test results is crucial in day to day procedures. A clinically useful test must provide reliable information that would not normally be derived from symptoms alone. Reliability of test results to NutriPATH Pathology means that a diagnostic test assay that is performed must be adequately sensitive and highly reproducible. For a test to be reliable, not only must it be reproducible over time, it must also vary with real change in the individual, either owing to treatment or some other factor, not just random variation of the assay. Reliability of a test is determined by both analytical and non-analytical factors.

Analytical factors such as: Accuracy, Precision, Sensitivity and Specificity.

Accuracy: A pathology test is accurate if it reflects the true value or detects the presence of the substance being measured. In analytical runs of multiple specimens, internal quality control (QC) as well as external quality control (EQC) samples of known values are included to check for the accuracy of the run each time the assay is performed.

Precision: Precision refers to the measure of variance of the assay. Imagine a target in a shooting range. If all of the hits form a tight cluster on the target, the shooter is very precise. If it hits the cluster around the centre, the shooter is also very accurate. Similarly, a test that is run on the same sample many times should yield results that are clustered in a small range. In some cases where a result is flagged outside of range, this is repeated at no charge for confirmation.

Sensitivity and Specificity: These terms refer to a diagnostic test's ability to reliably predict whether a person does or does not have a particular condition. A test with high sensitivity correctly identifies a high percentage of patients who actually have a particular condition.

What makes NutriPATH unique to other Laboratories?

NutriPATH has a strong specialty in microbiology testing. Our laboratory uses multiple diagnostic testing methods such as:

- GC-MS, automated chemistry for various stool biomarkers.
- Bacteria Culture/**MALDI-TOF Mass Spectrometry to identify organisms**
- Direct Microscopic Examination
- Enzyme-linked immunosorbent assay (ELISA)
- Microbial antibiotic susceptibility using key prescriptive agents
- Microbial natural susceptibility using key natural agents
- Polymerase Chain Reaction **(PCR DNA MULTIPLEX-ELIMINATES THE NEED FOR 3 DAY STOOL COLLECTIONS)**

NutriPATH
Integrative Pathology Services

Why Choose NutriPATH Pathology for stool testing?

- Technical support available Monday-Friday 8.30am - 5.00pm (multiple clinicians onsite).
- Comprehensive and easy to understand test reporting.
- Competitive pricing for you and your patients to achieve optimal health results.
- An easy and reliable portal ordering system for your clinics needs with 24-48 hour delivery.
- A comprehensive choice of test selection and customisation to your patient's needs.

The clinical relevance of CDSA Testing:

NutriPATH offers an extensive range of gastrointestinal test profiles to assist and provide the practitioner with flexibility in deciding the most appropriate test analyses for the patient.

With our range of Comprehensive Digestive Stool Analysis (CDSA) Profiles, the CDSA Test can assist physicians to develop earlier, more effective preventive interventions, improve the timing and precision of treatments and reduce the risk of clinical relapse in certain groups of patients. It will also allow physicians to better evaluate and document the medical necessity of more invasive procedures such as colonoscopy.

When Should a CDSA profile be considered?

A CDSA profile can reveal important underlying clinical information about many common symptoms such as gas, bloating, abdominal pain, diarrhoea and constipation. A standard stool test (micro culture and sensitivities) does not provide the vast information of a CDSA. The CDSA assesses the digestive tract and large intestine providing useful information and treatment protocols for your patients.

What can clinicians and patients expect from a CDSA profile compared to other diagnostics?

Evidence suggests that both localised and systemic health issues may begin as imbalances in Gastrointestinal function. The CDSA test provides immediate, actionable and clinical information for patients presenting with GI complaints. It aids clinicians in the identification of root cause(s) of digestive discomfort and supports identification of targeted treatments.

How should my patient thoroughly prepare for this Test?

Instruction:	Recommended Timeframe to Discontinue	Possible Biomarker(s) Impacted
The Diet		
General Dietary Requirements:	It is recommended to continue your routine diet as per usual	Not Applicable
High Protein	It is recommended to continue your routine diet as per usual	May see elevated putrefactive SCFAs
High Fat	It is recommended to continue your routine diet as per usual	May see elevated faecal fats
Foods with Beneficial Flora (Yogurt)	10-14 days	May influence the beneficial bacteria levels on culture, as well as metabolic markers; the presence of beneficial bacteria may alter levels of other bacteria, yeast and parasites
POSSIBLE INTERACTION: Anti-inflammatory and Immune Modulating Medications		
Aspirin and NSAIDs *(i.e. ibuprofen, etc.)*	2 days	May influence inflammation/immune biomarkers *(EPX, calprotectin)*
Steroid *(i.e. prednisone, etc.)*	No recommendation to discontinue	
Autoimmune medications *(i.e. Humira, etc.)*	No recommendation to discontinue	
POSSIBLE INTERACTION: Digestive Tract Medications and Supplements		
Probiotics *(beneficial bacteria)*	10-14 days	May influence the beneficial bacteria levels on culture, as well as metabolic markers; the presence of beneficial bacteria may alter levels of other bacteria, yeast and parasites
Antacids, PPI *(i.e. proton-pump inhibitors)*	3-14 days; if ordering CDSA 3+ or CDSA 4+, 14 days is recommended	May result in false negative H. pylori if PPI not discontinued for 14 days (H2 blockers do not interfere); additionally, acid-blocking medication may influence levels of the digestion and absorption markers; PPIs may clear the body relatively quickly, however the antacid effect may linger 3-5 days
Bismuth	CDSA 3+ or CDSA 4+, 14 days is recommended	May result in false negative H. pylori if bismuth not discontinued for 14 days; may affect other bacterial levels
Antacids *(i.e. Quick-Eze, Mylanta, H2 blockers)*	2 days	Acid-blocking medication may influence levels of the digestion and absorption markers
Bentonite clay		Bacteria and parasites that may be identified through microscopic examination and culture growth
Digestive enzymes		Enzymes are intended to improve digestion, therefore markers of digestion and absorption may be influenced
Laxatives		Laxatives are intended to alter transit time; if laxative use results in normalized transit, there may be no effect on biomarkers; however if transit time is rapid, the markers of digestion and absorption may be influenced
Rectal suppositories, enemas		These agents can alter the density of stool samples resulting in inaccurate biomarker findings
Please note that 4 weeks is recommended from colonoscopy or as it has been observed that 4 weeks is sufficient time for most patients to resume their previous level of gut function, considering elimination of all traces of barium, dietary/digestive normalisation and regeneration of microflora populations.		
POSSIBLE INTERACTION: Antimicrobial Agents		
Antibiotics	14 days (28 days may be preferred after antibiotics)	May influence levels of bacteria, yeast and parasites, as well as metabolic markers
Antifungals		
Antiparasitics		

NutriPATH
Integrative Pathology Services

Comprehensive Digestive Stool Analysis Collection Kit


NutriPATH Phone: 1300 688 522

CDSA STOOL KIT

Collection Instructions

COLLECTION REQUIREMENTS

If you are uncertain of the collection procedure after reading these instructions, please contact our Customer Service on 1300 688 522 who will clearly explain the procedure.

Before proceeding, please check the following guidelines.
Without taking these precautions your results may be inaccurate.

IMPORTANT NOTES

- DO NOT discard the fluid in the tubes as this is a preservative.
- AVOID contact of the fluid in the tubes with your eyes or skin (wash area thoroughly if this occurs).
- DO NOT inhale or ingest the fluid in the tubes.
- Keep out of reach of children.
- 2 weeks before the test, cease taking antibiotics and antifungal drugs, unless instructed otherwise by your practitioner.
- 3 days before the test, cease taking oral castor oil, probiotics, laxatives, aspirin or ibuprofen. Also cease taking all gut associated supplements like pre- and probiotics, digestive enzymes, detox/liver herbs and minerals, amino acids unless instructed otherwise by your practitioner.
- 2 days before the test, follow your normal diet, including the following foods if possible: meat, fish, poultry, vegetables (raw and cooked), carbohydrates (potatoes, rice, pasta, bread), fats (butter, margarine, oils). If you do not eat some of these foods, you can still do the test. Vegetarians and vegans should eat their normal diet.

PO Box 442, Ashburton, VIC, 3147

Each CDSA Level Explained:

When to use which profile

Each CDSA panel reports on sensitivities against microbial infection. The CDSA 3+ and the DNA Faecal PCR is a great starting point in assessing gastrointestinal dysfunction and microbiota status.

CDSA LEVEL 1- ASSESSES: Macroscopic & Microscopic Description, Beneficial and other Bacteria; Yeasts; Parasites (visual detection); Antibiotic/Natural agents sensitivities (bacteria & yeasts) **$120.00 or with PCR $170.00**

CLINICALLY: Ideal to identify the good and bad bugs or beneficial bacteria and pathogenic microbiota in the GUT.

CDSA LEVEL 2- ASSESSES: Macroscopic & Microscopic Description; Digestive, Absorption and Metabolic markers; Beneficial and other Bacteria; Yeasts; Parasites (visual detection); Antibiotic/Natural agents sensitivities (bacteria & yeasts) **$220.00 or with PCR $270.00**

CLINICALLY: Ideal to identify digestive and metabolic markers together with beneficial bacteria and pathogenic microbiota in the GUT.

CDSA LEVEL 3- ASSESSES: Macroscopic & Microscopic Description; Digestive, Absorption and Metabolic markers including PE1; Beneficial and other Bacteria; Yeasts; Parasites (visual detection); Antibiotic/Natural agents sensitivities (bacteria & yeasts) **$260.00 or with PCR $310.00**

CLINICALLY: Ideal to identify pancreatic function with digestive and metabolic markers together with beneficial bacteria and pathogenic microbiota in the GUT.

CDSA LEVEL 3+ ASSESSES: Macroscopic & Microscopic Description; Digestive, Absorption and Metabolic markers; Inflammation markers; Tumour/Ulcer markers; Beneficial and other Bacteria; Yeasts; Parasites (visual detection); Antibiotics/Natural agents sensitivities (bacteria & yeasts) **$370.00 or with PCR $420.00**

CLINICALLY: The ideal starting point test identifying inflammatory markers including Transglutaminase, a gluten sensitive marker and tumour markers; digestive and metabolic markers together with beneficial bacteria and pathogenic microbiota in the GUT.

CDSA LEVEL 4 - ASSESSES: Macroscopic & Microscopic Description; Digestive, Absorption and Metabolic markers including PE1; Beneficial & other Bacteria; Yeasts; Parasites (visual & chemical EIA detection); Antibiotic/Natural agents sensitivities (bacteria & yeasts) **$340.00 or with PCR $390.00**

CLINICALLY: Ideal to identify digestive and metabolic markers together with beneficial bacteria and pathogenic microbiota in the GUT. Parasites are measured in both visual and EIA technology.

CDSA LEVEL 4 + ASSESSES: Macroscopic & Microscopic Description; Digestive, Absorption and Metabolic markers; Inflammation markers; Tumour/Ulcer markers; Beneficial and other Bacteria; Yeasts; Parasites (visual & chemical EIA detection); Antibiotics/Natural agents sensitivities (bacteria & yeasts) **$450.00 or with PCR $500.00**

CLINICALLY: Ideal to identify inflammatory markers including Transglutaminase, a gluten sensitive marker and tumour markers; digestive and metabolic markers together with beneficial bacteria and pathogenic microbiota in the GUT. Parasites are measured in both visual and EIA technology.

CDSA LEVEL 5- ASSESSES: Macroscopic & Microscopic Description; Beneficial and other Bacteria; Yeasts; Parasites (visual & chemical EIA detection); Antibiotic/Natural agents sensitivities (bacteria & yeasts) **$200.00 or With PCR $250.00**

CLINICALLY: Ideal to identify the good and bad bugs or beneficial bacteria and pathogenic microbiota in the GUT. Parasites are measured in both visual and EIA technology.

Faecal DNA MULTIPLEX PCR - PCR detection and identification of 10 parasitic & bacterial organisms: Giardia intestinalis, Cryptosporidium, Dientamoeba fragilis, Entamoeba histolytica, Blastocystis hominis; Campylobacter spp, Salmonella spp, Shigella spp, Yersinia enterocolitica, Aeromonas spp. **$50.00**

CLINICALLY: ideal for the detected for 5 parasitic organisms and 5 bacterial organisms using an advanced DNA technology

---

Other Custom GIT markers for clinical utility:

Marker	Individual Pricing
Helicobacter Pylori	**$40.00**
M2-PK	**$40.00**
Calprotectin	**$40.00**
Transglutaminase IgA	**$40.00**
Pancreatic Elastase	**$40.00**

****Best Value****

Combination Pricing
1 Marker - **$40.00**
2 Markers - **$70.00**
3 Markers - **$105.00**
4 Markers - **$120.00**
5 Markers - **$150.00**

NutriPATH
Integrative Pathology Services

Stool Analyte Comparison Chart

	CDSA Level 1 Code 2003	CDSA Level 2 Code 2004	CDSA Level 3 Code 2005	CDSA Level 3+ Code 2006	CDSA Level 4 Code 2007	CDSA Level 4+ Code 2008	CDSA Level 5 Code 2009
MACROSCOPY Stool colour & form Mucous / Blood	✓	✓	✓	✓	✓	✓	✓
MICROSCOPY RBC, WBC, Food remnants, Fat globules, Starch	✓	✓	✓	✓	✓	✓	✓
DIGESTIVE MARKERS							
Chymotrypsin		✓	✓	✓	✓	✓	
Meat and Vege. fibres	✓	✓	✓	✓	✓	✓	✓
SCFA, putrefactive		✓	✓	✓	✓	✓	
Pancreatic elastase 1**			✓	✓	✓	✓	
ABSORPTION MARKERS							
Triglycerides (stool) Long chain fatty acids Cholesterol (stool) Phospholipids		✓	✓	✓	✓	✓	
METABOLIC MARKERS							
Beneficial SCFAs		✓	✓	✓	✓	✓	
pH	✓	✓	✓	✓	✓	✓	✓
Butyrate		✓	✓	✓	✓	✓	
Acetate		✓	✓	✓	✓	✓	
Proprionate		✓	✓	✓	✓	✓	
β-glucoronidase		✓	✓	✓	✓	✓	
INFLAMMATION MARKERS							
Transglutaminase IgA**				✓		✓	
Eosinophil Protein X				✓		✓	
Calprotectin **				✓		✓	
Bile acids - Lithocholic acid, Deoxycholic acid							
Lactoferrin, faecal							
TUMOUR/ULCER MARKERS							
M2 pyruvate kinase**				✓		✓	
H. pylori antigen **				✓		✓	
BACTERIOLOGY							
Bifidobacteria, Lactobacilli E. coli, Enterococci	✓	✓	✓	✓	✓	✓	✓
Klebsiella, Pseudomonas Campylobacter, Citrobacter, Yersinia, OTHER	✓	✓	✓	✓	✓	✓	✓
Candida, Yeasts	✓	✓	✓	✓	✓	✓	✓
Antibiotic/Natural Sensitivities	✓	✓	✓	✓	✓	✓	✓
PARASITOLOGY							
Cryptosporidium Giardia lamblia Entamoeba histolytica	✓	✓	✓	✓	✓+ EIA	✓+ EIA	✓+ EIA
Blastocystis hominis, OTHER	✓	✓	✓	✓	✓	✓	✓
PCR DNA- BACTERIA/PARASITES	**PCR ADD ON-$50.00 to any of the CDSA profiles offered by NutriPATH Pathology**						

CDSA Test Results Explained

These are explained in the format of:

Result range indication (Low, Normal, Elevated)

Explanation of the results (Suspect)

Recommendations of further diagnostics (Consider)

1. Digestive Markers

		Result	Suspect	Consider
Analyte, Related Profiles	**Chymotrypsin**	Low < 0.9 mcg/g	• Pancreatic insufficiency or hypochlorhdyria • Other factors include slow transit time	• Assess putrefactive SCFAs • Therapeutic Interventions: › Pancreatic enzyme supplementation and/or betaine HCL › Dietary fiber (insoluble) to improve transit time
		Normal 0.9-26.8 mcg/g 1 SD = 2.1-13.7	Adequate exocrine pancreatic function	1-2 SD = Results from 1-2 SD (yellow range) warrant clinical correlation even though within the "normal" reference range.
		Elevated > 26.8 mcg/g		Rule out false elevations from diarrhea (assess pancreatic elastase 1 levels) • Further Testing: › Faecal DNA PCR Code 2002 › SIBO Code 2025 • › Intestinal Permeability Code 2011 › IgG96 Foods Test Code 3206 › Coeliac Testing Code 2022
Analyte, Related Profiles	**Pancreatic Elastase 1 (PE1)**	Low 100-200 mcg/g	Mild to moderate pancreatic insufficiency	• Further Testing › Intestinal Permeability Code 2011 › Faecal DNA PCR Code 2002 › Coeliac Testing Code 2022 • Therapeutic Intervention › Pancreatic enzyme supplementation
		Very Low < 100 mcg/g	Moderate to severe pancreatic insufficiency	• Further Testing › N-Telopeptides Code 1218 › Insulin Resistance Index Code 1109 › Coeliac Testing Code 2022 › SIBO Code 2025 • Therapeutic Interventions › Pancreatic enzyme supplementation › Vitamin and mineral supplementation
		Normal > 200 mcg/g	Adequate exocrine pancreatic function	No further action necessary. Pancreatic supplementation may be of benefit in low normal (< 400 mcg/g) range
Analyte, Related Profiles	**Putrefactive Short-Chain Fatty Acids (SCFA's)**	Low < 1.3 micromol/g	Low protein diet	• Further Testing › Amino Acid Analysis Test Code 5004
		Normal 1.3-8.6 micromol/g 1 SD = 2.2-6.2	Adequate digestion and absorption of dietary protein	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
		Elevated > 8.6 micromol/g	• Hypochlorhydria, exocrine pancreatic insufficiency, or protein malabsorption • Other causes include bacterial overgrowth of the small bowel, gastrointestinal disease, and/or rapid transit time	Assess pancreatic elastase 1 to evaluate exocrine pancreatic function • Further Testing › SIBO Code 2025 › Helicobacter pylori Antibody Assessment Code 2010 • Therapeutic Interventions › Betaine HCL supplementation and/or pancreatic enzyme supplementation
Analyte, Related Profiles	**Meat Fibers/ Vegetable Fibers** NEG= 0 Rare= + Few= ++ Many =+++	Inside reference range • Meat › None • Vegetable fibers › None-Few	Adequate digestion and absorption of dietary protein (meat or fish) and vegetable fiber	Assess chymotrypsin and/or pancreatic elastase 1, putrefactive SCFAs
		Outside reference range • Meat › Rare-many • Vegetable fibers › Few-many	Pancreatic insufficiency, hypochlorhydria, inadeqate mastication, bile salt insufficiency	Assess chymotrypsin and/or pancreatic elastase 1, putrefactive SCFAs • Therapeutic Interventions › Pancreatic enzyme supplementation › Betaine HCL › Cholagogues

2. Absorption Markers

		Result	Suspect	Consider
Analyte, Related Profiles	**Triglycerides**	Low < 0.2 mg/g	Low dietary fat intake	Assess other markers of fat metabolism (LCFAs, phospholipids, cholesterol and fecal fat) • Further Testing › Essential & Metabolic Fatty Acid Analysis
		Normal 0.2-3.3 mg/g 1 SD = 0.4-1.7	Adequate fat hydrolysis	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
		Elevated > 3.3 mg/g	Incomplete fat hydrolysis • Rule out › Bile insufficiency › Reduced pancreatic function › High fat diet › Hypochlorhydria	Assess other markers of fat metabolism (LCFAs, phospholipids, cholesterol and fecal fat), chymotrypsin and/or pancreatic elastase 1 • Further Testing › Intestinal Permeability Assessment Code 2011 › Essential Fatty Acid Analysis Code 5011 • Therapeutic Interventions › Cholagogues, betaine HCL supplementation and/or pancreatic enzyme supplementation
Analyte, Related Profiles	**Long Chain Fatty Acids (LCFAs)**	Low < 1.3 mg/g	Low dietary fat intake	Assess other markers of fat metabolism (triglycerides, phospholipids, cholesterol and fecal fat) • Further Testing: › Essential Fatty Acid Analysis Code 5011
		Normal 1.3-23.7 mg/g 1 SD = 3.4-15.8	Adequate free fatty acid absorption	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
		Elevated > 23.7 mg/g	• Malabsorption • Increased mucosal cell turnover • Bacterial overgrowth of the small intestine • Bile insufficiency	Assess other markers of fat metabolism (triglycerides, LCFAs, phospholipids and fecal fat), chymotrypsin and/or PE1 • Further Testing: › SIBO Code 2025 › Intestinal Permeability Assessment Code 2011 › Essential Fatty Acid Analysis Code 5011 • Therapeutic Interventions: › Cholagogues, betaine HCL supplementation and/or pancreatic enzyme supplementation
Analyte, Related Profiles	**Cholesterol**	Low < 0.2 mg/g	Low dietary fat intake	Assess other markers of fat metabolism (triglycerides, phospholipids, cholesterol and fecal fat) • Further Testing: › Essential Fatty Acid Analysis Code 5011
		Normal 0.2-3.5 mg/g 1 SD = 0.4-2.0	Adequate free fatty acid absorption	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
		Elevated > 3.5 mg/g	• Malabsorption • Increased mucosal cell turnover • Bacterial overgrowth of the small intestine	Assess other markers of fat metabolism (triglycerides, LCFAs, phospholipids and fecal fat), chymotrypsin and/or pancreatic elastase1 • Further Testing: › SIBO Code 2025 › Intestinal Permeability Assessment Code 2011 › Essential Fatty Acid Analysis Code 5011 • Therapeutic Interventions: › Cholagogues, betaine HCL supplementation and/or pancreatic enzyme supplementation

2. Absorption Markers (cont.)

		Result	Suspect	Consider
Analyte, Related Profiles	**Phospholipids**	Low < 0.2 mg/g	• Insufficient dietary fat intake • Dietary phospholipid deficiency • Impaired gall bladder function	Assess other markers of fat metabolism (triglycerides, LCFAs, phospholipids and fecal fat), chymotrypsin and/or pancreatic elastase 1 • Further Testing › Essential Fatty Acid Analysis Code 5011 • Therapeutic Interventions: › Phosphatidyl choline (lecithin) › Phosphatidyl serine › Phosphatidyl inositol
		Normal 0.2-8.8 mg/g 1 SD = 0.4-4.7	Adequate dietary phospholipid intake and absorption	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
		Elevated > 8.8 mg/g	• Malabsorption • Reduced bile salt resorption • Increased mucosal cell turnover	Assess other markers of fat metabolism (triglycerides, LCFAs, cholesterol and fecal fat), chymotrypsin and/or pancreatic elastase 1, eosinophil protein X (EPX) and calprotectin • Further Testing › Intestinal Permeability Assessment Code 2011 › Essential Fatty Acid Analysis Code 5011 • Therapeutic Interventions › Cholagogues, betaine HCL supplementation and/or pancreatic enzyme supplementation
Analyte, Related Profiles	**Fecal Fat (Total)**	Low < 2.6 mg/g	Low dietary fat intake	Assess other markers of fat metabolism (triglycerides, LCFAs, cholesterol and phospholipids) • Further Testing › Essential Fatty Acid Analysis Code 5011
		Normal 2.6-32.4 mg/g 1 SD = 6.1-23	Adequate dietary fat absorption	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
		Elevated > 32.4 mg/g = + +	Malabsorption, increased mucosal cell turnover, bacterial overgrowth of the small intestine	Assess other markers of fat metabolism (triglycerides, LCFAs, cholesterol and phospholipids), chymotrypsin and/or pancreatic elastase 1, eosinophil protein X (EPX) and calprotectin • Further Testing › Intestinal Permeability Assessment Code 2011 › Essential Fatty Acid Analysis Code 5011 • Therapeutic Interventions › Cholagogues, betaine HCL supplementation and/or pancreatic enzyme supplementation

3. Metabolic Markers

		Result	Suspect	Consider
Analyte, Related Profiles	**Short-Chain Fatty Acids (SCFAs)**	Low < 13.6 micromol/g	Insufficient fiber Slow transit time Recent antibiotic therapy Low dietary fat intake	• Dietary and Therapeutic Interventions › Dietary fiber and resistant starch, prebiotics & probiotics, butyric acid (oral or rectal)
		Normal ≥ 13.6 micromol/g 1 SD 29.8	Suggests adequate energy for the colonocytes	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
Analyte, Related Profiles	**SCFA Distribution**	Inside reference range • Acetate › 44.5-72.4% • Proprionate › ≤ 32.1% • n-Butyrate › 10.8-33.5%	Adequate balance among anaerobic organisms in the colon	No further action necessary
		Outside reference range • Acetate › < 44.5% or › > 72.4% • Proprionate › > 32.1% n- • Butyrate › < 10.8 or › > 33.5%	Imbalance among anaerobic organisms in the colon. Elevated % recovery of acetate suggests an overgrowth of anaerobic flora, specifically *Clostridium*	Assess Bifidobacteria • Further Testing › *Clostridium dificile* EIA Code 2017
Analyte, Related Profiles	**n-Butyrate (as part of SCFAs)**	Low < 2.5 micromol/g	• Insufficient fiber • Slow transit time • Recent antibiotic therapy	• Dietary and Therapeutic Interventions › Dietary fiber and resistant starch, prebiotics and probiotics, butyric acid (oral or rectal)
		Normal ≥ 2.5 micromol/g 1 SD 5.6	Adequate energy for the colonocytes	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
Analyte, Related Profiles	**pH**	Low <6.3	Carbohydrate maldigestion or malabsorption, osmotic laxatives, rapid transit time, or small bowel bacterial overgrowth	• Further Testing › SIBO Code 2025 › Intestinal Permeability Assessment Code 2011 › Essential Fatty Acid Analysis Code 5011 • Therapeutic Interventions › Plant or pancreatic enzymes, betaine HCL and/or disaccharidases
		Normal 6.3-7.7 1SD=6.8-7.4	Balanced concentration between acids and bases witin the colon	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
		Elevated >7.7	• High protein and/or low fiber diet • Dysbiosis • Slow transit time • Hypochlorhydria • Increased bile flow rate • Pancreatic bicarbonate • Associated with increased risk for colorectal cancer	Assess putrefactive SCFAs • Dietary and Therapeutic Interventions › Reduce dietary fat and protein; increase fiber (particularly resistant starch) › Probiotic supplementation › Prebiotic supplementation

3. Metabolic Markers (cont.)

		Result	Suspect	Consider
Analyte, Related Profiles	**Beta-glucuronidase**	Low < 337 U/g	• Reduced enterohepatic recirculation and increased excretion of toxins, drugs, steroid hormones, and other compounds subject to glucuronidation • Rule out recent use of broadspectrum antibiotics	• Further Testing: › Liver Detoxification Profile Code 4010 › DetoxiGenomic™ Profile Code 8003
		Normal 337-4,433 U/g 1 SD = 647-2143	Balanced microbial activity from anaerobic organisms that produce this enzyme *(Bacteroides, Clostridia, E.coli, Peptostreptococcus)*	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
		Elevated > 4,433 U/g	Increased activation and enterohepatic recirculation of toxins, hormones, and various drugs within the body. Increased burden on glucuronidation pathway is associated with increased risk of colorectal, prostate and breast cancers	Assess stool pH (alkaline pH induces the activity of beta-glucuronidase) • Further Testing › Adrenocortex Profile Code 1001 › Estrogen Metabolites Level 2 Code 1308 › Female/Male Hormone Profile 1005/1007 › Thyroid Function Test Basic Code 1113 › Liver Detoxification Profile Code 4010 › DetoxiGenomic™ Profile Code 8003 • Dietary and Therapeutic Interventions: › Reduce fatty meat intake; increase insoluble dietary fiber › Probiotics (Lactobacilli and Bifodobacteria), Silybum marianum, calcium-D-glucarate
Analyte, Related Profiles	**Lithocholic: Deoxycholic Acid Ratio (LCA:DCA)**	Low < 0.39 mg/g	Imbalanced colonic ecology *(Clostridia, Bacteroides, Enterococcus,* and *Lactobacilli* modify primary bile acids intosecondary bile acids). Rule out recent broad spectrum antibiotic therapy	Acssess microbial ecology
		Normal 0.39-2.07 mg/g 1 SD = 0.66-1.55	Healthy ratio of secondary bile acids reflecting balance between dietary and endogenous cholesterol	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
		Elevated > 2.07	• Inhibition of glutathione-S-transferase with subsequent recirculation of procarcinogens. • Associated with increased risk of breast and colorectal cancer • Small bowel bacterial overgrowth, cholelithiasis and cholecystectomy	Consider the levels of calprotectin, betaglucuronidase, pH, n-butyrate and occult blood to assess overall neoplastic risk • Further Testing: › SIBO Code 2025 › Estrogen Metabolites Level 2 Code 1308 › DetoxiGenomic™ Profile Code 8003 • Dietary and therapeutic considerations: › Reduce fat intake; increase vegetable intake (beta-sitosterol); increase dietary fiber (insoluble fiber) › Probiotics *(Lactobacillius reuteri, Lactobacillius acidophilus)*

4. Immunology Markers

		Result	Suspect	Consider
Analyte, Related Profiles	**Eosinophil Protein X**	Normal ≤ 7.0 mcg/g 1 SD ≥ 1.0	No active inflammation of the GI tract, successful elimination diets	1-2 SD = Results from 1-2 SD may warrant clinical correlation even though within the "normal" reference range.
		Elevated > 7.0 mcg/g	Inflammation and/or tissue damage in the GI tract. This could be due to food allergy, protein sensitive enteropathy, helminthic infection, Inflammatory Bowel Disease (IBD), allergic colitis, or gastroesophageal reflux	Assess calprotectin levels Code 2001 • Further Testing › IgG Food Sensitivity Test Code 3206 › (Macroscopic Exam for Worms) › Coeliac Testing Code 2022 • Natural therapeutics to reduce inflammation › Probiotics, fish oils, N-acetylglucosamine › Anti-inflammatory agents such as the leukotriene inhibitors or TNF-alpha antagonists › Elimination Diet
Analyte, Related Profiles	**Calprotectin**	Mildly Elevated 50-100 mcg/g	Low-grade inflammation of the GI tract is present. This could be due to post-infectious Irritable Bowel Syndrome (IBS), infection, food allergies, polyps, neoplasia, nonsteroidal anti-inflammatory drugs (NSAIDs), or IBD (in remission)	Levels between 50-100 mcg/g require repeat testing in six weeks. If levels remain elevated after ruling out other etiologies, further investigative tests (endoscopic or radiographic) should be considered • Further Testing › Intestinal Permeability Assessment Code 2011 › IgG Food Sensitivity Test Code 3206 › Coeliac Testing Code 2022 • Therapeutic Interventions: › Probiotics , fish oils, N-acetylglucosamine, rutin
		Highly Elevated > 150 mcg/g	A value above 150 mcg/g indicates significant inflammation in the gastrointestinal tract. Possible causes include: Inflammatory Bowel Disease (IBD), infection, food allergies, NSAID use, polyps, adenomas, colorectal cancer, diverticulitis	• Unless source of inflammation is clear, further evaluation is recommended and may include endoscopy and/or colonoscopy • Assess microbiology/parasitology • Further Testing › Intestinal Permeability Assessment Code 2011 › IgG Food Sensitivity Test Code 3206 › Coeliac Testing Code 2022 • Therapeutic Interventions › Probiotics, fish oils, N-acetylglucosamine, rutin › Anti-inflammatory agents such as the leukotriene inhibitors or TNF-alpha antagonists
		Extremely Elevated > 250 mcg/g	In addition to the possible causes listed for calprotectin > 150 µg/g (see above): • In patients with Inflammatory Bowel Disease (IBD), levels > 250 indicate disease activity. Patients with IBD in remission who have levels > 250 mcg/g are at high risk of relapse within one year.	• In addition to the above recommendations for calprotectin > 150 mcg/g, the following is suggested: • Management of IBD with standard therapies, as directed by a qualified gastroenterologist when necessary • Therapeutic interventions › Probiotics, fish oils, N-acetylglucosamine, rutin › Anti-inflammatory agents such as the leukotriene inhibitors or TNF-alpha antagonists
Analyte, Related Profiles	**Lactoferrin** (ADD ON)	Negative	No acute inflammation	No further action necessary
		Positive	Significant mucosal inflammation from bacterial or parasitic infection, diverticulitis or active Inflammatory bowel disease (IBD)	Rule out enteric infection • Further Testing › Calprotectin Code 2001 › Eosinophil protein X › Intestinal Permeability Assessment Code 2011 • Therapeutic interventions › Probiotics, fish oils, N-acetylglucosamine, rutin Anti-inflammatory agents such as the leukotriene inhibitors or TNF-alpha antagonists

5. Microbiology Markers

		Result	Suspect	Consider
Analyte, Related Profiles	**Beneficial Bacteria**	Within normal levels *Lactobacilli* > 2+ *Bifidobacteria* > 4+	Suggests healthy levels of beneficial flora	No further action necessary
		Below normal levels *Lactobacilli* < 2+ *Bifidobacteria* < 4+ *E.coli* < 4+	Increased susceptibility to pathogenic bacterial infection, increased toxic enzyme exposure, increased risk for mucosal barrier defects and immune dysregulation	Assess SCFAs, beta-glucuronidase, pH and mycology • Further Testing › Intestinal Permeability Assessment Code 2011 › SIBO Code 2025 • Therapeutic Interventions › Probiotics (supplementation with *Lactobacilli* and *Bifidobacteria* to help balance deficient flora)
Analyte, Related Profiles	**OTHER BACTERIA**	Non-Pathogenic (NP) <4+	Organisms that constitute normal aerobic flora or commensal flora, and have not been recognized as etiological agents of disease	Although some organisms require no further action. Growth of certain organisms may still cause symptoms. Therefore, follow natural/prescriptive agents for treatment.
		Potential Pathogen (PP) 4+	Organisms that have the potential in certain hosts to be opportunistic pathogens	• If clinical symptoms persist in the absence of any other clearly defined infection, treatment may be considered • Refer to the **Pathogenic Organsim Chart*** for therapeutic recommendations
		Pathogen (P) >4+	Organisms that have the potential in certain hosts to be opportunistic pathogens	• Refer to the **Pathogenic Organsim Chart*** for therapeutic recommendations
Analyte, Related Profiles	**YEASTS**	Candida species	Organisms that may be involved in gastrointestinal symptoms	• Further Testing › Intestinal Permeability Assessment Code 2011 › SIBO Code 2025 › Candida Antibodies Code 3002 • Refer to the **Pathogenic Organsim Chart*** for clinical significance and therapeutic recommendations
		Yeast, not Candida Includes Cryptococcus, Geotrichum, and Rhodotorula species	Rare, opportunistic organisms usually isolated only in immunocompromised hosts	• Further Testing › Intestinal Permeability Assessment Code 2011 • Refer to the **Pathogenic Organsim Chart*** for clinical significance and therapeutic recommendations
Analyte, Related Profiles	**H. pylori Stool Antigen**	Negative	No active Helicobacter pylori infection, or successful eradication (after at least 7 days of treatment)	No further action necessary
		Positive	Active Helicobacter pylori infection or partially treated H. pylori infection (antibiotic failure/ resistant strain)	• Therapeutic Interventions › Triple or quadruple therapy - see the HpSA whitepaper*. Retest patient in 7-14 days for test-ofcure, particularly if symptoms continue › Consider addition of probiotics to improve treatment tolerance & effectiveness › Consider mastic gum and/or zinc carnosine as alternative/ adjunctive therapy › Refer to the **Pathogenic Organsim Chart*** for clinical significance and therapeutic recommendations

5. Microbiology Markers (cont.)

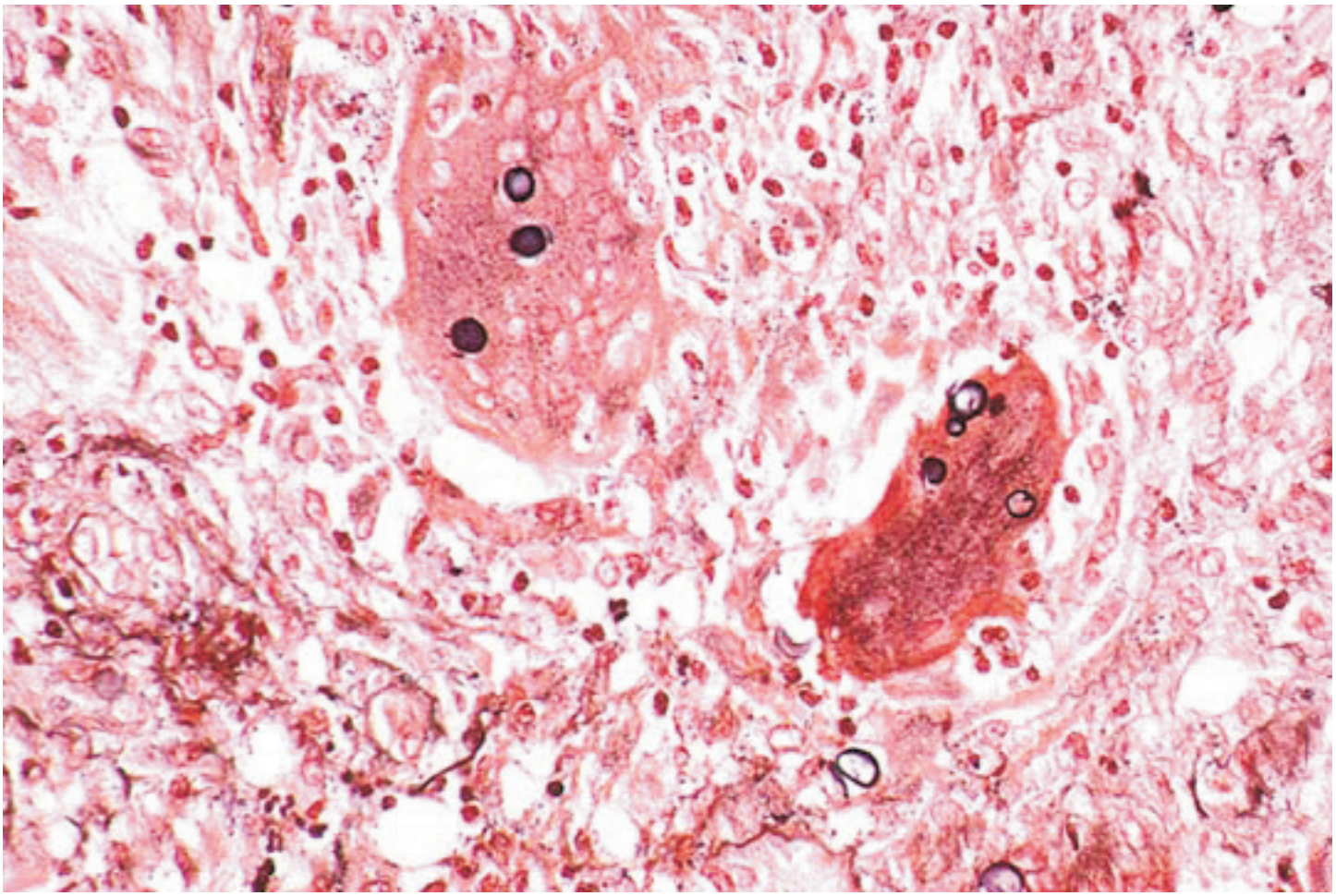
		Result	Suspect	Consider
Analyte, Related Profiles	**Shiga Toxin E.coli (STEC)**	Negative	No active infection	No further action necessary
		Positive	Active STEC infection	• Antibiotics are NOT effective (unless etiological role in cystitis or pyelonephritis) • Probiotics may help to prevent infection, but cannot nullify the effects of STEC once it has attached and released its toxin • Refer to the **Pathogenic Organsim Chart*** for clinical significance and therapeutic recommendations
Analyte, Related Profiles	**Campylobacter specific antigen**	Negative	No active infection	No further action necessary
		Positive	Active *Campylobacter* infection	• Infections are usually self-limiting and do not require antibiotic therapy. Patients with persistent diarrhea secondary to *Camplyobacter* infection require antibiotic therapy (erythromycin or ciprofloxacin are the preferred drugs of choice). Activated charcoal may decrease symptoms. • Refer to the **Pathogenic Organsim Chart*** for clinical significance and therapeutic recommendations
Analyte, Related Profiles	**Clostridium difficile Toxins A & B**	Negative	Absence of both toxins A and B, or an extremely low toxin level below the assay's detection limit	No further action necessary
		Positive	Active *Clostridium difficile* infection	• Oral vancomycin or metronidazole are the drugs of choice for severe infection, though disease relapse can occur • Probiotics such as *Lactobacillus rhamnosus* (GG), *Bifidobacterium bifidum,* and *Saccharomyces boulardii* may help prevent infection and/or the recurrence of *C.difficile* • Probiotics will NOT nullify the effects of C.difficile once the toxins have been released and the mucosal barrier has been compromised • Refer to the **Pathogenic Organsim Chart*** for clinical significance and therapeutic recommendations
Analyte, Related Profiles	**Occult Blood**	Negative	No hemoglobin detected in the stool	Rule out ingestion of vitamin C above 250 mg/day (inactivates test)
		Positive	Suggests abnormal amounts of hemoglobin from excessive blood loss. Suspect ulcers, polyps, diverticulitis or colorectal cancer	• Rule out false positive results from non-intestinal sources of bleeding (hemorrhoids, menstruation, hematuria) or use of rectal suppositories, oral medications, including aspirin and corticosteroids • Repeat positive results should be followed up with other diagnostic procedures such as protosigmoidoscopic examination, full colonoscopy, barium enema, or other examinations

5. Microbiology Markers (cont.)

		Result	Suspect	Consider
Analyte, Related Profiles	**PARASITES**	Positive	Parasite infection and Dysbiosis	Assess calprotectin, EPX and/or Lactoferrin • Further Testing › Intestinal Permeability Assessment Code 2011 › IgG Food Sensitivity Test Code 3206 › Adrenocortex test Code 1001 › Extensive Neurotransmitters Code 4026 • Refer to the **Parasitic Organism Chart*** for clinical significance and therapeutic recommendations

If symptoms persist after routine parasitology investigations have yielded negative results for parasites, we suggest faecal Multiplex DNA PCR testing Code 2002 . This is a more sensitive technique that can identify the presence of specific DNA of 10 commonly observed parasites/bacteria.

Bacterial Pathogenic Organism Chart



In this section you will find the following about each organism:

- Description
- Habitat/Source of Isolation
- Pathogenicity
- Common Symptoms
- Possible Treatment Options

Pathogenic Organism Chart

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Aeromonas (PP):	*Aeromonas* is a gram-negative rod belonging to the *Vibrionaceae* family. There are at least four species of *Aeromonas* with *A. hydrophilia* being the most common isolated species in the U.S.[1] [2]	Aeromonads are ubiquitous in fresh water environments. The number present is dependant on the extent of sewage pollution and the ambient temperature. Recent studies have directly attributed *Aeromonas* as the cause of food-borne infections. The following foods may harbor the organism: raw meat, freshwater fish, shellfish and other seafood. Raw milk can also be a source of infection.[3]	Definitive experimental evidence for the causative role of *Aeromonas* in gastrointestinal disorders is still lacking. Although human volunteer studies are inconclusive, epidemiological evidence has shown that the presence of these organisms in stools is significantly more often associated with diarrhea than with the carrier state.[4] [5]	*Aeromonas* gastroenteritis may affect both children and adults with the highest seasonal incidence occurring in the summer months. Symptoms tend to be generally mild, self-limiting diseases with watery diarrhea.[6] Bloody stools have been reported. *Aeromonas* infections tend to be more acute in children and more chronic in adults.[7] [8]	Most *Aeromonas species* are generally susceptible to cephalosporins, aminoglycosides, carbapenems, tetracyclines, trimethoprim-sulfamethoxazole and quinolones.[9] Susceptibility must guide testing.
**Aeromonas hydrophilia/caviae*					
**Aeromonas veronii biovar sobria*					
**Aeromonas biovar veronii*					
**Aeromonas species*					

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Bacillus (PP):	*Bacillus species* are spore forming, gram-positive rods belonging to the *Bacillaceae* family.[10] [11] There are currently 50 valid species within the genus.[12]	Sources of the diarrheal type of *B. cereus* food poisoning include: meats, pasta, vegetable dishes, desserts, cakes, sauces and milk.[13] The emetic type of infection is predominately associated with oriental rice dishes. Pasteurized cream, milk pudding and pasta have occasionally been implicated.[14] The incidence of *B. cereus* infection is increased during the summer months.[15]	Although part of the normal flora, *B.cereus* has been established as an opportunistic pathogen.[16] The gram-positive spore forming rods of *B.cereus* elaborate enterotoxins.[17] Both types of food poisoning result from spores that have survived cooking, then germinated, producing vegetative cells that have multiplied.[18] *NB, it is estimated that only half the isolated strains of B. cereus are enterotoxin positive.*[19]	*B. cereus* is the etiological agent of two distinct types of food poisoning: 1) The diarrheal type, which is caused by a heat-labile enterotoxic complex. Symptoms include abdominal pain, and diarrhea 8-12 hours after ingestion of the organism.[20] [21] 2) The emetic type, caused by a heat-stable enterotoxin. Nausea and vomiting usually occur 1-5 hours after ingestion.[22] [23]	*B. cereus* is almost always susceptible to clindamycin, erythromycin and vancomycin.[24]
**Bacillus cereus*					
**Bacillus species*		Meat dishes are a common source of infection in other species of *Bacillus* such as *B. subtilis* and *B. licheniformis*.[25]	As yet, no toxins or other virulence factors have been identified in association with the symptoms that accompany non-*B. cereus species*.[26]	*B. licheniformis* and *B. subtilis* are associated with food-borne diarrheal illness.[27]	

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Campylobacter jejuni (P)	*Campylobacter* are gram-negative, non-spore forming rods belonging to the *Campylobacterace ae* family.[28] In total there are 18 species and subspecies within the genus.[29]	Poultry is a key source of infection, in particular chicken. Red meat and shellfish can also harbor the organism.[30] Other sources include unpasteurized milk, and water contaminated by wild birds.[31]	Recognized as the principle cause of diarrhea in humans. *C. jejuni* and *C. coli* are the most common species associated with diarrheal illness.[32] The infective dose as yet has not been clearly defined, but it is thought that as little as 1000 organisms are capable of causing infection.[33]	The incubation period can be 2 to 10 days, though is usually 2 to 5 days.[34] Symptoms can include fever, abdominal cramping, diarrhea (often bloody) abdominal pain and fever. Relapses may occur in 5%-10% of untreated cases.[35]	Erythromycin is the drug of choice for treating *C. jejuni* infections. Ciprofloxacin may be an alternative drug.[36]

Pathogenic Organism Chart

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Candida (PP): **Candida albicans* **Candida famata* **Candida glabrata* **Candida guilliermondii* **Candida krusei* **Candida lambica* **Candida lusitaniae* **Candida parapsilosis* **Candida paratropicalis* **Candida pseudotropicalis* **Candida rugosa* **Candida species* **Candida stellatoidea* **Candida tropicalis* **Candida zeylanoides*	The genus *Candida* is comprised of approximately 200 different species.[37] *C. albicans* is the most commonly isolated strain from the GI tract.[38]	Most sources of Candida infection are thought to be of endogenous origin.[39] While yeast are ubiquitous in the environment and are found on fruits, vegetables and other plant materials, contamination from external sources is linked to patients and health care workers.[40 41]	A normal inhabitant of the GI tract. May become an opportunistic pathogen after disruption of the mucosal barrier, imbalance of the normal intestinal flora and/or impaired immunity.[42 43 44 45] Risk factors for colonization include: Antibiotics, corticosteroids, antacids, H2 blockers, oral contraceptives, irradiation, GI surgery, Diabetes mellitus, burns, T cell dysfunction, chronic stress and chronic renal disease.[46 47 48 49]	The most common symptom attributable to non-invasive yeast overgrowth is diarrhea.[50] Symptoms of chronic candidiasis affect four main areas of the body: **Intestinal system** – symptoms include: diarrhea, constipation, abdominal discomfort, distention, flatulence and rectal itching. **Genital Urinary system** – symptoms include: menstrual complaints, vaginitis, cystitis and urethritis. **Nervous system** – symptoms include: severe depression, extreme irritability, inability to concentrate, memory lapses and headaches. **Immune system** – symptoms include urticaria, hayfever, asthma, and external otitis. Sensitivities to tobacco, perfumes, diesel fumes and other chemicals.[51 52]	Currently, standard texts provide no specific antifungal guidelines for GI overgrowth of *Candida*. Oral azoles have been recommended for extra intestinal infections. Susceptibility testing is advised due to increasing drug resistance.[53 54]

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Citrobacter (PP): **Citrobacter amalonaticus* **Citrobacter braakii* **Citrobacter diversus* **Citrobacter freundii* **Citrobacter freundii/youngae* **Citrobacter freundii complex* **Citrobacter koseri* **Citrobacter species*	*Citrobacter* is a gram-negative rod belonging to the *Enterobacteriaceae* family.[55] Citrobacter contains 9 named species and two unnamed genomospecies.[56]	Common in the environment and may be spread by person-to person contact. Several outbreaks have occurred in babies in hospital units.[57 58] Isolated from water, fish, animals and food.[59]	*Citrobacter* is considered an opportunistic pathogen and therefore can be found in the gut as part of the normal flora.[60]	*Citrobacter* has occasionally been implicated in diarrheal disease, particularly *C. freundii* and *C. diversus* and *C. koseri*.[61]	Currently, standard texts provide no specific antimicrobial guidelines for GI overgrowth of *Citrobacter*.[62 63] Carbapenems and fluroquinolones are the recommended antibiotics for extra-intestinal sites.[64 65]
Clostridium difficile (PP)	The genus *Clostridium* are anaerobic gram-positive, spore-forming bacteria.[66]	The organism has many natural habitats including hay, soil, cows, horses and dogs.[67] Almost 50% of neonates carry this organism asymptomatically as part of their gastrointestinal flora during the first year of life. This rate decreases sequentially to about 3% in adults and less in children over two years of age.[68 69]	*C. difficile* is the major cause of antibiotic-associated diarrhea and pseudomembranous colitis and the most common cause of hospital-acquired diarrhea.[70] Isolation of *C. difficile* without a positive toxin test has little clinical value. It is important to test for both toxins A and B in the stool. Toxin A is an enterotoxin and toxin B is a cytotoxin that inhibits bowel motility. It is thought that both toxins are important in the pathogenesis.[71 72]	Mild cases of *C. difficile* disease are characterized by frequent, foul-smelling, watery stools. More severe symptoms, indicative of pseudomembranous colitis, include diarrhea that contains blood and mucous, and abdominal cramps.[73]	Severe *C. difficile* intestinal disease is usually treated with oral vancomycin or metronidazole. However, antimicrobial therapy often results in relapse of the disease.[74] In addition, there is concern that oral vancomycin can lead to the emergence of vancomycin-resistant *Enterococci*.[75]

Pathogenic Organism Chart

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Cryptococcus (PP): **Cryptococcus albidus* **Cryptococcus humicolus* **Cryptococcus laurentii* **Cryptococcus luteolus* **Cryptococcus neoformans* **Cryptococcus species* **Cryptococcus terreus* **Cryptococcus uniguttulatus*	*Cryptococcus* is a yeast-like fungus, which closely resembles the genus *Candida*.[76] The genus contains a number of species, of which only *C. neoformans* is considered to be a human pathogen.[77]	Found in the excreta of pigeons and other birds in most parts of the world. The yeast is associated with aged bird droppings that have accumulated over a long period of time on window ledges, vacant buildings and other roosting sites.[78]	Can be an opportunistic pathogen, predominately in the immunocompromised host.[79] *Cryptococcus* is considered one of the defining diseases of AIDS. Patients with *Cryptococcus* and serologic evidence of HIV are considered to have AIDS.[80]	Diarrhea has been associated with Cryptococcal infection.[81] Usually infection occurs in the tissue of the central nervous system but occasionally can produce lesions in the skin, bones, lungs, or other internal organs.[82]	Currently, standard texts provide no specific antimicrobial guidelines for GI overgrowth of *Cryptococcus*.[83 84] Fluconazole is considered the primary antimicrobial agent in extraintestinal sites.[85]
Edwardsiella tarda(P)	The genus *Edwardsiella* is a gram-negative rod that belongs to the *Enterobacteriaceae* family.[86] To date there are three species, though only *E. tarda* is associated with human disease.[87]	Isolated from cold-blooded animals such as fish and reptiles and their environment.[88] Infection is more common in tropical and subtropical environments and developing countries.[89]	*E. tarda* is considered an opportunistic pathogen, occasionally causing acute gastroenteritis.[90 91]	Diarrheal disease is associated with infection, with a clinical picture similar to *Salmonella* enteritis.[92] Isolation of the *E. tarda* is more common in young children and the elderly.[93]	If antibiotic treatment is required, ampicillin, trimethoprim-sulfamethoxazole and ciprofloxacin have all been found to be effective agents.[94]

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
E.coli Shiga-like toxin	Shigatoxin-producing *E. coli* strains are referred to as STEC. This includes the 0157 and many other STEC serogroups.[95] **In total, at least 100 serotypes have been isolated from persons with diarrhea.[96]**	0157 STEC colonize dairy and beef cattle, which is why ground beef is the most common infection vehicle. However, raw milk, sausage, roast beef, unchlorinated water, apple cider, and raw vegetables have also been implicated.[97]	*E. coli* 0157:H7 and 0157:non-motile (0157 STEC) produce one or more Shiga toxins and, are the most commonly identified diarrheagenic *E.coli* isolates in North America and Europe.[98] Non-toxin-producing strains are normal in the human intestine. 0157 STEC spreads easily from person to person because the infectious dose is low.[99]	The STEC strains cause a spectrum of illness that can present as mild non-bloody diarrhea, severe bloody diarrhea (hemorrhagic colitis), and hemolytic uremic syndrome (HUS).[100] About 6% of 0157 STEC patients develop HUS.[101]	Antimicrobial therapy for 0157 STEC has NOT been demonstrated to be effective or safe, except for cases of cystitis and pyelonephritis.[102] Antimicrobial therapy for intestinal disease may enhance toxin release and predispose for HUS.[103]
Enterobacter cloacae (PP)	Gram-negative rod that is part of the *Enterobacteriaceae* family.[104] There are 14 species in the genus, though only *E. cloacae* has been associated with GI infection.[105 106]	Widely distributed in the environment. Water, soil, sewage and cornstalks have all been identified as sources of contamination.[107 108]	Usually considered a commensal organism; however, strains of *E. cloacae* have been shown to produce a heat-stable toxin similar to that produced by *E.coli*.[109]	Has been associated with diarrhea in children.[110 111]	Currently, standard texts provide no specific antimicrobial guidelines for GI overgrowth of *Enterobacter*.[112 113] Carbapenems are recommended for extra-intestinal sites.[114]
Geotrichum (PP): **Geotrichum candidum* **Geotrichum capitum* **Geotrichum species*	*Geotrichum* are yeast belonging to the *Endomyceteaceae* family. There are several species within the genus, of which *G. candidum* is the most common.[115]	This organism can be found in soil, dairy products and in human skin and mucosae.[116]	Usually only considered an opportunistic pathogen in immune-compromised hosts.[117 118] *Geotrichum candidum* is the etiological agent of Geotrichosis.[119] *Geotrichum* may also play a role in IBS.[120]	Symptoms of *Geotrichum* infection have been associated with diarrhea and enteritis.[121 122] Symptoms of Geotrichosis may resemble those of candidiasis.[123]	Currently, standard texts provide no specific antifungal guidelines for GI overgrowth of *Geotrichum*. Oral azoles and have been recommended for extra intestinal infections. Susceptibility testing is advised owing to increasing drug resistance.[124 125]

Pathogenic Organism Chart

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Hafnia alvei (PP)	*Hafnia* is a gram-negative rod considered part of the *Enterobacteriaceae* family. There is only one species of Hafnia– *H. alvei*–which was previously a member of the *Enterobacter* genus.[126]	Commonly found in warm-blooded animals, particularly birds. Other environmental sources include contaminated water, sewage, food, and dairy products.[127]	This organism is a natural inhabitant of the GI tract in humans. *Hafnia* strains are opportunistic pathogens; community and hospital outbreaks have been associated with GI infection.[128]	Diarrheal illness has been associated with outbreaks and virulence factors similar to toxigenic *E.coli* have been described.[129]	*Hafnia* strains are usually susceptible to piperacillin, imipenum, quinolones and the newer cephalosporins.[130]
Helicobacter pylori (P)	The genus *Helicobacter* are gram-negative, non-spore forming rods. There are currently 19 species within the genus.[131] Seroprevalence of *H. pylori* varies from 20% in young adults in developed countries to sometimes more than 90% in developing countries.[132]	Reservoirs of infection include the intestinal tract of mammals and birds. Mode of transmission is usually via the fecal-oral or oral-to-oral route.[133]	*H. pylori* causes chronic gastritis and predisposes to gastric and duodenal ulcers. Increased risk of gastric carcinoma is associated with infection.[134] It is estimated that 50% of the world's population is infected with *H. pylori*.[135]	Those infected with *H. pylori* may develop acute gastritis with symptoms of abdominal pain, nausea and vomiting, usually within two weeks of infection. Many patients have recurrent abdominal symptoms (non-ulcer dyspepsia) without ulcer disease.[136]	Cure rates require multi-drug regimens along with antacid medications.[137] The most successful treatment includes a combination of metronidazole, omeprazole and clarithromycin.[138]

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Klebsiella (PP): **Klebsiella ornithinolytica* **Klebsiella oxytoca* **Klebsiella ozaenae* **Klebsiella pneumoniae* **Klebsiella rhinoscleromatis* **Klebsiella species*	*Klebsiella* is part of the *Enterobacteriaceae* family and as such is a gram-negative rod.[139] There are 7 species of *Klebsiella* within the genus, though only 2 have been associated with GI infection.[140]	Isolated from foods and environmental sources.[141] *Klebsiella* appears to thrive in individuals on a high starch diet. Avoiding carbohydrates such as rice, potatoes, flour products and sugary foods reduces the amount of *Klebsiella* in the gut.[142]	Part of the normal GI flora in small numbers, but can be an opportunistic pathogen. *Klebsiella* is capable of translocating from the gut when in high numbers.[143 144] Certain strains of *K. oxytoca* have demonstrated cytotoxin production.[145 146 147 148 149] Of the 77 *Klebsiella* capsular polysaccharides, only 3 are associated with ankylosing spondylitis: K26, K36 and K50.[150 151]	*K. pneumoniae* and *K. oxytoca* have been associated with diarrhea in humans.[152 153 154 155 156 157] Cytotoxin-producing strains are associated with acute hemorrhagic enterocolitis.[158 159 160 161 162] Increased colonization of *Klebsiella* in the stool has been found in HLA-B27 + AS patients.[163 164]	Currently, standard texts provide no specific antimicrobial guidelines for GI overgrowth of *Klebsiella*.[165 166] Third generation cephalosporins and fluroquinolones are the recommended antimicrobial agents for extra-intestinal sites.[167]

Pathogenic Organism Chart

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Listeria monocytogenes (PP)	The genus *Listeria* are gram-positive cocciod- to rod-shaped bacteria of which there are 7 species in total.[168] The only species associated with infection in humans is *L. monocytogenes*.[169]	Dairy products are sources of *Listeria* infection. The organism has been found in raw milk, pasteurized milk, cream, butter, cheese and ice cream.[170] The use of manure as fertilizers on salad and vegetable crops have been associated with *Listeria* infection.[171] Fish and seafood may also be a reservoir of infection.[172]	GI symptoms have been associated with infection, though are not usually related to the ingestion of contaminated food.[173] A transient intestinal carrier state exists in 2%-20% of humans.[174] Development of an invasive infection depends on several factors, namely: host susceptibility, gastric acidity and the virulence of the organism.[175]	Symptoms of diarrhea have been noted with *Listeria* infection.[176]	*Listeria* is usually susceptible to penicillin, ampicillin, gentamycin, erythromycin, and tetracycline.[177]
Moellerella wisconsensis (PP)	*Meollerella* is a gram-negative rod that is part of the *Enterobacteriaceae* family.[178] Currently, there is only one species in the genus.[179]	Contaminated water supplies are the main reservoir of infection.[180]	The exact role of *Moellerella* in causing diarrhea has not yet been fully elucidated.[181]	Diarrhea and gastroenteritis have been associated with *M. wisconsensis*.[182 183]	Currently, standard texts provide no specific antimicrobial guidelines for GI overgrowth of *Moellerella*.[184 185] MIC studies have demonstrated susceptibility to cephalothin, gentamicin and naladixic acid.[186]

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Morganella morganii (PP)	*Morganella* is gram-negative rod belonging to the *Enterobacteriaceae* family.[187] Currently, there are 3 species within the genus.[188]	*M. morganii* originates from the gill and skin of fish. It is possible that it may cross-contaminate during handling of fish in processing plants and restaurants.[189]	The role of *Morganella* as an etiological agent in diarrheal disease is controversial. Although *Morganella* constitutes part of the normal flora, in certain hosts it may be a potential pathogen.[190 191] Recently it was shown that the majority of clinical isolates of *Morganella* belonged to the *subsp Morganii*.[192]	Diarrhea has been associated with infection of this organism.[193 194]	Currently, standard texts provide no specific antimicrobial guidelines for GI overgrowth of *Morganella*.[195 196] Carbapenems, 3rd and 4th generation cephalosporins and fluroquinolones are the agents recommended for extra-intestinal infections.[197]
Plesiomonas shigelloides (PP)	*Plesiomonas* is a gram-negative rod belonging to the *Vibrionaceae* family, though it does contain the *Enterobacteriaceae* antigen. *P. shigelloides* is the only species in the genus.[198 199]	Usually found in fresh water or estuarine water. Occurs in fish, shellfish, oysters, toads, snakes, monkeys, dogs, cats, goats, pigs, poultry, and cattle.[200] There is a low incidence of *Plesiomonas shigelliodes* in the US and Europe.[201] In Asia, however, the organism contributes to a significant proportion of traveler's diarrhea.[202 203]	*P. shigelloides* is not a natural inhabitant of the GI tract.[204] Although feeding studies with humans resulted in the excretion of the organism (but not diarrhea) from about one third of the volunteers, several epidemiological studies suggest that *Plesiomonas* is a possible agent in GI disease. It has been isolated from human stool specimens in the absence of symptoms and may be difficult to attribute as the cause of diarrhea in some cases.[205]	Symptoms range from short-lived episodes of watery stools to several days of dysentery-like diarrhea. Has not been reported to affect specific age groups more often than others.[206 207] Accompanying symptoms vary and may include abdominal pain, nausea, vomiting, chills, headaches and dehydration..[208] Infections with *P. shigelloides* are usually self-limiting, lasting up to 7 days and occasionally longer.[209 210]	*P. shigelliodes* is susceptible to most major classes of antibiotics, including trimethoprim, cephalosporins, and quinolones.[211]

Pathogenic Organism Chart

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Proteus (PP): **Proteus mirabilis* **Proteus penneri* **Proteus vulgaris*	*Proteus* is a gram-negative rod belonging to the *Enterobacteriaceae* family.[212] 10 species in total are attributed to the genus of which *P. mirabilis* is considered the most important.[213]	Food has been implicated as a vehicle of infection.[214]	Part of the normal flora of the GI tract, though has been shown to be an independent causative agent of intestinal disorders.[215] May also play a role as an opportunistic organism in enteric infection due to other pathogens.[216]	Occasionally implicated in diarrheal disorders.[217 218] Recently, it has been suggested that *P. mirabilis* may be an etiological agent in rheumatoid arthritis. The mechanism may be related to the molecular cross reactivity between *P. mirabilis* and the HLA antigens, specifically HLA-DR4.[219]	Currently, standard texts provide no specific antimicrobial guidelines for GI overgrowth of *Proteus*.[220 221] Ampicillin is recommended for extra-intestinal infections of *P. mirabilis*, followed by trimethoprim-sulfamethoxazole.[222]
Provedencia alcalifaciens (PP)	*Provedencia* is a member of the *Enterobacteriaceae* family of which there are 5species.[223 224]	GI tract infection with *P. alcalifaciens* has been associated with overseas travel.[225]	*Provedencia* is not normally present in a healthy GI tract.[226] Its pathogenic role may lie in the ability of the organism to take advantage of conditions created by other infectious microbes.[227]	This organism has been implicated as a cause of diarrhea.[228 229] *P. alcalifaciens* is thought to induce invasive diarrhea in patients by invading cells in the intestine, thus producing inflammatory changes in the ileum.[230]	Currently, standard texts provide no specific antimicrobial guidelines for GI overgrowth of *Providencia*.[231 232] 3rd generation cephalosporins and fluroquinolones are recommended for extra-intestinal sites.[233 234]

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Pseudomonas (PP): **Pseudomonas aeruginosa* **Pseudomonas species*	*Pseudomonas species* are aerobic, non-spore forming gram-negative rods.[235] There are 10 species in the genus, though *P. aeruginosa* is considered the most important pathogen.[236]	Found in water and soil as well as fruits and vegetables. Bottled water can be a common source of infection. Because the organism is able to survive aqueous environments, it is an important nosocomial pathogen. *Pseudomonas* can also be found on a number of surfaces and in aqueous solutions.[237]	*Pseudomonas* is considered an opportunistic pathogen.[238] Animal studies have isolated an enterotoxin thought to be responsible for causing diarrhea.[239]	Associated with diarrheal infection, particularly in the immunocompromised host.[240 241] *Pseudomonas* can also be an etiological agent of antibiotic-associated diarrhea.[242]	Ciprofloxacin is recommended for the treatment of *Pseudomonas*-induced antibiotic-associated colitis.[243] *Pseudomonas* is usually susceptible to antipseudomonal penicillins, aminoglycosides, carbapenems, 3rd generation cephalosporins and gentamycin.[244 245]
Saccharomyces cerevisiae (PP)	*Saccharomyces* are yeast belonging to the *Sacccharomycetaceae* family. Currently there are 18 species within the genus of which *S. cerevisiae* is the most common.[246 247]	*S. cerevisiae* is a commonly used industrial microorganism and is ubiquitous in nature, being present on fruits and vegetables. Also known as Baker's Yeast or Brewer's Yeast, this organism has been used for centuries as leavening for bread and as a fermenter of alcoholic beverages.[248 249]	*S. cerevisiae* commonly colonizes mucosal surfaces, and is rarely considered an opportunistic pathogen.[250 251 252] Severe immunosuppression, prolonged hospitalization, and antibiotic therapy are all associated with*Saccharomyces* infection..[253] Overgrowth may be associated with dietary ingestion of *S. cerevisiae* and/or *S. boulardii* as part of a "health food" regimen.	Studies have shown that patients with *S. cerevisiae* overgrowth usually have an underlying disease.[254] Disseminated infections are thought to arise from the gastrointestinal tract.[255]	Currently standard texts provide no specific antifungal guidelines for GI overgrowth of *Saccharomyces*.

Pathogenic Organism Chart

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Salmonella (P):	*Salmonella* are members of the *Enterobacteriaceae* family and as such are gram-negative rods.[256]	Animals and birds utilized for meat are subject to contamination with *Salmonella*. Eggs, cereals and cereal products are other sources of contamination.[257] The incidence of infection increases over the summer, and is predominantly associated with acute diarrhea in infants.[258]	*Salmonella* are considered frank pathogens in humans. These organisms are NOT part of the normal bowel flora. *Salmonella species* are acid-sensitive, invasive, and produce enterotoxins in the GI tract. Several thousand cells may be needed to cause infection.[259]	Gastroenteritis and diarrhea are caused by more than 2000 serotypes producing infections limited to the mucosa and submucosa of the GI tract. *S. typhimurium* and *S. enteritidis* are the serotypes most common in the US. Bacteremia and extraintestinal infections occur by spread from the GI tract, and any serotype is capable of causing bacteremia.[260] [261]	Antimicrobial therapy is not recommended for uncomplicated *Salmonella* gastroenteritis.[262] Antimicrobial therapy is warranted in cases of bacteremia. Enteric fever (typhoid fever) is characterized by prolonged fever and multisystem involvement. This is a life-threatening infection caused by *S. typhi* or *S. paratyphi*. Antimicrobial therapy is needed in cases of typhoid fever.[263]
**Salmonella Group C and D*					
**Salmonella arizonae*					
**Salmonella group A*					
**Salmonella group B*					
**Salmonella group C*					
**Salmonella group D*					
**Salmonella group E*					
**Salmonella group E + G*					
**Salmonella paratyphi A*					
**Salmonella paratyphi B*					
**Salmonella paratyphi C*					
**Salmonella species*					
**Salmonella typhi*					
Serratia marcesens (PP)	*Serratia* is a gram-negative rod belonging to the *Enterobacteriaceae* family.[264]	*Serratia* is more often associated with nosocomial infection, and seldom occurs in the community. The most common route of transmission is hand-to-hand spread via nurses, physicians and other healthcare workers.[265]	A natural inhabitant of the GI tract, though on occasion can become an opportunistic pathogen.[266]	In neonates the gastrointestinal system is an important source of the organism.[267]	Currently, standard texts provide no specific antimicrobial guidelines for GI overgrowth of *Serratia*.[268] [269] Third generation cephalosporins, carbapenems, and fluroquinolones are the recommended antibiotics for extra-intestinal infections.[270]

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
***Shigatoxin-producing E.coli (STEC)-* See E.coli Shiga-like toxin.**					
Shigella (P):	*Shigella* are members of the *Enterobacteriaceae* family. There are 4 serogroups that have historically been treated as species: -*S. dysenteriae* (Serogroup A) -*S. flexneri* (Serogroup B) -*S. boydii* (Serogroup C) -*S. sonnei* (Serogroup D).[271]	Spread from person to –person by the fecal-oral route, especially in overcrowded areas and areas with poor sanitary conditions. Ingestion is also a primary source of infection.[272] A predominant organism responsible for acute diarrheal disease in infants and children.[273]	*Shigella* is only found in humans at times of infections and is NOT part of the normal bowel flora. All species are considered frank pathogens in humans.[274]	Symptoms can range from mild to explosive diarrhea. It is somewhat acid-resistant, invades epithelial cells, and produces toxins. Less than 100 cells are required to initiate infection.[275] [276] *S. dysenteriae* is rare in the US and causes classic dysentery, producing the Shiga toxin. *S. sonnei* is most common in the US, and usually produces only a watery diarrhea.[277]	*Shigella* infections are often treated with antibiotics, and antimicrobial susceptibility testing is recommend owing to widespread resistance.[278] Resistant strains are usually susceptible to the fluroquinolones.[279]
**Shigella boydii*					
**Shigella dysenteriae*					
**Shigella flexneri*					
**Shigella sonnei*					
**Shigella species*					

Pathogenic Organism Chart

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Staphylococcus aureus (PP)	Members of the genus *Staphylococcus* are gram-positive cocci. Currently, the genus is composed of 32 species and 15 subspecies.[280]	Foods that require considerable handling during preparation or that are kept at slightly elevated temperatures after preparation are frequently involved in staphylococcal food poisoning. The key foods associated with staphylococcal food poisoning include meat and meat products; poultry and egg products; salads such as egg, tuna, chicken, potato, and macaroni; bakery products such as cream-filled pastries, cream pies, and chocolate éclairs; sandwich fillings; and milk and dairyproducts.[281 282]	Food poisoning is often attributed to the staphylococcal enterotoxin.[283] The toxin produced by the bacteria is very heat-stable and therefore not easily destroyed by heat at normal cooking temperatures. The toxin can remain, despite the organism being destroyed.[284] There is considerable variation in susceptibility to the enterotoxin in adults. Children and the elderly have the highest degree of susceptibility.[285]	Symptoms of staphylococcal food poisoning usually appear within 1 to 6 hours after ingestion. The individual response to the toxin may vary and depends upon the amount of contaminated food eaten, the amount of toxin ingested, and general health status.[286] Nausea, vomiting, abdominal cramping, and diarrhea are the most common symptoms. In more severe cases, headache, muscle cramping, and changes in blood pressure and pulse rate may occur.[287] Recovery generally takes two days. It is not unusual for complete recovery to take three days and sometimes longer.[288 289]	In most cases, treatment for *S. aureus* infection is not necessary and complete recovery usually occurs after cessation of symptoms.[290]

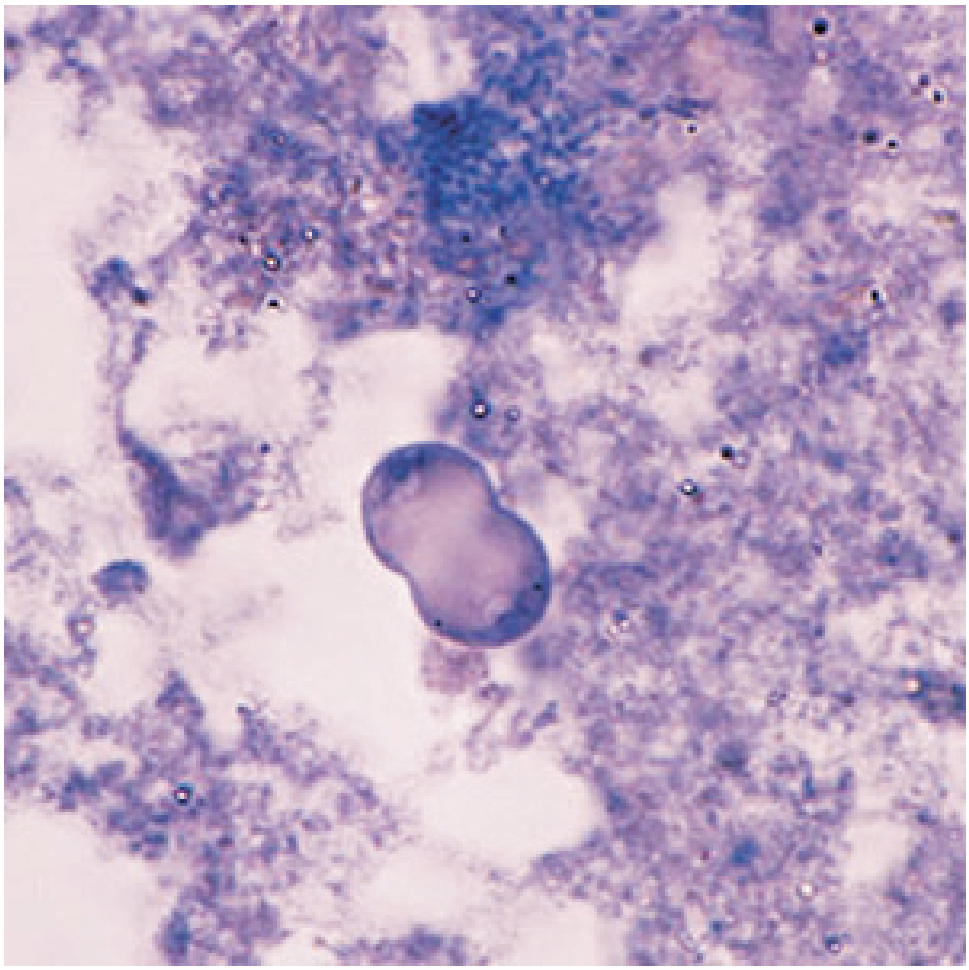
Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Vibrio (PP): **Vibrio cholerae* **Vibrio fluvialis* **Vibrio furnissii* **Vibrio hollisae* **Vibrio metschnikovii* **Vibrio mimicus* **Vibrio parahaemolyticus* **Vibrio species*	*Vibrio* are members of the *Vibrionaceae* family and as such are gram negative rods.[291] There over 35 species within the genus, of which only about one third are pathogenic for humans.[292]	Pathogenic *Vibrio* are part of the autochthonous microbial flora in brackish and marine environments in temperate or tropical regions. *V. cholerae* and *V. mimicus* may be found in fresh water and in birds and herbivores.[293]	*Cholera* is caused by *V. cholerae* 01.[294] Gastroenteritis is classically associated with *V. cholerae* non-01, *V. parahaemolyticus*, *V. hollisae*, *V. mimicus*, *V. fluvialis*, *V metschnikovii*, and *V. furnissii*.[295]	While classic cholera is rare in the US, the rice-water stool remains the characteristic symptom, among others and its infectious dose is quite large.[296] Gastroenteritis caused by other *Vibrio* sp. presents as diarrhea and may be accompanied by cramps, nausea, vomiting and fever.[297]	Antimicrobial therapy reduces the frequency and duration of the diarrhea and shortens the post-infective period of shedding of *V.cholerae*.[298] Tetracycline or less commonly furazolidone are drugs of choice, though antibiotic resistance is increasing.[299 300]
Yeast not candida (PP): ***Blastoschizomyces:*** **Blastoschizomyces capitatus* ***Hansenula anomala*** ***Pichia ohmeri*** ***Rhodotorula*** **Rhodotorula glutinis* **Rhodotorula rubra* **Rhodotorula species* ***Trichosporon*** **Trichosporon pullulans* **Trichosporon species*	Yeast are unicellular, budding cells and are usually round to oval in shape, though some forms have demonstrated elongated and irregular shapes.[301]	Yeast are ubiquitous in the environment and can be found on fruits, vegetables and other plant materials.[302] They can also live as normal inhabitants both within and on the body.[303]	Less common yeast such as those outlined in this section should only be considered opportunistic pathogens in the immunocompromised host.[304 305 306 307 308 309 310 311 312]	Disseminated infections may include the intestinal tract and are usually associated with immunosuppressive diseases or conditions such as leukemia, organ transplant, multiple myeloma, aplastic anemia, diabetes mellitus with ketoacidosis, ICU patients, lymphoma, solid tumors and AIDS.[313 314] Immunosuppressive therapy such as corticosteroids, chemotherapeutic agents and cyclosporine can also enhance fungal overgrowth.[315]	Currently, standard texts provide no specific antifungal guidelines for GI overgrowth of the fungi mentioned.[316 317] Treatment is at the discretion of the practitioner, and should be based upon clinical symptoms and a positive reculture of the organism.

Pathogenic Organism Chart

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Yersinia (PP):	*Yersinia* are gram-negative enteropathogenic bacilli that belong to the *Enterobacteriaceae* family.[318] At present, there are at least 10 species within the *Yersinia* genus.[319]	*Y. pseudotuberculosis* is found naturally in numerous wild and domestic mammals and birds. *Y. enterocolitica* can be found in all warm-blooded wild, domestic and pet animals and occasionally in some fish. Pigs are important reservoirs for the human strains of *Y. enterocolitica*.[320] Infections may be acquired by ingestion of contaminated food or water, or, rarely by direct person-to-person transmission in schools and hospitals.[321]	Intestinal yersiniosis may present in three clinical forms: enteritis, terminal ileitis, or mesenteric lymphadenitis causing "pseudoappendicitis" and septicemia.[322] *Y. entercolitica* and *Y. pseudotuberculosis* are most commonly isolated from cases of gastroenteritis. Both would be considered significant isolates from stool. Both of these organisms show preference for lymphatic tissue and can spread via the bloodstream.[323] *Yersinia* infection has been shown to induce chronic inflammatory bowel disorders such as chronic diarrhea and IBD. Rheumatoid arthritis, reactive arthritis and unspecified arthralgias have also been noted after *Yersinia* infection.[324 325]	Watery and sometimes bloody stools, fever, vomiting, abdominal pain are common with *Y. enterocolitica*, particularly in adults and less frequently in children but rarely in *Y. pesudotuberculosis* infection which is more common in children exhibiting terminal ileitis, lymphadenitis, and pseudoappendicitis.[326] Animal and in-vitro studies have isolated an antigen designated *Yersinia pseudotuberculosis* mitogen (YPM) that is capable of increasing epithelial permeability.[327] Chronic GI disease (eg intermediate colitis, UC, CD may follow *Y. enterocolitica* infection, though the exact role this organism plays has not been fully elucidated.[328 329 330 331]	Intestinal infections with *Y. enterocolitica* and *Y. pseudotuberculosis* are usually self-limiting and do not require antibiotic therapy. In cases of complicated gastroenteritis, doxycycline or trimethoprim-sulfmethoxazole are the antibiotics of choice.[332]
**Yersinia enterocolitica*					
**Yersinia pseudotuberculosis*					
**Yersinia species*					

* Susceptibility testing must guide treatment for all microbial and fungal organisms.

Parasitic Organism Chart Explained



In this section you will find the following about each organism:

- Description
- Habitat/Source of Isolation
- Pathogenicity
- Common Symptoms
- Possible Treatment Options

Parasite Organism Chart

Parasite	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	*Treatment
Blastocystis hominis	*B. hominis* has recently been reclassified as a protozoan, of which there are thought to be four separate serologic groups.[1]	This organism is transmitted via the fecal-oral route or from contaminated food or water.[2] Prevention can be enhanced by improving personal hygiene and sanitary conditions.[3]	The role of *B. hominis* in terms of colonization and disease is still considered controversial. When this organism is present in the absence of any other parasites, enteric organisms or viruses, it may be considered the etiological agent of disease.[4]	Symptoms can include: diarrhea, cramps, nausea, fever, vomiting and abdominal pain.[5] *B. hominis* has been associated with irritable bowel syndrome, infective arthritis and intestinal obstruction.[6]	Currently, **Metronidazole** (Flagyl) is considered the most effective drug (750 mg tid x 10 days).[7] **Iodoquinol** (Yodoxin) is also an effective medication (650 mg tid x 20 days).[8] Recommended therapy can also eliminate *G. lamblia*, *E. histolytica* and *D. fragilis*, all of which may be concomitant undetected pathogens and part of patient symptamology.[9,10]

Parasite	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	Treatment
Cryptosporidium spp	*Cryptosporidium spp* are coccidian parasites that belong to the *Cryptosporidiidae* family.[11]	Infection is thought to occur by environmentally resistant oocysts, zoonotic transmission, nosocomial transmission and direct person-to-person contact.[12] Contamination of public water supply has been associated with outbreaks. Raw foods such as unpasturized milk and raw meat can also harbor the organism.[13]	*Cryptosporidium* is an important agent of diarrhea in both the immunocompetant and immunocompromised host.[14] The organism inhabits the intestinal mucosa causing diarrhea.[15] Infection in the immunocompromised host may cause life threatening disease and can disseminate from the intestinal tract.[16] *Cryptosporidium* is considered an important opportunistic pathogen in patients with AIDS, and detection is associated with a poor prognosis.[17]	Acute infections can mimic Crohn's disease with villus atrophy, enlarged crypts, and infiltration of the lamina propria by inflammatory cells.[18] Clinical symptoms in the immunocompetant host include nausea, low-grade fever, abdominal cramps, anorexia and up to 5-10 watery bowel movements a day, which may be followed by constipation.[19] Immunocompetent hosts can also be asymptomatic.[20] *Cryptosporidium* in the immunocompromised host may be ongoing and severe. The length and severity depend on the ability to reverse the immunosuppression. Extra-intestinal infections can occur with respiratory symptoms, cholecystitis, hepatitis and pancreatitis[21] Chronic cryptosporidiosis in infants is associated with failure to thrive[22]	Cryptosporidiosis is generally self-limiting in immunocompetent patients, lasting approximately 2 weeks.[23] Currently, there is no totally effective therapy for cryptosporidiosis. Refer to the Medical Letter and/or Sanford Guide for therapeutic protocols in the immunocompromised host.[24]

Parasite Organism Chart

Parasite	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	Treatment
Dientamoeba fragilis	*D. fragilis* has recently been reclassified as an ameboflagellate (previously ameba) and is closely related to *Histomonas* and *Trichomonas* species[25]	Because this organism does not have a cyst stage, there is uncertainty of the mode of transmission.[26] Fecal oral transmission thus far has not been documented.[27] Higher incidences have been reported for mental institutions, missionaries and Native Americans of Arizona. *D. fragilis* is also common in pediatric populations and patients under the age of 20.[28]	*D. fragilis* is known to cause non-invasive diarrheal illness in humans. 90% of children are symptomatic, whereas only 15-20% of adults are.[29]	The most common symptoms associated with *D. fragilis* are intermittent diarrhea, fatigue, abdominal pain, fatigue, nausea, anorexia, malaise and unexplained eosinophilia.[30] Diarrhea is predominately seen during the first 1-2 weeks of infection and abdominal pain may persist for 1-2 months.[31]	**Iodoquinol** (650 mg tid x 20 days) or **Tetracycline** (500 mg qid x 10 days) or **Metronidazole** (500-750 mg tid x 10 days) have been used to treat *D. fragilis*.[32] Another alternative is **Paromomycin** (500 mg tid x 7 days).[33]
Entamoeba coli	This organism is a protozoan belonging to the amebae family.[34]	*Entamoeba coli* has a worldwide distribution, the prevalence is generally greater in warmer climates.[35] The cyst which is the infectious form is ingested from contaminated food and water. Direct transmission can also occur via the fecal-oral-route.[36]	While *Entamoeba coli* is the most common ameba isolated in humans, it is considered non-pathogenic.[37]	*Entamoeba coli* is not associated with intestinal symptoms.	The Medical Letter and Sanford Guide provide no therapeutic recommendations for *Entamoeba coli*. Treatment is not recommended for non-pathogenic amebae. Improving sanitary conditions and personal hygiene help to prevent infection.[38]

Parasite	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	Treatment
Entamoeba dispar	*E. dispar* is a protozoan that belongs to the amebae family.[39]	Transmission is from the ingestion of infective cysts in contaminated food or water. Person-to-person contact is also a source of transmission.[40]	*Entameoba dispar* is considered to be non-pathogenic in humans.[41]	This particular species of *Entameoba* is not known to produce intestinal symptoms, nor is it invasive in humans.[42]	The Medical Letter and Sanford Guide provide no therapeutic recommendations for *Entamoeba dispar*. Treatment is generally not recommended for non-pathogenic amebae, however this recommendation is based upon being able to accurately differentiate *E. dispar* from pathogenic *E. histolytica*.[43]
Entamoeba hartmanni	This organism belongs to the amebae family.[44]	Transmission is related to the ingestion of cysts from contaminated food or water.[45]	*Entamoeba hartmanni* is not considered a pathogen in humans. While early research identified this organism as a potential pathogen, subsequent studies were unable to adequately confirm.[46]	*Entamoeba hartmanni* is not routinely associated with clinical symptoms.[47]	Treatment for *E. hartmani* is usually not recommended, accordingly the Medical Letter and the Sanford guide have no therapeutic recommendations.[48]

Parasite Organism Chart

Parasite	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	Treatment
Entamoeba histolytica	*E. histolytica* belongs to the ameba family of protozoa.[49]	This organism has been recovered worldwide, though is more prevalent in the tropics and subtropics. In unsanitary conditions, infection rates are equivalent to tropical regions despite colder climates.[50] Humans are the reservoir for *E. histolytica* and can transmit the parasite to other humans, primates, cats, dogs and possibly pigs.[51]	*E. histolytica* is pathogenic for humans, causing invasive intestinal and extraintestinal amebiasis.[52] In 2-8% of infected individuals, invasion of the intestinal mucosa occurs with dissemination to other organs (most frequently the liver).[53] The organism is capable of inducing both humoral and cellular immune responses.[54]	While a large number of people worldwide are infected with *E. histolytica*, only a few manifest clinical symptoms.[55] Asymptomatic patients may excrete cysts for only a short period of time and are essentially unaffected and never experience symptoms.[56] Some patients may experience symptoms that mimic ulcerative colitis. Others still may have a gradual onset of symptoms including diarrhea, colicky abdominal pain, and tenesmus. The incubation time for those symptomatic can vary from 1-4 weeks. With the onset of dysentery, diarrhea can occur with up to 10 movements a day that are characterized by blood-tinged mucus.[57]	*E. histolytica* should be treated even if patients are asymptomatic. **Paromomycin** 500 mg tid x 7 days) or **Iodoquinol** (650 mg tid x 20 days) **Diloxanide Furoate** (500 mg tid x 10 days are used for asymptomatic patients with cysts in the gut lumen, but are ineffective for extraintestinal infections.[58] **Metronidazole** (500-750 mg tid x 10 days) or **Tinidazole** (1g q12h x 3 days), or **Ornidazole** 500 mg q12h x 5 days) followed by either **Paromomycin** 500 mg tid x 10 days) or **Iodoquinol** (650 mg tid x 20 days) are used for patients with mild to moderate disease.[59] Severe extraintestinal infection requires IV therapy, refer to the Sanford guide for therapeutic guidelines.[60]

Parasite	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	Treatment
Giardia lamblia	*Giardia lamblia* is the most commonly diagnosed flagellate in the intestinal tract.[61] *Giardia intestinalis* and *Giardia duodenalis* are also used as names for this organism.[62]	Infection occurs via fecal-oral transmission or from food and water contaminated with the cysts.[63] *Giardia lamblia* has a worldwide distribution, though is more common in warmer climates than cooler ones. Isolation of the organism is more prevalent in children or those living in close quarters with poor sanitary conditions.[64]	*Giardia lamblia* is considered a pathogen in humans.[65]	Most people infected with *G. lamblia* are asymptomatic. For those symptomatic, there can be an acute and a chronic phase of infection.[66] After an incubation period of 2-20 days, symptoms of watery diarrhea, nausea, low grade fever and chills can occur lasting only a few days.[67] Acute infection can mimic food poisoning, bacillary dysentery, viral enteritis, acute intestinal amebiasis or travelers diarrhea. One point of differentiation is the lack of blood, mucus and cellular exudates in the stool with *G. lamblia*.[68] In the chronic phase, symptoms can include recurrent foul smelling diarrhea, abdominal distention, belching and heartburn.[69] Chronic Giardiasis may lead to dehydration, malabsorption and impaired pancreatic function.[70]	The drug of choice is **Metronidazole** (250 mg tid x 5 days) and is recommended also for immunocompetent hosts with self limiting infections. Treatment helps prevent transmission of the organism and reduce the duration of symptoms.[71] Other therapeutic alternatives include **Furazolidone** (100 mg qid x 7-10 days) or **Tinidazole** (2g once).[72] **Paromomycin** (500 mg 4x/day x 7 days) is the alternative for treating *G. lamblia* during pregnancy.[73]

Parasite Organism Chart

Parasite	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	Treatment
Helminths					
Ascaris lumbricoides	*Ascaris lumbricoides* is the largest and most prevalent of all the human intestinal nematodes.[74]	This organism is more prevalent in warm, moist climates, though it can survive in temperate regions.[75] Infection is acquired through the ingestion of embryonated eggs in contaminated soil.[76]	This organism is a clearly defined pathogen in humans with infection rates as high as 45% in Central and South America.[77] The pathogenesis of *A. lumbricoides* is attributed to (i) the immune response of the host (ii) the effects of larval migration (iii) the effects of adult worms (iv) nutritional deficiencies resultant from the adult worms.[78]	Symptoms relate to the migration of the worm after hatching in the stomach, penetrating the intestinal wall and migrating through the liver to the lungs. When in the intestine, patients are usually asymptomatic, unless the worm burden is high. Migration can result in intestinal blockage, entry into the bile or pancreatic duct, or liver or peritoneal cavity. Repeated infections or those with a large volume of eggs can result in pneumonitis (Loeffler's syndrome) during the larval migration phase through the lungs. Symptoms include cough, dyspnea, wheezing or coarse rales, fever and transient eosinophilia.[79] Infection can be terminated by the spontaneous passage of the adult worms from the anus, mouth or nares.[80]	**Mebendazole** (100 bid x 3 days) is considered the most effective drug and is suitable for both children and adults. **Pyrantel pamoate** (11 mg/kg once (maximum 1 gram), repeat after two weeks) or **Albendazole** (400 mg once) are alternatives, however these drugs are still considered investigational.[81]

Parasite	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	Treatment
Enterobius vermicularis (Pinworm)	*E. vermicularis* is a nematode, and is the most prevalent parasitic infection in the world.[82]	Infection is more common in the cooler, temperate regions, and thought to be related to reduced bathing and changing of underclothes.[83] Infection is more prevalent in children and occurs more commonly in females.[84]	*E. vermicularis* is considered a pathogenic organism.[85]	Those infected may be asymptomatic or experience pruritus from the migration of the worms from the anus to the perianal skin where the eggs are deposited.[86] Other symptoms found in infected children include insomnia, nervousness, irritability, nightmares and convulsions.[87]	Treatment is with **Pyrantel pamoate** (11 mg/kg once (maximum once), repeat after two weeks) or **Mebendazole** (100 mg once, repeat after two weeks), or **Albendazole** (400 mg once, repeat after two weeks). Therapy should always be based upon evidence of infection and symptomology.[88]

Parasite Organism Chart

Parasite	Description	Habitat/Sources of Isolation	Pathogenicity	Symptoms	Treatment
Strongyloides stercoralis	*S. stercoralis* is classified as a nematode.[89]	The organism is more prevalent in tropics and subtropics, though can survive colder climates.[90] The first stage larvae are contaminated in the soil, and infection occurs from skin penetration where the organism then travels to the intestine via the blood, lungs, trachea and upper Gastro-intestinal tract.[91]	*S. stercoralis* is considered a pathogen in humans.[92]	Individuals can be asymptomatic, or exhibit symptoms in three key areas relative to the life cycle of the parasite and a heavy infective dose.[93] Cutaneous penetration may result in pruritis and erythema when the larvae are in high numbers. With larval migration through the lungs, infected hosts may develop a cough, shortness of breath, wheezing, fever, and pneumonia. When there is intestinal infestation, symptoms can mimic peptic ulcer and there may be damage to the intestinal mucosa with villous atrophy and crypt hyperplasia. Radiographic findings may be akin to Crohn's disease of the proximal small intestine.[94] Reactive arthritis has also been associated with a heavy *Strongyloides* infection.[95]	Treatment options for *Strongyloides* include **Ivermectin** (200 ug/kg/day x 1-2 days) or **Thiabendazole** (25mg/kg.day bid (maximum of 3g/day).[96]

*** Treatment protocols sourced from the Medical Letter (03) or the Sanford Guide (03).**

[1] Garcia, LS. *Diagnostic Medical Parasitology*. 4th ed. Washington DC: ASM; 2001; 28.

A full list of references is available upon request by calling NutriPATH on 1300 688 522.

Notes

NutriPATH

INTEGRATIVE PATHOLOGY SERVICES

16 Harker Street,
Burwood, Vic 3125
P: 1300 688 522
info@nutripath.com.au
www.nutripath.com.au